NEVADA STATE BOARD of DENTAL EXAMINERS

Anesthesia Committee Teleconference Meeting

Thursday, July 25th, 2024

6:00 p.m.

PUBLIC BOOK

Nevada State Board of Dental Examiners

2651 N. Green Valley Pkwy, Ste. 104 • Henderson, NV 89014 • (702) 486-7044 • (800) DDS-EXAM • Fax (702) 486-7046

Anesthesia Committee Teleconference Meeting

Meeting Date & Time

Thursday, July 25, 2024
6:00 P.M.

Meeting Location:

Nevada State Board of Dental Examiners
2651 N. Green Valley Pkwy., Suite 104
Henderson, NV 89014

Video Conferencing / Teleconferencing Available

To access by phone, call Zoom teleconference Phone Number: (669) 900 6833
To access by video webinar, visit www.zoom.com or use the Zoom app

Zoom Webinar/Meeting ID#: **886 8568 8090**
Zoom Webinar/Meeting Passcode: **816148**

PUBLIC NOTICE:

Public Comment by pre-submitted email/written form and Live Public Comment by teleconference is available after roll call (beginning of meeting and prior to adjournment (end of meeting). Live Public Comment is limited to three (3) minutes for each individual.

Members of the public may submit public comment in written form to: **Nevada State Board of Dental Examiners, 2651 N. Green Valley Pkwy, Ste. 104, Henderson, NV 89014; FAX number (702) 486-7046; e-mail address nsbde@dental.nv.gov.** Written submissions received by the Board on or before **Wednesday, July 24, 2024 by 4:00 p.m.** may be entered into the record during the meeting. Any other written public comment submissions received prior to the adjournment of the meeting will be included in the permanent record.

The Nevada State Board of Dental Examiners may 1) address agenda items out of sequence to accommodate persons appearing before the Board or to aid the efficiency or effectiveness of the meeting; 2) combine items for consideration by the public body; 3) pull or remove items from the agenda at any time. The Board may convene in closed session to consider the character, alleged misconduct, professional competence or physical or mental health of a person. *See* NRS 241.030. Prior to the commencement and conclusion of a contested case or a quasi-judicial proceeding that may affect the due process rights of an individual the board may refuse to consider public comment. *See* NRS 233B.126.

Persons/facilities who want to be on the mailing list must submit a written request every six (6) months to the Nevada State Board of Dental Examiners at the address listed in the previous paragraph. With regard to any board meeting or telephone conference, it is possible that an amended agenda will be published adding new items to the original agenda. Amended Nevada notices will be posted in compliance with the Open Meeting Law.

We are pleased to make reasonable accommodations for members of the public who are disabled and wish to attend the meeting. If special arrangements for the meeting are necessary, please notify the Board, at (702) 486-7044, no later than 48 hours prior to the meeting. Requests for special arrangements made after this time frame cannot be guaranteed.

Pursuant to NRS 241.020(2) you may contact at (702) 486-7044, to request supporting materials for the public body or you may download the supporting materials for the public body from the Board's website at http://dental.nv.gov In addition, the supporting materials for the public body are available at the Board's office located at 2651 N Green Valley Pkwy, Ste 104, Henderson, Nevada 89014.

Note: Asterisks (*) **"For Possible Action"** denotes items on which the Board may take action.
Note: Action by the Board on an item may be to approve, deny, amend, or tabled.

1. **Call to Order**

   - Roll call/Quorum

2. **Public Comment (Live public comment, teleconference, and pre-submitted email/written form):**
The public comment period is limited to matters specifically noticed on the agenda. No action may be taken upon the matter raised during public comment unless the matter itself has been specifically included on the agenda as an action item. Comments by the public may be limited to three (3) minutes as a reasonable time, place, and manner restriction, but may not be limited based upon viewpoint. The Chairperson may allow additional time at his/her discretion.

   Members of the public may submit public comment via email to nsbde@dental.nv.gov, or by mailing/faxing messages to the Board office. Written submissions received by the Board on or before **Wednesday, July 24, 2024 by 4:00 P.M.** may be entered into the record during the meeting. Any other written public comment submissions received prior to the adjournment of the meeting will be included in the permanent record.

   In accordance with Attorney General Opinion No. 00-047, as restated in the Attorney General's Open Meeting Law Manual, the Chair may prohibit comment if the content of that comment is a topic that is not relevant to, or within the authority of, the Nevada State Board of Dental Examiners, or if the content is willfully disruptive of the meeting by being irrelevant, repetitious, slanderous, offensive, inflammatory, irrational, or amounting to personal attacks or interfering with the rights of other speakers.

*3. **Chairman's Report**: Joshua Branco, DMD (For Possible Action)

   *a. **Request to Remove Agenda Item(s)** (For Possible Action)

   *b. **Approve Agenda** (For Possible Action)

*4. **Old Business**: (For Possible Action)

   *a. **Approval/Rejection of Permanent Anesthesia Permit – NRS 631.190 & NAC 631.2235**
   (For Possible Action)

      (1) Nathan Antoine, DMD – Moderate Sedation

*5. **New Business**: (For Possible Action)

   *a. **Discussion, Consideration, and Possible Recommendation to the Board to Approve or Deny DOCS EDUCATION as a Moderate Sedation Board Approved Course of Study and to Specifically Address Whether Such Course is for Patients 13 years of Age or Older NRS 631.190 & NAC 631.2213** (For possible action.)

*6. **Public Comment (Live public comment and by teleconference):** This public comment period is for any matter that is within the jurisdiction of the public body. No action may be taken upon the matter raised during public comment unless the matter itself has been specifically included on the agenda as an action item. Comments by the public may be limited to three (3) minutes as a reasonable time, place and manner restriction, but may not be limited based upon viewpoint. The Chairperson may allow additional time at his/her discretion.

   Members of the public may submit public comment via email to nsbde@dental.nv.gov, or by mailing/faxing written messages to the Board office. Written submissions should be received by the Board on or before **Wednesday, July 24, 2024 by 4:00 p.m.** may be entered into the record during the meeting. Any other written public comment submissions received prior to the adjournment of the meeting will be included in the permanent record.

   In accordance with Attorney General Opinion No. 00-047, as restated in the Attorney General's Open Meeting Law Manual, the Chair may prohibit comment if the content of that comment is a topic that is not relevant to, or within the authority of, the Nevada State Board of Dental Examiners, or if the content is willfully disruptive of the meeting by being irrelevant, repetitious, slanderous, offensive, inflammatory, irrational, or amounting to personal attacks or interfering with the rights of other speakers.

*7. **Announcements**

*8. **Adjournment** (For Possible Action)

Agenda Item 4(a):

Approval/Rejection of Permanent Anesthesia Permit NRS 631.190 & NAC 631.2235

(For Possible Action)

NRS 631.190 Powers and duties. [Effective January 1, 2020.] In addition to the powers and duties provided in this chapter, the Board shall:

1. Adopt rules and regulations necessary to carry out the provisions of this chapter.

2. Appoint such committees, review panels, examiners, officers, employees, agents, attorneys, investigators and other professional consultants and define their duties and incur such expense as it may deem proper or necessary to carry out the provisions of this chapter, the expense to be paid as provided in this chapter.

3. Fix the time and place for and conduct examinations for the granting of licenses to practice dentistry, dental hygiene and dental therapy.

4. Examine applicants for licenses to practice dentistry, dental hygiene and dental therapy.

5. Collect and apply fees as provided in this chapter.

6. Keep a register of all dentists, dental hygienists and dental therapists licensed in this State, together with their addresses, license numbers and renewal certificate numbers.

7. Have and use a common seal.

8. Keep such records as may be necessary to report the acts and proceedings of the Board. Except as otherwise provided in NRS 631.368, the records must be open to public inspection.

9. Maintain offices in as many localities in the State as it finds necessary to carry out the provisions of this chapter.

10. Have discretion to examine work authorizations in dental offices or dental laboratories.

[Part 4:152:1951; A 1953, 363] — (NRS A 1963, 150; 1967, 865; 1993, 2743; 2009, 3002; 2017, 989, 2848; 2019, 3205, effective January 1, 2020)

NAC 631.2235 Inspections and evaluations: Grading; report of recommendation of evaluator; issuance of permit for passing; failure to pass; request for reevaluation; issuance of order for summary suspension. (NRS 631.190, 631.265)

1. The persons performing an inspection or evaluation of a dentist and his or her office for the issuance or renewal of a general anesthesia permit or moderate sedation permit shall grade the dentist as passing or failing to meet the requirements set forth in NAC 631.2219 to 631.2231, inclusive. Within 72 hours after completing the inspection or evaluation, each evaluator shall report his or her recommendation for passing or failing to the Executive Director, setting forth the details supporting his or her conclusion.

2. If the dentist meets the requirements set forth in NAC 631.2219 to 631.2231, inclusive, the Board will issue the general anesthesia permit or moderate sedation permit, as applicable.

3. If the dentist does not meet the requirements set forth in NAC 631.2219 to 631.2231, inclusive, the Executive Director shall issue a written notice to the dentist that identifies the reasons he or she failed the inspection or evaluation.

4. A dentist who has received a notice of failure from the Board pursuant to subsection 3:

(a) Must cease the administration of any general anesthesia, deep sedation or moderate sedation until the dentist has obtained the general anesthesia permit or moderate sedation permit, as applicable; and

(b) May, within 15 days after receiving the notice, request the Board in writing for a reevaluation. The request for a reevaluation must state specific grounds supporting it.

5. If the reevaluation is granted by the Board, it will be conducted by different persons in the manner set forth by NAC 631.2219 to 631.2231, inclusive, for an original evaluation.

6. No dentist who has received a notice of failing an inspection or evaluation from the Board may request more than one reevaluation within any period of 12 months.

7. Pursuant to subsection 3 of NRS 233B.127, if an inspection or evaluation of a dentist or his or her office indicates that the public health, safety or welfare imperatively requires emergency action, the President of the Board may, without any further action by the Board, issue an order of summary suspension of the license of the dentist pending proceedings for revocation or other action. An order of summary suspension issued by the President of the Board must contain findings of the exigent circumstances which warrant the issuance of the order of summary suspension. The President of the Board shall not participate in any further proceedings relating to the order.

(Added to NAC by Bd. of Dental Exam’rs, eff. 10-21-83; A by R005-99, 9-7-2000; R004-17, 5-16-2018)

Agenda Item 4(a)(1):

Nathan Antoine, DMD - Moderate Sedation

Nevada State Board of Dental Examiners

2651 N. Green Valley Pkwy, Ste. 104 • Henderson, NV 89014 • (702) 486-7044 • (800) DDS-EXAM • Fax (702) 486-7046

(TEMPORARY)
MODERATE SEDATION ADMIN PERMIT APPLICATION
(Administration of Moderate Sedation restricted to patients 13 years of age and older)

QUALIFICATIONS OF APPLICANTS

Nathan M Antoine, DMD		APPLICANT NAME LIC #S4-133 – licensed 03/06/2023) (Previous license # 6915 – licensed 06/09/2017)
Yes,	No	COMPLETED APPLICATION
Yes	No	PAYMENT RECEIVED (CHK#1019 $ 750.00)
SEE ATTACHED		CERTIFICATION OF MINIMUM 60 HOURS APPROVED COURSE STUDY DEDICATED EXCLUSIVELY TO THE ADMINISTRATION OF MODERATE SEDATION: **Program: St. Louis University – Periodontics Center for Advanced Dental Education**
SEE ATTACHED		CERTIFICATION OF THE ADMINISTRATION OF A MINIMUM OF 20 SEDATION CASES SUCCESSFULLY MANAGED BY THE APPLICANT **Location: St. Louis University – Periodontics Center for Advanced Dental Education**
Yes	No	CERTIFICATION OF SPECIALTY PROGRAM COMPLETION APPROVED BY ADA CODA WHICH INCLUDES EDUCATION/TRAINING IN MS ADMINISTRATION (EQUIVALENT TO 60 HOURS/20 CASES)
Specialty:		
Yes	No	ACLS CERTIFICATION IN COMPLIANCE WITH AMERICAN HEART ASSOCIATION STANDARDS ACLS VALID DATES: **03/09/2022 – 03/2024**

CERTIFICATION CAN INCLUDE LETTER FROM PROGRAM DIRECTOR ON INSTITUTION'S LETTERHEAD (W/SEAL) OR CERTIFICATE OF COMPLETION BY RECOGNIZED SPECIALTY BOARD PURSUANT TO NAC 631.190.

REVIEW CONTINUED – APPLICANT: NATHAN M ANTOINE, DMD

Review by Chair of Anesthesia Committee:

RECOMMEND APPROVAL: YES NO

IF NO,
Reasons/Concerns:

8/15/23

William T Thompson, DMD
Chair of Anesthesia Committee

Date

Received
AUG 16 2023
NSBDE

Review by Secretary-Treasurer:

APPLICATION APPROVED: YES NO

IF REJECTED,
Reasons/Concerns:

8-29-23

Tejpaul Ashi, DDS
Secretary-Treasurer

Date

Nevada State Board of Dental Examiners

6010 S. Rainbow Blvd., Bldg. A, Ste. 1
Las Vegas, NV 89118
(702) 486-7044 • (800) DDS-EXAM • Fax (702) 486-7046

MODERATE SEDATION ADMIN PERMIT APPLICATION

(Administration of **Moderate Sedation** to patients 13 years of age or older)

Office Site Permit

Check box if you are applying for a Site Permit for this same office location as well

Name: Nathan Antoine License Number: ~~S4-133~~ 6915

Dental Practice Name Team Perio

Office Address: [redacted]

Office Telephone [redacted]

Office Fax: _____

DENTAL EDUCATION

University/ College: Midwestern University - AZ College of Dental Medicine

Location: Glendale, AZ

Dates attended: 08/2013 to 05/2017

Degree Earned: DMD

BOARD APPROVED PROGRAM

Name/ Instructor: St. Louis University - Periodontics Center for Advanced Dental Education

Location: 3320 Rutger St. St. Louis, MO 63104

Dates attended: 07/2020 to 06/2023

Certificate Granted: MS Dentistry + Certificate in periodontics

The following information and documentation must be received by the Board office prior to consideration of a **MODERATE SEDATION** permit:

1) *Completed and signed application form;*
2) *Non-refundable application fee in the amount of $750.00;*
3) *Certification of completion of a course of study, subject to the approval of the Board, of not less than sixty (60) hours of course study dedicated exclusively to the administration of moderate sedation to patients 13 years of age or older and proof of successful management as the operator of moderate sedation to not less than twenty (20) patients who are 13 years of age or older.*

Received
AUG 14 2023
NSBDE

4) *Valid certification in Advance Cardiac Life Support by the American Heart Association or the completion of a course approved by the Board that provides instruction on medical emergencies and airway management*

I hereby make application for a Moderate Sedation Permit to administer moderate sedation *to patients 13 years of age or older* from the Nevada State Board of Dental Examiners. I understand that if this permit is issued, I am authorized to administer moderate sedation **ONLY** *to patients 13 years of age or older* at the address listed above. If I wish to administer moderate sedation *to patients 13 years of age or older* at another location, I understand that each site must be inspected and a "Moderate Sedation Site Permit" must be issued by the Board prior to administration of moderate sedation to *patients 13 years of age or older* . I understand that this permit, if issued, allows only *me* to administer moderate sedation to *patients 13 years of age or older* .

I also understand that this permit does **NOT** allow for the administration *of moderate sedation to patients 12 years of age or younger* or the administration of deep sedation or *general anesthesia* by me, a physician, nurse anesthetist, or any other person. I have read and am familiar with the provisions and requirements of NRS 631 and NAC 631 regarding the administration of moderate sedation.

I, hereby acknowledge the information contained on this application is true and correct and I further acknowledge any omissions, inaccuracies, or misrepresentations of information on this application are grounds for the revocation of a permit which may have been obtained through this application. It is understood and agreed that the title of all certificates shall remain in the Nevada State Board of Dental Examiners and shall be surrendered by order of said Board.

Signature of Applicant Nathan Austin

Date 8/10/23

NOTE: *In order to administer moderate sedation to patients 12 years of age or younger, you must meet the requirements set forth in NAC 631.2213 and submit an application for a "Pediatric Moderate Sedation Admin Permit"*

APPLICATION FOR MODERATE SEDATION ADMINISTRATION

Pursuant to NAC 631.2213; Applicants must submit *certification of completion of a course of study, subject to the approval of the Board, of not less than sixty (60) hours of course study dedicated exclusively to the administration of moderate sedation to patients 13 years of age or older and proof of successful management as the operator of moderate sedation to not less than twenty (20) patients who are 13 years of age or older*

SUBMISSION OF NO LESS THAN 20 CASES OF MODERATE SEDATION ADMINISTRATION

Received
AUG 14 2023
NSBDE

Nevada State Board of Dental Examiners
2651 N. Green Valley Parkway, Suite 104, Henderson, NV 89014
(702) 486-7044 • (800) DDS-EXAM • Fax (702) 486-7046
nsbde@dental.nv.gov

MODERATE SEDATION
INSPECTION AND EVALUATION REPORT

☑ ON-SITE/ADMINISTRATOR EVALUATION	☐ SITE ONLY INSPECTION
Name of Practitioner: NATHAN ANTOINE	Proposed Dates: 5-15-24
Location to be Inspected: 855 W 7th ST, SUITE 130 RENO, NV 89503	Telephone Number: [redacted]
	Email Address:
Date of Evaluation: 5-15-24	Time of Evaluation/Inspection: Start Time: 08:45 Finish Time: 12:45 PM

Evaluators

1.	EDWARD GRAY
2.	THOMAS MYATT
3.	

INSTRUCTIONS FOR COMPLETING MODERATE SEDATION ON-SITE INSPECTION AND EVALUATION FORM

1. Prior to evaluation, review criteria and guidelines for Moderate Sedation (MS) On-Site/Administrator and Site Only Inspection in the Examiner Manual.
2. Each evaluator should complete a MS On-Site/Administrator or Site Only Inspection report independently by checking the appropriate answer box to the corresponding question or by filling in a blank space.
3. After answering all questions, each evaluator should make a separate overall "pass" or "fail" recommendation to the Board. "Fail" recommendations must be documented with a narrative explanation.
4. Sign the evaluation report and return to the Board office within **72 hours** after evaluation has been completed.

SITE INSPECTION

OFFICE FACILITIES AND EQUIPMENT	YES	NO
1. Operating Room		
a. Is operating room large enough to adequately accommodate the patient on a table or in an operating chair?	✓	
b. Does the operating room permit an operating team consisting of at least three individuals to freely move about the patient?	✓	
2. Operating Chair or Table		
a. Does operating chair or table permit the patient to be positioned so the operating team can maintain the airway?	✓	
b. Does operating chair or table permit the team to quickly alter the patient's position in an emergency?	✓	
c. Does operating chair or table provide a firm platform for the management of cardiopulmonary resuscitation?	✓	
3. Lighting System		
a. Does lighting system permit evaluation of the patient's skin and mucosal color?	✓	
b. Is there a battery powered backup lighting system?	✓	
c. Is backup lighting system of sufficient intensity to permit completion of any operation underway at the time of general power failure?	✓	
4. Suction Equipment		
a. Does suction equipment permit aspiration of the oral & pharyngeal cavities?	✓	
b. Is there a backup suction device available which can operate at the time of general power failure?	✓	
5. Oxygen Delivery System		
a. Does oxygen delivery system have adequate full face masks and appropriate connectors and is capable of delivering oxygen to the patient under positive pressure?	✓	
b. Is there an adequate backup oxygen delivery system which can operate at the time of general power failure?	✓	
6. Recovery Area (Recovery area can be operating room)		
a. Does recovery area have available oxygen?	✓	
b. Does recovery area have available adequate suction?	✓	
c. Does recovery area have adequate lighting?	✓	
d. Does recovery area have available adequate electrical outlets?	✓	

SITE INSPECTION

OFFICE FACILITIES AND EQUIPMENT	YES	NO
7. Ancillary Equipment in Good Operating Condition?		
a. Are there oral pathways?	✓	
b. Is there a tonsilar or pharyngeal type suction tip adaptable to all office suction outlets?	✓	
c. Is there a sphygmomanometer and stethoscope?	✓	
d. Is there adequate equipment for the establishment of an intravenous infusion?	✓	
e. Is there a pulse oximeter?	✓	

DRUGS	DRUG NAME	EXPIRES	YES	NO
1. Vasopressor drug available?	EPI SYRINGE	6-30	✓	
2. Corticosteroid drug available?	DEXAMETHASONE	5-25	✓	
3. Bronchodilator drug available?	ALBUTEROL MDI	12-25	✓	
4. Appropriate drug antagonists available?	FLUMAZENIL NALOXONE	3-25 2-25	✓ ✓	
5. Antihistaminic drug available?	BENADRYL	6-25	✓	
6. Anticholinergic drug available?	ATROPINE	7-25	✓	
7. Coronary artery vasodilator drug available?	NTG	9-24	✓	
8. Anticonvulsant drug available?	MIDAZOLAM	10-25	✓	
9. Oxygen available?	✓	✓	✓	

RECORDS – Are the following records maintained?	YES	NO
1. An adequate medical history of the patient?	✓	
2. An adequate physical evaluation of the patient?	✓	
3. Sedation records show patient's vital signs?	✓	
4. Includes American Society of Anesthesiologists physical status classification?	✓	
5. Sedation records listing the drugs administered, amounts administered, and time administered?	✓	
6. Sedation records reflecting the length of the procedure?	✓	
7. Sedation records reflecting complications of the procedure, if any?	✓	
8. Written informed consent of the patient, or if the patient is a minor, their parent's or guardian's consent for administration of sedation?	✓	

SITE INSPECTION

	YES	NO
Is there moderate sedation administered at the dentist office to a patient of 12 years of age or younger? (If yes, complete the section below)		✓
ADDITIONAL EQUIPMENT FOR 12 YEARS OF AGE AND YOUNGER	**YES**	**NO**
1. Bag valve mask with appropriate size masks		✓
2. Appropriate size blood pressure cuffs		✓
3. Appropriate size oral and nasal airways		✓
ADDITIONAL EMERGENCY DRUG FOR 12 YEARS OF AGE AND YOUNGER	**YES**	**NO**
1. Appropriate dosages of epinephrine or a pediatric epinephrine auto-injector		✓
ADDITIONAL RECORDS FOR 12 YEARS OF AGE AND YOUNGER	**YES**	**NO**
1. Sedation records reflecting monitoring of patient that is consistent with the guidelines of the American Academy of Pediatric Dentistry		✓

Evaluator Overall Recommendation of Site Inspection

☑ Pass ☐ Fail ☐ Pass Pending*

**If Pass Pending, please list all deficiencies*

Comments: _____

Page 4 of 6

Signature of Evaluator

5-15-25

Date

THIS CONCLUDES THE SITE INSPECTION REPORT.

FOR AN EVALUATION OF AN ADMINISTERING PERMIT, CONTINUE TO NEXT SECTION

EVALUATION

DEMONSTRATION OF MODERATE SEDATION	YES	NO
1. Who administered moderate sedation? Dentist's Name: NATHAN ANTOINE	✓	
2. Was sedation case demonstrated within the definition of moderate sedation?	✓	
3. While sedated, was patient continuously monitored during the procedure with a pulse oximeter?	✓	
If not, what type of monitoring was utilized? CRITA CARE		
4. Was the patient monitored while recovering from sedation? Monitored by whom: SARA / EMMA Title: SURG ASSISTANT	✓	
5. Is this person a licensed health professional experienced in the care and resuscitation of patients recovering from moderate sedation?	✓	
6. Were personnel competent?	✓	
7. Are all personnel involved with the care of patients certified in basic cardiac life support?	✓	
8. Was dentist able to perform the procedure without any action or omission that could have resulted in a life-threatening situation to the patient?	✓	
9. What was the length of the case demonstrated? 1 HR	✓	

SIMULATED EMERGENCIES – Was dentist and staff able to demonstrate knowledge and ability in recognition and treatment of:	YES	NO
1. Laryngospasm?	✓	
2. Bronchospasm?	✓	
3. Emesis and aspiration of foreign material under anesthesia?	✓	
4. Angina pectoris?	✓	
5. Myocardial infarction?	✓	
6. Hypotension?	✓	
7. Hypertension?	✓	
8. Cardiac arrest?	✓	

SITE INSPECTION

SIMULATED EMERGENCIES – Was dentist and staff able to demonstrate knowledge and ability in recognition and treatment of:	YES	NO
9. Allergic reaction?	✓	
10. Convulsions?	✓	
11. Hypoglycemia?	✓	
12. Asthma?	✓	
13. Respiratory depression?	✓	
14. Local anesthesia overdose?	✓	
15. Hyperventilation syndrome?	✓	
16. Syncope?	✓	

Evaluator Overall Recommendation of Site Inspection

☑ Pass ☐ Fail

5-15-24
Date

Comments:

Signature of Evaluator

5-15-24
Date

Agenda Item 5(a):

Discussion, Consideration, and Possible Recommendation to the Board to Approve or Deny DOCS EDUCATION as a Moderate Sedation Board Approved Course of Study and to Specifically Address Whether Such Course is for Patients 13 Years of Age or Older NRS 631.190 & NAC 631.2213

(For Possible Action)

NRS 631.190 Powers and duties. [Effective January 1, 2020.] In addition to the powers and duties provided in this chapter, the Board shall:

1. Adopt rules and regulations necessary to carry out the provisions of this chapter.

2. Appoint such committees, review panels, examiners, officers, employees, agents, attorneys, investigators and other professional consultants and define their duties and incur such expense as it may deem proper or necessary to carry out the provisions of this chapter, the expense to be paid as provided in this chapter.

3. Fix the time and place for and conduct examinations for the granting of licenses to practice dentistry, dental hygiene and dental therapy.

4. Examine applicants for licenses to practice dentistry, dental hygiene and dental therapy.

5. Collect and apply fees as provided in this chapter.

6. Keep a register of all dentists, dental hygienists and dental therapists licensed in this State, together with their addresses, license numbers and renewal certificate numbers.

7. Have and use a common seal.

8. Keep such records as may be necessary to report the acts and proceedings of the Board. Except as otherwise provided in NRS 631.368, the records must be open to public inspection.

9. Maintain offices in as many localities in the State as it finds necessary to carry out the provisions of this chapter.

10. Have discretion to examine work authorizations in dental offices or dental laboratories.

[Part 4:152:1951; A 1953, 363] — (NRS A 1963, 150; 1967, 865; 1993, 2743; 2009, 3002; 2017, 989, 2848; 2019, 3205, effective January 1, 2020)

NAC 631.2213 Permit required; qualifications of applicants. (NRS 631.190, 631.265)

1. Except as otherwise set forth in NAC 631.2211 to 631.2256, inclusive, no dentist may:

(a) Use general anesthesia or deep sedation for dental patients, except in a facility for which a permit is held as required by NRS 449.442, unless he or she first:

(1) Obtains a general anesthesia permit; or

(2) Employs a dentist who is licensed in this State and who holds a general anesthesia permit to administer general anesthesia to his or her patients, and obtains a certificate of site approval for each location at which general anesthesia, deep sedation or moderate sedation is administered to his or her patients;

(b) Use moderate sedation for dental patients who are 13 years of age or older, except in a facility for which a permit is held as required by NRS 449.442, unless he or she first:

(1) Obtains a general anesthesia permit or a moderate sedation permit pursuant to paragraph (a) of subsection 2; or

(2) Employs a dentist who is licensed in this State and who holds a general anesthesia permit or a moderate sedation permit pursuant to paragraph (a) of subsection 2 to administer moderate sedation to his or her patients who are 13 years of age or older, and obtains a certificate of site approval for each location at which moderate sedation is administered to his or her patients who are 13 years of age or older; or

(c) Use moderate sedation for dental patients who are 12 years of age or younger, except in a facility for which a permit is held as required by NRS 449.442, unless he or she first:

(1) Obtains a moderate sedation permit pursuant to paragraph (b) of subsection 2; or

(2) Employs a dentist who is licensed in this State and who holds a general anesthesia permit or a moderate sedation permit pursuant to paragraph (b) of subsection 2 to administer moderate sedation to his or her patients who are 12 years of age or younger, and obtains a certificate of site approval for each location at which moderate sedation is administered to his or her patients who are 12 years of age or younger.

2. To obtain a general anesthesia permit or moderate sedation permit, a dentist must apply to the Board for such a permit on a form prescribed by the Board, submit any fees that are set by the Board pursuant to NRS 631.345 and produce evidence showing that he or she is a dentist who is licensed in this State, and:

(a) For a moderate sedation permit to administer moderate sedation to a patient 13 years of age or older, the applicant must show evidence of:

(1) The completion of a course of study, subject to the approval of the Board, of not less than 60 hours dedicated exclusively to the administration of moderate sedation, and the successful administration as the operator of moderate sedation to not less than 20 patients; or

(2) The completion of a program for specialty training which is approved by the Commission on Dental Accreditation of the American Dental Association and which includes education and training in the administration of moderate sedation that is equivalent to the education and training described in subparagraph (1) and:

(I) Valid certification in Advanced Cardiac Life Support by the American Heart Association; or

(II) The completion of a course approved by the Board that provides instruction on medical emergencies and airway management.

(b) For a moderate sedation permit to administer moderate sedation to a patient 12 years of age or younger, the applicant must show evidence of:

(1) The completion of a course of study, subject to the approval of the Board, of not less than 60 hours dedicated exclusively to the administration of moderate sedation to patients 12 years of age or younger, and the successful administration as the operator of moderate sedation to not less than 25 patients who are 12 years of age or younger; or

(2) The completion of a program for specialty training which is approved by the Commission on Dental Accreditation of the American Dental Association and which includes education and training in the administration of moderate sedation that is equivalent to the education and training described in subparagraph (1) and:

(I) Valid certification in Pediatric Advanced Life Support by the American Heart Association; or

(II) The completion of a course approved by the Board that provides instruction on medical emergencies and airway management.

(c) For a general anesthesia permit, the applicant must show evidence of the completion of an Advanced Cardiac Life Support course given by the American Heart Association or a course providing similar instruction that is approved by the Board, and:

(1) The completion of a program, subject to the approval of the Board, of advanced training in anesthesiology and related academic subjects beyond the level of undergraduate dental school in a training program as described in the *Guidelines for Teaching Pain Control and Sedation to Dentists and Dental Students*, published by the American Dental Association, 211 East Chicago Avenue, Chicago, Illinois 60611, and available, free of charge, at the Internet address **http://www.ada.org/~/media/ADA/Education%20and%20Careers/Files/ADA_Sedation_Teaching_Guidelines.pdf?la=en**; or

(2) The completion of a graduate program in oral and maxillofacial surgery or dental anesthesiology which has been approved by the Commission on Dental Accreditation of the American Dental Association.

3. A holder of a general anesthesia permit may administer general anesthesia, deep sedation or moderate sedation to a patient of any age.

(Added to NAC by Bd. of Dental Exam'rs, eff. 10-21-83; A by R005-99, 9-7-2000; R159-08, 4-23-2009; R004-17, 5-16-2018)

MODERATE SEDATION PROGRAM PROVIDER APPROVAL REQUEST
(Patients 13 years of age or older)
SUBMISSION GUIDELINES

Please comply with the following:

I certify that the program if granted Board approval will be conducted as an education program and will meet the following minimum requirements:

1) That instruction shall be conducted on the same educational standards of scholarship and teaching as that required of a true university discipline.

2) The course or topic of instruction shall conform to the purpose and method of higher education.

3) The completion of a course of study, subject to the approval of the Board, of not less than 60 Hours dedicated exclusively to the administration of moderate sedation, and the successful administration as the operator of moderate sedation to no less than 20 patients

FEE (FOR "FOR PROFIT" ORGANIZATIONS): $150.00 FOR THE FIRST CREDIT HOUR REQUESTED, $50.00 FOR EACH ADDITIONAL CREDIT HOUR. **THIS FEE IS FOR THE PROCESSING AND REVIEW OF YOUR REQUEST FOR PROVIDER APPROVAL AND MUST ACCOMPANY THIS FORM UPON SUBMISSION OF THE REQUEST.**

ALL PROVIDER APPROVAL REQUESTS MUST BE SUBMITTED TO THE BOARD FOR REVIEW NO LATER THAN 45 DAYS PRIOR TO THE BEGINNING DATE OF THE COURSE.

Nevada State Board of Dental Examiners
6010 S. Rainbow Blvd., Bldg. A, Ste. 1
Las Vegas, NV 89118
(702) 486-7044 • (800) DDS-EXAM • Fax (702) 486-7046

MODERATE SEDATION PROGRAM PROVIDER APPROVAL REQUEST
(Patients 13 years of age or older)

Pursuant to NAC 631.2213(2)(a) which states: The completion of a course of study, subject to the approval of the Board, of not less than 60 Hours dedicated exclusively to the administration of moderate sedation, and the successful administration as the operator of moderate sedation to no less than 20 patients:

Business Name: [redacted]
Business Address: [redacted]
City, State & Zip: [redacted]
Business Telephone: [redacted]
Comprehensive Course Materials and Objectives: **Please submit copies of all course materials.**

Hours of Actual Instruction: 90
Location/Facility Name, Address and instructors Name: [redacted]

Date(s) of Course: Monthly, please see attached schedule
Individual Submitting Request: David Palmer, Esq.
Business Address: [redacted]
City, State & Zip: [redacted]
Business Telephone: [redacted]
Date of Request: 6/10/2024

Signature of Person Authorized to Represent Program Provider

PLEASE ATTACH NAMES AND BRIEF BIOGRAPHICAL SKETCHES OF INSTRUCTORS AND OUTLINE OF COURSE, INCLUDING METHOD OF PRESENTATION TO THIS FORM.

FOR OFFICE USE ONLY - DO NOT WRITE BELOW THIS LINE.

Approved by:
Number of Hours Approved:
Effective Date of Approval:
Disapproved [Explanation]:

D·O·C·S EDUCATION

LEADERSHIP · COMMUNITY

2024 IV SEDATION CERTIFICATION COURSE

In Affiliation with

Idaho State University

MEHARRY® MEDICAL COLLEGE

Table of Contents:

1	Introduction	Pages 3-5
2	Complete faculty list with formal CV of each faculty member	Exhibit A 6-63
3	Complete course breakdown including teaching and lecture assignments Course Topics, and Objectives	Exhibit B 64-68
4	Certificates for ISU and Meharry	Exhibit C 69-75

RE: IV SEDATION CERTIFICATION COURSE
FROM DOCS EDUCATION

Thank you very much for reviewing the DOCS Education IV Sedation course for dentistry for approval in Nevada

DOCS Education is excited to offer this unique, comprehensive course in affiliation with ADA-CODA educational institutions, Idaho State University and Meharry Medical College.

After successful completion of the course, a dentist will qualify to provide IV Sedation in almost every U.S. state and Canadian province.

Course Description

The course is designed to provide the participant with the knowledge necessary to safely administer IV Sedation in the dental office setting. The course meets or exceeds the "Guidelines for Teaching Pain Control and Sedation to Dentists and Dental Students" (Oct. 2016) of the American Dental Association. It consists of a total of ninety (90) CE units as follows:

1. Sixty (60) hours of didactic instruction. This includes 50 hours of home-study divided into four modules, each with its own test, plus four corresponding 1-hour Office Hours sessions via Zoom/Skype with direct two-way audio/visual contact with the faculty.

2. A clinical module, during which each dentist personally administers IV Sedation to at least twenty (20) dental patients, while providing dental care in a one-to-one instructor-participant ratio. Class size is limited to 7 doctors, plus their assistants. All patients are carefully pre-screened with a thorough health history, and only ASA I or II are selected. The clinical module also includes emergency medical training on a high-fidelity patient simulator.

DOCS Education Provides the Following Equipment, Supplies, and Services:

1. **Home-Study Course:** Four modules with six chapters in each, including three hours of opioid training.
2. **Course Manual:** 351 pages detailing each chapter of the home-study that follows along with each session.
3. **Office Hours:** Video conferencing with faculty in four required sessions, one for each module.
    a. Module quizzes help faculty assess completion of required study.
4. **Final Exam:** Proctored final exam to verify competence.
5. **Pre-Clinical Session (Lexington, KY; Salt Lake City, UT):** For practicing dentists to prepare for three days of providing IV Sedation to 20 patients.
    a. Medical Emergency training with a high-fidelity patient simulator.
    b. Doctor and at least one assistant training on IV access and all equipment used during treatment.

6. Clinical Session: (Lexington, KY; Salt Lake City,UT): Three intense days of treating patients with one-to-one oversight by faculty.

a. The student-instructor ratio is 1:1 in the early states of participation. In later states, student-instructor ratio is 2:1.

b. Participants will personally perform pre-anesthetic evaluation on each patient, perform the sedation and dentistry, monitor the patient, and follow them through to discharge.

c. The following agents are taught:

Midazolam (IV)
Fentanyl (IV)
Triazolam (oral)
Lorazepam (oral)
Diazepam (IV [emergency anticonvulsant] & oral)

The Course Can be Offered on the Following Suggested Schedule:

1. Didactic Home-Study: May be started at any time

a. Office Hours are provided each month after business hours for convenience.

2. Clinical Sessions (for practicing dentists): Run once a month for groups of seven dentists with at least one team member.

a. Remaining 2024 Clinic Dates

August 7-10 (Lexington)
September 18-21 (Salt Lake City)
September 25-28 (Lexington)
October 9-12 (Lexington)
October 23-26 (SLC)
November 6-9 (Lexington)
2025 dates-TBD

Taught by World-Class Educators

Our faculty includes renowned dental educators, Dr. Anthony Feck, Dr. Leslie Fang, Dr. Taylor Tate, Dr. Carol Wilson, Dr. Ray Coleman, Dr. Steve Yun, and James Bovia, EMT. (A full formal CV of each faculty member is attached as **Exhibit A**. Faculty state licenses and NPI Self-Queries with malpractice histories with NPI numbers are available upon request.) Anthony Feck, DMD, is the primary educational director in charge of and responsible for determining that participants have been trained to competence.

The complete course breakdown including teaching and lecture assignments as well as clinical teaching assignments of all faculties and the time spent in each activity, course topics, and objectives is attached as **Exhibit B**.

The course is taught under the auspices of ADA-CODA educational institutions, Idaho State University Department of Dental Sciences and Meharry Medical College School of Dentistry. Certifications for ISU and Meharry are attached as **Exhibit C.**

Thank You

Thank you for your time and consideration of our course. If you have any questions or require any additional information, please contact me at (206) 350-4889 or david@docsedu.com.

Kindest Regards,

Michael Silverman, DMD, President & Founder DOCS Education

Fellow International Academy for Dental Facial Esthetics
Fellow American College of Dentistry
Fellow International College of Dentistry
Diplomate International Congress of Oral Implantologists

EXHIBIT A

ANTHONY S. FECK, DMD

Education:

1983	DMD University of Kentucky College of Dentistry - one of only three to graduate with High Distinction. Omicron Kappa Upsilon Honorary Dental Fraternity
1979	University of Kentucky

Postdoctoral Training:

2010	Duquesne University – IV Sedation Training
2009	University of Kentucky – IV Sedation Mini Residency
2007 - Present	Midwest Implant Institute
2002 - Present	Advanced Cardiac Life Support Certified
2002 - Present	John Kois – Kois Center for Advanced Dental Learning
2000	Montefiore Hospital IV Sedation Mini Residency, Bronx, NY
1998 - 1999	Misch Implant Institute
1995 - 1997	Peter Dawson – Center for Advanced Dental Studies
1983 - 2008	Over 2300 Hours of Continuing Education spanning the spectrum of the Dental Profession from Anesthesia to Implantology, Cosmetics, Endodontics, and Complex Restorative Dentistry.

Licensure and Certification:

1983 – Present	Licensed to practice Dentistry in Kentucky
2002 - Present	Advanced Cardiac Life Support
2006 - Present	Anesthesia Permit in Kentucky

Faculty:

2018 - Present	Idaho State University
2018 - Present	Meharry Medical College
2018 - 2018	DOCS Education IV Sedation Clinical Instructor
2017 – 2021	Oregon Health Sciences University
2016 - 2018	On-Call Urgent Dental
2013 - 2019	Little Implant Co. (Formerly OsteoReady) Clinical Educator
2011 – 2016	Six Month Smiles Certified Clinical Instructor
2011 – 2013	University of Florida College of Dentistry
2019 – 2012	American Academy of Facial Esthetics
2006 - 2014	gIDE Global Institute for Dental Education
2004 - 2018	New York University College of Dentistry, C.E. Division
2004 - Present	Sunrise Dental Solutions, Owner & Director of Education
2000 - Present	Dean of Faculty - Dental Organization for Conscious Sedation
1996 - 2004	MasterPlan Alliance
1996 - 2000	Dental Boot Kamp Continuum
1983 - 1987	University of Kentucky College of Dentistry, Department of Periodontics

Study Clubs:

2018 - Present	Sunrise Dental Solutions Study Club
2007 - 2011	Band of Brothers Mastermind
2004 - 2007	Mile High Mastermind Soaring Eagles
2002 - 2004	Mastermind

Associations:

2013 – 2016	Academy of Periointegration
2013 – 2016	American Academy of Dental Sleep Medicine
2009 – 2012	American Academy of Facial Esthetics
2008 – 2020	American Association of Dental Legal Consultants
2003 - Present	American Dental Society of Anesthesiology
2003 - 2007	Crown Council Qualified Member
2000 - Present	Dental Organization for Conscious Sedation
1994 - 2000	International Congress of Oral Implantologists
1985 - 2020	Academy of General Dentistry
1983 - 2020	American Dental Association

Awards & Recognition:

2021	Dentistry Today, Listed among the "Leaders in CE"
2020	Inducted into the International College of Dentists
2019	For contributions to dental implantology education, dental Implant from The Little Implant Co. named "The Tony Feck"
2010	Outstanding Education Award – DOCS Education
2010	American Academy of Facial Esthetics Diplomate
2006	Dental Organization for Conscious Sedation Diplomate
2003	Dental Organization for Conscious Sedation Fellowship
2000	MAP recognition for Outstanding Performance
1999	MAP recognition for Dental Leadership

Community Service:

2018 – Present	Giving Smiles Back Lexington, KY
2010 - 2011	Dentistry from The Heart Chickasha, OK (2010) Bowling Green, KY (2011) Lions Club International
1983 - 1996	Lions Club International

Family:

[redacted]

Avocations:

Reading, Writing
Sports - Boating, Golf
Computers

Professional Experience:

1995 – Present	Business Solutions Consultant and Coach
1988 - Present	Speaker, Educator, Author on Dental Topics
1983 - 2023	Private Practice Dentistry

Positions Held:

2017 – 2019	Advisory Board, Little Implant Co. (Formerly OsteoReady LLC)
2016 – 2017	Faculty, Transition Time
2016 – 2018	Faculty, On-Call Urgent Care
2013 – 2019	Faculty, Little Implant Co. (Formerly OsteoReady)
2012 – 2016	Member, American Board of Facial Esthetics
2012 – 2015	Clinical Director – Six Month Smiles

2010 - Present	Director of IV Sedation – DOCS Education
2008 - Present	Chairman, Dentcubator General Dentistry Committee
2007- Present	Editor, DOCS Digest
2006 - Present	Dental Organization for Conscious Sedation, Dean of Faculty
2004 - Present	Sunrise Dental Solutions, President
2003 - 2006	Dental Organization for Conscious Sedation Vice-President
1997 - 1998	Managing Director of the Academy of Dental Consultants
1983 - 1987	Associate Professor, University of Kentucky College of Dentistry

Media Interviews:

2017	The Wellness Hour – Implants, Sedation, TransitionReady
2012	The Wellness Hour – Implants, Six Month Smiles
2009	Dental Tribune International – Sedation Dentistry
2008	Dental Tribune International – Sedation Dentistry
2005	WLEX TV, Lexington, KY - Oral Cancer Detection
2001	WVLK TV, Lexington, KY - Digital Radiographs

Published Manuals/Books:

2022	"Building the Prosperous Dental Practice – Financial Considerations". EDRA Publishing
2016	"Implants: Simplified, OsteoReady Institute Mini-Residency Course"
2013	"Six Month Smiles Provider Training – Level 2"
2012	"Six Month Smiles Provider Training – Level 1"
2009	"IV Sedation Update'
2005	"Sedation Solutions"
2003	"Complex Adult Oral Sedation"
2002	"Adult Oral Sedation"
2002	"Anxiolysis – Beyond Valium"
2001	"Complex Sedation Patient"
2000	"The Essentials of Oral Sedation"

Dental Board Presentations:

2009	Texas
2009	Missouri
2009	Florida
2008	Missouri
2007	Arkansas
2007	North Carolina
2002	Texas

Clinics and Courses Presented To Universities or Professional Organizations:

November 15-18, 2023	IV Sedation Dentistry Lexington, KY
October 11-14, 2023	IV Sedation Dentistry Lexington, KY
October 6-7, 2023	Adult Oral Sedation Dentistry Recertification Lexington, KY
September 13-16, 2023	IV Sedation Dentistry Lexington, KY
August 25-26, 2023	Master Series – Advanced Sedation Memphis, TN
August 25-26, 2023	Adult Oral Sedation Dentistry Recertification Memphis, TN
August 9-12, 2023	IV Sedation Dentistry Lexington, KY
July 12-15, 2023	IV Sedation Dentistry Lexington, KY
June 14-17, 2023	IV Sedation Dentistry Lexington, KY
May 24-27, 2023	IV Sedation Dentistry Lexington, KY
May 19-20, 2023	Light Sedation: N2O & Oral Sedatives Live Streaming Event
May 19-20, 2023	Master Series – Advanced Sedation Live Streaming Event
May 19-20, 2023	Master Series – Advanced Sedation Live Streaming Event
May 19-20, 2023	Adult Oral Sedation Dentistry Recertification Live Streaming Event
May 19-20, 2023	Adult Oral Sedation Dentistry Live Streaming Event
April 12-15, 2023	IV Sedation Dentistry Lexington, KY
March 10-11, 2023	Master Series – Advanced Sedation Chicago, IL
March 10-11, 2023	Adult Oral Sedation Dentistry Recertification Chicago, IL
March 1-4, 2023	IV Sedation Dentistry Lexington, KY
February 24-25, 2023	Light Sedation: N2O & Oral Sedatives Live Streaming Event
February 24-25, 2023	Master Series – Advanced Sedation Live Streaming Event
February 24-25, 2023	Master Series – Advanced Sedation Live Streaming Event

February 24-25, 2023	Adult Oral Sedation Dentistry Recertification Live Streaming Event
February 24-25, 2023	Adult Oral Sedation Dentistry Live Streaming Event
Feb 8-11, 2023	IV Sedation Dentistry Lexington, KY
Jan 11-14, 2023	IV Sedation Dentistry Lexington, KY
November 9-12, 2022	IV Sedation Dentistry Lexington, KY
October 12-15, 2022	IV Sedation Dentistry Lexington, KY
October 7-8, 2022	Light Sedation: N2O & Oral Sedatives Live Streaming Event
October 7-8, 2022	Master Series – Advanced Sedation Live Streaming Event
October 7-8, 2022	Master Series – Advanced Sedation Live Streaming Event
October 7-8, 2022	Adult Oral Sedation Dentistry Recertification Live Streaming Event
October 7-8, 2022	Adult Oral Sedation Dentistry Live Streaming Event
September 7-10, 2022	IV Sedation Dentistry Lexington, KY
August 19-20, 2022	Adult Oral Sedation Dentistry Recertification Atlanta, GA
August 10-13, 2022	IV Sedation Dentistry Lexington, KY
July 13-16, 2022	IV Sedation Dentistry Lexington, KY
June 22-25, 2022	IV Sedation Dentistry Lexington, KY
June 4, 2022	Adult Oral Sedation Dentistry Recertification Irvine, CA
May 20-21, 2022	IV Sedation Dentistry Recertification Live Streaming Event
May 20-21, 2022	Light Sedation: N2O & Oral Sedatives Live Streaming Event
May 20-21, 2022	Master Series – Advanced Sedation Live Streaming Event
May 20-21, 2022	Adult Oral Sedation Dentistry Recertification Live Streaming Event
May 20-21, 2022	Adult Oral Sedation Dentistry Live Streaming Event
May 11-14, 2022	IV Sedation Dentistry Lexington, KY
April 6-9, 2022	IV Sedation Dentistry Lexington, KY

March 16-19, 2022	IV Sedation Dentistry Lexington, KY
February 18-19, 2022	IV Sedation Dentistry Recertification Live Streaming Event
February 18-19, 2022	Light Sedation: N2O & Oral Sedatives Live Streaming Event
February 18-19, 2022	Master Series – Advanced Sedation Live Streaming Event
February 18-19, 2022	Adult Oral Sedation Dentistry Recertification Live Streaming Event
February 18-19, 2022	Adult Oral Sedation Dentistry Live Streaming Event
February 9-12, 2022	IV Sedation Dentistry Lexington, KY
January 12-15, 2022	IV Sedation Dentistry Lexington, KY
November 20-21, 2021	Adult Oral Sedation Dentistry Live Streaming Event for Afdent
November 10-13-, 2021	IV Sedation Dentistry Lexington, KY
October 1-2, 2021	Master Series – Advanced Sedation Live Streaming Event
October 1-2, 2021	Light Sedation: N2O & Oral Sedatives Live Streaming Event
October 1-2, 2021	IV Sedation Dentistry Recertification Live Streaming Event
October 1-2, 2021	Adult Oral Sedation Dentistry Live Streaming Event
October 1-2, 2021	Adult Oral Sedation Dentistry Recertification Live Streaming Event
October 6-9, 2021	IV Sedation Dentistry Lexington, KY
September 15-18, 2021	IV Sedation Dentistry Lexington, KY
August 20-21, 2021	Adult Oral Sedation Dentistry Recertification Nashville, TN
August 11-14, 2021	IV Sedation Dentistry Lexington, KY
July 14-17, 2021	IV Sedation Dentistry Lexington, KY
June 23-26, 2021	IV Sedation Dentistry Lexington, KY
May 21-22, 2021	Adult Oral Sedation Dentistry Live Streaming Event

May 21-22, 2021	Adult Oral Sedation Dentistry Recertification Live Streaming Event
May 21-22, 2021	Master Series – Advanced Sedation Live Streaming Event
May 21-22, 2021	Light Sedation: N2O & Oral Sedatives Live Streaming Event
May 21-22, 2021	IV Sedation Dentistry Recertification Live Streaming Event
May 12-15, 2021	IV Sedation Dentistry Lexington, KY
April 14-17, 2021	IV Sedation Dentistry Lexington, KY
March 24-27, 2021	IV Sedation Dentistry Lexington, KY
March 22, 2021	Master Series – Advanced Sedation Chicago, IL
February 24-27, 2021	IV Sedation Dentistry Lexington, KY
February 19-20, 2021	Adult Oral Sedation Dentistry Live Streaming Event
February 19-20, 2021	Adult Oral Sedation Dentistry Recertification Live Streaming Event
February 19-20, 2021	Master Series – Advanced Sedation Live Streaming Event
February 19-20, 2021	Light Sedation: N2O & Oral Sedatives Live Streaming Event
February 19-20, 2021	IV Sedation Dentistry Recertification Live Streaming Event
January 20-23, 2021	IV Sedation Dentistry Lexington, KY
December 12, 2020	Adult Oral Sedation Dentistry Recertification Live Streaming Event
November 11-14-, 2020	IV Sedation Dentistry Lexington, KY
October 14-17, 2020	IV Sedation Dentistry Lexington, KY
October 2-3, 2020	IV Sedation Dentistry Recertification Live Streaming Event
October 2-3, 2020	Adult Oral Sedation Dentistry Live Streaming Event
October 2-3, 2020	Adult Oral Sedation Dentistry Recertification Live Streaming Event
October 2-3, 2020	Master Series – Advanced Sedation Live Streaming Event
October 2-3, 2020	Light Sedation: N2O & Oral Sedatives Live Streaming Event

September 16-19, 2020	IV Sedation Dentistry Lexington, KY
August 12-15, 2020	IV Sedation Dentistry Lexington, KY
July 22-25, 2020	IV Sedation Dentistry Lexington, KY
May 15-16, 2020	Adult Oral Sedation Dentistry Live Streaming Event
May 15-16, 2020	Adult Oral Sedation Dentistry Recertification Live Streaming Event
May 15-16, 2020	Master Series – Advanced Sedation Live Streaming Event
May 15-16, 2020	Light Sedation: N2O & Oral Sedatives Live Streaming Event
May 15-16, 2020	IV Sedation Dentistry Recertification Live Streaming Event
March 11-14, 2020	IV Sedation Dentistry Lexington, KY
February 21-23, 2020	Adult Oral Sedation Dentistry San Francisco, CA
February 21-23, 2020	Adult Oral Sedation Dentistry Recertification San Francisco, CA
February 21-23, 2020	Master Series – Advanced Sedation San Francisco, CA
February 21-23, 2020	Light Sedation: N2O & Oral Sedatives Live Streaming Event
February 21-23, 2020	IV Sedation Dentistry Recertification Live Streaming Event
February 5-8, 2020	IV Sedation Dentistry Lexington, KY
January 15-18, 2020	IV Sedation Dentistry Lexington, KY
December 11-14, 2019	IV Sedation Dentistry Lexington, KY
November 20-23, 2019	IV Sedation Dentistry Lexington, KY
November 9-10, 2019	IV Sedation Dentistry Recertification Seattle, WA
November 9-10, 2019	Master Series – Advanced Sedation Seattle, WA
November 9-10, 2019	Light Sedation: N2O & Oral Sedatives Seattle, WA
November 8-10, 2019	Adult Oral Sedation Dentistry Seattle, WA
November 8-10, 2019	Adult Oral Sedation Dentistry Recertification Seattle, WA

Anthony S. Feck, DMD continued

August 2019	"Master Series: Advanced Sedation" "Oral Sedation Dentistry" "IV Sedation Recertification" Memphis, TN
July 2019	"IV Sedation Certification" Lexington, KY
June 2019	"IV Sedation Certification" Lexington, KY
May 2019	"IV Sedation Certification" Lexington, KY
May 2019	"Oral Sedation Dentistry" "Master Series: Advanced Sedation" "IV Sedation Recertification" Arlington, VA
April 2019	IV Sedation Certification Lexington, KY
February 2019	"Oral Sedation Dentistry" "Master Series: Advanced Sedation" San Diego, CA
December 2018	"Little Implant Co. Mini-Residency" Lexington, KY
November 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
November 2018	"Oral Sedation Dentistry" "Masters Series" Scottsdale, AZ
October 2018	"Nitrous Oxide & Single Dose Enteral Sedation" Las Vegas, NV
October 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
October 2018	"Enteral Sedation Recertification for California Dentists" Irvine, CA
September 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
August 2018	"Oral Sedation Dentistry" "Masters Series" "IV Sedation Recertification" Atlanta, GA
July 2018	"Sedation Dentistry" – for Implant Educators Tampa, FL
July 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
June 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
May 2018	"Oral Sedation Dentistry" "Masters Series" "IV Sedation Recertification" Chicago, IL

Anthony S. Feck, DMD continued

May 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
April 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
March 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
February 2018	"Oral Sedation Dentistry" "Masters Series" San Francisco, CA
February 2018	"OsteoReady Institute Mini-Residency" Lexington, KY
December 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
November 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
November 2017	"Oral Sedation Dentistry" "Masters Series" Seattle, WA
October 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
October 2017	"Nitrous Oxide/Single Dose Enteral Sedation" Dallas, TX
September 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
August 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
August 2017	"Oral Sedation Dentistry" "Masters Series" "IV Sedation Recertification" Memphis, TN
July 2017	"Sedation Dentistry" – for Implant Educators Tampa, FL
July 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
June 2017	"Oral Sedation Dentistry" California Implant Institute San Diego, CA
June 2017	"IV Conscious Sedation Recertification – Tennessee" Chattanooga Dental Care
May 2017	"Oral Sedation Dentistry" "Masters Series" "IV Sedation Recertification" Reston, VA
April 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
March 2017	"OsteoReady Institute Mini-Residency" Lexington, KY
February 2017	"OsteoReady Institute Mini-Residency" Lexington, KY

February 2017	"Oral Sedation Dentistry" "Advanced Sedation" Newport Beach, CA
February 2017	"Strategic Winning Attitude Team Training" Sunrise Dental Solutions Dallas, TX
January 2017	"OsteoReady Institute Mini-Residency" Lexington, KY

Dr. Feck has presented hundreds of courses between 2008 and 2020. For a full list, ask DOCS Education.

Sponsorship Affiliations

- Aurum Dental Laboratories
- Demandforce
- American Eagle Instruments
- Golden Misch – Physics Forceps
- Dental Technology Consultants
- The Health Chair
- Darby Dental Supply
- Center for Employee Dispute Resolution
- Spectro-Shade Matching Instrument

Communication/Practice Management Lectures:

- Seattle Study Club Annual Symposium 2022 – "The Prosperous Dental Practice: Strategies for Practices that Don't Want to Go Corporate"
- Seattle Study Club – "From Economic Uncertainty to Financial Security Building an Updated Financial Plan"
- Annual Speaker for the Sunrise Dental Solutions Summit (2005-Present)
- Speaker & Instructor for "Strategic Winning Attitude Team Training"
- Creator and Speaker for "CEO – Challenging and Encouraging Ownership" for Sunrise Dental Solutions
- Creator and Speaker for "Production Explosion" for Sunrise Dental Solutions
- Creator and Speaker for "The Ultimate Team" for Sunrise Dental Solutions
- Creator and Speaker for "Starting Point" for Sunrise Dental Solutions
- Creator and Speaker for "Own Your Market" for Sunrise Dental Solutions
- Instructor for "Hands-On Dental Boot Kamp"
- Co-Creator & Lead Speaker for "The Leadership Challenge"
- Co-Creator & Lead Speaker for "MAP II" of the Master Plan Alliance

Articles Published:

2020	New.Dentist.com – "5 Game-Changing Strategies for Practice Growth"
2019	New.Dentist.com – "Antiresorptive Medications & Medication Related Osteonecrosis of the Jaw"
2015	DOCS Digest (January) – "Make More. Keep More. & Enjoy It More –Developing A Master Plan for a Successful 2015"
2014	Dental Economics (July) – "Four Game Changers For Your Practice In 2014"
2013	The New Dentist – "A New Era of Biocompatible Dental Implants –Achieving Periointegration with Zirconium Nitride Coating"
2012	2012 Dental Economics (December) – "Preparing for Medical Emergencies in the Dental Office"
2012	2012 American Academy of Cosmetic Dentistry (March, Vol.17, Issue 6)) – "Short-Term Orthodontics, The Platinum Rule And The Business Of Dentistry"
2010	2010 Dental Economics (October) – "Predictable, Atraumatic Dental Extractions"
2010	2010 Dental Products Report (August) – "Wondering what's next? – Botox and dermal filler therapy could represent the next big opportunity for growth in cosmetic dentistry"
2010	Australian Dentist (May/June) – "The One-Minute Atraumatic Extraction"

2010	Dentistry Today (April) – "Total Facial Esthetics For Every Dental Practice"
2010	California Dental Association Journal – "Evaluating the Potential Benefits of Combining Oral and IV Sedation Protocols in Treating the Anxious Dental Patient"
2009	Aurum Ceramic Continuum - "Be An Agent of Change – Become a "Dentrepreneur"
2009	Dental Economics – "Profitability Comparison of Three Forms of Sedation"
2009	Dental Products Shopper – "The Sedation Driven Practice – A Business Model"
2008	Dental Tribune UK - "Thriving In An Economic Downturn"
2008	Dental Economics – "Sedation Architectonics"
2008	DOCS Digest – "Post-Operative Analgesia for Sedation Patients"
2008	Dental Tribune – "Recession Proof Your Practice"
2008	Dental Tribune – "The Five Words To More High Quality New Patients"
2008	Dental Economics – "Incorporating Sedation Dentistry Into Your Practice"
2006	Compendium of Continuing Dental Education – "Local Anesthesia Aided by Oral Conscious Sedation"
2005	Journal of the Academy of General Dentistry – "Oral Conscious Sedation"
2003	Dentistry Today – "Anxiolysis in General Dental Practice"
1999	Dental Products Report – "Marketing for the Dental Practice"
1998	Dental Economics – "Treatment Planning"
1983	Journal of the American Student Dental Association – "Acupuncture"

Leslie Shu-Tung Fang, MD, PhD

Education

1967	BSc	University of Illinois, Champaign-Urbana, Illinois (Bronze Tablet)
1969	MSc	University of Illinois, Champaign-Urbana, Illinois
1971	PhD	University of Illinois, Champaign-Urbana, Illinois Physiology and Biophysics
1974	MD	Harvard Medical School, Boston Massachusetts

Postdoctoral Training

Internship and Residencies:

1974-1975	Internship in Medicine, Massachusetts General Hospital
1975-1976	Assistant Resident in Medicine, Massachusetts General Hospital
1978-1979	Senior Resident in Medicine, Massachusetts General Hospital
1979-1980	Chief Resident in Medicine, Massachusetts General Hospital

Fellowship:

1976-1977	Clinical Fellow in Medicine (Nephrology), Massachusetts General Hospital
1977-1978	Clinical and Research Fellow in Medicine (Nephrology), Massachusetts General Hospital

Licensure and Certification:

1975	Massachusetts License Registration
1975	American Board of Internal Medicine, Diplomate
1980	American Board of Internal Medicine, Diplomate in Subspecialty of Nephrology

Academic and Hospital Appointments

Academic Appointments:

1967-1971	Teaching Assistant, Department of Physiology and Biophysics, University of Illinois, Champaign-Urbana, Illinois
1971	Instructor, Department of Physiology and Biophysics University of Illinois, Champaign-Urbana, Illinois
1974-1979	Teaching Fellow in Medicine, Harvard Medical School
1979-1983	Instructor in Medicine, Harvard Medical School
1983- Present	Assistant Professor in Medicine, Harvard Medical School

Hospital Appointments:

1980-1984	Assistant in Medicine, Massachusetts General Hospital
1980-1997	Clerkship Director, Core Clerkship in Medicine,

	Massachusetts General Hospital
1980-Present	Staff Physician, Renal Unit, Massachusetts General Hospital
1984-1990	Assistant Physician, Massachusetts General Hospital
1990-1993	Associate Physician, Massachusetts General Hospital
1993-Present	Physician, Massachusetts General Hospital

Hospital Service Responsibilities:

1980-Present	Attending Physician, Medical Service Massachusetts General Hospital
1980-Present	Attending Physician, Renal Service, Massachusetts General Hospital
1980-Present	Attending Physician, Primary Care Unit, Massachusetts General Hospital
1980-1990	Assistant in Medicine, Massachusetts General Hospital
1980-1997	Associate Director, Hemodialysis Unit, Massachusetts General Hospital
1984-1988	Co-Director, Chronic Ambulatory Peritoneal Dialysis Unit, Massachusetts General Hospital
1980-1997	Associate Director, Hemodialysis Unit Massachusetts General Hospital
1989-1997	Firm Chief, Bauer Firm, Medical Services, Massachusetts General Hospital

Major Administrative Responsibilities

1986-1987	Acting Program Director, Department of Medicine, Massachusetts General Hospital
1989-1997	Clerkship Director, Advanced Clerkship in Medicine, Massachusetts General Hospital
1989-1997	Firm Chief, Walter Bauer Firm, Medical Service, Massachusetts General Hospital
1991-1994	Coordinator, Private Service Team IV, Medical Service, Massachusetts General Hospital
1991-1992	Coordinator, Private Service Team V, Medical Service, Massachusetts General Hospital

Major Committee Assignments

Harvard Medical School:

1981-1997	Committee on Clinical Clerkship, Harvard Medical School
1984	Committee on Minority Recruitment, Harvard Medical School
1985	Committee on Student Evaluation, Harvard Medical School
1986	Committee on Stress in Residency Training, Harvard Medical School
1987-1988	Committee on Educational Evaluation, Surgery Core Clerkship Site Visits, Harvard Medical School
1987	Program on Student Evaluation, New Pathway Program Harvard Medical School
1988-1997	Committee of Medical Clerkship Directors, Harvard Medical School
1989	Chairman, Committee on Education, Core Clerkship in Surgery, Harvard Medical School
1991-1997	Committee on Years III and IV, New Pathway Program, Harvard Medical School
1991-1997	Committee on Years I and II, New Pathway Program,

	Harvard Medical School
1992	Faculty Council, Harvard Medical School
1993-1996	Curriculum Committee, Health Science and Technology Program (Harvard-MIT)
1994-1996	Committee on Enrichment of Clinical Clerkship Health Science and Technology Program (Harvard-MIT)
1994-1996	Screening Committee Harvard Medical School
1994	Faculty Council Harvard Medical School
1994	Docket Committee, Faculty Council, Harvard Medical School
1995	Faculty Council Harvard Medical School
1995-96	Curriculum Committee, Year III and IV Harvard Medical School
1996	Faculty Council Harvard Medical School
1997	Faculty Council Harvard Medical School
1998	Faculty Council Harvard Medical School
1999	Faculty Council Harvard Medical School

Hospital:

1980-1997	Internship Selection Committee, Massachusetts General Hospital
1982-1985	Committee on Teaching and Education, Massachusetts General Hospital
1982-1985	Executive Committee on Teaching and Education, Massachusetts General Hospital
1983-1993	Physician Advisory Committee, Utilization Review Committee, Massachusetts General Hospital
1983-1994	Executive Committee, Utilization Review, Massachusetts General Hospital
1985-1997	Clinical Advisory Committee, Medical Service, Massachusetts General Hospital
1989-1997	Chairman, Internship Selection Committee, Medical Service, Massachusetts General Hospital
1989-1997	Residency Selection Committee, Psychiatry Department Massachusetts General Hospital
1989-1997	Residency Selection Committee, Medicine-Pediatrics Program, Massachusetts General Hospital
1989-1997	Committee on Minority Recruitment, Massachusetts General Hospital
1989-1997	Committee on Clinical Teaching, Massachusetts General Hospital
1990-1997	Training Program Committee, Medical Service, Massachusetts General Hospital
1991-1993	Emergency Department Task Force, Massachusetts General Hospital
1991-1997	Clinical Practice Advisory Committee, Massachusetts General Hospital
1991-1992	Tower II Space Allocation Committee, Massachusetts General Hospital
1991-1992	Emergency Department Space Allocation Committee, Massachusetts General Hospital
1991-1993	Clinical Practice Information Committee,

	Massachusetts General Hospital
1992-1994	Executive Committee, Massachusetts General Professional Corporation Massachusetts General Hospital
1992-1994	Chairman, Information Resources Committee, Massachusetts General Professional Corporation Massachusetts General Hospital
1992-1994	US Healthcare Negotiating Team, Massachusetts General Professional Corporation Massachusetts General Hospital
1992-1996	Global Fee Analysis Team Massachusetts General Professional Corporation Massachusetts General Hospital
1992-1997	Committee on Promotions Clinician-Teacher Track Massachusetts General Hospital
1993-1995	Information Technology Committee (ITAB) Massachusetts General Hospital
1993-1994	Executive Committee, Massachusetts General Professional Corporation Massachusetts General Hospital
1993-1995	Executive Committee on Information System Massachusetts General Hospital
1994-1996	Steering Committee, Alumni Association Massachusetts General Hospital
1994-1997	Clinical Practice Council, Department of Medicine Massachusetts General Hospital
1994-1997	Task Force on Integrated Health Care Delivery System Massachusetts General Hospital
1994-1997	Clinical Practice Committee, Massachusetts General Hospital
1994-1997	Task Force on the Evaluation of Laboratory Systems Massachusetts General Hospital
1995-1997	Physician Information Resource Committee Massachusetts General Hospital
1995-1997	Governing Board, Medical Services Associates Massachusetts General Hospital
1995-1997	Executive Board, Medical Services Associates Massachusetts General Hospital
1996-1997	Clinical Director, Medical Services Associates Massachusetts General Hospital

Professional Societies

1980-Present	International Society of Nephrology
1980-Present	American Medical Association
1982-Present	American Society of Transplant Physicians
1990-Present	Association of Clerkship Directors in Internal Medicine
1993-Present	American Society of Nephrology

Awards and Honors

1965	Phi ETA Sigma
1966	Phi Kappa Phi
1966	Gregory Scholar, University of Illinois
1967	Phi Beta Kappa
1965-1967	James Scholar, University of Illinois
1967	Bronze Tablet, University of Illinois
1969	Sigma Xi
1970	Phi Sigma
1974	Alpha Omega Alpha

1983	Boylston Society Award for Excellent in Clinical Teaching, Harvard Medical School
1989	Teaching Award for Excellence in Clinical Teaching, Harvard Medical School
1993	Faculty Award for Excellence in Clinical Teaching Harvard Medical School
1994	Teaching Award for Excellence in Clinical Teaching Harvard Medical School
1996	Teaching Award for Excellence in Clinical Teaching in Primary Care Massachusetts General Hospital
1998	The Alfred Kranes Award for Excellence in Clinical Teaching Massachusetts General Hospital
1999	Psychiatry Resident Award for Contribution to the Residency Selection Process Massachusetts General Hospital
2015	John R. Gallagher III and Katherine A. Gallagher Endowed Chair in Clinical Nephrology
2022	P-I Branemark Award for Lifetime Achievement

Part II. Research, Teaching and Clinical Contributions

I maintain a busy international practice in medicine and nephrology and continue to teach from the clinical forum. In clinical teaching, my major focus has involved the development of an interactive platform to facilitate education. Using case-based method and an audience response system, students would participate interactively to manage patients through a large variety of clinical scenarios. Pre- and post-tests would allow teachers and participants alike to gauge the effectiveness of any educational session. Interactive teaching has been extended from the education of students, residents and fellows to physicians in the community, and, most recently, to dental professionals who are interested in optimizing management of medically complex patients.

Internship and Residency: Massachusetts General Hospital
Chief Residency: Massachusetts General Hospital
Fellowship in Nephrology: Massachusetts General Hospital

Board Certification in Medicine
Board Certification in Nephrology
Board Certification in Emergency Medicine

International Practice in Nephrology and Internal Medicine at Massachusetts General Hospital

Teaching Portfolio:

February 17, 2017	Sedation Voyage Dental Organization for Conscious Sedation Newport Beach, California
February 17, 2017	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Newport Beach, California
February 17, 2017	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation Newport Beach, California
February 17, 2017	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Newport Beach, California
February 17, 2017	Airway Evaluation and Management Dental Organization for Conscious Sedation Newport Beach, California
February 17, 2017	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Newport Beach, California

February 17, 2017	Cardiac Rhythm Review Dental Organization for Conscious Sedation Newport Beach, California
February 17, 2017	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Newport Beach, California
February 18, 2017	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Newport Beach, California
February 18, 2017	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Newport Beach, California
February 18, 2017	Assessment of Pediatric Patient Dental Organization for Conscious Sedation Newport Beach, California
February 18, 2017	Assessment of Complicated Patient Dental Organization for Conscious Sedation Newport Beach, California
February 19, 2017	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Seattle, Washington
February 19, 2017	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Newport Beach, California
February 19, 2017	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Newport Beach, California
February 19, 2017	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Newport Beach, California
February 19, 2017	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Newport Beach, California
February 19, 2017	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Newport Beach, California
February 19, 2017	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Newport Beach, California
March 4, 2017	Science of Sedation New York University New York, New York
March 4, 2017	Local Anesthetics: Practical Vignettes New York University New York, New York
March 4, 2017	Patient Assessment in the Dental Office New York University New York, New York
March 4, 2017	Pharmacology of Sedation Drugs New York University New York, New York

March 4, 2017	Management of Patients with Cardiovascular Disease New York University New York, New York
March 5, 2017	Metabolism of Drugs and Drug-Drug Interactions New York University New York, New York
March 5, 2017	Antibiotic Management of Odontogenic Infections New York University New York, New York
March 5, 2017	Antibiotic Prophylaxis: Myths and Reality New York University New York, New York
March 5, 2017	Management of Patients with Renal Disease New York University New York, New York
March 5, 2017	Management of Patients with Coagulopathy New York University New York, New York
March 5, 2017	Dental Management of Patients with Liver Disease New York University New York, New York
March 5, 2017	Medical Emergencies in the Dental Office New York University New York, New York
May 19, 2017	Sedation Voyage Dental Organization for Conscious Sedation Washington, DC
May 19, 2017	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Washington, DC
May 19, 2017	Evaluation of the Pediatric Patient Dental Organization for Conscious Sedation Washington, DC
May 19, 2017	Drug-Drug Interactions in Sedation Dentistry Dental Organization for Conscious Sedation Washington, DC
May 19, 2017	Evaluation of the Complicated Pediatric Patient Dental Organization for Conscious Sedation San Diego, California
May 19, 2017	Airway Evaluation and Management Dental Organization for Conscious Sedation Washington, DC
May 20, 2017	General Principles of Pharmacology in Pediatric Sedation Dental Organization for Conscious Sedation Washington, DC
May 20, 2017	Pharmacology of Selected Drugs Dental Organization for Conscious Sedation Washington, DC
May 20, 2017	Management of Pediatric Emergencies Dental Organization for Conscious Sedation San Diego, California

May 20, 2017	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Washington, DC
May 20, 2017	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Washington, DC
May 20, 2017	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Washington, DC
May 20, 2017	Management of Patients with Diabetes Dental Organization for Conscious Sedation Washington, DC
May 20, 2017	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Washington, DC
May 21, 2017	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Washington, DC
May 21, 2017	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Washington, DC
May 21, 2017	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Washington, DC
May 21, 2017	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Washington, DC
May 21, 2017	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Washington, DC
May 21, 2017	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Washington, DC
August 18, 2017	Sedation Voyage Dental Organization for Conscious Sedation Memphis, TN
August 18, 2017	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Memphis, TN
August 18, 2017	Evaluation of the Pediatric Patient Dental Organization for Conscious Sedation Memphis, TN
August 18, 2017	Drug-Drug Interactions in Sedation Dentistry Dental Organization for Conscious Sedation Memphis, TN
August 18, 2017	Evaluation of the Complicated Pediatric Patient Dental Organization for Conscious Sedation Memphis, TN
August 18, 2017	Airway Evaluation and Management in Pediatric Patient Dental Organization for Conscious Sedation Memphis, TN

August 19, 2017	Airway Evaluation and Management in IV Sedation Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Cardiac Rhythm Review Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Management of Pediatric Emergencies Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Memphis, TN
August 19, 2017	Management of Patients with Diabetes Dental Organization for Conscious Sedation Memphis, TN
August 20, 2017	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Memphis, TN
August 20, 2017	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Memphis, TN
August 20, 2017	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Memphis, TN
August 20, 2017	Management of Patients with Renal Disease Dental Organization for Conscious Sedation San Francisco, California
August 20, 2017	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation San Francisco, California
August 20, 2017	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Memphis, TN
August 20, 2017	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Memphis, TN
November 10, 2017	Sedation Voyage Dental Organization for Conscious Sedation Seattle, Washington

November 10, 2017	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Seattle, Washington
November 10, 2017	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation Seattle, Washington
November 10, 2017	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Seattle, Washington
November 10, 2017	Airway Evaluation and Management Dental Organization for Conscious Sedation Seattle, Washington
November 10, 2017	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Seattle, Washington
November 10, 2017	Cardiac Rhythm Review Dental Organization for Conscious Sedation Seattle, Washington
November 10, 2017	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Seattle, Washington
November 11, 2017	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Seattle, Washington
November 11, 2017	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Seattle, Washington
November 11, 2017	Assessment of Pediatric Patient Dental Organization for Conscious Sedation Seattle, Washington
November 11, 2017	Assessment of Complicated Patient Dental Organization for Conscious Sedation Seattle, Washington
November 11, 2017	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Seattle, Washington
November 11, 2017	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Seattle, Washington
November 12, 2017	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Seattle, Washington
November 12, 2017	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Seattle, Washington
November 12, 2017	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Seattle, Washington
November 12, 2017	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Seattle, Washington

November 12, 2017	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Seattle, Washington
February 23, 2018	Sedation Voyage Dental Organization for Conscious Sedation San Francisco, California
February 23, 2018	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation San Francisco, California
February 23, 2018	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation San Francisco, California
February 23, 2018	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation San Francisco, California
February 23, 2018	Airway Evaluation and Management Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Cardiac Rhythm Review Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Assessment of Pediatric Patient Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Assessment of Complicated Patient Dental Organization for Conscious Sedation San Francisco, California
February 24, 2018	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation San Francisco, California
February 25, 2018	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation San Francisco, California
February 25, 2018	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation San Francisco, California
February 25, 2018	Management of Patients with Renal Disease Dental Organization for Conscious Sedation San Francisco, California

February 25, 2018	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation San Francisco, California
February 25, 2018	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation San Francisco, California
February 25, 2018	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation San Francisco, California
March 3, 2018	Science of Sedation New York University New York, New York
March 3, 2018	Local Anesthetics: Practical Vignettes New York University New York, New York
March 3, 2018	Patient Assessment in the Dental Office New York University New York, New York
March 3, 2018	Pharmacology of Sedation Drugs New York University New York, New York
March 3, 2018	Management of Patients with Cardiovascular Disease New York University New York, New York
March 3, 2018	New Drugs with Dental Implications New York University New York, New York
March 3, 2018	New Oral Anticoagulants (NOACs) New York University New York, New York
March 3, 2018	New Anti-platelet Drugs New York University New York, New York
March 3, 2018	Management of Patients with Hepatitis C New York University New York, New York
March 3, 2018	Management of Patients with Congestive Heart Failure New York University New York, New York
March 4, 2018	Metabolism of Drugs and Drug-Drug Interactions New York University New York, New York
March 4, 2018	Antibiotic Management of Odontogenic Infections New York University New York, New York
March 4, 2018	Antibiotic Prophylaxis: Myths and Reality New York University New York, New York
March 4, 2018	Management of Patients with Renal Disease New York University New York, New York

March 4, 2018	Management of Patients with Coagulopathy New York University New York, New York
March 4, 2018	Dental Management of Patients with Liver Disease New York University New York, New York
March 4, 2018	Medical Emergencies in the Dental Office New York University New York, New York
May 18, 2018	Sedation Voyage Dental Organization for Conscious Sedation Chicago, Illinois
May 18, 2018	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Chicago, Illinois
May 18, 2018	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation Chicago, Illinois
May 18, 2018	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Chicago, Illinois
May 18, 2018	Airway Evaluation and Management Dental Organization for Conscious Sedation Chicago, Illinois
May 19, 2018	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Chicago, Illinois
May 19, 2018	Cardiac Rhythm Review Dental Organization for Conscious Sedation Chicago, Illinois
May 19, 2018	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Chicago, Illinois
May 19, 2018	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Chicago, Illinois
May 19, 2018	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Chicago, Illinois
May 19, 2018	Assessment of Pediatric Patient Dental Organization for Conscious Sedation Chicago, Illinois
May 19, 2018	Assessment of Complicated Patient Dental Organization for Conscious Sedation Chicago, Illinois
May 20, 2018	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Chicago, Illinois
May 20, 2018	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Chicago, Illinois

May 20, 2018	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Chicago, Illinois
May 20, 2018	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Chicago, Illinois
May 20, 2018	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Chicago, Illinois
May 20, 2018	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Chicago, Illinois
May 20, 2018	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Chicago, Illinois
August 17, 2018	Sedation Voyage Dental Organization for Conscious Sedation Atlanta, Georgia
August 17, 2018	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Atlanta, Georgia
August 17, 2018	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation Atlanta, Georgia
August 17, 2018	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Atlanta, Georgia
August 17, 2018	Airway Evaluation and Management Dental Organization for Conscious Sedation Atlanta, Georgia
August 18, 2018	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Atlanta, Georgia
August 18, 2018	Cardiac Rhythm Review Dental Organization for Conscious Sedation Atlanta, Georgia
August 18, 2018	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Atlanta, Georgia
August 18, 2018	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Atlanta, Georgia
August 18, 2018	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Atlanta, Georgia
August 18, 2018	Assessment of Pediatric Patient Dental Organization for Conscious Sedation Atlanta, Georgia
August 18, 2018	Assessment of Complicated Patient Dental Organization for Conscious Sedation Atlanta, Georgia

August 19, 2018	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Atlanta, Georgia
August 19, 2018	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Atlanta, Georgia
August 19, 2018	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Atlanta, Georgia
August 19, 2018	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Atlanta, Georgia
August 19, 2018	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Atlanta, Georgia
August 19, 2018	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Atlanta, Georgia
August 19, 2018	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Atlanta, Georgia
November 9, 2018	Sedation Voyage Dental Organization for Conscious Sedation Scottsdale, Arizona
November 9, 2018	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Scottsdale, Arizona
November 9, 2018	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation Scottsdale, Arizona
November 9, 2018	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Scottsdale, Arizona
November 9, 2018	Airway Evaluation and Management Dental Organization for Conscious Sedation Scottsdale, Arizona
November 10, 2018	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Scottsdale, Arizona
November 10, 2018	Cardiac Rhythm Review Dental Organization for Conscious Sedation Scottsdale, Arizona
November 10, 2018	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Scottsdale, Arizona
November 10, 2018	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Scottsdale, Arizona
November 10, 2018	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Scottsdale, Arizona

November 10, 2018	Assessment of Pediatric Patient Dental Organization for Conscious Sedation Scottsdale, Arizona
November 10, 2018	Assessment of Complicated Patient Dental Organization for Conscious Sedation Scottsdale, Arizona
November 11, 2018	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Scottsdale, Arizona
November 11, 2018	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Scottsdale, Arizona
November 11, 2018	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Scottsdale, Arizona
November 11, 2018	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Scottsdale, Arizona
November 11, 2018	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Scottsdale, Arizona
November 11, 2018	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Scottsdale, Arizona
November 11, 2018	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Scottsdale, Arizona
February 22, 2019	Sedation Voyage Dental Organization for Conscious Sedation San Diego, California
February 22, 2019	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation San Diego, California
February 22, 2019	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation San Diego, California
February 22, 2019	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation San Diego, California
February 22, 2019	Airway Evaluation and Management Dental Organization for Conscious Sedation San Diego, California
February 23, 2019	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation San Diego, California
February 23, 2019	Cardiac Rhythm Review Dental Organization for Conscious Sedation San Diego, California
February 23, 2019	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation San Diego, California

February 23, 2019	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation San Diego, California
February 23, 2019	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation San Diego, California
February 23, 2019	Assessment of Pediatric Patient Dental Organization for Conscious Sedation San Diego, California
February 23, 2019	Assessment of Complicated Patient Dental Organization for Conscious Sedation San Diego, California
February 24, 2019	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation San Diego, California
February 24, 2019	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation San Diego, California
February 24, 2019	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation San Diego, California
February 24, 2019	Management of Patients with Renal Disease Dental Organization for Conscious Sedation San Diego, California
February 24, 2019	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation San Diego, California
February 24, 2019	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation San Diego, California
February 24, 2019	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation San Diego, California
May 17, 2019	Sedation Voyage Dental Organization for Conscious Sedation Arlington, Virginia
May 17, 2019	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Arlington, Virginia
May 17, 2019	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation Arlington, Virginia
May 17, 2019	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Arlington, Virginia
May 17, 2019	Airway Evaluation and Management Dental Organization for Conscious Sedation Arlington, Virginia
May 18, 2019	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Arlington, Virginia

May 18, 2019	Cardiac Rhythm Review Dental Organization for Conscious Sedation Arlington, Virginia
May 18, 2019	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Arlington, Virginia
May 18, 2019	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Arlington, Virginia
May 18, 2019	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Arlington, Virginia
May 18, 2019	Assessment of Pediatric Patient Dental Organization for Conscious Sedation Arlington, Virginia
May 18, 2019	Assessment of Complicated Patient Dental Organization for Conscious Sedation Arlington, Virginia
May 19, 2019	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Arlington, Virginia
May 19, 2019	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Arlington, Virginia
May 19, 2019	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Arlington, Virginia
May 19, 2019	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Arlington, Virginia
May 19, 2019	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Arlington, Virginia
May 19, 2019	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Arlington, Virginia
May 19, 2019	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Arlington, Virginia
August 16, 2019	Sedation Voyage Dental Organization for Conscious Sedation Memphis, Tennessee
August 16, 2019	Pharmacology of Pediatric Sedation Drugs Dental Organization for Conscious Sedation Memphis, Tennessee
August 16, 2019	Drug Metabolism and Drug-Drug Interactions Dental Organization for Conscious Sedation Memphis, Tennessee
August 16, 2019	Patient Assessment in the Dental Office Dental Organization for Conscious Sedation Memphis, Tennessee

August 16, 2019	Airway Evaluation and Management Dental Organization for Conscious Sedation Memphis, Tennessee
August 17, 2019	Patient Assessment for IV Sedation Dental Organization for Conscious Sedation Memphis, Tennessee
August 17, 2019	Cardiac Rhythm Review Dental Organization for Conscious Sedation Memphis, Tennessee
August 17, 2019	Medical Emergencies in IV Sedation Dental Organization for Conscious Sedation Memphis, Tennessee
August 17, 2019	Pharmacology of Sedation Drugs Dental Organization for Conscious Sedation Memphis, Tennessee
August 17, 2019	Management of Patients with Cardiovascular Disease Dental Organization for Conscious Sedation Memphis, Tennessee
August 17, 2019	Assessment of Pediatric Patient Dental Organization for Conscious Sedation Memphis, Tennessee
August 17, 2019	Assessment of Complicated Patient Dental Organization for Conscious Sedation Memphis, Tennessee
August 18, 2019	Metabolism of Drugs and Drug-Drug Interactions Dental Organization for Conscious Sedation Memphis, Tennessee
August 18, 2019	Antibiotic Management of Odontogenic Infections Dental Organization for Conscious Sedation Memphis, Tennessee
August 18, 2019	Antibiotic Prophylaxis: Myths and Reality Dental Organization for Conscious Sedation Memphis, Tennessee
August 18, 2019	Management of Patients with Renal Disease Dental Organization for Conscious Sedation Memphis, Tennessee
August 18, 2019	Management of Patients with Coagulopathy Dental Organization for Conscious Sedation Memphis, Tennessee
August 18, 2019	Dental Management of Patients with Liver Disease Dental Organization for Conscious Sedation Memphis, Tennessee
August 18, 2019	Medical Emergencies in the Dental Office Dental Organization for Conscious Sedation Memphis, Tennessee

November 8-10, 2019	Adult Oral Sedation Dentistry Seattle, WA
November 8-10, 2019	Adult Oral Sedation Dentistry Recertification Seattle, WA
November 9-10, 2019	Master Series – Advanced Sedation Seattle, WA
November 20-23, 2019	IV Sedation Dentistry Lexington, KY
December 11-14, 2019	IV Sedation Dentistry Lexington, KY
January 15-18, 2020	IV Sedation Dentistry Lexington, KY
February 5-8, 2020	IV Sedation Dentistry Lexington, KY
February 21-23, 2020	Adult Oral Sedation Dentistry San Francisco, CA
February 21-23, 2020	Adult Oral Sedation Dentistry Recertification San Francisco, CA
February 21-23, 2020	Master Series – Advanced Sedation San Francisco, CA
March 11-14, 2020	IV Sedation Dentistry Lexington, KY
May 15-16, 2020	Adult Oral Sedation Dentistry Live Streaming Event
May 15-16, 2020	Adult Oral Sedation Dentistry Recertification Live Streaming Event
May 15-16, 2020	Master Series – Advanced Sedation Live Streaming Event
July 22-25, 2020	IV Sedation Dentistry Lexington, KY
August 12-15, 2020	IV Sedation Dentistry Lexington, KY
September 16-19, 2020	IV Sedation Dentistry Lexington, KY
October 2-3, 2020	Adult Oral Sedation Dentistry Live Streaming Event
October 2-3, 2020	Adult Oral Sedation Dentistry Recertification Live Streaming Event

October 2-3, 2020	Master Series – Advanced Sedation Live Streaming Event
October 14-17, 2020	IV Sedation Dentistry Lexington, KY
November 11-14-, 2020	IV Sedation Dentistry Lexington, KY
Dec 12, 2020	Adult Oral Sedation Dentistry Recertification Live Streaming Event
January 20-23, 2021	IV Sedation Dentistry Lexington, KY
February 19-20, 2021	Adult Oral Sedation Dentistry Live Streaming Event
February 19-20, 2021	Adult Oral Sedation Dentistry Recertification Live Streaming Event
February 19-20, 2021	Master Series – Advanced Sedation Live Streaming Event
February 24-27, 2021	IV Sedation Dentistry Lexington, KY
March 22, 2021	Master Series – Advanced Sedation Chicago, IL
March 24-27, 2021	IV Sedation Dentistry Lexington, KY
April 14-17, 2021	IV Sedation Dentistry Lexington, KY
May 21-22, 2021	Adult Oral Sedation Dentistry Live Streaming Event
May 21-22, 2021	Adult Oral Sedation Dentistry Recertification Live Streaming Event
May 21-22, 2021	Master Series – Advanced Sedation Live Streaming Event
May 12-15, 2021	IV Sedation Dentistry Lexington, KY
June 23-26, 2021	IV Sedation Dentistry Lexington, KY
July 14-17, 2021	IV Sedation Dentistry Lexington, KY
August 11-14, 2021	IV Sedation Dentistry Lexington, KY

August 20-21, 2021	Adult Oral Sedation Dentistry Recertification Nashville, TN
September 15-18, 2021	IV Sedation Dentistry Lexington, KY
October 6-9, 2021	IV Sedation Dentistry Lexington, KY
October 1-2, 2021	Adult Oral Sedation Dentistry Live Streaming Event
October 1-2, 2021	Adult Oral Sedation Dentistry Recertification Live Streaming Event
October 1-2, 2021	Master Series – Advanced Sedation Live Streaming Event
November 10-13, 2021	IV Sedation Dentistry Lexington, KY
November 20-21, 2021	Adult Oral Sedation Dentistry Live Streaming Event for Afdent
January 12-15, 2022	IV Sedation Dentistry Lexington, KY
February 9-12, 2022	IV Sedation Dentistry Lexington, KY
February 18-19, 2022	Adult Oral Sedation Dentistry Live Streaming Event
February 18-19, 2022	Adult Oral Sedation Dentistry Recertification Live Streaming Event
February 18-19, 2022	Master Series – Advanced Sedation Live Streaming Event
March 16-19, 2022	IV Sedation Dentistry Lexington, KY
March 25-26, 2022	Advanced Medical Training Chicago, IL
April 6-9, 2022	IV Sedation Dentistry Lexington, KY
May 11-14, 2022	IV Sedation Dentistry Lexington, KY
May 20-21, 2022	Adult Oral Sedation Dentistry Live Streaming Event
May 20-21, 2022	Adult Oral Sedation Dentistry Recertification Live Streaming Event

May 20-21, 2022	Master Series – Advanced Sedation Live Streaming Event
June 4, 2022	Adult Oral Sedation Dentistry Recertification Irvine, CA
June 22-25, 2022	IV Sedation Dentistry Lexington, KY
July 13-16, 2022	IV Sedation Dentistry Lexington, KY
August 10-13, 2022	IV Sedation Dentistry Lexington, KY
August 19-20, 2022	Adult Oral Sedation Dentistry Recertification Atlanta, GA
September 7-10, 2022	IV Sedation Dentistry Lexington, KY
October 7-8, 2022	Adult Oral Sedation Dentistry Live Streaming Event
October 7-8, 2022	Adult Oral Sedation Dentistry Recertification Live Streaming Event
October 7-8, 2022	Master Series – Advanced Sedation Live Streaming Event
October 12-15,2022	IV Sedation Dentistry Lexington, KY
November 9-12, 2022	IV Sedation Dentistry Lexington, KY
Jan 11-14, 2023	IV Sedation Dentistry Lexington, KY
Feb 8-11, 2023	IV Sedation Dentistry Lexington, KY
February 24-25, 2023	Adult Oral Sedation Dentistry Live Streaming Event
February 24-25, 2023	Adult Oral Sedation Dentistry Recertification Live Streaming Event
February 24-25, 2023	Master Series – Advanced Sedation Live Streaming Event
March 1-4, 2023	IV Sedation Dentistry Lexington, KY
March 10-11, 2023	Adult Oral Sedation Dentistry Recertification Chicago, IL
March 10-11, 2023	Master Series – Advanced Sedation Chicago, IL

Leslie Shu-Tung Fang, MD, PhD continued

April 12-15, 2023	IV Sedation Dentistry Lexington, KY
May 19-20, 2023	Adult Oral Sedation Dentistry Live Streaming Event
May 19-20, 2023	Adult Oral Sedation Dentistry Recertification Live Streaming Event
May 19-20, 2023	Master Series – Advanced Sedation Live Streaming Event
May 24-27, 2023	IV Sedation Dentistry Lexington, KY
June 14-17, 2023	IV Sedation Dentistry Lexington, KY
July 12-15, 2023	IV Sedation Dentistry Lexington, KY
August 9-12, 2023	IV Sedation Dentistry Lexington, KY
August 25-26, 2023	Adult Oral Sedation Dentistry Recertification Memphis, TN
August 25-26, 2023	Master Series – Advanced Sedation Memphis, TN
September 13-16, 2023	IV Sedation Dentistry Lexington, KY
October 6-7, 2023	Adult Oral Sedation Dentistry Live Streaming Event
October 6-7, 2023	Adult Oral Sedation Dentistry Recertification Live Streaming Event
October 6-7, 2023	Master Series – Advanced Sedation Live Streaming Event
October 11-14, 2023	IV Sedation Dentistry Lexington, KY
October 25-28, 2023	IV Sedation Dentistry Salt Lake City, UT
November 15-18, 2023	IV Sedation Dentistry Lexington, KY
Nov. 29 – Dec. 2, 2023	IV Sedation Dentistry Salt Lake City, UT

Dr. Fang has presented hundreds of courses between 2007 and 2020. For a full list, ask DOCS Education.

Academic and Hospital Appointments

Academic Appointments:

Assistant Professor in Medicine, Harvard Medical School

Hospital Appointments:

Former Firm Chief, Walter Bauer Firm, Medical Service,
Massachusetts General Hospital

Physician, Massachusetts General Hospital

Publications in Dentistry

1. Sonis, ST, Fazio, RC, Fang, LST. Principles and Practive of Oral Medicine. Third Edition. WB Saunders, Philadelphia, in preparation
2. Sonis, ST, Fazio, RC, Fang LST. Oral Medicine Secrets. Hanley. 2003
3. Fang, LST and Fazio, RC. Antibiotics in Dentistry. CE Magic 2002
4. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2007
5. Fang, LST and Menhall, TW. The Ultimate Basic Drug Kit for Medical Emergencies in the Dental Office. UCS 2007
6. Fang, LST and Menhall, TW. The Ultimate Advanced Drug Kit for Medical Emergencies in the Dental Office. UCS 2008
7. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2008
8. Fang, LST and Menhall, TW. The Ultimate Basic Drug Kit for Medical Emergencies in the Dental Office. UCS 2009
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16. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2015
17. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2016
18. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2017
19. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2018
20. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2019
21. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2020
22. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2021
23. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2022
24. Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2023

Non-Peer Reviewed Scientific or Medical Publications/Materials in Print or Other Media

1. Fang LST. Emergency Preparedness: Do you have a plan? April 2019. The New Dentist
2. Fang LST. New Drugs you MUST Learn to Master. August 2019. The New Dentist
3. Fang LST. Coronavirus Practical Recommendations. March 2020. The New Dentist
4. Fang LST. New Update: The Pandemic and the Dentist. April 2020. The New Dentist
5. Fang LST. The Pandemic and the Dentist. April 9, 2020. The Incisor
6. Fang LST. Fighting a STAR WAR: The SARS-CoV-2 Aerosol Transmission Response. June 2020. The New Dentist
7. Fang LST. S.T.A.R. WARS. June 24, 2020. The Incisor
8. Fang LST. COVID Essentials: Solid Tools to Keep Your Practice Safe. July 29, 2020. The Incisor
9. Fang LST. Calls after Visit to Inform You that They are COVID positive. November 10, 2020. The Incisor

Fellowship in Dentistry

Fellow of the Dental Organization for Conscious Sedation

Contributions in Dentistry

Bridging the disciplines of medicine and dentistry
Empowering the dental professional to consider the patient as a whole

Awards and Honors

- Boylston Society Award for Excellent in Clinical Teaching
  Harvard Medical School
- Teaching Award for Excellence in Clinical Teaching,
  Harvard Medical School
- Faculty Award for Excellence in Clinical Teaching
  Harvard Medical School
- Teaching Award for Excellence in Clinical Teaching
  Harvard Medical School
- Teaching Award for Excellence in Clinical Teaching in Primary Care
  Massachusetts General Hospital
- The Alfred Kranes Award for Excellence in Clinical Teaching
  Massachusetts General Hospital
- Psychiatry Resident Award for Contribution to the Residency Selection Process
  Massachusetts General Hospital
- Best Doctor In America 2008
- Best Doctor In Boston 2007
  John R. Gallagher III and Katherine A. Gallagher Endowed Chair in Clinical
- Nephrology 2015
- P-I Branemark Award for Lifetime Achievement 2022

Awards in Dentistry:

CRE Product of the Year 2004: Fang, LST and Fazio, RC. Antibiotics in Dentistry. CE Magic 2002

CE Best Educational Product of the Year 2008: Fang, LST, Fazio, RC and Menhall, TW. Ultimate Cheat Sheets. The Practical Guide for Dentists. UCS 2008

Narrative Report

I maintain an international practice in medicine and nephrology and continue to teach from the clinical forum. In clinical teaching, my major focus has involved the development of an interactive platform to facilitate education. Using case-based method and an audience response system, students would participate interactively to manage patients through a large variety of clinical scenarios. Pre- and post-tests would allow teachers and participants alike to gauge the effectiveness of any educational session. Interactive teaching has been extended from the education of students, residents and fellows to physicians in the community, and to dental professionals who are interested in optimizing management of medically complex patients.

Professional Biography

In medicine:
Leslie S.T. Fang received his PhD degree in Physiology and Biophysics from the University of Illinois, Champaign-Urbana. At the University of Illinois, he was a Gregory Scholar and James Scholar and was elected to Phi Eta Sigma, Phi Kappa Phi, Phi Beta Kappa, Sigma Xi and Phi Sigma. He graduated as a Bronze Tablet Scholar. He did his medical training at Harvard Medical School and was elected to Alpha Omega Alpha as a junior. He did his residency training at the Massachusetts General Hospital where he served as Chief Medical Resident. He did his fellowship training in Nephrology at the Massachusetts General Hospital and is board certified in Internal Medicine and Nephrology.

He has been on the staff of Massachusetts General Hospital and on the faculty of Harvard Medical School. A clinician and a teacher, he maintains an active international practice in Internal Medicine Medicine and Nephrology and is a leading teacher at Harvard Medical School. At the Massachusetts General Hospital, he served as Chief of the Walter Bauer Firm and as the Clinical Director of the Governing Board of the Medical Services Associates. He has received numerous awards for excellence in teaching from Harvard Medical School and has served and chaired the Medical Internship Selection Committee at the Massachusetts General Hospital for two decades.

In the dental field:
He is the co-author of the major textbook Principle and Practice of Oral Medicine, Oral Medicine Secrets and has been heavily involved in the clinical teaching of Oral Medicine. He is the co-author of an interactive DVD on Antibiotics in Dentistry in the EndoMagic Interactive Teaching Series, which won the CRE product of the year award for 2004. He is the co-author of the recently released Ultimate Cheat Sheets: Practical Guide for the Dentist, The Ultimate Basic Drug Kit and The Ultimate Advanced Drug Kit for Medical Emergencies in the Dental Office.

The Ultimate Cheat Sheets won the CE Award for Best Educational Product of 2008.
He has been the featured speaker at many of the major dental meetings. He has been integrally involved in the EndoMagic course, providing an important bridge between the dental and the medical profession. He has also been involved with teaching of oral medicine at the Las Vegas Institute of Advanced Dentistry where he runs a one-day seminar on Medical Emergencies in the Dental Office. He is on the board of LVI Global. He is on the faculty of Dental Organization for Conscious Sedation and is instrumental in the design of the curriculum and the teaching of oral medicine pertinent to sedation.

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26. Fang LST. Management of urinary tract infections in women. Comprehensive Therapy, 1987; 13: 3-7.
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Diuretic therapy
Hepatorenal syndrome

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5. Sonis, ST, Fazio, RC, Fang LST. Oral Medicine Secrets. Hanley and Balsey, Philadelphia 2004.

6. Fang, LST and Fazio, RC. Antibiotics in Dentistry. CE Magic 2004.

7. Fang, LST, Fazio, RC and Menhall, T. Ultimate Cheatsheets: The Practical Guide for Dentists. UCS Boston 2006.

8. Fang, LST, Fazio, RC and Menhall, T. Ultimate Cheatsheets: The Practical Guide for Dentists. Canada. UCS Boston, 2007, 2008, 2009

9. Fang, LST and Menhall, T. Ultimate Drug Kit, Basic Kit for Medical Emergencies in the Dental Office. UCS Boston, 2007, 2008, 2009

10. Fang, LST and Menhall, T. Ultimate Drug Kit, Advanced Kit for Medical Emergencies in the Dental Office. UCS Boston, 2007, 2008, 2009

CAROL A. WILSON, DMD

[redacted]

Education:

2006 DMD University of Kentucky College of Dentistry
With Distinction- Top 20% of Class, GPA 3.81

2002 Morehead State University
Summa Cum Laude, GPA 4.0

Postdoctoral Training:

Externship Oral and Maxillofacial Surgery, University of Kentucky
University of Kentucky College of Dentistry
Chandler Medical Center
Lexington, KY 40536
859.323.5749

Externship Jack Lenihan, DMD
800 Violet Road
Crittenden, KY 41030
859.428.3100

2023 Molis Coaching

2023 Invisalign Training

2022 Empire Medical Training Botox and Dermal Filler

2021 AIE/Foundry Clinical Implant Education

2021 AIE Implant Education

2021 Implant Educators Implant Continuum

Carol A. Wilson, DMD continued

2019 ACLS/BLS Recertification
2019 Renewal of IV Sedation
2018 Rutkowski PRP/PRF Bone Grafting Training
2017 DOCS & OHSU IV Sedation Training
2017 SWAT- Patient Relation
2016 DOCS Conscious Oral Sedation Training
2016 OsteoReady Implant Mini Residency (Now Little Implant Company)
2016 Advanced Cardiac Life Support Certified
2015 Facially Generated Treatment Planning, Spear Institute Seminar
2015 Dental Bootcamp
2015 Cerec Three Dimensional Imaging and Milling
2014 Sybron Endodontic Hands on Course
2010 Brasseler Endodontic Hands on Course
2006 Maintain CE for each renewal cycle courses include OSA, practice management, restorative dentistry, restoring the worn dentition, Implant restoration, OSHA and HIPAA compliance.

Licensure & Certification:

2006 - Present Licensed to Practice Dentistry in Kentucky
2006 - Present DEA Licensed
2006 - Present Basic Life Support
2010 Dental Expert Witness - Nicholasville, KY
2016 - Present Advanced Cardiac Life Support
2016 - Present Anesthesia Permit in Kentucky - Moderate Parenteral

Faculty:

2019 - Present DOCS Education IV Sedation Clinical Instructor
2018 - Present Associate Clinical Instructor, Little Implant Company
2006 - 2007 Part time Prosthodontic Clinical Instructor, UKCD

Associations:

2002 - 2006 American Student Dental Association
2002 - 2006 American Association of Women Dentists
2006 - 2008 American Dental Association
2006 - 2008 Kentucky Dental Association
2006 - 2007 Lexington Dental Implant Society

Study Clubs:

2015 - Present Spear Study Club
2022- Present AACA Study Club

Carol A. Wilson, DMD continued

Awards & Recognitions:

2006	Excellence in Prosthodontics
2006	Outstanding Radiology Student
2006	Omicron Kappy Upsilon Dental Honor Society
2004	Susan McEvoy Scholarship
2003 - 2006	Dean's Scholarship
2001 - 2002	Phi Kappa Phi Honoree and Scholarship Recipient
2001	Cardinal Key Inductee
2000	Harrison Electric Cooperative Scholarship
2000	General Human Science Scholarship
1999 - 2002	Presidential Scholarship, Governor's Scholar
1999 - 2002	Academic Honor's Scholarship

Institutional Committees:

2005 - Present	Associate Member of the UKCD Admissions Committee
2015 - 2016	Brookside Elementary Site Based Decision Making Council
2013 - 2014	President Jessamine Early Learning Village PTO
2004 - 2006	President American Association of Women Dentists
2004 - 2006	UKCD Student Clinical Advisory Committee
2004 - 2006	Voting Member of UKCD Admissions Committee
2003	Physiology Review Committee, Dental School Member
2002 - 2009	Professional Education Preparation Program (PEPP) Dental Experiential Coordinator
2000 - 2002	Baptist Student Union Council Member
2000 - 2002	Student Alumni Ambassador, Secretary

National Presentations:

2000	"Application of Plato's 'Capital Ideas'", National Honors Conference, Washington, D.C.
2000	"Euthanasia", National Honors Conference, Washington, D.C.

Presentations To Predoctoral Students:

2007	Authored Prosthodontic Unit Exam, UKCD
2001	"Food Mythology, Food Symbolism, and the Manipulation of the American Consumer, Honor's Round Table
2001	"Beauty and Aesthetic Quality", Honor's Round Table

Media Content:

2022	Monitoring Article for the Incisor
2019	Created YouTube Videos to Assist Participating Dentists in the DOCS IV Sedation Curriculum with record keeping and EKG Monitoring

Carol A. Wilson, DMD continued

Professional Experience:

2022 - Present	Fifty Percent Owner of Dental Wellness of Lexington
2016 - Present	Associate Dentist, Dental Wellness of Lexington
2017 - 2019	Locum Tenen Dentist, Dentistry for Today
2015 - 2017	Locum Tenen Dentist, Susan Couzens, DMD
2014 - 2016	Associate Dentist, Immediadent
2010 - 2014	Owner, Jessamine Dental
2009 - 2010	Locum Tenen Dentist, Malcolm Miracle, DMD, Jessamine Dental
2006 - 2007	Part Time Faculty Member of Prosthodontics, UKCD
2006 - 2009	Associate Dentist, Jack Lenihan, DMD

Clinics & Courses Presented:

May 2018	IV Sedation Clinical Instruction for DOCS/OHSU Course
Sept 2018	IV Sedation Clinical Instruction for DOCS/OHSU Course
Jan 2019	IV Sedation Clinical Instruction for DOCS/OHSU Course
April 2019	IV Sedation Clinical Instruction for DOCS/OHSU Course
May 2019	IV Sedation Clinical Instruction for DOCS/OHSU Course
June 2019	IV Sedation Clinical Instruction for DOCS/OHSU Course
July 2019	IV Sedation Clinical Instruction for DOCS/OHSU Course
Sept 2019	IV Sedation Clinical Instruction for DOCS/OHSU Course
Nov 2019	Monitoring During Sedation, DOCS Conference Seattle, WA
July 2020	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Aug 2020	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Sept 2020	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Oct 2020	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Nov 2020	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Jan 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Feb 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Feb 2021	DOCS Sedation Training for the Team, ILS
Feb 2021	DOCS Light Sedation, Nitrous Oxide, ILS
March 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
April 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
May 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
May 2021	DOCS Sedation Training for the Team, ILS
May 2021	DOCS Light Sedation, Nitrous Oxide, ILS
June 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
July 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Aug 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Sep 2021	IV Sedation Clinical Instruction for DOCS/ISU Meharry Course

Carol A. Wilson, DMD continued

Oct 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Oct 2021	DOCS Sedation Training for the Team, ILS
Oct 2021	DOCS Light Sedation, Nitrous Oxide, ILS
Nov 2021	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Jan 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Feb 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Feb 2022	DOCS Sedation Training for the Team, ILS
Feb 2022	DOCS Light Sedation, Nitrous Oxide, ILS
March 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
April 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
May 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
May 2022	DOCS Sedation Training for the Team, ILS
May 2022	DOCS Light Sedation, Nitrous Oxide, ILS
June 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
July 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Aug 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Sept 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Oct 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Oct 2022	DOCS Sedation Training for the Team, ILS
Oct 2022	DOCS Light Sedation, Nitrous Oxide, ILS
Nov 2022	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Jan 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Feb 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Feb 2023	DOCS Sedation Training for the Team, ILS
Feb 2023	DOCS Light Sedation, Nitrous Oxide, ILS
March 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
April 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
May 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
May 2023	DOCS Sedation Training for the Team, ILS
May 2023	DOCS Light Sedation, Nitrous Oxide, ILS
June 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
July 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Aug 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Sept 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Oct 2023	IV Sedation Clinical Instruction for DOCS/ISU Meharry Course
Oct 2023	DOCS Sedation Training for the Team, ILS
Oct 2023	DOCS Light Sedation, Nitrous Oxide, ILS
Nov 2023	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Jan 2024	IV Sedation Clinical Instruction for DOCS/ISU/Meharry Course
Jan 2024	Washington AGD, Which Level of Sedation Fits You Best?

Carol A. Wilson, DMD continued

Community Service:

2022, 2023	East Jessamine Middle Lady Jaguar Basketball President
2017 - 2019	Southland Christian Church Small Group Leader
2018, 2019	Volunteer at Smiles From the Heart, Bowling Green, KY
2017	Volunteer at Gift of Smiles, Lexington, KY
2016 - 2017	YMCA Assistant Soccer Coach
2015 - 2016	Council of Councils Site Based Council Jessamine County
2013 - 2014	Southland Christian Church Weekend Story Teller Youth Ministry
2012	Refuge Clinic
2013	RJ Corman Health Fair
2012, 2015	Presentation on Oral Health, Brookside Elementary
2007	Women's Health Fair, Nicholasville, KY
2006 - 2011	Southland Christian Church "The Zone" Ministry
2004 - 2005	Sealant Project at Kentucky State Fair
2005	University of Kentucky Summer Dental Camp Counselor
2003, 2016	Jesus Prom
2002 - 2006, 2018	University of Kentucky College of Dentistry Fall Open House
2003 - 2005	Race for the Cure
2003	God's Pantry
2002, 2006	Saturday Morning Clinic

TAYLOR TATE, DDS

Education:

2018 DDS University of Utah School of Dentistry
Top 10% of Class

2014 Bachelors in Exercise Science from the University of Utah

Postdoctoral Training:

2018 Fast track to Digital Dentistry

2018 Dr Choukroun PRF/PRP training

2021 Zero Bone Loss Concepts

2022 Advanced Cardiac Life Support Certified

Licensure & Certification:

2018-Present Licensed to Practice Dentistry if Utah

2018-Present DEA Licensed

2010-Present Basic Life Support

2022-Present Advanced Cardiac Life Support

Faculty:

2023-Present DOCS Education IV Sedation Clinical Instructor

Associations:

2014-2018 American Student Dental Association

Awards & Recognitions:

2018 Excellence in Oral Pathology

2018 Excellence in Prosthodontics

2018 Excellence in Periodontics

2010-2014 Dean's List

2018 Omicron Kappa Upsilon Dental Honor Society

Professional Experience:

2018-2021 Associate at Coleman Family Dental

2021-Present Owner at Coleman Tate Dental

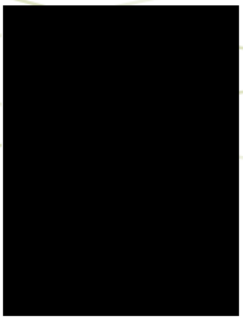
Taylor Tate, DDS continued

Clinics & Courses Presented:

Oct 2023	IV Sedation Clinical Instruction for DOCS/ISU/MMC Course
Nov 2023	IV Sedation Clinical Instruction for DOCS/ISU/MMC Course
Jan 2024	IV Sedation Clinical Instruction for DOCS/ISU/MMC Course

Community Service:

2015-2017	ASDA Community Outreach Chair



C. RAY COLEMAN

Education:

1985 Brigham Young University
1989 Oklahoma College of Dentistry (OUCOD)

Licensure & Certification:

1991-Present Licensed to Practice Dentistry if Utah
1988-Present Basic Life Support
2016-Present Advanced Cardiac Life Support

Faculty:

2023-Present DOCS Education IV Sedation Clinical Instructor

Awards & Recognitions:

1998-2020 Philippine Smile Clinic

Professional Experience:

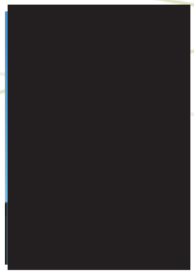
1989 William Beaumont Army Dental Clinic
1994-2021 Owner at Coleman Family Dental
2021-Present Owner at Coleman Family Dental
2009-Present Owner of Digital Contour Dental Lab in Salt Lake City
2020-Present Founder of Incredicreds Dental Credentialing Software and Service

Clinics & Courses Presented:

Oct 2023 IV Sedation Clinical Instruction for DOCS/ISU/MMC Course
Nov 2023 IV Sedation Clinical Instruction for DOCS/ISU/MMC Course
Jan 2024 IV Sedation Clinical Instruction for DOCS/ISU/MMC Course

Community Service:

Utah Food Bank



STEVE C. YUN, M.D.

Board Certification:

2001 American Board of Anesthesiology

2011 Re-certified by the American Board of Anesthesiology

2021 Re-certified by the American Board of Anesthesiology

Training/Education:

2000 Residency in Anesthesiology
UCLA Medical Center, Los Angeles, CA

1997 Internship in General Surgery
University of Illinois-Chicago Medical Center

1996 M.D. with Honors (Dean's Scholar)
University of Southern California

1991 B.S. with Honors (Dean's Scholar)
University of Wisconsin-Madison

Professional Work Experience:

2000-2011 Self-employed as a Sole Proprietor
In full-time clinical practice as an anesthesiologist at various hospitals, medical centers, outpatient surgical facilities, and dental/oral surgery offices.

2011-Present Employed by Steve C. Yun, M.D., Inc.
In full-time clinical practice as an anesthesiologist at various hospitals, medical centers, outpatient surgical facilities, and dental/oral surgery offices

Academic Appointments:

2016-Present Conscious Sedation & General Anesthesia Examiner
Dental Board of California

2016-Present Surveyor
American Association for Accreditation of Ambulatory Surgery Facilities

2014-Present Medical Director
Beverly Hills Integrated Surgery Center

2011-2014 Medical Director
Alicia Surgery Center, Laguna Hills, CA

2008-2011 Medical Director
Four Seasons Surgery Center, Anaheim, CA.

2010-2017 California Society of Anesthesiologists District Delegate

2020-Present California Society of Anesthesiologists District Delegate

Steve C. Yun, M.D. continued

Other Professional Activity:

2020-Present	Speaker and Faculty for DOCS Education - Selected Teaching Portfolio:	
	June 2020	Emergency Medications in Dentistry: Taming the Unknown
	Oct 2020	Cognitive Errors in Dental Sedation: How to Identify and prevent hidden hazards to patient safety
	Apr 2021	Medical Emergencies in the Dental Office - Licensure Renewal Training
	July 2022	Levels of Sedation and Monitoring for Pediatrics
	July 2022	Pediatric Sedation Case Selection
	July 2022	Pediatric Dentistry: Basic Pediatric Anatomy, Physiology and Pharmacology
	Apr 2023	DEA MATE Compliance: Treatment and Management of Patients with Opioid or Other Substance Abuse Disorders
	Oct 2023	Pre-Sedation Medical Evaluation for Dentists with Steven Yun MD
	Oct 2023	Medical Emergencies in the Dental Office - MD Licensure Renewal Training
	Nov 2023	Medical Emergencies with Steve Yun, MD
	Jan 2024	Anesthesia Monitoring for WA Team Members
	Jan 2024	Oral Sedation Recertification: Pre-Sedation Evaluation, Medical Emergencies & Preparing for a Dental Board Inspection
2019-Present	Speaker and Consultant for AcelRx Pharmaceuticals	
2019-Present	Consultant for Koa Accel	
2019-Present	Medical Advisory Board member, HealthFirst	

Honors/Awards/Publications:

2021	Physician of Excellence Award by the Orange County Medical Association. Also received this same award in 2007, 2010, 2013, 2016, and 2019.
2007	Anesthesiologist of the Year Fresh Start Surgical Gifts, Encinitas, CA
2001	"Practice Guidelines for Adult Sedation and Analgesia." With R. Steadman. In Sedation Analgesia for Diagnostic & Therapeutic Procedures. Edited by S. Malyviya. Humana Press.

Clinical Research Experience:

2010	A Randomized, Double-Blind, Parallel Pharmacokinetic and Safety Study of 3 Doses of *** versus *** in Healthy Normal Volunteers (WCCT).
2011	Randomized, Double-Blind, Placebo-Controlled, Ascending Single-Dose Study to Evaluate the Safety, Tolerability, Pharmacokinetics, and Pharmacodynamics of *** in Healthy Young and Elderly Subjects (WCCT).
2012	Placebo-Controlled, Ascending Multiple-Dose Study to Evaluate the Safety, Pharmacokinetics and Pharmacodynamics of *** in Healthy Young and Elderly non-Japanese Subjects, and Healthy Young Japanese Subjects (WCCT).
2013	A Two-Part, Randomized, Double-Blind, Placebo-Controlled, Single Dose Study to Assess the Safety,

Steve C. Yun, M.D. continued

Tolerability, Pharmacokinetics and Lung Levels of *** Inhalation Solution (*** inhalation solution) Administered to Healthy Volunteers (WCCT).

2016 A Randomized, Double-Blind, Placebo-Controlled, Single Ascending Dose Study of Intravenously Administered *** in Healthy Subjects (WCCT).

2016 A Phase 3, Multicenter, Randomized, Double-Blind, Placebo- and Active-Controlled Study of *** for the Treatment of Moderate to Severe Acute Pain after Bunionectomy (AGMG).

2016 A Randomized, Double-Blind, Placebo-Controlled, Parallel Group, Multiple Ascending Dose Study to Assess the Safety, Tolerability, Pharmacokinetics, and Pharmacodynamics of Orally Administered *** in Healthy Subjects (WCCT).

Hospital Appointments:

Saddleback Memorial Medical Center, Laguna Hills, CA. (Active)

Rady's Children's Hospital, San Diego, CA. (Courtesy)

Placentia Linda Medical Center, Placentia, CA. (Courtesy)

Chapman Medical Center, Orange, CA (Courtesy)

Memberships:

American Society of Anesthesiologists

California Society of Anesthesiologists

American Dental Society of Anesthesiology

California Dental Society of Anesthesiology

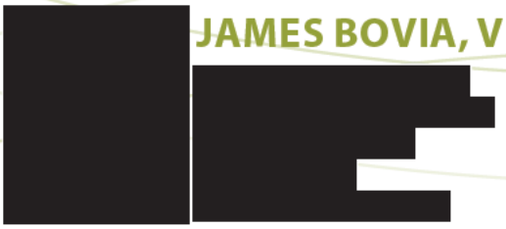
California Society of Pediatric Dentistry

Certifications:

Advanced Cardiac Life Support

Pediatric Advanced Life Support

General Anesthesia Dental Permit (2/2011 to Present)



JAMES BOVIA, VP OF EDUCATION

Objective:

It is Jim's objective to enhance the ability of primary care physicians and dentists to deliver effective emergency treatment when presented with the rare occurrence until EMS arrives.

Employment:

1995-Present	Vice President – Life Support Services, Inc. Oversees the annual training of 16,000 medical professionals in Basic Life Support, Advanced Cardiac Life Support, Pediatric Advanced Life Support, and trauma management.
1994-2000 2004-2015	Paramedic – Huron Valley Ambulance.
2000-2004	Emergency room technician - University of Michigan Hospital .
1989-1994	Basic EMT Washtenaw county jail medical unit .
1989-1994	Fire fighter Sumpter twp fire fighter.

University of Michigan Emergency Department

Education:

1985	Lincoln High school
1988 - 1992	Life Support Services Inc. EMT and Paramedic training.

Consultant in Pre-Hospital Care and Acute Intervention:

2016 - Current	Lincoln High school
2010 - Current	Life Support Services Inc. EMT and Paramedic training.

Program Development:

2015	Designed an Emergency Management Course for Dentists incorporating advanced airway manuvers.
2007	Customized the American Heart Association's Advanced Cardiac Life Support to fit the challenges of the free standing Dental office.

JAMES BOVIA, VP OF EDUCATION continued

Has Provided Emergency Medical Training to the following organizations:

- Berry Center LLC
- Blake Woods Surgery Center
- Bloomfield Children's Dentistry
- Fred Bonine, DDS, Brighton, MI
- Cardiology & Vascular Associates
- Central Michigan Community Hospital
- Central Michigan University
- Chelsea Community Hospital
- Dental Organization for Conscious Sedation
- Eaton Rapids Medical Center
- Anthony Feck, DDS Lexington, KY
- Great Lakes Heart & Vascular Institute
- Harbor Beach Medical Center
- Henry Ford Hospital
- Independent Anesthesia
- Ingham Regional Medical Center Emergency Department
- LaRabida Children's Hospital
- NorthWest Anesthesia Seminars
- Oakwood Healthcare System
- Orthopedic Associates
- St Joes Mercy Livingston Hospital, Ann Arbor, Livingston, Pontiac, MI
- Government of Turks & Caicos
- University of Michigan Health Systems
- UnaSource Surgery Center
- VA Healthcare System
- Woods Mill Anesthesia

Home Address:

[redacted]

EXHIBIT B

2024

Module 1 (Chapters 1-6)	Instructor	Segment Length
INTRODUCTION TO IV SEDATION DENTISTRY 1	Tony Feck	1:31:00
Workbook pages: I 1 - 8		0:12:00
IV SEDATION VOYAGE (ANATOMY & PHYSIOLOGY) 2	Tony Feck	1:07:42
Workbook pages: II 1 - 26		0:52:00
PHARMACOLOGY FOR THE IV SEDATION PROCEDURE 3	Tony Feck	2:21:29
Workbook pages: III 1 - 28		0:56:00
MONITORING FOR THE IV SEDATION PROCEDURE 4	Tony Feck	0:28:36
Workbook pages: IV 1 - 15		0:30:00
LOCAL ANESTHETICS 5	Tony Feck	0:27:23
Workbook pages: V 1 - 19		0:38:00
PRE-OPERATIVE ENTERAL SEDATION FOR IV SEDATION 6	Tony Feck	0:24:43
Workbook pages: VI 1 - 16		0:32:00
Test. End of Session 1		0:30:00
Office Hour Session 1 Review test and questions with instructor	Taylor Tate	1:00:00

Module 2 (Chapters 7-12)	Instructor	Part 1 - Segment Length
NITROUS OXIDE 7	Tony Feck	1:13:01
Workbook pages: VII 1 - 36		1:12:00
AIRWAY MAINTENANCE & MANAGEMENT 8	Tony Feck	1:54:50
Workbook pages: VIII 1 -25		0:50:00
VENIPUNCTURE 9	Tony Feck	0:58:43
Workbook pages: IX 1 - 8		0:16:00
IV SEDATION PROTOCOLS 10	Tony Feck	0:30:03
Workbook pages: X 1 - 12		0:24:00
IV SEDATION COMPLICATIONS 11	Tony Feck	0:20:47
Workbook pages: XI 1 - 8		0:16:00
IV SEDATION CHALLENGES 12	Tony Feck	0:21:02
Workbook pages: XII 1 - 11		0:22:00
Test. End of Session 2		0:30:00
Office Hour Session 2 Review test and questions with instructor	Taylor Tate	1:00:00

Module 3 (Chapters 13-19)	Instructor	Part 1 - Segment Length
NEW DRUGS 2018 13	Leslie Fang	1:17:40
Workbook pages: XIII 1 - 8		0:16:00
PATIENT ASSESSMENT 14	Leslie Fang	3:48:35
Workbook pages: XIV 1 - 35		1:10:00
PROSTHETIC JOINT 15	Leslie Fang	0:34:19
Workbook pages: XV 1 - 7		0:14:00
POST-SEDATION ANALGESIA 16	Leslie Fang	0:32:09
Workbook pages: XVI 1 - 4		0:08:00
BLEEDING DIATHESIS 17	Leslie Fang	0:32:16
Workbook pages: XVII 1 - 12		0:22:00
ANTIBIOTICS FOR ODONTOGENIC INFECTIONS 18	Leslie Fang	0:36:02
Workbook pages: XVIII 1 - 10		0:20:00
CARDIAC RHYTHM REVIEW 19	Leslie Fang	0:58:25
Workbook pages: XIX 1 - 19		0:38:00

NEW ADA POLICY FOR OPIOID PRESCRIPTIONS (to include T. C. A. § 63-1-402)	Leslie Fang	2:24:04
		0:30:00
	Taylor Tate	1:00:00
Test. End of Session 3		
Office Hour Session 3 Review test and questions with instructor		
	Instructor	Part 1 - Segment Length
	Leslie Fang	1:13:14
Module 4 (Chapters 20-24)		0:42:00
CASE EXAMPLES I 20	Leslie Fang	0:45:47
Workbook pages: XX 1 - 21		0:40:00
CASE EXAMPLES II 21		
Workbook pages: XXI 1 - 20	Tony Feck	0:50:45
IV SEDATION RECORD-KEEPING, DISPENSING, AND DEA COMPLIANCE 22		0:32:00
	Tony Feck	0:56:41
Workbook pages: XXII 1 - 16		0:32:00
IV SEDATION CASE EXAMPLES 23	Tony Feck	0:40:44
Workbook pages: XXIII 1 - 16		0:16:00
OFFICE INSPECTION 24		0:22:31
Workbook pages: XXIV 1 - 8		0:28:00
Clinic instruction videos		0:30:00
Workbook page: Appendix Final Test. End of Session Didactic		
Office Hour Session 4 Review test and questions with instructor - Clinic instruction and review videos and full review of any chapters that they want to go back over before the clinical session. They will still have access to the full course. (Many will probably want to go back to chapters 10-12 to review)	Taylor Tate	1:00:00
	Jim Bovia	3:30:00
Medical Emergency Training Video		
	Feck/Bovia	10:00:00
Didactic lectures at clinic site (day 1 session)	Feck	30:00:00
20 Patients - clinical 3 days		
		2:00:00
Final Exam - 60 Questions		

IV Sedation Certification Topics and Objectives

Curriculum Topics:

- The sedation spectrum
- Mechanisms of orofacial pain and analgesia
- Psychological aspects of pain and anxiety
- Principles of pharmacology
- Pharmacokinetics, pharmacodynamics, and pharmacotherapeutics of conscious sedation medications used in dentistry
- Drug interactions
- Patient assessment
- Advanced airway maintenance & management
- Monitoring
- IV sedation protocol
- Venipuncture
- Record-keeping
- Sedation blunders
- Advanced Cardiac Life Support (ACLS), including ACLS certification

Course Objectives: Upon completion of a competency course in enteral sedation, the dentist must be able to:

1. Incorporate methodologies to safely provide both enteral (oral) and parenteral (IV) minimal and moderate sedation in an outpatient setting.
2. Expand your confidence and ability to treat medically complex patients safely, and realizing the synergies that arise—for both doctor and patient—when you strategically combine safe oral and IV sedation.
3. Understand the ADA guidelines for Teaching Pain Control and Sedation to Dentists and Dental Students (Adopted by the ADA House of Delegates in Oct. 2016).
4. Fulfill the ADA-recommended advanced airway management and opioid prescription training, and qualify for ACLS certification.
5. Understand the advantages and disadvantages of minimal and moderate sedation and the sedation spectrum.

6. Learn prevention, recognition, and management of complications from venipuncture and other parenteral techniques; oral sedation and other enteral techniques; and minimal and moderate sedation with particular attention to airway maintenance and support of the respiratory and cardiovascular systems.
7. Review abuse potential, occupational hazards, and other detrimental effects of the agents utilized to achieve minimal and moderate sedation.
8. Techniques of intravenous access, intramuscular injection, and other parenteral techniques through demonstration.
9. Study Pharmacokinetics, pharmacodynamics, and pharmacotherapeutics of sedation medications used in dentistry for administration, including the precautions, indications, contraindications, and adverse reactions associated with them.
10. Understand complications and potential missteps associated with techniques of minimal and moderate sedation.
11. Learn protocols for management of most commonly encountered emergencies in the dental office, and the drugs and equipment required for prevention and management; demonstrate proficiency.
12. Study historical, philosophical, and psychological aspects of anxiety and pain control, mechanisms of orofacial pain and analgesia.
13. Understand patient evaluation, assessment, selection, monitoring and recordkeeping through a detailed review of medical history taking, physical diagnosis, and psychological considerations.
14. Description of the sedation anesthesia continuum with special emphasis on the distinction between the conscious and the unconscious state.
15. Pharmacology of local anesthetics and agents used in IV Sedation, including drug interactions and contraindications.
16. Indications and contraindications for use of IV Sedation.
17. Review of dental procedures possible under IV Sedation.
18. Patient monitoring using observation and monitoring equipment with particular attention to vital signs and reflexes related to consciousness.
19. Safely and effectively administer the selected drug(s) to dental patients in a clinical setting.

EXHIBIT C

Idaho State
UNIVERSITY

Department of Dental Sciences
Idaho Dental Education Program
921 South 8th Avenue, Stop 8088 • Pocatello, Idaho 83209-8088

October 09, 2019

To whom it may concern,

Since 2019, the DOCS Education IV Sedation Certification course has been taught under the auspices of the Idaho State University Department of Dental Sciences. The Idaho Advanced Education in General Dentistry is an ADA-recognized educational and CODA accredited program.

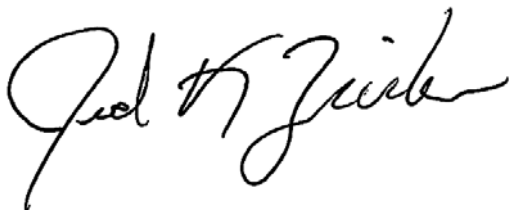
Respectfully,

Brian R. Crawford, DDS, FICD
Chair, Department of Dental Science
Program Director, Idaho AEGD
Idaho State University
921 S. 8th Ave, Stop 8088
Pocatello, ID 83209
(208)282-5275

Idaho State University
Department of Dental Sciences
921 S. 8th Ave, Stop 8088
Pocatello, ID 83209
(208) 282-3289

ATTENDANCE AND COMPLETION VERIFICATION

Participant's Name:
Location: Happy Smiles, Lexington Kentucky

Course Date:	Course Title	Duration:	Educational Method:	CDE Credits:
June 11-12, 2023	IV Sedation for Dentistry	12.5 hours	Didactic Lecture	12.5
June 25-26, 2023	IV Sedation for Dentistry	12.5 hours	Didactic Lecture	12.5
July 9-10, 2023	IV Sedation for Dentistry	12.5 hours	Didactic Lecture	12.5
July 23-24, 2023	IV Sedation for Dentistry	8.5 hours	Didactic Lecture	8.5
July 23-24, 2023	Airway Management	4 hours	Didactic Lecture	4
August 9, 2023	IV Sedation for Dentistry	7 hours	Didactic Lecture	7
August 9, 2023	Hands-On Simulated Competence in Airway Management of the Compromised Airway	3 hours	Participation/ Hands-on	3
August 10, 2023	IV Sedation for Dentistry-Clinical	9 hours 7 pt. cases	Participation/ Hands-on	9
August 10, 2023	IV Sedation for Dentistry	1 hour	Didactic Lecture	1
August 11, 2023	IV Sedation for Dentistry-Clinical	9 hours 7 pt. cases	Participation/ Hands-on	9
August 11, 2023	IV Sedation for Dentistry	1 hour	Didactic Lecture	1
August 12, 2023	IV Sedation for Dentistry-Clinical	9 hours 6 pt. cases	Participation/ Hands-on	9
August 12, 2023	IV Sedation for Dentistry	1 hour	Didactic Lecture	1
			Didactic Hours	60
			Participation/Hands-on	30
			TOTAL HOURS	**90**
Signature verifying attendance	 Jed Zirker, DMD- AEGD Program Director			

Please keep this form for your records.

CODA
Commission on Dental Accreditation

Idaho State University
Department of Dental Sciences
921 S. 8th Ave, Stop 8088
Pocatello, ID 83209
(208) 282-3289

Please keep this form for your records.

This course is verified to be consistent with the ADA Guidelines for Teaching Pain Control and Sedation to Dentists and Dental Students (2016) previously referred to as Guidelines for Teaching the Comprehensive Control of Anxiety and Pain in Dentistry 2000 edition.

The Idaho AEGD is a CODA accredited AEGD program with clinics in Meridian and Pocatello, Idaho, and Lexington, Kentucky.

Each attendee is responsible for maintaining their own records and for reporting their own CDE activities to all appropriate bodies in accord with any jurisdictional and/or membership requirements. The Idaho AEGD and Idaho State University will maintain information for no more than seven years from date of course.

MEHARRY MEDICAL COLLEGE
Office of Lifelong Learning

10/23/19

To Whom It May Concern:

Since 2019, the DOCS Education IV Sedation Certification course has been taught under the auspices of Meharry Medical College School of Dentistry an ADA-CERP educational institution.

Sincerely,

Allyson F. Fleming, Ed.D, Director
Meharry Medical College
Office of Lifelong Learning
Continuing Medical Education
Continuing Dental Education
[redacted]

ATTENDANCE AND COMPLETION VERIFICATION

Participant's Name:
Location: Happy Smiles, Lexington Kentucky

Course Date:	**Course Title**	**Duration:**	**Educational Method:**	**CDE Credits:**
February 11-12, 2024	IV Sedation for Dentistry	12.5 hours	Didactic Lecture	12.5
February 25-26, 2024	IV Sedation for Dentistry	12.5 hours	Didactic Lecture	12.5
March 10-11, 2024	IV Sedation for Dentistry	12.5 hours	Didactic Lecture	12.5
March 24-25, 2024	IV Sedation for Dentistry	8.5 hours	Didactic Lecture	8.5
March 24-25, 2024	Airway Management	4 hours	Didactic Lecture	4
April 3, 2024	IV Sedation for Dentistry	7 hours	Didactic Lecture	7
April 3, 2024	Hands-On Simulated Competence in Airway Management of the Compromised Airway	3 hours	Participation/ Hands-on	3
April 4, 2024	IV Sedation for Dentistry-Clinical	9 hours 7 pt. cases	Participation/ Hands-on	9
April 4, 2024	IV Sedation for Dentistry	1 hour	Didactic Lecture	1
April 5, 2024	IV Sedation for Dentistry-Clinical	9 hours 7 pt. cases	Participation/ Hands-on	9
April 5, 2024	IV Sedation for Dentistry	1 hour	Didactic Lecture	1
April 6, 2024	IV Sedation for Dentistry-Clinical	9 hours 6 pt. cases	Participation/ Hands-on	9
April 6, 2024	IV Sedation for Dentistry	1 hour	Didactic Lecture	1
			Didactic Hours	60
			Participation/Hands-on	30
			TOTAL HOURS	**90**
Signature verifying attendance	Joyce A Barbour, DDS- Asst Professor - OMFS Department, Director - General Practice Residency Prg			

Please keep this form for your records.

Please keep this form for your records.

This course is verified to be consistent with the ADA Guidelines for Teaching Pain Control and Sedation to Dentists and Dental Students (2016) previously referred to as Guidelines for Teaching the Comprehensive Control of Anxiety and Pain in Dentistry 2000 edition.

The Meharry Medical College School of Dentistry GPR program is CODA accredited with clinics in Nashville, Tennessee, Lexington, Kentucky, and Salt Lake City, Utah.

Each attendee is responsible for maintaining their own records and for reporting their own CDE activities to all appropriate bodies in accord with any jurisdictional and/or membership requirements. The Meharry Medical College School of Dentistry will maintain information for no more than seven years from date of course.

NOTES

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CELEBRATING 20 YEARS
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EDUCATION
LEADERSHIP · COMMUNITY
1999·2019

IV SEDATION – PRESENTED BY DOCS EDUCATION

INTRODUCTION TO IV SEDATION DENTISTRY 1

IV SEDATION VOYAGE (ANATOMY & PHYSIOLOGY) 2

PHARMACOLOGY FOR THE IV SEDATION PROCEDURE 3

MONITORING FOR THE IV SEDATION PROCEDURE 4

LOCAL ANESTHETICS 5

PRE-OPERATIVE ENTERAL SEDATION FOR IV SEDATION 6

NITROUS OXIDE 7

AIRWAY MAINTENANCE & MANAGEMENT 8

VENIPUNCTURE 9

IV SEDATION PROTOCOLS 10

IV SEDATION COMPLICATIONS 11

IV SEDATION CHALLENGES 12

NEW DRUGS 2018 13

PATIENT ASSESSMENT 14

PROSTHETIC JOINT 15

POST-SEDATION ANALGESIA 16

BLEEDING DIATHESIS 17

ANTIBIOTICS FOR ODONTOGENIC INFECTIONS 18

CARDIAC RHYTHM REVIEW 19

CASE EAMPLES I 20

CASE EXAMPLES II 21

IV SEDATION RECORD-KEEPING, DISPENSING, AND DEA COMPLIANCE 22

IV SEDATION CASE EXAMPLES 23

OFFICE INSPECTION 24

WELCOME TO *"IV SEDATION DENTISTRY"*

Presented by

DOCS EDUCATION in Collaboration with ISU & MMC

To the dental professional team:
Fear has no boundaries. It can affect all types of people. There is no age, sex, economic, or race discrimination guidelines. Research shows that almost 30% of our population avoids going to the dentist due to fear. Our personal estimate is closer to 50% where anxiety is the culprit for avoiding needed care. When you realize that a huge segment of the population is in desperate need of our service but are too afraid to seek us out, then it is easy to see why a safe, non-judgmental, pain-free experience would be a welcome change to the anxiety-stricken patient.

This intravenous conscious sedation course was designed to bring to your attention the incredible need in our profession to offer sedation dentistry to this un-served mass of patients. A depth of information will be presented to help you incorporate this much-needed care into your practice. Specific requirements will be presented in order to make you aware of the important commitment necessary to safely and effectively treat patients with IV conscious sedation.

This course is not intended as an exhaustive study of all the implications of sedation dentistry. The learning curve for any technique is different for each of us. To be successful in offering sedation dentistry you need to make a commitment to explore the educational opportunities available to advance your understanding and expertise in this specialized field. It is no secret that Success is a Journey, not a Destination.

To be totally successful with sedation dentistry in your practice you must learn more than just the technical skills. You must understand the emotional state of the high-fear patient. This will involve taking the time to actively listen to those who have avoided dentistry for years because of intense, realistic, palm-sweating, sick-to-the-stomach, eye-popping, and sleep depriving fear.

It is a well-known fact that many people are often afraid to tell anyone about their fear, so they feel isolated. You must be their rock in these situations and let them know that you understand their fear and that is okay for them to feel the way they do. Using compassion and verbal skills you will be able to convey to them that you do take their fear seriously, that you know they are not being silly, and that you care about their emotional needs as well as their dental needs.

If the patient fears one visit to the dentist, imagine their frustration when they find out that because they have not attended to their dental health over the years,

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that they may have to have numerous appointments, over many months, to complete their care. This is always received as discouraging news and often becomes the deciding factor in **NOT** having their needed dentistry done.

So, we pose the obvious question and ask, “If there was a way, with sedation, that our patients could have their treatment accomplished in minimal visits, with no pain and little memory of this experience, would this be more encouraging?” And the answer is an obvious and resounding *yes*.

It is our sincere hope that by offering this course, you will be able to treat patients in the most comfortable and caring way possible, which will help to improve your patients’ health and self-esteem, while at the same time improving the public’s view of our profession.

Imagine how powerful introducing sedation into your practice would be!

Anthony S. Feck, DMD
Dean of Education for DOCS Education
IV Sedation Course Director

Leslie Shu-Tung Fang, MD, PhD
Medical Director for DOCS Education

Jim Bovia, CMT
Director for ACLS & Airway Management Education

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Introduction

The workbook follows the agenda and slides, and contains additional reference information for the course attendee's use following the course.

This CODA-approved course meets the certification requirements of all U.S. states and Canadian provinces*. Specifically, these requirements include:

1. Attending 60 hours of didactic instruction
2. Passing a competency test examination on the material from the didactic instruction
3. Passing a competency hands-on examination in airway management and venipuncture
4. Completing 20 patient experiences in which the student established a patent IV line, appropriately administered medications, monitored the patient from beginning until recovery, and supervised the dental procedure until completion.

*Individual states or provincial regulatory agencies have the right to accept or reject any educational offering.

Agenda for "IV Sedation Dentistry"

Session 1

Day One

- Introduction
- Anatomy & Physiology
- Pharmacology
- Monitoring
- Local Anesthesia
- Enteral Sedation

Day Two

- Nitrous Oxide
- Airway Maintenance & Management
- Venipuncture
- IV Sedation Protocols
- Case Examples I

Day Three

- Medical History
- New Drugs of Importance
- Cardiovascular Diseases

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- Diabetes
- Respiratory Disease
- Liver Disease
- Kidney Disease
- Bleeding Diathesis
- Intraoperative Monitoring

Day Four

- Post-Operative Analgesia
- Prosthetic Joint
- Antibiotics in Odontogenic Infections

Session 2

Day Five

- Review of Session 1
- IV Sedation Complications
- IV Record Keeping
- IV Sedation Case Examples II
- Competency Test

Day Six

- IV Setup
- Venipuncture Practical
- Clinical Practical
- Airway & Monitoring Clinical
- Airway & Medical Emergencies

Day Seven

- Airway Emergency Training
- Cardiovascular Emergency Training

Day Eight

- Cardiovascular Emergency Training

Clinical Sessions

- Patient Care
- Satisfaction of Clinical Case Requirements

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The Purpose of IV Sedation Dentistry

The standard by which we must evaluate everything we do must be measured against keeping our patients **safe and comfortable** at all times.

Safety

When it comes to planning for a safe outcome for the IV sedation patient, it is meaningful to look at sedation outcomes that resulted in adverse outcomes. While such occurrences are uncommon, and the data from those occurrences difficult to obtain, there are reports, often from liability insurance carriers such as the one from The Dentists Insurance Company of California, titled *"3 Best Ways to Avoid Adverse Outcomes".* The following were the findings of this report:

1. Inadequate preoperative evaluation of the patient
2. Lack of knowledge of the pharmacology of the drugs being administered
3. Inadequate monitoring during the procedure

This course will take these recommendations and others into consideration to insure the patient is safe at all times.

Efficacy

When it comes to efficacy, protocols will be presented that increase the chances to achieve the ultimate goal of sedation dentistry – "To create a calm, comfortable environment for the patient to receive the care they need and want".

Candidates for IV Sedation Dentistry

While any patient who desires and is otherwise acceptable by the standards of case selection can receive sedation for their treatment, the following patient characteristics are especially suited for sedation dentistry:

- Severe Gag Response
- Anxious, Phobic, Fearful Patient
- Local Anesthetic Resistant
- Time Constraints
- Complex Restorative Patient
- Post-Operative Pain, Sensitive Patient

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Benefits of IV Sedation Dentistry

The benefits to the dentist include the following:

- Increased Quality of Treatment
- Increased Quantity of Treatment
- Increased Gratitude from Patients & Team Members
- Decreased Stress! Everywhere
- Decreased Failed Appointments
- Decreased Rejected Treatment Plans

Advantages of IV Sedation Dentistry

Within the realm of sedation alternatives (Parenteral or Enteral), IV sedation has some significant advantages:

- Titrateable
- Fast onset of action
- The ability to deliver both sedative and analgesic drugs
- The ability to address the needs of the hyporesponder
- The ability to address hypoglycemia in the patient who has been NPO for several hours
- The ability to address dehydration in the patient who has been NPO for several hours

Disadvantages of IV Sedation Dentistry

- Like any dental discipline there can be disadvantages as well:
- Duration of action may require frequent dosing
- Potency of medication (requires, careful, conservative titration dosing)
- Needle phobia on the part of the patient, and the dental team
- Complications of venipuncture
- Cost of administration, monitoring, and training

Key Definitions:

Enteral – refers to the enteric membranes of the gastrointestinal tract. The absorption of swallowed, sublingual, or rectally administered forms of sedation medication are considered enteral.

Parenteral – means other than the enteric membranes of the gastrointestinal tract. The absorption of intramuscular, intravenous, subcutaneous (other than sublingual), and inhalation forms of sedation medication are considered parenteral.

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The **route of administration** is the principle factor governing:

- Onset of action
- Potency
- Duration of action

The Sedation Dentistry Guidebook contains tools to promote successful case acceptance by reducing buyer's remorse and provide increased justification to support the patient's decision to seek and complete treatment.

How can we tell if a patient is awake or asleep?

- Our goal with anxiolysis is not to put the patient to sleep. Our goal is to create the most relaxed and comfortable appointment possible, while the patient remains awake and conscious. However, many sedated patients feel like they slept through the entire appointment since they have little or no memory of the appointment. We create a relaxed comfortable environment where a patient will experience little or no anxiety with little or no memory of the appointment.

Our goal is not to create sleep.

How do we tell if our patient is properly sedated?

- Their eyes?
- Their skin color?
- The only way to properly assess the sedation level of our patients is to talk to them, confirming their consciousness by their ability to verbally interact.

The Spectrum of Sedation

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Anxiolysis – A pharmacologically induced state of consciousness where an individual is awake but has decreased anxiety to facilitate coping skills, retaining interaction ability.

Conscious Sedation – A minimally depressed level of consciousness whereby the patient maintains their own airway and reflexes, and responds to physical and *verbal stimuli.*

Minimal Sedation – a minimally depressed level of consciousness, produced by a pharmacological method, that retains the patient's ability to independently and continuously maintain an airway and respond *normally* to tactile stimulation and verbal command. Although cognitive function and coordination may be modestly impaired, ventilatory and cardiovascular functions are unaffected**.**

Moderate Sedation - a drug-induced depression of consciousness during which patients respond *purposefully* to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

Deep Sedation – A depressed level of consciousness whereby the patient may not respond to physical or verbal stimuli, may not maintain their own reflex mechanisms, and needs assistance in maintaining their own airway.

General Anesthesia – An unconscious patient that does not maintain their own reflexes, cannot maintain their own airway, cannot respond to physical or verbal stimuli, and cannot breathe on their own.

*****This Spectrum of Sedation shown is for healthy adults (18 – 64 y.o.) without other CNS depressants on board. This course is designed to teach concepts consistent with the above definitions for anxiolysis for healthy adult dental patients.

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Dental Organization for Continuing Sedation Education

Benefits of Membership

New Patients!

Reach hundreds of thousands potential patients with a listing on SedationCare.com; the #1 positioned consumer site that allows people to search for sedation dentists by zip code. Member profiles were viewed 150,000 times in the past 12 months by those looking for a sedation dentist.

EliteDOCS Forum

Receive answers to your unique patient questions 24 hours a day including the latest clinical and management issues revolving around sedation. Continue the educational process with support and information from your sedation colleagues and DOCS Education faculty.

Mentorship

As a stepping-stone for new sedation dentists, the Mentorship Program connects the dentist with an experienced member to ask questions about anything from patient care and implementation to marketing. Find or become a mentor with this one-of-a-kind experience.

SS Memo

A weekly email covering a complex case study that the faculty found interesting and valuable. This memo will keep your clinical skills sharp and up-to-date.

DOCS Digest

Published quarterly and delivered to your door, the DOCS Digest features articles written by dentists sharing their sedation knowledge and experiences as well as faculty and staff updates**.**

Members Only Information at www.DOCSeducation.org

Gain access to the DOCS Digest, drug dispensing and office forms, team training materials, anesthesia worksheets, guidelines, and many other helpful resources.

DEA Compliant Drug Logbook

Meet the Drug Enforcement Administration's requirements for inventory and the dispensing of medications with this easy-to-use log. Receive a new book with each renewing year.

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Continuing Education

Experience the ever-expanding DOCS Education curriculum for a special member rate giving you, on average, 10% on additional courses. Members typically take at least one course from DOCS annually making the course savings ALONE worth the dues!

Product Discounts and access to New Products at special savings DOCS constantly uses the power of our membership co-op for the best rates. Enjoy tremendous savings on many materials, products, services and equipment. We offer everything you need to incorporate and market sedation dentistry at great savings and find many products guarantee to match prices with any competitor.

Member pricing will save you thousands of dollars in start-up and renewable products to keep you saving throughout your sedation career.

Credentialing: Fellowship/Diplomat Programs

Many doctors love to be proficient in their field – and be recognized for it. Submit case studies in an easy-to-follow format and become a Fellow or Diplomat.
Fellows need to submit 50 pt. cases/75 hours CE
Diplomats need to submit 200 pt. cases/200 hours CE

Regulatory Support

Our entire goal is to keep sedation accessible to qualified practitioners and their patients. Your dues give you access to DOCS Education's full-time Regulatory Counsel to help you stay compliant to your state's sedation and advertising regulations. Receive support regarding your licensure, permits, certifications, and the changing laws that affect your sedation practice.

Oral Sedation Research Fund

Our mission is to document the safe, effective and proven use of incremental oral administration for conscious sedation through performance of clinical studies and surveys. It is our intent to establish
irrefutable evidence of the safety of the incremental oral administration technique. Studies are currently in progress and new grant requests are invited.

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Gold class membership benefits include the above PLUS:

Bring an additional team member and save up to $738

Bring one team member to a course and the second team member is free (one per year). Extend your team training.

SedationCare.com Premier Listing

Your primary office gets preferred listing with a Gold Class Member icon appearing at the top of all searches within the patients' specifications.

Additional office listing: $99

Equipment Pre-sale Notification

Receive first notification of any equipment sale prior to regular members. Get deeper savings on specified products and access to limited quantities first.

Free Online Courses as Available – Save up to $500!

PACE-approved CE from the comfort of your home or office.

Examples include:

Science of Sedation (2 CE)

Incremental (2 CE)

Patient Assessment (2 CE)

Top Dose (1 CE)

Airway Training (1 CE)

Infomercial Customization w/preferred media reservations

Show potential patients the quality of dental care available in their area.

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SEDATION VOYAGE
ANATOMICAL & PHYSIOLOGICAL CONSIDERATIONS

A *basic* understanding of the anatomical structures and associated physiology as it relates to the use of pharmacological agents is necessary for the safe and effective administration of sedation dentistry. This section follows the voyage of sublingually delivered benzodiazepine as it journeys through various organ system. It is an overview of the most critical organ systems associated with conscious sedation in the dental office with an emphasis on normal anatomy and physiology in the healthy adult patient.

The organ systems that will be discussed are:

- Sublingual absorption
- Gastrointestinal
- Cardiovascular and Circulatory
- Central Nervous System
- Respiratory
- Hepatic
- Renal Systems

I. ORAL CAVITY: SUBLINGUAL ABSORPTION

Sedation drugs should be administered sublingually with medications crushed by a pill crusher.

Orally administered medications enter the body through the oral cavity and are either swallowed to pass into subsequent portions of the gastrointestinal tract where they are primarily absorbed in the small intestine, or pass through the sublingual mucosal membranes into the general circulation.

The sublingual plexus of blood vessels approximate the mucosal surface, allowing for soluble medications to pass through the mucosa, into the interstitial space, and into blood vessels, namely, the deep lingual vessels and their associated branches.

The sublingual space is exceedingly vascularized and, for medications that can dissolve in that space, provides an extremely efficient mode of delivery of drug. Sublingual administration permits very rapid absorption via the highly vascularized buccal mucosa. The active ingredients are absorbed directly into the blood stream. This then passes into the jugular vein and the superior vena cava.

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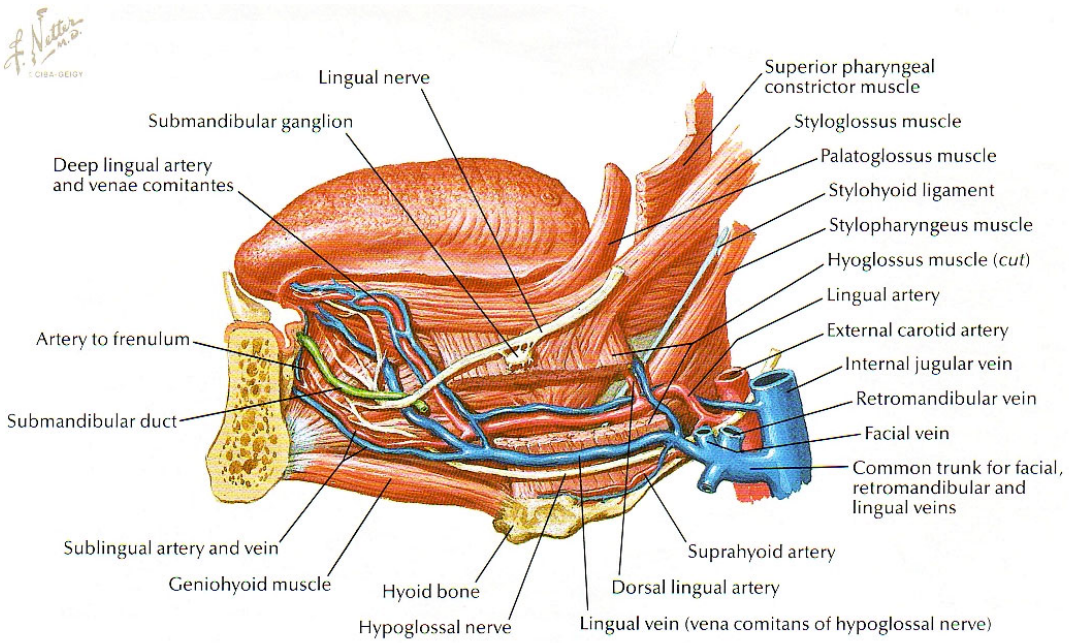
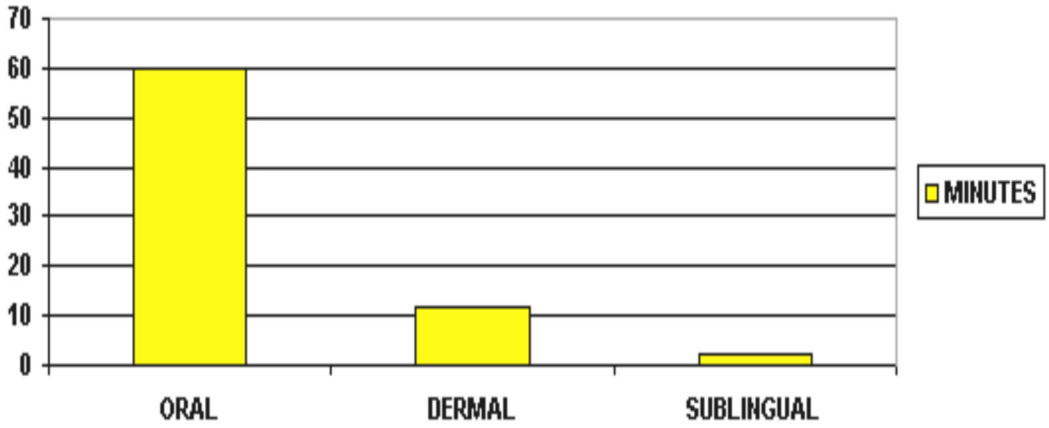
This allows for bypassing the gastrointestinal tract and the liver and therefore avoids the first pass phenomenon of drugs normally absorbed through the gastrointestinal tract.



Many drugs that need rapid mode of action are designed to be absorbed sublingually, the most prominent of which is nitroglycerin. Onset of action of drugs that can be absorbed sublingually is often within a minute with rapid peaking of action.



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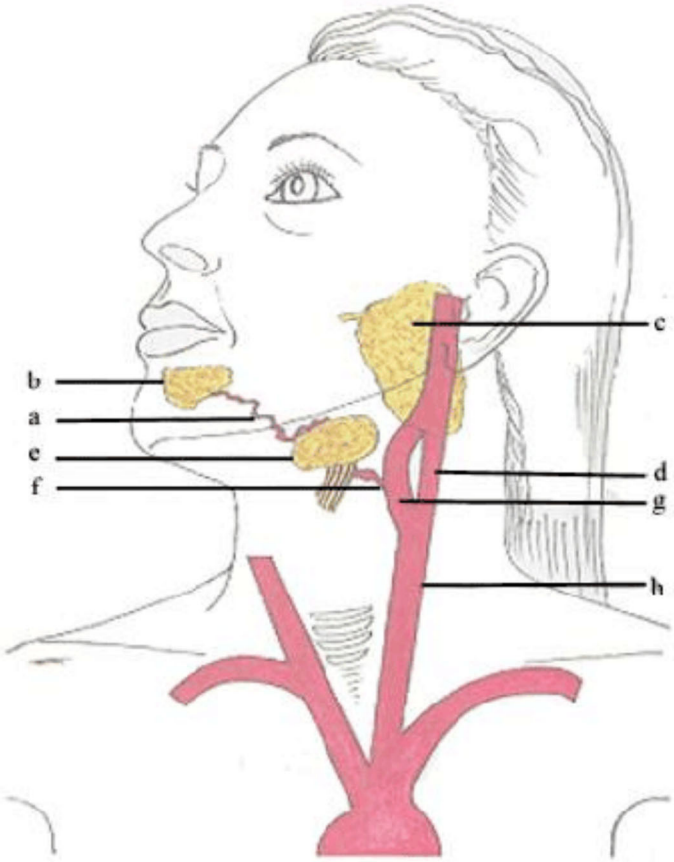

a — sublingual artery
b — sublingual gland
c — parotid gland
d — internal carotid artery
e — submandibular gland
f — lingual artery
g — external carotid artery
h — common carotid artery

II. GASTROINTESTINAL SYSTEM

The function of the gastrointestinal system is to transfer food and water from the external environment to the internal environment, where they can be distributed to the cells of the body by the circulatory system.

THE STOMACH

Very little absorption takes place in the stomach. The most important function of the stomach is to regulate the rate at which substances enters the small intestine, where most of the process of digestion and absorption takes place. In the absence of the stomach, a normal-sized meal moves so rapidly

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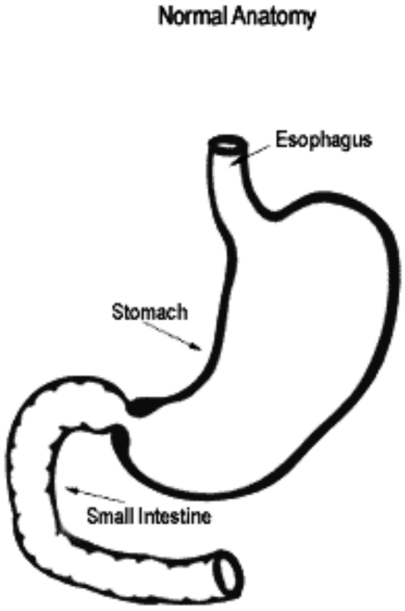
through the small intestine that only a fraction of the food has time to be digested and absorbed.

The activity of the stomach is controlled by the autonomic nervous system. The most important factor controlling gastric emptying is the chemical composition and amount of partially digested food in the upper part of the small intestine (duodenum). When the duodenum contains fat, acid, or hypertonic solutions, or when it is distended, gastric motility is reflexly inhibited. Of these factors, fat in the duodenum is the most potent stimulus for the inhibition of gastric motility. The high fat content of eggs and milk may inhibit gastric emptying to the point where some of the meal may be found in the stomach after 6 hr. In contrast, a meal of meat and potatoes (protein and carbohydrate) may empty in 3 hr. It is therefore important to make the patient NPO after midnight to facilitate the transfer of the stomach content into the small intestines.



From a practical standpoint, asking the patient to be NPO after midnight would optimize the absorption of drugs administered.

THE SMALL INTESTINE

The small intestine is approximately 9 feet in length and can be divided three segments: an initial short 8 inch segment, the duodenum, two much longer segments, the jejunum (3 ft.) and the terminal segment, the ileum. It is in the small intestine that most digestion and absorption occur.

V-3

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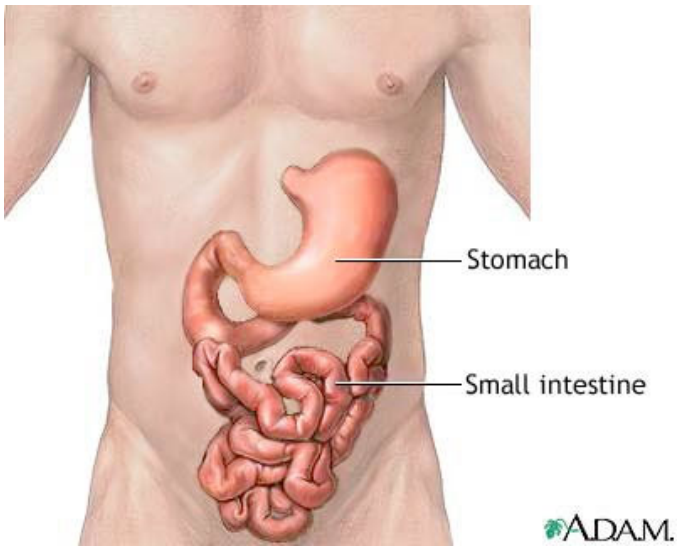
The blood flow to the small intestine of a normal man at rest averages about 1 liter/min. or one-fifth the resting cardiac output. Much of what is absorbed from the small intestine enters the portal circulation for transport to the liver and its actions.



LIVER

Drugs absorbed throughout the gastrointestinal tract enters the portal circulation and is delivered to the liver via the portal system. Here, benzodiazepines are partially metabolized ("First pass " phenomenon). The remainder of the drugs enters the central circulation to be distributed throughout the body.

Liver is the major site of metabolism of all benzodiazepines. Detailed discussion of the metabolism of all of the drugs used in sedation protocols will be undertaken on Day 3 under the section of Pharmacology II.

III. CARDIOVASCULAR & CIRCULATORY SYSTEM

The cardiovascular system's critical role in the maintenance of life lies in its ability to adequately perfuse vital organs and tissues with oxygenated blood, nutritive substances, as well as route blood to organs for purification and waste removal. Sedative drugs can affect the functioning of the primary organ of the cardiovascular system, the heart, through their effects on the respiratory and central nervous systems.

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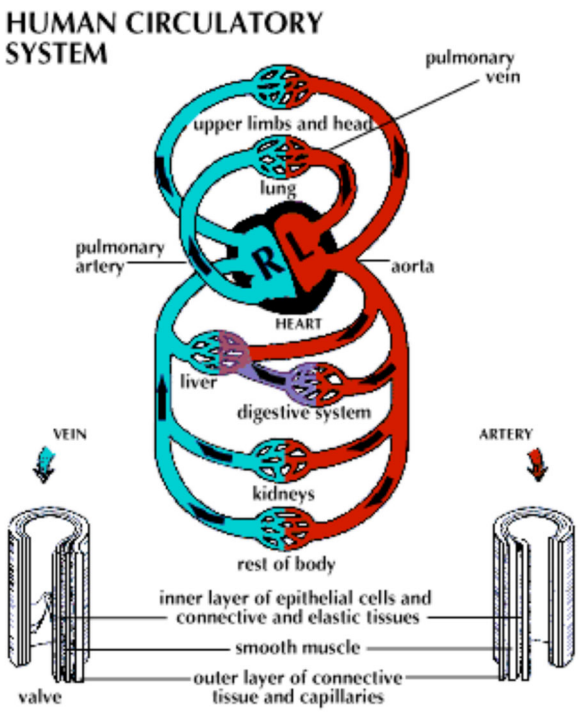
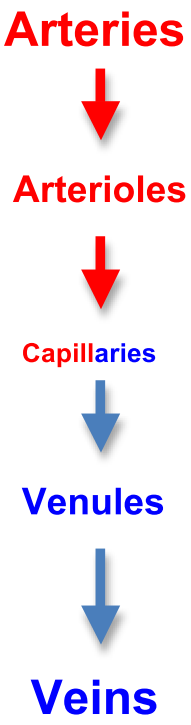
PERIPHERAL CIRCULATION

The peripheral circulatory system consists of a system of tubes called the arteries and veins and further subdivide into smaller vessels called arterioles and veinules and finally into fine vessels called capillaries.





Capillaries absorbed the nutrients and gather into the tubular beginnings of a return system with the formation of the venules. Venules converge to form larger vessels, the veins, which run parallel to the arteries. Two or more veins of equivalent size approach each other to form a larger one. The same principle applies to the formation of the larger veins of the extremities, head, and trunk until two major venous vessels are formed. These are the venae cavae, which conduct the blood from the superior and inferior body regions into the heart.

From the heart, blood is pumped into the systemic circulation by way of the large vessels, arteries. The arteries gradually become smaller and simpler as they diverge and repeatedly branch within all parts of the body. They continue as microscopic vessels, the arterioles, which finally divide into a network of tiny vessls, the capillaries, within the tissues and in proximity with the intercellular substance.

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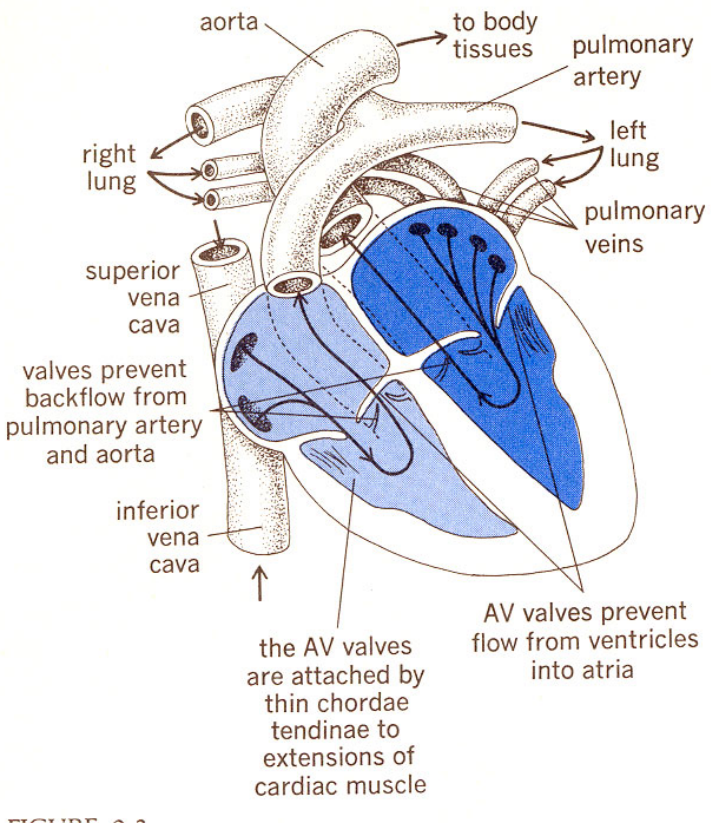
THE HEART

The heart consists of four chambers, two atria, and two ventricles. Blood coming into the heart from the superior and inferior vena cava will go into the right atrium. Blood would then go into the right ventricle and is pumped out via the pulmonary artery into the lungs. Here the blood becomes oxygenated and returns back to the heart via the pulmonary veins.

Blood would now enter into the left atrium and then the left ventricle. Contraction of the left ventricle would lead to pumping of the blood out of the heart into the systemic circulation via the aorta.

V-5



A. THE ELECTRICAL SYSTEM

The contraction of the heart within a cardiac cycle must be carefully synchronized if function is to be normal. This responsibility is attributed to a specialized system of tissues that depolarizes spontaneously. These

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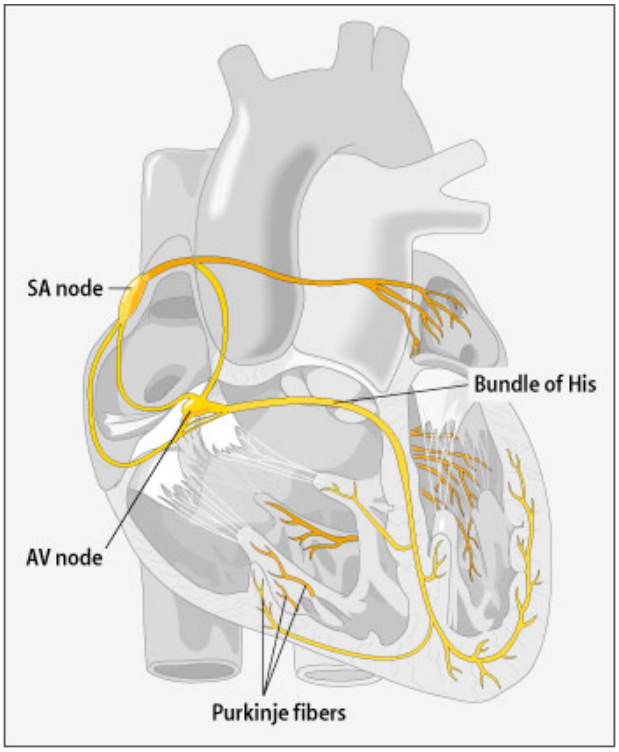
impulses spread directly onto myocardial cells, igniting them to depolarize and contract. Essential components of this specialized system, as well as the sequence of their conduction, commence with the sinoatrial node (SA node), spreading through the atrioventricular node (AV node), bundle of His, and Purkinje fibers.



Because its spontaneous firing rate is greater than that of the others, the SA node normally functions as the heart's pacemaker. The intrinsic firing rates of all these tissues are influenced by autonomic innervations: sympathetic fibers increase the rates, and parasympathetic fibers decrease the rates. Diseases in the SA node can cause rhythm abnormalities such as atrial fibrillation.

Atrial fibrillation is a relatively common cardiac arrhythmia. It is usually related to ischemia to the SA node, with resultant chaotic rhythm because of multiple random firings from different parts of the atria. Patients with atrial fibrillation are often on anticoagulants such as Coumadin to decrease the likelihood of thrombus formation in the atrium and embolic strokes from these thrombi.

Atrial fibrillation is the most common reason for a patient to be on Coumadin. In these patients, concerns over hemostasis need to be addressed.

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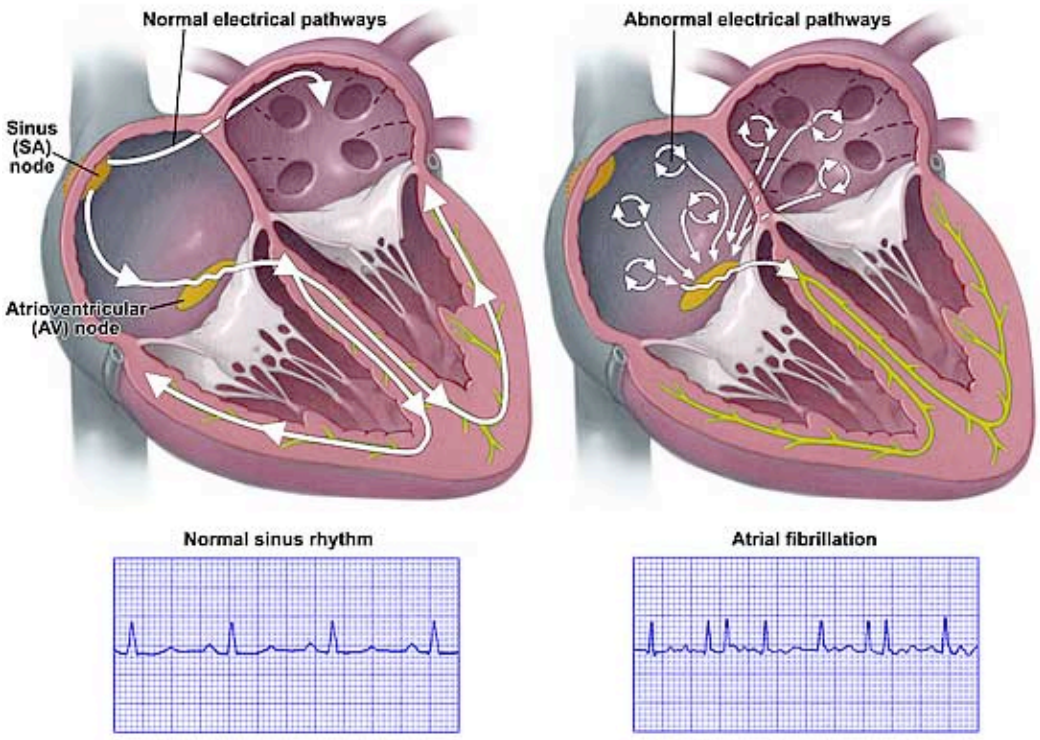

The electrical event has to go down the conduction system to reach the ventricle. Abnormalities in the conduction system can result in heart block. A very slow heart rate can obviously cause significant problems with perfusion of vital organ. The tracing below shows a patient with complete heart block and a heart rate of 14 beats per minute:

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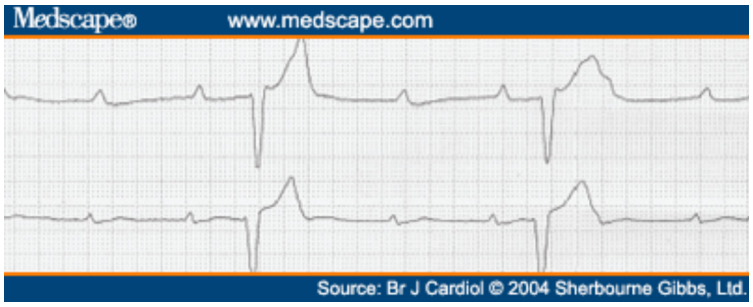

Heart rate is controlled by the autonomic nervous system with the sympathetic system responsible for increasing the heart rate and the parasympathetic nervous system responsible for decreasing the heart rate

The key component dictating the integrity of cardiac function is the perfusion of the heart via the coronary arteries. Coronary artery disease causes angina, myocardial infarction, heart block and arrhythmia.

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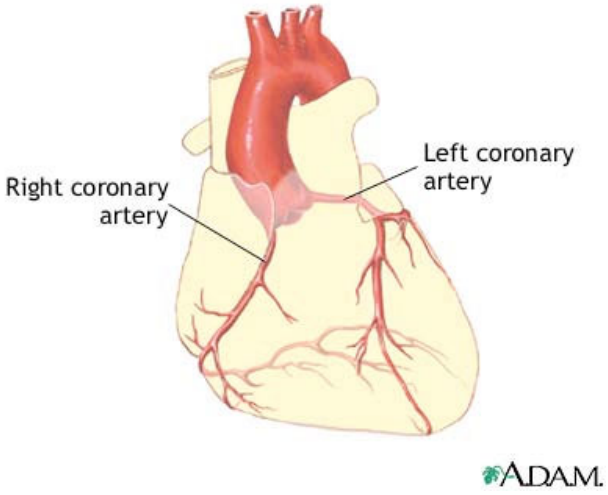
B. CORONARY PERFUSION

Coronary arteries are responsible for delivery of oxygen to the heart. Inadequate perfusion results in angina or myocardial infarction as well as arrhythmia, including lethal arrhythmias such as ventricular tachycardia or ventricular fibrillation.



The major determinants of adequacy of coronary perfusion include:

1. Patency of the coronary arteries
2. Oxygenation of the blood (pO2 and, indirectly, SO2)
3. Hemoglobin content: anemia would decrease the oxygen carrying capacity.

Progression of a plaque or rupture of the cap of a plaque can result in acute blockage of the artery, with resultant myocardial infarction.

Acute myocardial infarction is a life-threatening event and can be complicated by congestive heart failure, arrhythmia, heart blocks and cardiopulmonary arrest.

It is therefore critical to carefully assess patients with respect to risk factors associated with coronary artery disease.

If critical coronary artery disease can be detected before complete occlusion, coronary intervention such as coronary stents and coronary artery bypass graft can be undertaken.

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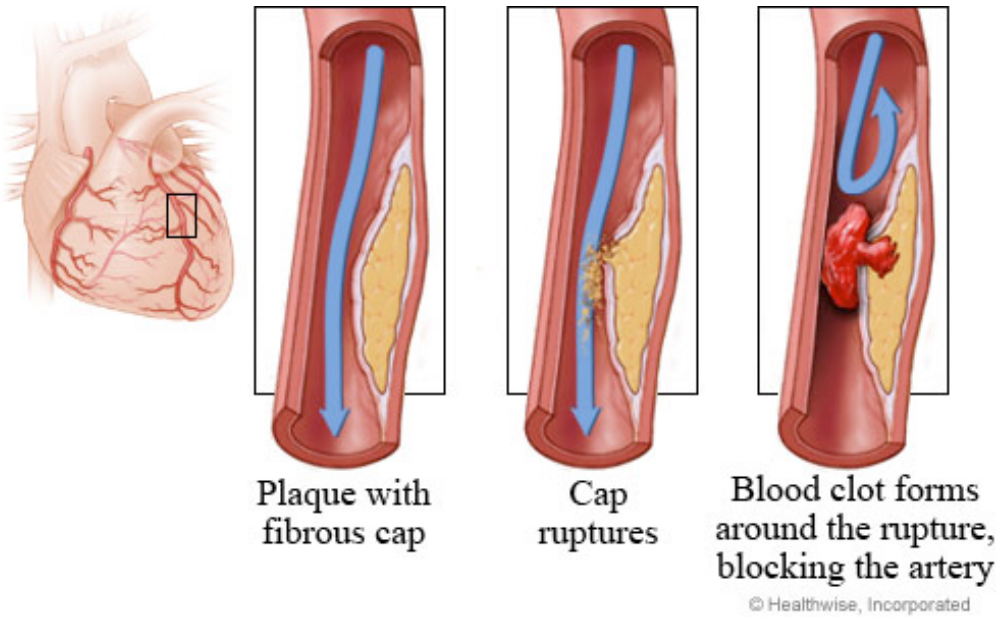

C. CORONARY DEMAND: BLOOD PRESSURE AND PULSE

The energy requirement of the heart is dependent upon:

1. Heart rate: increased rate increases energy demand
2. Pre-load: filling pressure of the heart determines adequacy of the circulatory volume
3. Afterload: Blood pressure

Arterial blood pressure fluctuates according to specific events in each cardiac cycle. The pressure is greatest during ventricular systole (systolic blood pressure) and is attributed to ventricular ejection. At the completion of systole the ventricles enter a period of rest (diastole), and their pressure decreases to zero. However, arterial pressure does not decline this far because, on closure of the aortic valve, resistance within the arterial system maintains a diastolic pressure. Vessel diameter and elasticity are the principal factors that determine the degree of arterial resistance and therefore diastolic pressure.

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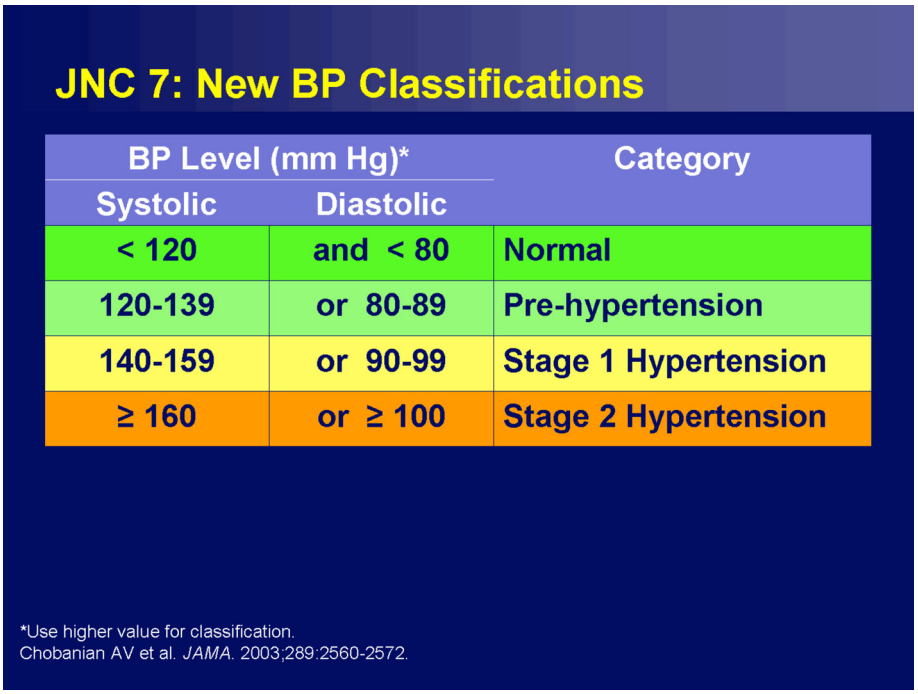
Normal Blood Pressure Numbers



Latest Classification of blood pressure for adults JCN VII guidelines

These guidelines have significant impact on dental interventions on hypertensive patients:

BP Classification	Systolic mm Hg	Diastolic mm Hg	Lifestyle Modification	Dental Rx
Normal	< 120	< 80	Encourage	No Limitations
Pre-Hypertension	120-139	80-89	Yes	No Limitations
Stage I HTN	140-159	90-99	Yes	No Limitations inform patient
Stage II HTN	>159	>99	Yes	Selective non-stressful treatment only; Immediate MD Referral

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D. IMBALANCE BETWEEN DEMAND AND SUPPLY

Coronary ischemia results when the cardiac demand outstrips supply.
Angina is the result of reversible ischemia.
Myocardial infarction is the result of irreversible ischemia.

Coronary ischemia promotes heart blocks and arrhythmias, including lethal arrhythmias that can result in cardiac arrest.

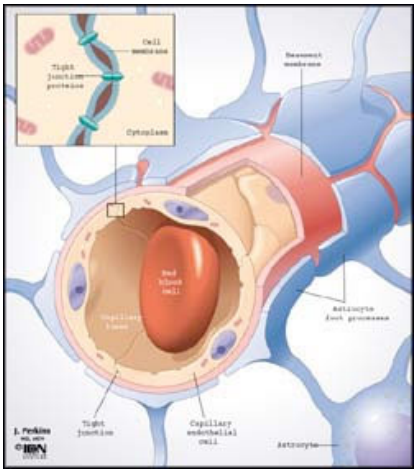
MONITORING OF CARDIAC FUNCTION

Blood pressure
Pulse rate
Rhythm
Should be recorded every 5 minutes during sedation

IV. NERVOUS SYSTEM: CENTRAL NERVOUS SYSTEM

THE BLOOD-BRAIN BARRIER

A complex group of blood-brain barrier mechanisms closely controls both the kinds of substances which enter the extra-cellular space of the brain and the rate at which they enter. The blood brain barrier is formed by fusion of foot processes from astrocytes of the brain. The barrier controls entry of substances into the central nervous system.



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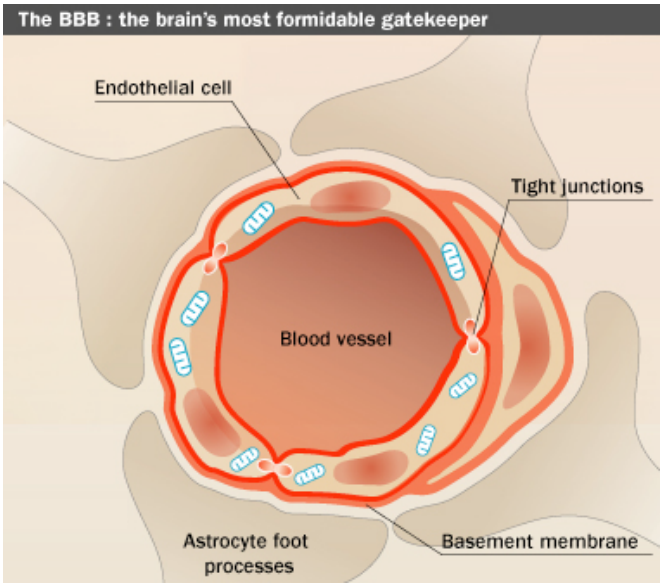
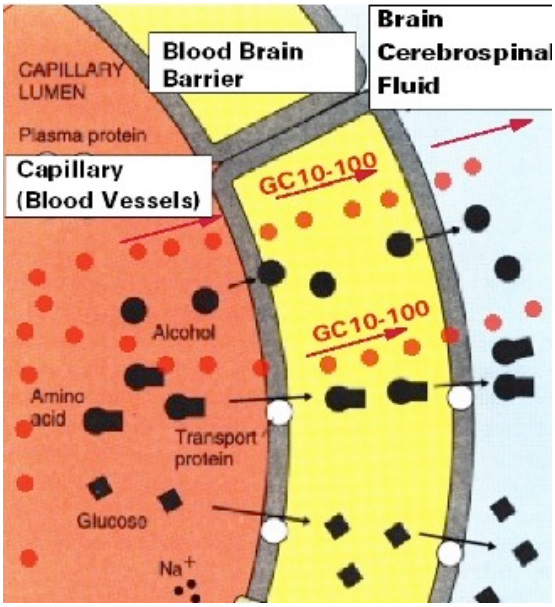


The barrier comprises both anatomical structures and physiological transport systems which handle different classes of substances in different ways. The blood-brain barrier mechanisms precisely regulate the chemical composition of the extra-cellular space of the brain and prevent harmful substances from reaching neural tissue.



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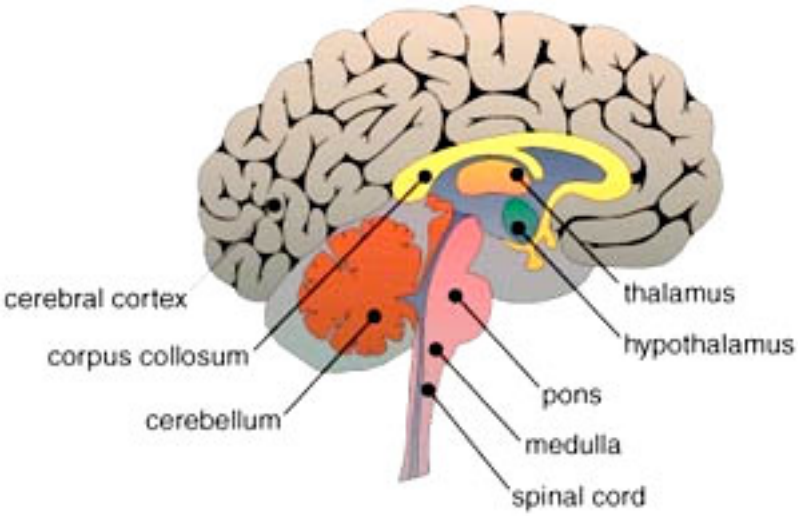
THE BRAIN

The nervous system regulates the function of other systems of the body and provides for the awareness of changes which occur within the body or in its environment. It can be further divided into the central and peripheral nervous systems. The Central Nervous System is the site of action where sedative medications exert their pharmacologic effect, and consists of two main parts, the spinal cord and the brain. The brain is the portion of the central nervous system that concerns us most as sedation dentists. The primary divisions of the brain are the brainstem, cerebellum, and cerebrum. The brainstem is composed of the medulla, pons, and midbrain.



RETICULAR ACTIVATING SYSTEM

Running though the entire brainstem is a core of tissue called the reticular activating system. The reticular activating system is made up of the cerebral cortex, basal ganglia, limbic system, and other regions deep within the cerebrum. Some reticular activating system neurons are clustered together, forming “centers”. Reticular activating system is responsible for a variety of functions, including the state of consciousness. Within the reticular activating system are cardiovascular (blood pressure, heart rate, vascular tone), respiratory, and vomiting centers.

Sedation drugs exert its action by suppressing the reticular activating system. The goal is to decrease the state of consciousness.

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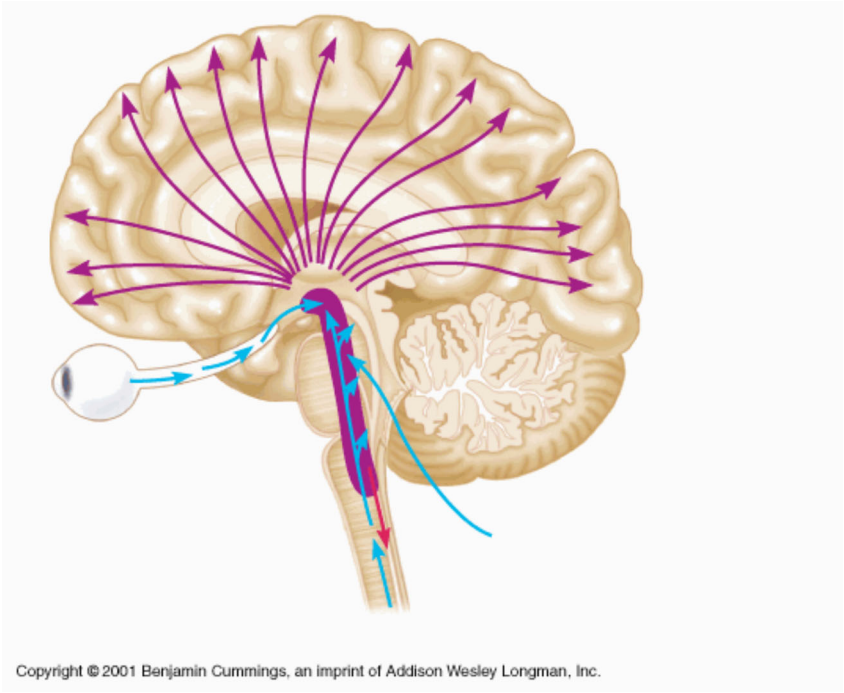
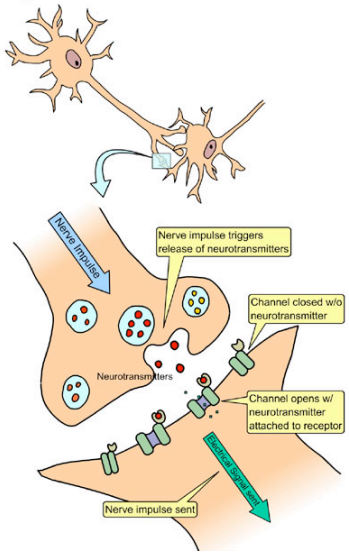


However, since the RAS is also responsible for control of blood pressure, heart rate, vascular tone and respiratory function, these have to be carefully monitored during the sedation process.

Gamma-aminobutyric acid (GABA)

Most neurological transmission is mediated by neurotransmitters. These are chemical mediators that travel from the pre-synaptic neuron to the receiving cell (the post-synaptic neuron).

Figure D-2: Neurotransmitters



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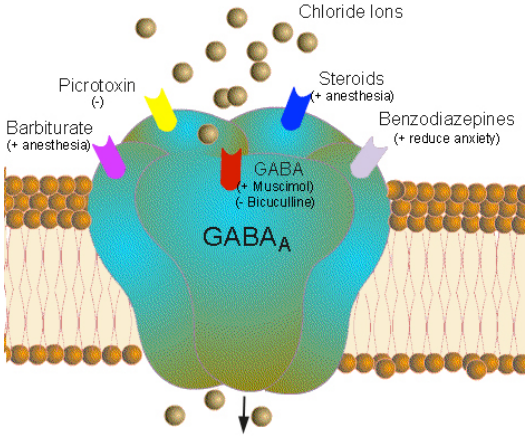
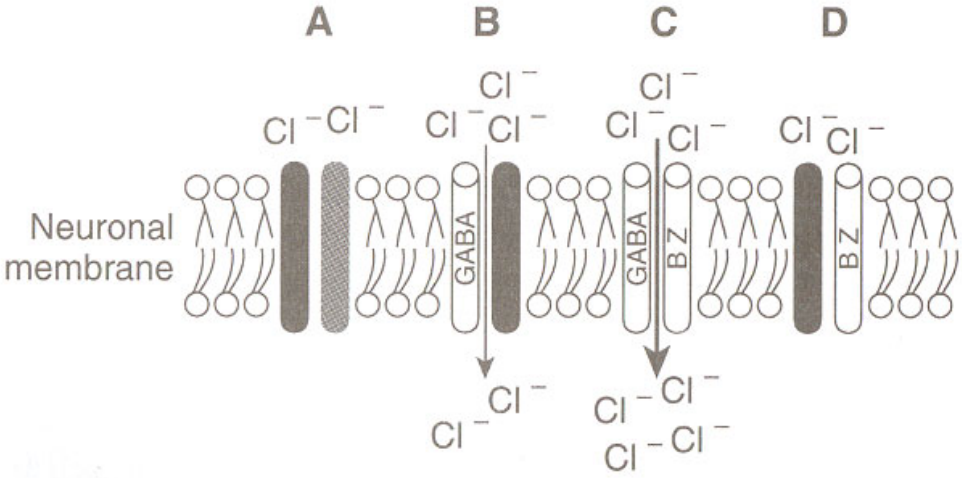
The transmission of signals by chemical neurotransmitters allows the body a means of fine regulation of nerve activation or inhibition.

The principal inhibitory neurotransmitter in the brain is gamma-aminobutyric acid (GABA).

The GABA receptor is located within a large protein complex that surrounds chloride ion channels (chloride ionophores) found in neuronal cell membranes. When GABA binds to this receptor, the channel opens, allowing greater numbers of chloride ions to enter the cell. This influx of negative ions hyperpolarizes the neuron, rendering it less responsive to excitatory signals. Some sedative agents can bind to the GABA-chloride inonophore complex and potentiate the influence of GABA-mediated opening of the chloride channels.





A. No GABA: chloride is on the outside of the neuron
A. GABA binds to receptor and causes influx of chloride ions
B. Benzodiazepine (BZ) enhances action of GABA
C. In the absence of GABA, BZ has no effect

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Benzodiazepines act on GABA receptors in all parts of the brain and accentuates the inhibitory effects of GABA.

MONITORING SEDATION

Response to verbal commands
Blood pressure
Pulse rate
Rhythm
Respiratory mechanics
Pulse oximetry

V. RESPIRATORY SYSTEM

The respiratory system is the most susceptible of all organ systems to adverse outcome associated with conscious sedation. Drugs used for these procedures can depress cardiovascular function directly, but adverse cardiovascular events are more likely caused by respiratory depression. Medications used for sedation can also cause loss of muscle tone in the posterior pharynx, resulting in upper airway obstruction by the tongue over the larynx.

A. ANATOMY AND PHYSIOLOGY

The airways are a system of tubes that connect the outside atmosphere and the gas exchanging surfaces of the lungs. The upper portion of the respiratory tree is the nasal and oral cavities, down through the larynx and trachea. The airway then divides into the right and the left mainstem bronchi. From here, air would travel down to the smaller bronchioles and eventually to the alveoli, which are tiny airsacs within the lungs where gas exchange takes place.

At its most simple level, external respiration is the diffusing of gases between the alveoli and the pulmonary circulation, which is separated by the wall of each alveolus and each capillary. There are approximately 300,000,000 alveoli in the average adult human.

The diffusion of these gases (oxygen and carbon dioxide) is mediated by the partial pressures of the gases on either side of the membranes. In general, oxygen will diffuse from the external air through the membrane into the capillary blood of the alveoli. CO_2, on the other hand, will diffuse from the capillary blood into the air in the alveolar sac. These partial pressures of

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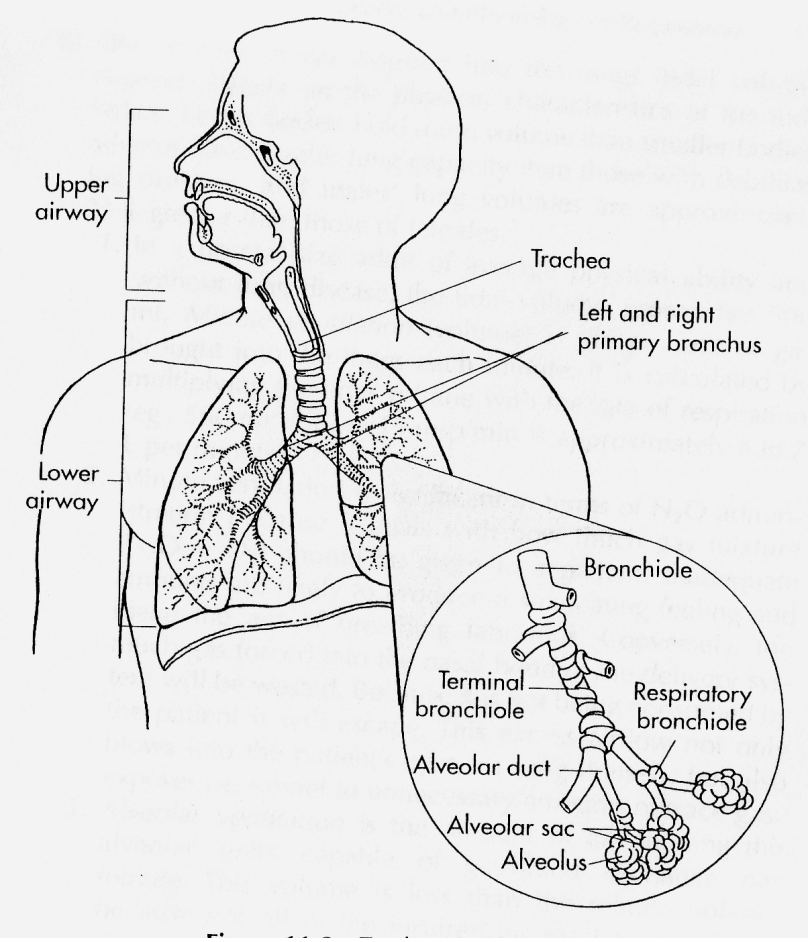
gases will lead to oxygenation of the blood in the pulmonary vessels and removal of CO_2 from the blood into the exhaled air.



Anatomy of upper and lower airways

Oxygen is transported from the lung capillaries to the systemic circulation, whereas the byproduct of cellular metabolism, carbon dioxide, is transported back to the lungs for expiration. Hemoglobin is a protein found in erythrocytes, the predominant cellular component of the blood, and is responsible for oxygen transportation throughout the body. High concentrations of oxygen in the lung capillaries favor the combination of oxygen and hemoglobin, conversely low oxygen tension in the peripheral tissues favors oxygen release. 85% of carbon dioxide is carried in the blood as sodium bicarbonate; the remainder is transported in the form of carboxyhemoglobin. Since venous circulation is characterized by a low O2 tension and a relatively high CO2 tension, the pressure gradient favors expiration of CO2 and diffusion of oxygen back into the lung capillaries.

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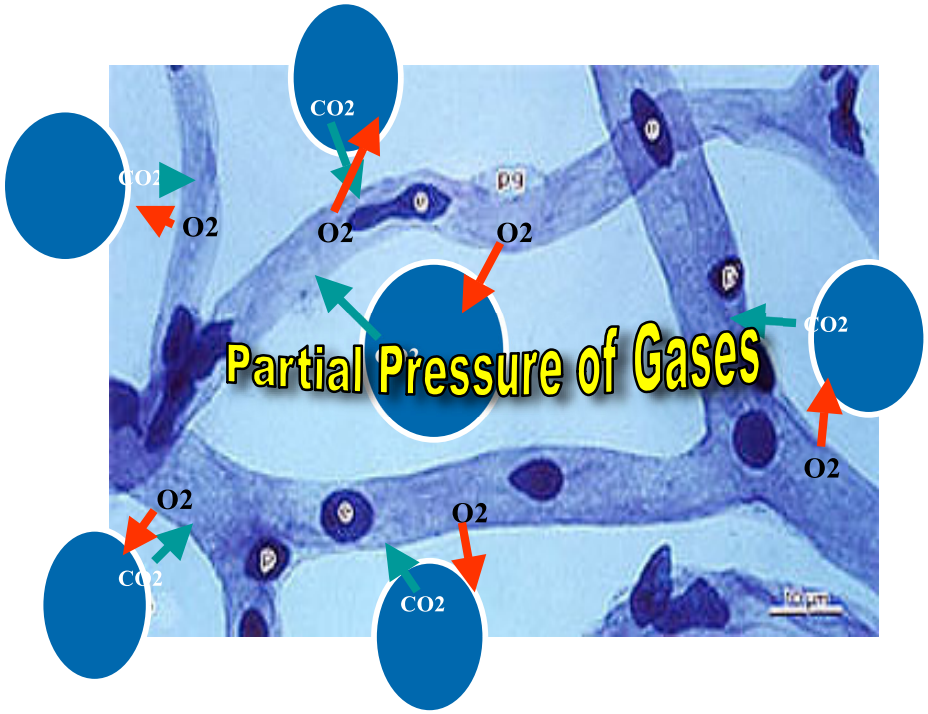
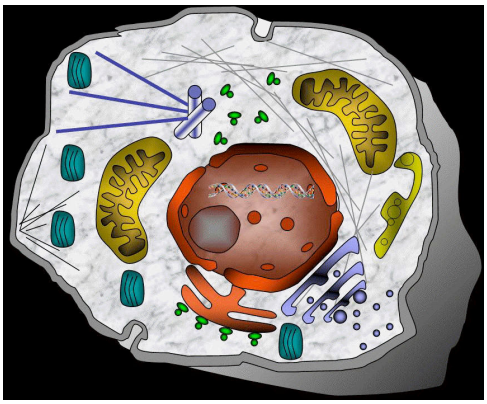


The normal partial pressures of gases in the arterial system are:

pO_2	100 mm of Hg
pCO_2	40 mm of Hg
pH	7.40

Intracellular respiration involves the exchange of gases between the blood and the tissue cells. Normal metabolic functions within the cells require oxygen for energy production and produce carbon dioxide as a byproduct. As intracellular oxygen levels decrease, and carbon dioxide levels rise, the pressure gradient is favorable for the diffusion of gases and the re-oxygenation of the cells. Energy production in the cell, utilizing oxygen and producing carbon dioxide, occurs in the mitochondria.



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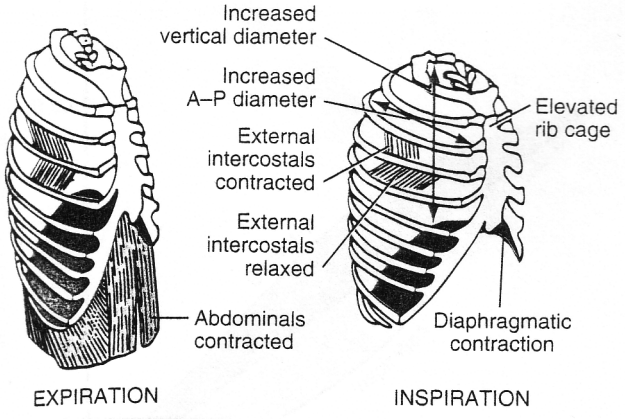
B. MECHANICS OF RESPIRATION

Normal in/outflow of gases into the respiratory system is mediated by differences in partial pressures and the rhythmic expansion of the chest cavity. During inspiration, the chest cavity expands through outward movement of the ribs and a downward movement of the diaphragm. This expansion of the lung creates a negative pressure gradient with the outside atmosphere and air enters the lungs and equilibrates the system. Upon expiration the opposite is true, the lung muscles relax and the diaphragm moves upward increasing intra-thoracic pressure and forcing air outward. Inspiration is characterized by muscular effort while expiration most commonly is passive, unless forced.



Expansion and contraction of the thoracic cavity during inspiration and expiration

C. STIMULUS OF RESPIRATION

The respiratory center is located in the medulla and initiates neural impulses to respiratory muscles that generate inspiratory effort. The respiratory center is stimulated by two principal mechanisms, so-called hypercapnic and hypoxemic drives.

Receptors for the hypercapnic drive are located within the respiratory center and are triggered by hydrogen ions produced by ionization of carbonic acid. In essence, the central receptors would drive respiration whenever the pCO2 level exceeds 45 mm Hg. This would result in CNS acidosis and would trigger respirations.

$$CO_2 + H_20 \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO3^-$$

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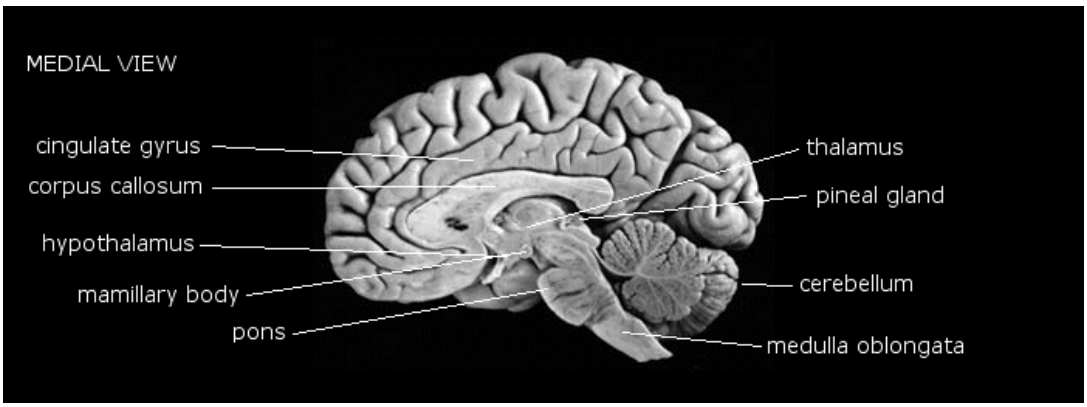
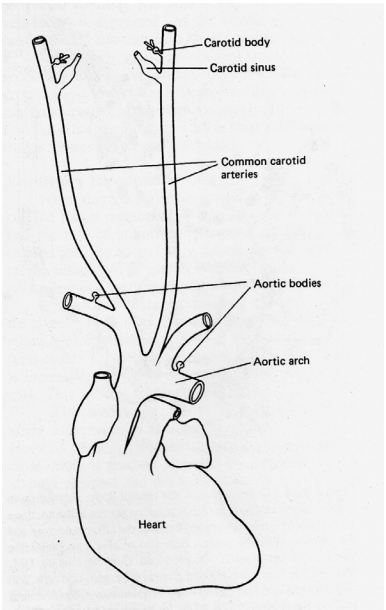


Sedation drugs would blunt the central hypercapneic drive to breathe. However, sedation drugs do not blunt the peripheral hypoxemic drive. This is mediated via receptors located peripherally, within the carotid and aortic bodies, and sense arterial oxygen tensions below 60 mm Hg (Oxygen saturation of <90%)



Locations of aortic and carotid bodies

As sedation dentists we administer medications that can create hypoxic situations. It is therefore critical to keep oxygen saturation above 90%. This would allow the more sensitive central hypercapneic drive to guide respiration and avoid using the less sensitive peripheral hypoxemic drive to dictate the safety margin.

Different drug classes can produce differing levels of respiratory depression, as well as upper airway compromise related to loss of muscle tone in the posterior pharynx. For this reason the maintenance of a patent airway must always be paramount.

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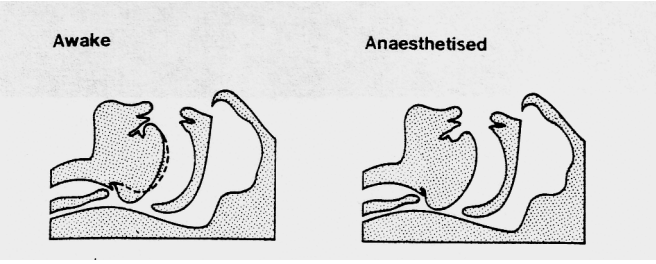
UPPER AIRWAY CONCERNS

Upper airway obstruction can be accentuated by improper head position. This can be corrected by repositioning the patient, proper use of a chair pad and appropriate positioning of the chin.



Upper airway difficulty is more commonly seen in obese patients.

Patients with sleep apnea are particularly prone to upper airway difficulties. Sleep apnea patients are ASA III patients and are not candidates for sedation until the dentist has had extensive experience and training.

D. MONITORING OF RESPIRATION

1. Look: watch rise and fall of the thorax during the procedure
2. Hear: listen for signs of airway obstruction
3. Pulse oximeter: keep SaO_2 above 90%, with the understanding that levels below 90% reflects pO_2 below 60 mm of Hg.

PULSE OXIMETER is a critical piece of equipment for sedation since changes in SaO_2 is an exceedingly sensitive indicator of respiratory compromises.

It is important to point out that pulse oximetry measures oxygen saturation (SaO_2), which is related to PaO_2 indirectly via the oxyhemoglobin dissociation curve.

Oxyhemoglobin Dissociation Curve

SaO_2 = oxygen saturation of the blood as defined as % of heme sites occupied by an oxygen molecule. Expressed in %. Measured by the Pulse Oximeter

PaO_2 = partial atmospheric pressure of oxygen that is dissolved in the blood. Measured in mmHg. Oxygen molecules pass from alveoli to blood and from

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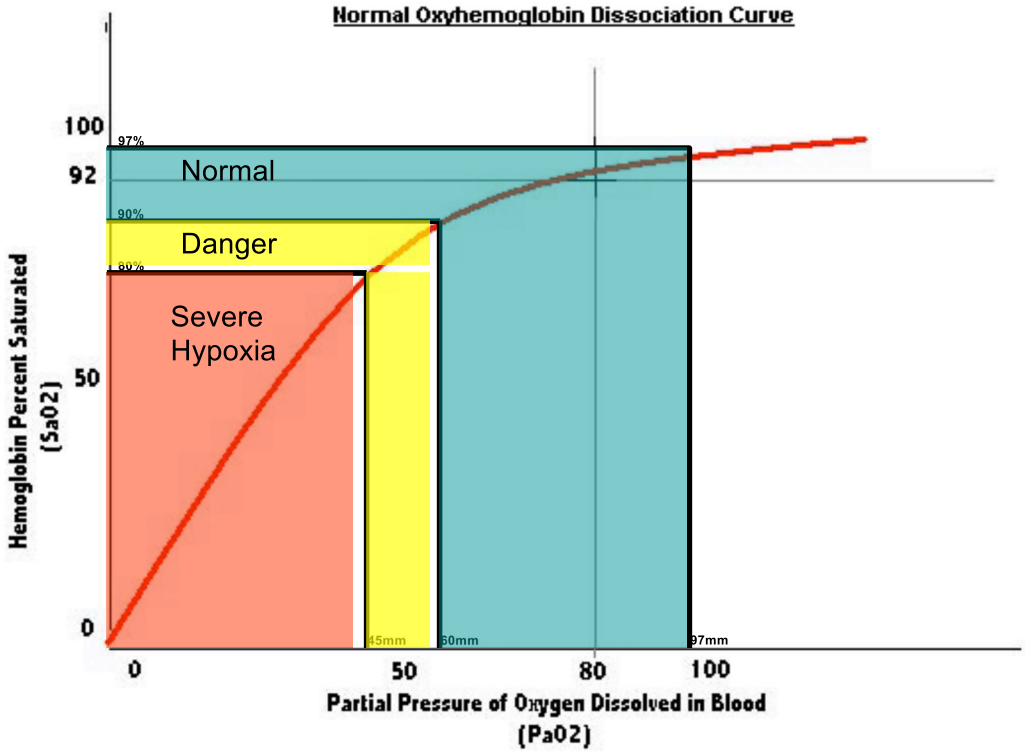
blood to other oxygen poor cells across a passive diffusion gradient. Measured in mm Hg.



The relationship between the amount of oxygen dissolved in the blood and the amount attached to the hemoglobin is called the oxyhemoglobin dissociation curve.

97% saturation = 97 mmHg (PaO_2) – Normal
Below 90% saturation = 60 mmHg (PaO_2) – Danger!
Below 80% saturation = 45 mmHg (PaO_2) – Severe Hypoxia!

When the SaO_2 drops below 90%, the PaO_2 drops steeply, resulting in a substantial drop in the pressure that drives oxygen from the blood into oxygen poor tissues.

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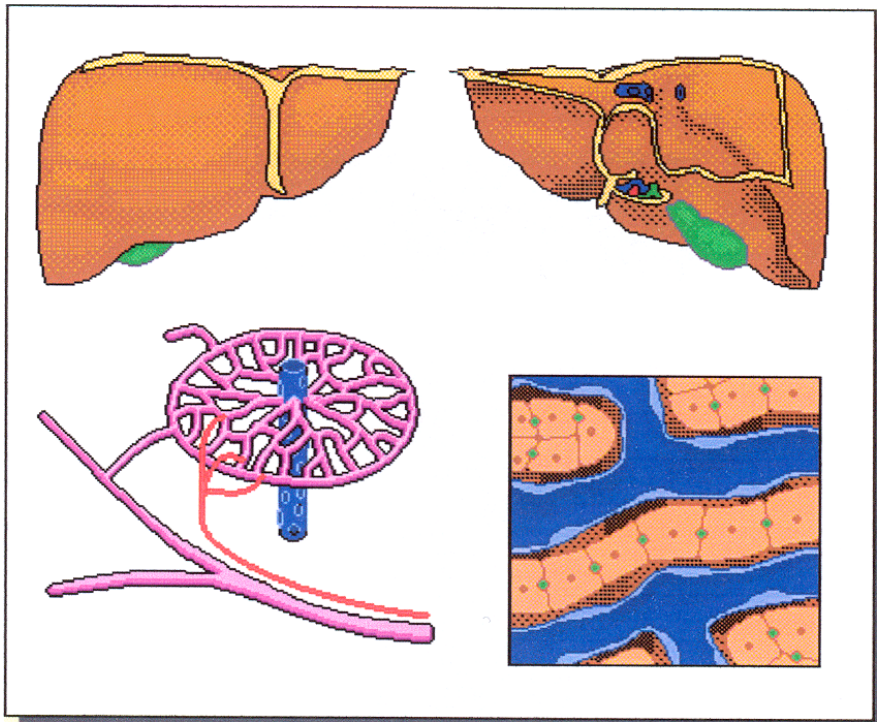
VI. HEPATIC SYSTEM

THE LIVER

The liver is the largest gland of the body, made up of myriads of microscopic hepatic lobules. The hepatic lobule is a hexagonal structure made up of anastomosing plates of hepatic cells which radiate outward from a central vein. At the angles of the hexagons are strands of connective tissue through which course branches of the portal vein and the hepatic artery. During its passage the blood nourishes the liver cells and the contained food substances are withdrawn or acted upon by the hepatic cells. Many metabolic actions are performed simultaneously within the cytoplasm of the hepatic cells.
Hepatic functions of concern to us as sedation dentists include:

1. Synthetic function
   - Proteins
   - Cholesterol
   - Hormones
   - Clotting factors
2. Biotransformation
   - Detoxification
   - Metabolism



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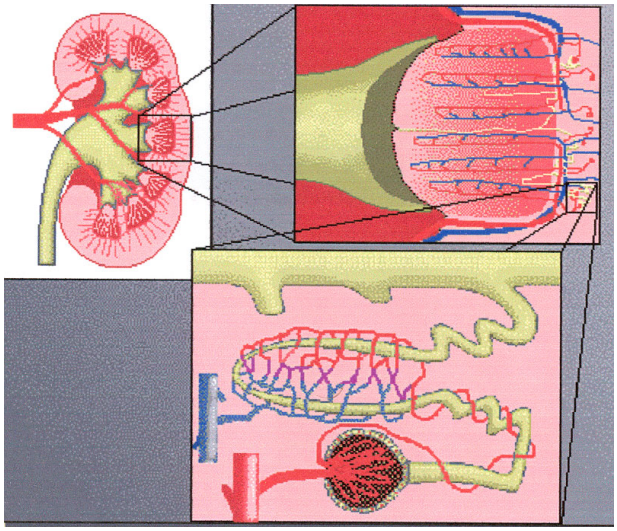
VII. RENAL SYSTEM

THE KIDNEY

The typical human kidney is composed of approximately 1 million tiny units, nephrons, all similar in structure and function. Each nephron consists of a vascular component (the glomerulus), and a tubular component. The renal glomeruli form a tremendous blood filtering mechanism. All of an individual's blood passes through the kidneys about 15 times an hour.



Metabolic wastes in the form of compounds such as urea are removed from the blood by filtration into the glomerular fluid. Many drugs, as well as substances toxic to the body, are also removed from the blood by this route.

The appearance of large numbers of epithelial cells, blood cells, or cellular debris is an indication of disease of the urinary system. In microscopic and chemical examinations of the urine (urinalysis) these elements are watched for. The appearance of large molecular proteins, such as albumin, is an indication that damage to the renal filter by disease is permitting them to escape from the blood.

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IV Sedation Pharmacology

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Dean of Faculty, DOCS Education

PHARMACOLOGY

It can be argued that the three most important subjects when it comes to the safety and efficacy of sedation are pharmacology, patient selection, and airway management. All three subjects are tied intimately to patient safety, while pharmacology also has a great deal to do with sedation effectiveness. Understanding the mechanisms of how drugs act and how the body responds to their action is the key to their safe and effective use.

Pharmacology, from the Greek meaning the study (logos), of drugs (pharmacy), is the science that deals with the study of drugs and their interaction with living systems.

There are numerous subtopics with the general overall topic of Pharmacology. The three subtopics that are most applicable to IV Sedation are:

- Pharmacokinetics – the interaction between drugs and the body as they move within the body, and consists of:
    - Absorption
    - Metabolism
    - Distribution, Redistribution
    - Elimination
- Pharmacodynamics – the interaction between drugs and the body at the site of pharmacologic action, often a receptor for a naturally occurring substance. In the case of sedation, the receptors are in the Central Nervous System.
- Pharmacotherapeutics – the selection of a remedial agent for a desired action. The action of these agents can also produce an undesired action(s).

Pharmacokinetics

Potency & Efficacy

The **dose-effect curve** (see below) plots the dose of a drug versus the intensity of its effect. The basic shape of most drugs on this graph is a sigmoidal or “s-shaped” curve that depicts the increase in intensity as the dose of the drug increases. There is a plateau or “ceiling-effect” that most drugs have where the intensity of effect does not increase with escalating dose.

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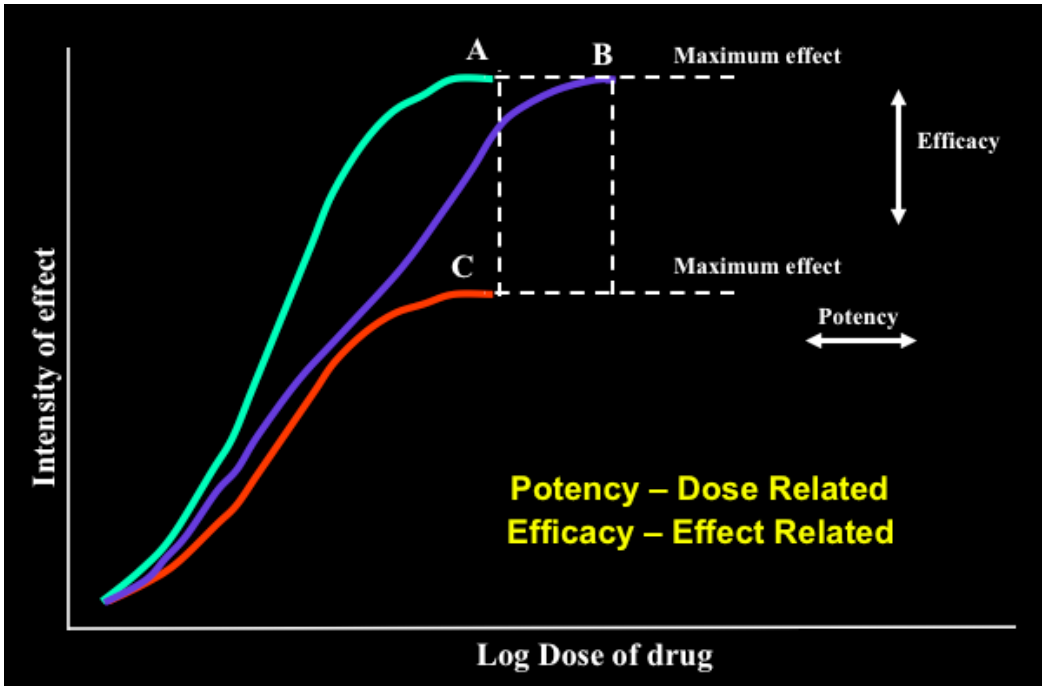
Different drugs with a similar action will likely have the same basic shape plotted on the dose-effect curve but will be positioned differently relative to the x- and y-axis. The difference between two drugs that reflects the y- value (vertical height of curve) represents **efficacy** and maximum effect.



The difference between the two drugs that reflects the x- value (horizontal dimension) represents **potency**, or the relative dose size required to cause the drug effect. Potency increases as the dose required to cause an effect decreases.

The effective dose of a drug is the amount of a drug that produces the desired therapeutic response. A drug's **ED50** (Effective Dose 50) is the average dose given to a general population that produces the desired effect in 50% of that population. In toxicology, the median lethal dose, or **LD50** (Lethal Dose 50) is the dose that causes death in 50% of the subjects taking it. The **Therapeutic Index** is a measure of drug safety as it describes the amount of difference (distance on the dose-effect curve) between the drug's LD50 and ED50. It can be arrived at mathematically by dividing a drug's LD50 by its ED50.

The concentration of a drug in the patient's plasma can be plotted over time on the **Time-Concentration Curve** (see below). The shape of the curve depicts the pharmacokinetic properties of absorption, distribution, metabolism, and elimination. The plasma concentration at which enough drug first causes observable signs of the desired action is called the **minimum effective concentration ($M_{in}EC$)**. The concentration at which no more of the desired effect is noticed despite increasing drug is called the **maximum effective concentration ($M_{ax}EC$)**. When the dose of drug exceeds this concentration, untoward effects (side effects) increase in frequency and severity. The distance

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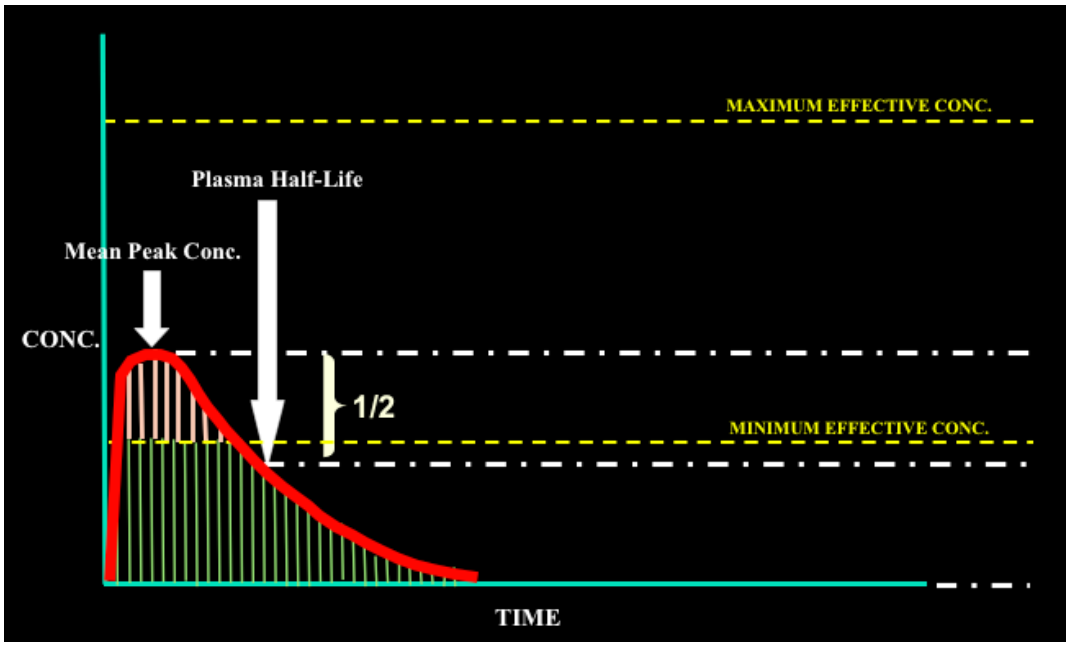
on the Time-Concentration Curve between the minimum effective concentration and the maximum effective concentration is called the **Therapeutic Range**.

The time between time zero (when drug is administered) and when the $M_{in}EC$ is reached is the **onset of action**. After a single dose of a drug, there will be a maximum concentration, which in a group of subjects the average maximum concentration is called the **mean peak concentration**. This concentration in the plasma corresponds roughly with the maximum clinical effects from that single dose.



Due to distribution, metabolism, and elimination of the drug, the concentration will eventually fall below the $M_{in}EC$. The time that the concentration of the drug was above the $M_{in}Ec$ is the drug's **duration of action**. As the concentration continues to fall, it will reach a point at which half the drug has been eliminated from the plasma. This point is called the **plasma half-life**, and is useful in determining how long it will take for the body to eliminate the drug. As a general rule, at four times the half-life of a drug, 90% of the drug will be cleared from the plasma.

This gradual elimination (4X half-life) from the plasma represents **First-Order Kinetics** (elimination). This occurs because the rate of elimination is affected by the remaining concentration of the drug. Some drugs are eliminated through **Zero-Order Kinetics**, where the drug elimination is not affected by the concentration of the drug used, resulting in accelerated removal of the drug (~2X half-life).

The time concentration curve does not differ between enteral (oral) and parenteral (eg. IV), administration in general shape, but does differ in the time involved in reaching each of the parameters measured by the curve (onset of action, duration of action, time to mean-peak concentration, and half-life).

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Compared to oral administration, IV administration is faster and shorter regarding these parameters.

Whenever a drug or multiple drugs are administered together by two different methods of absorption (eg. oral + IV), there is a mixture of pharmacologic characteristics. This combination of effects can be used to take advantage of the desirable clinical characteristics of both methods of administration.

Metabolism

Chemical characteristics of a drug impact its pharmacologic characteristics (potency and efficacy). Lipophilic characteristics promote absorption (reducing onset of action and increasing potency). In order to eliminate a drug, the body needs to transform it into a lipophobic (hydrophilic) compound by making it more polar so that it can be more easily excreted by the renal route.

Phase 1 Metabolism

- Also called functionalization reaction
- Removes or introduces a functional group on the parent compound
- Usually causes an inactive compound, but can result in an existing active drug being metabolized to another active drug
- A **Prodrug** is an inactive compound that is converted to an active form through metabolism. Examples include:
    - Acetylsalicylic Acid (Aspirin)
    - Levodopa
    - Enalapril
    - Prednisone

Phase I Processes

- Enzymatic process carried out by the Cytochrome P450 Monooxygenase System (“Microsomal enzymes”) of the liver and gut
    1. Oxidation – adding oxygen (majority of reactions)
    2. Reduction – removing oxygen
    3. Methylation
    4. Demethylation
    5. Hydrolysis- addition of water molecule to cleave ester drugs

An example is the Phase I Metabolism of Flumazenil. Flumazenil is metabolized into three different metabolites through methylation, oxidation, and demethylation.

Cytochrome P450 Monooxygenase System

- 50 active enzymes in humans
- Majority of all drug metabolism occurs as a result of these isoenzymes:

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  - CYP1A2, CYP1A2, CYP2C19, CYP2C8/9, CYP2E1, CYP2B6, CYP2A6, CYP3A4
- Predominant enzyme in this family of enzymes is CYP3A4
- The Cytochrome family of isoenzymes are named according to the family (CYP), sub-family (eg. 3A), and gene (eg. 4)
- Mostly located in the liver and mucosa of the gastrointestinal tract
- After the drug passes through the stomach, it enters the duodenum of the small intestine. From there it is absorbed into the enterocytes of the small intestine where it interacts with CYP enzymes. After that it is transported through the portal circulation where it is absorbed into the hepatocytes of the liver where it interacts with CYP enzymes. The net result of these metabolic interactions is that of the original 100% of drug that exits the stomach, 15% enters the general circulation as unmetabolized drug.

Induction and Inhibition of CYP3A4

The activity of CYP450 enzymes can be altered by medications the patient is taking in one of two general ways:

Induction – associated with an increase in enzyme activity. Enzyme induction occurs when hepatic blood flow is increased, or the synthesis of more CYP450 enzymes is stimulated. For drugs that are substrates of the isoenzyme induced, the effect is to lower the concentration of these substrates. The clinical consequence of the presence of an inducing agent and the resultant decrease in concentration of the substrate may mean loss of efficacy.

Examples of drugs known to cause induction of CYP3A4 include: Rifampin, Phenytoin, Barbiturates, St. John's Wort, Carbamezapine, and Dexamethasone.

Agents Known To Cause Induction of CYP3A4

Generic/Class	Brand Name	Degree of Interaction[1]	Rank in Top 100 Drugs[2]
Theophylline Derivatives	Uniphy, Theo-24	D	N/A
Rifampin	Rifadin	D	N/A
Phenytoin	Dilantin	C	N/A
Barbiturates	Luminal (Phenobarbital)	C	N/A
St. John's Wort	Tipton's Weed	C	OTC
Carbamazepine	Tegretol	C	N/A
Dexamethosone	DexPak	C	N/A

[1]Lexi-Comp Dental Drug Reference Library
[2]Top 100 Most Prescribed Drugs, USA National Pharmaceutical Magazine

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Inhibition – refers to the decrease in metabolic enzyme activity due to the presence of an inhibitor substrate. This may result in higher concentrations and/or prolonged half-life of another substrate drug, which enhances the potential for toxic side effects.

Drug metabolism by CYP450 can be inhibited by any of the following mechanisms:

- Competitive Inhibition (most common and occurs as a result of competitive binding at the enzyme's normal or orthosteric binding site). Agents known to act as competing substrates of the CYP3A4 enzyme are the Protease Inhibitors, Tetracycline, Cimetidine, and Cyclosporine.

Agents Known To Act As Competing Substrates

Generic/Class	Brand Name	Degree of Interaction[1]	Rank in Top 100 Drugs[2]
Protease Inhibitors	Norvir (Ritonavir)	D	N/A
Tetracycline	Doxycycline	C	51
Cimetidine	Tagamet	C	OTC
Cyclosporine	Neoral	C	N/A

- [1]Lexi-Comp Dental Drug Reference Library
- [2]Top 100 Most Prescribed Drugs, USA National Pharmaceutical Magazine

  - The **Protease Inhibitors** were first approved by the FDA in 1995 for treatment of HIV and Hepatitis C. They include drugs such as Indinavir (Crixivan), Ritonavir (Norvir), and Saquinavir (Invarase). They have a very high affinity for the CYP-450 enzymes, significantly inhibiting the metabolism of other drugs. In the time concentration curve, this results in an increased area under the curve (AUC). Practically, it means shorter onset of action, greater relatve potency, greater relative efficacy, longer duration of action, and in some cases, a longer half-life. For this reason, many oral sedatives are contraindicated in conjunction with protease inhibitors.

- Noncompetitive Inhibition occurs when an inhibitor binds to a different or allosteric site on the enzyme resulting in a conformational change making it unavailable for metabolism of another drug.

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Agents Known To Inhibit CYP3A4

Generic/Class	Brand Name	Degree of Interaction[1]	Rank in Top 100 Drugs[2]
Diltiazem	Cardizem	D	95
Verapamil	Calan, Isoptin	D	59
Clarithromycin	Biaxin	D	N/A
Fluconazole	Diflucan	D	48
Itraconazole	Sporanox	D	N/A
Ketaconazole	Nizoral	D	N/A
Nefazadone	Serzone	D	N/A
Grapefruit Juice	N/A	D	N/A

[1]Lexi-Comp Dental Drug Reference Library
[2]Top 100 Most Prescribed Drugs, USA National Pharmaceutical Magazine

- **Diltiazem and Verapamil** are non-dihydropyridine calcium channel blockers prescribed to treat hypertension and angina. They inhibit the metabolism of other drugs metabolized by the CYP-450 enzymes, resulting in increased serum concentration of those drugs, and prolonging their duration of action. If oral sedatives are planned, using a sedative not metabolized by the CYP-450 system is appropriate.
- The **Macrolide Antibiotics** such as Erythromycin, Clarithromycin, and Dirithromycin inhibit CYP3A4 metabolism. Therefore, oral sedative drugs that are metabolized by this enzyme (eg. Triazolam) should be avoided. Oral Lorazeapm, however, since it is not metabolized by CYP-450 enzymes is an acceptable alternative.
- **Systemic Azole Derivatives** such as Fluconazole (Diflucan), Itraconazole (Sporanox), and Ketaconazole (Nizoral), used to treat fungal infections, are major substrates of CYP3A4. They result in an increased AUC for other substrates of that enzyme. If oral sedatives are contemplated, agents that are not metabolized by CYP3A4 (eg. Lorazepam) should be considered.
- **Gemfibrozil** (Lopid), an antilipemic agent, is known to inhibit CYP3A4 and CYP2C19. The latter enzyme is predominantly responsible for Phase I metabolism of Diazepam.
- **Grapefruit Juice** is known as a "Suicide Inhibitor" of the CYP3A4 within the enterocytes of the small intestine, completely destroying these enzymes for 48-72 hours. This results in a relative increase in the circulating concentration of drugs metabolized by CYP3A4, but not an increase in half-life. The chemical within grapefruit responsible for this action is furanocoumarin.

CYP-450 enzyme inhibition can lead to toxic concentration of a substrate of that enzyme. An example is the drug Terfenadine (Seldane), a peripheral acting anti-histamine. While originally synthesized as a potential tranquilizer, it was found to be inactive for that purpose due to its inability to enter the CNS. However, the

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similarity of its chemical structure to diphenhydramine led researchers to explore its potential use as an antihistamine and was brought to market in the U.S. in 1985 as the first non-sedating antihistamine for the treatment of allergic rhinitis.

In the early 1990s, evidence of serious ventricular arrythmias (prolonged QT interval causing *torsades de pointes*) resulted in the FDA issuing a black box warning, and in 1997 terfenadine was withdrawn from the market. It was determined that a metabolite of terfenadine metabolism was the culprit through the blockage of potassium channels in the cardiovascular system. A metabolite of that agent however was determined to not have that effect, and thus fexofenadine (Allegra), was introduced in 1996 as a non-sedating antihistamine.

Polymorphism

- √ The presence or absence of an enzyme can be idiosyncratic or associated with certain patient subsets
- √ Absence of an enzyme means that the patient will have a lesser ability to metabolize the drug which may have serious consequences
- √ Hyperactivity of an enzyme means that the patient will have accelerated metabolism of the drug which will limits its efficacy

CYP2D6 Deficiency

- Increased potential for drug interactions and side effects
- May result in accumulation of CYP2D6 metabolized drugs and higher serum drug concentrations
- Patient Populations: 5%-10% of whites

Garcia-Barcelo M. *Clin Chem*. 200;46:18–23.
Bertilsson L. *Clin Pharmacokinet*. 1995;29:192–209.

CYP2D6 Hyperactivity

- Ultra-rapid metabolizer phenotype
- Overactive CYP2D6 activity
- Patient populations
- Northern European countries, 2%-4%
- Mediterranean area, 7%-12%
- Ethiopia, 29%
- Saudi Arabia, 21%

Kaiser R et al. *J Clin Oncol*. 2002;20:2805-2811.

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Top 100 Prescribed Drugs That Are Major Substrates Of CYP2D6

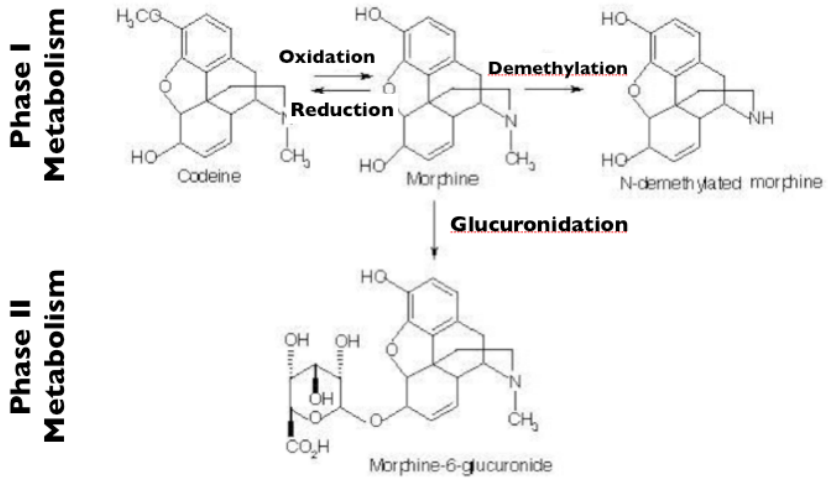
Generic	Brand Name	Rank
Sertraline	Zoloft	12
Metoprolol	Lopressor, Toprol	13
Fluoxetine	Prozac	19
Tramadol	Ultram	31
Paroxetine	Paxil	36
Propranolol	Inderal	78
Mirtazapine	Remeron	86
Hydroxyzine	Atarax, Vistaril	N/A

Phase II Metabolism

- Formation of a covalent linkage between functional group on active compound or phase I metabolite with endogenously derived:
  - Glucuronic acid
  - Sulfate
  - Glutathione
  - Amino acids
  - Acetate
- The resulting metabolite will be more polar and more easily excreted by the kidneys

Phase I and II metabolism often occur together. An example is the metabolism of Morphine. Morphine can be metabolized by Phase I metabolism through methylation (Codeine), and demethylation (N-demethylated morphine). It can also be metabolized by Phase II metabolism through glucuronidation to an inactive metabolite (Morphine-6-glucoronide).

Metabolism Of Morphine



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Metabolism is often more complicated than depicted here. For example, Tryptophan, an essential amino acid, involves numerous metabolic reactions, resulting in literally hundreds of metabolites, both active and inactive

Bioavailability

Plasma Protein Binding

A drug's potency may be affected by the degree to which it binds to the proteins within blood plasma. This phenomenon likely developed as a defense against potentially harmful foreign substances that found their way into the bloodstream, as it is the unbound fraction of drug in the plasma which exhibits pharmacodynamic actions. The less bound a drug is, the more efficiently it can traverse cell membranes or diffuse. Common blood proteins that drugs bind to are human serum albumin, lipoprotein, glycoprotein, and globulin.

Another defense mechanism against harmful substances involves **P-Glycoprotein** (P-gp, or Pgp). Pgp acts as an efflux pump within various body compartments including the CNS, kidneys, and intestinal walls, resulting in reduced bioavailability. Blocking Pgp to increase the effectiveness of chemotherapy has been a focus of cancer treatment.

Pharmacodynamics

Drugs cause their effect by attaching to and causing an effect at the receptor site. Receptors are macromolecules with protein-like properties and three-dimensional shapes. They are usually embedded into a cell membrane and when stimulated by a neurotransmitter or drug (ligand), an action occurs or is prevented from occurring within the cell.

Drugs "mimic" or prevent the activity of a neurotransmitter by having a shape that is similar to the transmitter, allowing it to bind to the receptor site. There are four basic types of receptors based on their action when stimulated (agonism):

1. Receptors that allow for conductance of ions
2. Receptors that result in G-Protein activation that allows for generation of a second messenger within the cell
3. Receptors that cause phosphorylation of key signaling molecules
4. Receptors that allow for transport of substances to the interior of the cell that activate transcription and translation

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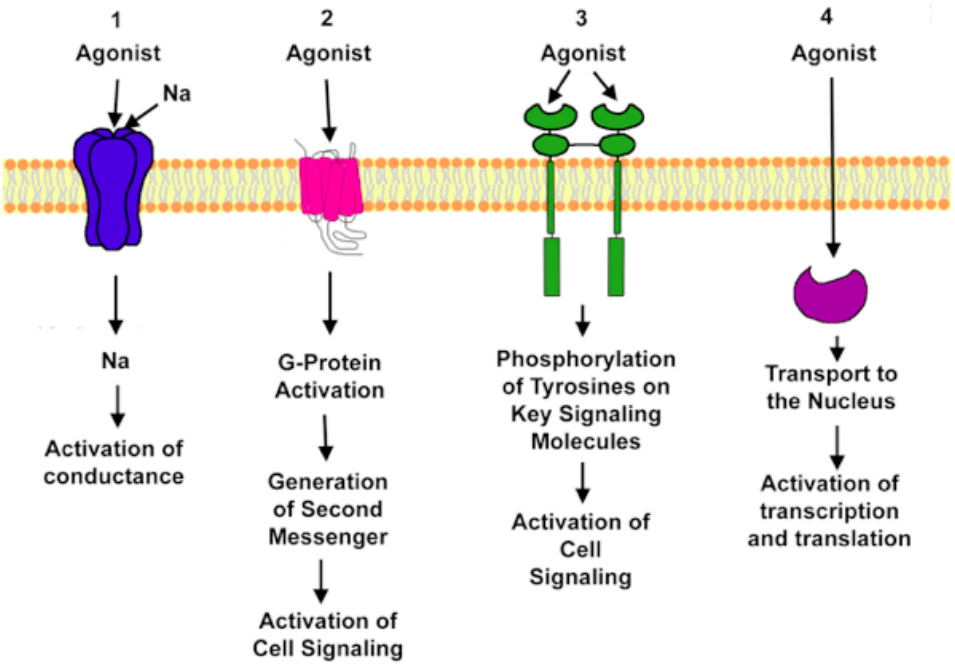
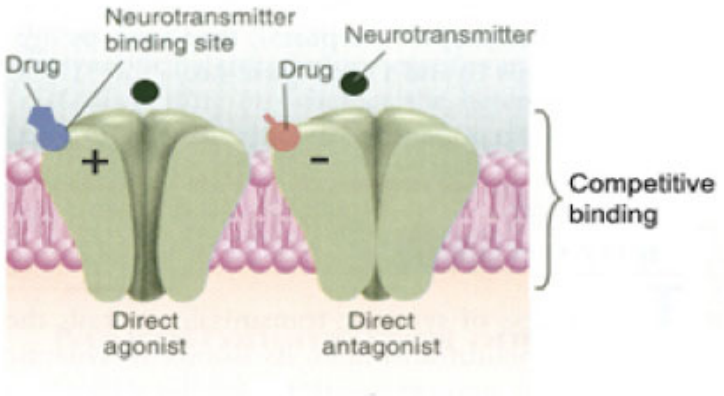
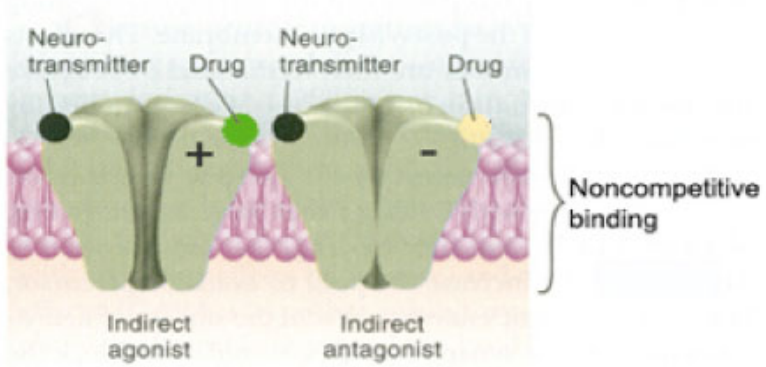
Types Of Receptors



Drug Effects on a Receptor





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Agonists/Antagonists

Agonists:

- Have affinity for, combine with, & **activate** receptors
- Competitive (Direct) agonists bind to the "natural" or orthosteric site
- Non-Competitive (Indirect) agonists bind to an "unnatural" or allosteric site
- Physiologic (Direct)
    - Affinity for a different receptor than agonist
    - Increases maximal response of agonist by producing a similar effect via different receptors
        - Synergism – when two or more drugs with similar pharmacologic effects act together to produce a greater effect than either drug alone
            - Additive Synergism – The combined drug effects are essentially the sum of their individual effects (1 + 1 = 2)
            - Potentiating Synergism – The combined drug effects are greater than the sum of their individual effects (1 + 1 > 2)

Antagonists deactivate the receptor's actions and can be:

- Competitive (Direct)
    - Combines with, has affinity for, and makes the receptor unavailable for combination with an agonist
    - No intrinsic activity
- Non-Competitive (Indirect)
    - Binds to different receptor site than agonist and reduces response
- Physiologic (Indirect)
    - Affinity for a different receptor than agonist
    - Decreases maximal response of agonist by producing an opposite effect via different receptor

GABA & The GABA Receptor

Gamma Amino Butyric Acid (GABA) is the primary inhibitory neurotransmitter in the central nervous system (CNS). It is found throughout the CNS in about 1/3 of all brain synapses. Derived from glutamate, GABA is the endogenous ligand for the GABA receptor.

When GABA receptors in the Limbic system of the brain (portion of the CNS responsible for emotions such as anxiety and responses to stimuli) are activated by the orthosteric agonist GABA, this portion of the CNS is depressed, causing a decreased level of consciousness and anxiety.

The GABA receptor consists of five subunits arranged around a central pore. The receptor sits in the membrane of its neuron, usually localized at a synapse, postsynaptically.

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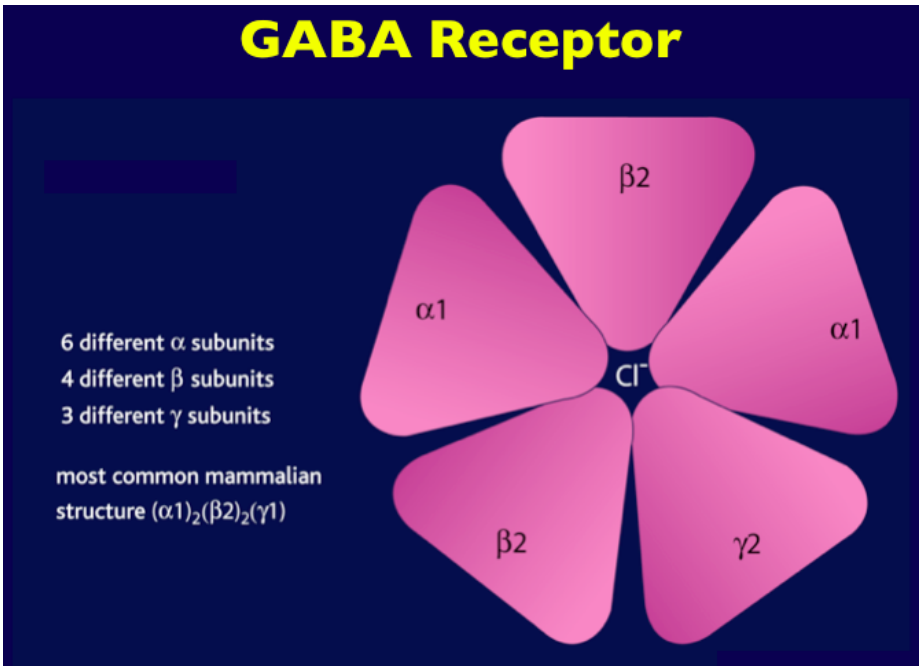

The receptor binds two GABA molecules, at the interface between the α and ß subunit. Binding of GABA molecules to their binding sites on the extracellular portion of the GABA Receptor triggers the opening of a chloride ion-selective pore. The increased chloride conductance causes hyperpolarization of the neuron, inhibiting the firing of new action potentials.

Substrates of the GABA Receptor may act as:

- Agonists – Direct or Indirect
- Antagonists – Direct or Indirect

Direct Agonists - Bind to orthosteric site (Competitive Binding)

Examples:

- GABA (natural ligand)
- Gaboxadol
- Ibotenic Acid
- Muscimol
- Progabide

Indirect Agonists - Bind to allosteric site (Non-Competitive Binding)

- Cause increased efficiency of main site
- Also called Positive Allosteric Modulators

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Examples:

- •Barbiturates
- •Benzodiazepines
- •Carisoprodol
- •Ethanol
- •Etomidate
- •Glutethimide
- •Kavalactones
- •Meprobamate
- •Methaqualone
- •Neuroactive steroids
- •Niacin/niacinamide
- •Non-benzodiazepines
- •Propofol
- •Theanine
- •Valerenic acid
- •Volatile anesthetics

Direct Antagonists - Bind to orthosteric site (Competitive Binding)

Examples:

- Bicuculline
- Gabazine

Indirect Antagonists - Bind to allosteric site (Non-Competitive Binding)

- Cause decreased efficiency of main site
- Also called Negative Allosteric Modulators

Examples:

- Flumazenil
- Ro-4513
- Sarmazenil
- Non-competitive channel blockers
- Cicutoxin
- Oenathotoxin
- Pentylenetetrazole
- Picrotoxin
- Thujone

BZs Pharmacologic Effects

- Sedative/Hypnotic
- Muscle Relaxant
- Anxiolytic
- Anticonvulsant
- Antidepressant

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BZs Adverse Effects – Like other drugs, many of the adverse effects of the benzodiazepines are extensions of their desirable effects, and are dose-related

- CNS Depression
- Anterograde Amnesia
- Visual Effects
- Cardiovascular Effects
- Respiratory Depression
    - Extremely rare, yet:
        - If not recognized – patient not properly monitored
        - If not appropriated treated – not administering the reversal agent
    - Can be the cause of the most serious complication
        - Avoid the very young, the very old, and the very sick

Abuse and Tolerance

- Physical dependence
- Cross tolerance with other CNS depressants
- Oral over-dosage is rare and requires extremely large doses

Benzodiazepine Withdrawal Symptoms

- Confusion
- Clouded sensorium
- Heightened sensory perception
- Dysosmia (abnormal taste and smell)
- Paresthesias
- Muscle cramps
- Muscle twitches
- Blurred vision
- Diarrhea
- Decreased appetite
- Weight loss

Severe withdrawal:

- Hallucinations
- Other psychotic symptoms
- Delirium
- Seizures

Risk Factors for Dependence:

- Longer Term Treatment
- Higher Dosage

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- Higher Potency
- Shorter Half-Life
- More Abrupt Discontinuation

Medical Condition Contraindications

- Acute narrow angle glaucoma
- Known hypersentivity
- Psychosis
- Pregnancy
- Excreted in human milk

Drug Cautions

- Antidepressants cause additive CNS depression
- Cimetidine (Tagamet) – inhibits CYP 3A4
- Antacids (pH affects absorption and inhibitor of p450 enzymes)

Drug Contraindications (Oral Sedation)

- Macrolide Antibiotics (Erythromycin and Clarithromycin) – inhibitor of CYP 3A4
  - But not Azithromycin
- Protease Inhibitors – inhibitors of CYP
- Cyclosporine (anti-rejection) – inhibits CYP
- Grapefruit Juice – suicide inhibitor of CYP in gut
- St. John's Wort -inducer of CYP
- Systemic Azole Antifungals (ketaconazole, itraconazole, fluconazole) – inhibitors of CYP

Cautions and Contraindications (Drug Interactions with Oral Sedation)

Three Pathways Where Interactions Can Occur:

1. Additional CNS Depression (additive or potentiating synergism)
2. Induction of metabolism resulting in decreased serum concentration
3. Inhibition of metabolism resulting in increased serum concentration

Pharmacotherapeutic Recommendations involving Drug Interactions with sedative medications (utilizing the Lexi-Comp Drug Interaction Analysis severity ranking system):

1. Sedate "Over Top" of any [A], [B], OR [C] interaction
2. Sedate "Over Top" of any [D] OR [X] interaction involving additional CNS Depression (both oral & IV)
3. Sedate "Around" [D] or [X] induction/inhibition interactions with oral agents (utilizing Lorazepam)
4. Sedate "Over Top" of [D] or [X] induction/inhibition interactions with IV agents titrating appropriate doses and appropriate intervals (low and slow)

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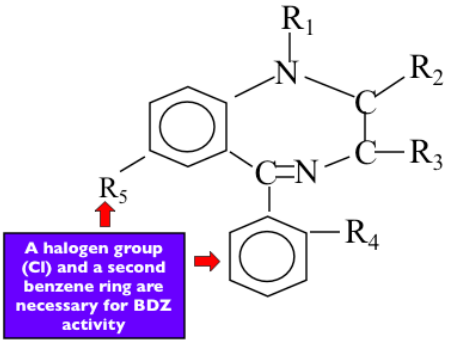
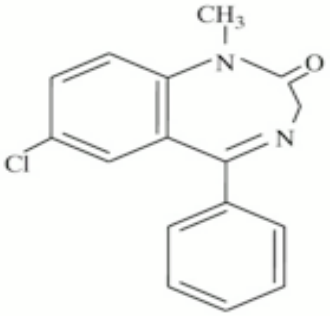
Pharmacology software products like Lexi-Comp Online provide comprehensive, up-to-date information on medications the patient is taking and their interactions with sedation medications.

Benzodiazepine Molecular Structure



The basic benzodiazepine structure includes a benzene ring connected to a two-nitrogen, three-carbon ring (diazepine ring). The seven-sided diazepine ring contains nitrogen atoms in the 1 and 4 positions. All "active" benzodiazepines have a second benzene ring and a halogen group (Cl) connected to the first benzene ring.

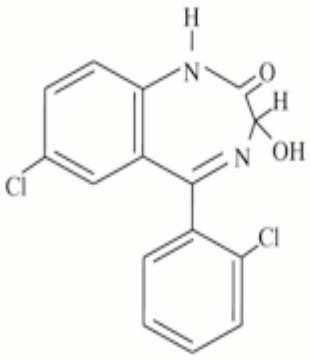
DOCS Education IVCS Protcol Benzodiazepines



Diazepam
Lipophilic
Relatively insoluble in water
pK_a 3.4 (basic)

V_d 0.8–2.6 l/kg
$T_{1/2\alpha}$ 20–240 minutes
$T_{1/2\beta}$ 24–48 hours
Cl 0.02–0.03 ml/kg/h

Active metabolite: nordiazepam



Lorazepam
Moderately lipid soluble
Insoluble in water (unionised at pH 7.4)
pK_a 1.3/11.5 (amphoteric)

V_d 1–2 l/kg
$T_{1/2\alpha}$ 2–3 hours
$T_{1/2\beta}$ 8–24 hours
Cl 0.01–0.1 ml/kg/h

Active metabolite: none

Non-Polar – Relatively insoluble in Water
Moderately Lipid Soluble – Slow Onset of Action
Active Metabolites – Long Half-Life and moderate duration of action due to active metabolites

Non-Polar – Moderately lipid soluble
Insoluble in water – Moderate Onset of Action
No Active Metabolites – Moderate Half-Life and moderate duration of action due to only Phase II metabolism

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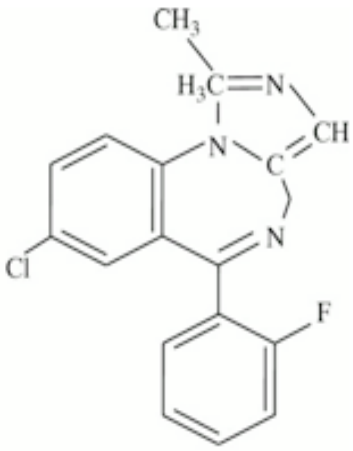
Polar – Water Soluble
Lipid Soluble – Fast Onset of Action
Few Active Metabolites – Short duration of action and half-life



Midazolam
Lipid soluble
Water soluble
pK_a 6.2 (basic)

V_d 0.6–1.7 l/kg
$T_{1/2\alpha}$ 15 minutes
$T_{1/2\beta}$ 1.5–3.5 hours
Cl 0.4–0.7 ml/kg/h

Active metabolite: αhydroxymidazolam

Midazolam

- Binds to GABA-A receptor
- Brand name - Versed
- Anterograde amnesia
- Water soluble
- Short half-life
- Reversible

Midazolam Dosage

Moderate sedation: I.V.: Initial: 1 mg slow I.V. test dose. If needed additional doses over at least 3 minutes; slowly titrate to effect by r1-2mg can be administered every 3 minutes; usual total dose: 2-5 mg; use decreased doses in elderly. If narcotics or other CNS depressants are administered concomitantly, the total midazolam dose should be reduced by 30-50%.

The amount of Midazolam needed to achieve moderate sedation decreases as the age of the patient increases. For example, a 2 and ½ year old may need

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12mg of midazolam to get moderately sedated, while an 80 year old may only need 0.25mg to achieve the same level of sedation.

Midazolam - Adverse Effects:

>10%:

- Respiratory: Decreased tidal volume and/or respiratory rate decrease, apnea (3% children)

1% to 10%:

- Cardiovascular: Hypotension (3% children)
- Central nervous system: Drowsiness (1%), oversedation, headache (1%), seizure-like activity (1% children)
- Gastrointestinal: Nausea (3%), vomiting (3%)
- Local: Pain and local reactions at injection site (4% I.M., 5% I.V.; severity less than diazepam)
- Ocular: Nystagmus (1% children)
- Respiratory: Cough (1%)
- Miscellaneous: Physical and psychological dependence with prolonged use, hiccups (4%, 1% children), paradoxical reaction (2% children)

<1%:

- Agitation, amnesia, bigeminy, bronchospasm, emergence delirium, euphoria, hallucinations, laryngospasm, rash

Remimazolam

- Benzodiazepine
- Marketed as Byfavo®
- Approved in the U.S. in July 2020
- Maintenance of procedural IV sedation in adults lasting 30 minutes or less
- Developed to have equivalent sedative effects to midazolam, but designed as an ester-based drug with a shorter duration of action

Remimazolam – Pharmacology

- Onset of action 3 minutes
- Median time to fully alert following last dose 11-14 minutes
- Terminal half-life 37-53 minutes
- Not metabolized by CYP450 enzymes
    - Low potential for drug interactions
- Reversible with Flumazenil

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Remimazolam – Side Effects

- Common
  - Hypotension
  - Hypertension
  - Hypoxia
  - Tachycarda
- Serious
  - Severe and life-threatening allergic reactions

Remimazolam – Dosing

- Initial dose
  - 5mg (2cc) push over 10-15 seconds
- Supplemental doses prn
  - 2.5mg (1cc) push over 10-15 seconds
  - 2-minute intervals between doses

Lorazepam

- Binds to GABA-A receptor
- Brand name - Ativan
- Anterograde amnesia
- Insoluble in water
  - Solvent – Propylene Glycol, must be refrigerated
- Intermediate half life
- Reversible
- Onset of Action - Hypnosis: I.M.: 20-30 minutes; Sedation: I.V.: 5 minutes; Anticonvulsant: I.V.: 5 minutes, oral: 30-60 minutes

Lorazepam Dosage

Adults: I.V.: 0.04 mg/kg q5m titrated to effect (usual maximum: 2 mg/dose)
Preprocedural anxiety: Adults - Oral: 1-2 mg 1 hour before procedure
Caution in elderly due to intermediate half-life

Lorazepam Significant Adverse Effects

>10%:
Central nervous system: Sedation
Respiratory: Respiratory depression
1% to 10%:
Cardiovascular: Hypotension
Central nervous system: Akesthisia, amnesia, ataxia, confusion, depression, disorientation, dizziness, headache
Dermatologic: Dermatitis, rash
Gastrointestinal: Changes in appetite, nausea, weight gain/loss
Neuromuscular & skeletal: Weakness

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Ocular: Visual disturbances
Respiratory: Apnea, hyperventilation, nasal congestion

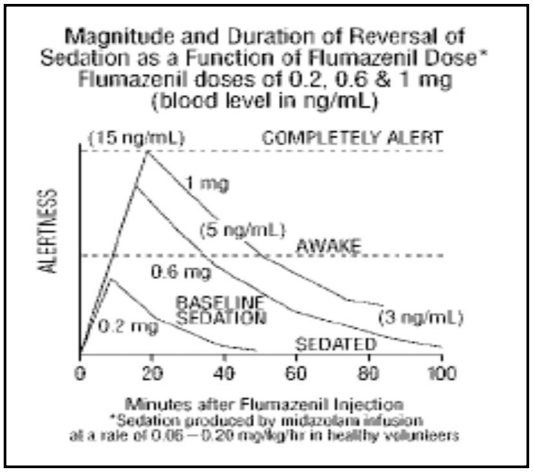
<1% or frequency not defined (Limited to important or life-threatening): Asthenia, blood dyscrasias, disinhibition, euphoria, fatigue, increased salivation, menstrual irregularities, physical and psychological dependence (with prolonged use), reflex slowing, polyethylene glycol or propylene glycol poisoning (prolonged I.V. infusion), suicidal ideation, seizure, vertigo

Flumazenil- Romazicon

- Indirect (Non-Competitive) Antagonist (negative allosteric modulator) of GABA
- Direct (Competitive) Antagonist of other benzodiazepines
- Effectively reduces somnolence and respiratory depression mediated at BZ receptors
- Available as injectable: 0.1 mg/ml in 5ml and 10 ml vials
- Intended for IV administration but may also be used submucosally
- IV - 0.2 mg increments up to 1mg (20 minutes)
- Contraindicated in benzodiazepine dependent patients and those with a seizure disorder

Onset of Action1-3 minutes; 80% response within 3 minutes; Peak effect: 6-10 minutes

Duration of Action: ~1 hour; duration related to dose given and benzodiazepine plasma concentrations; reversal effects of flumazenil may wear off before effects of benzodiazepine being reversed



Protein Binding - 40% to 50%

Metabolism is Hepatic; dependent upon hepatic blood flow

Half-life Elimination - Adults: Alpha (distribution): 7-15 minutes; Beta (metabolism): 41-79 minutes; Moderate hepatic dysfunction: 1.3 hours; Severe hepatic impairment: 2.4 hours

Benzodiazepine Reversal Protocol

1. Reasons for reversal confirmed:
    a. Respiratory depression
        i. Confirm accurate monitoring
    b. Level of sedation deeper than intended
2. Verify IV access patent
3. Administer flumazenil 0.2mg (2 cc) IV over 15 seconds

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4. Evaluate vital signs/level of consciousness at 1 minute post-administration
5. Repeat flumazenil doses:
    a. If desired results are not obtained, 0.2 mg may be repeated IV at 1-minute intervals
6. Maximum total cumulative dose 1 mg (usual dose: 0.6-1.0 mg)
    a. In the event of re-sedation: Repeat doses may be given at 20-minute intervals with maximum of 1 mg/dose and 3 mg/hour
7. Evaluate vital signs and level of consciousness after each dose
8. 911 if unresponsive and/or signs of respiratory depression remain

Flumazenil – Side Effects

- Amnesia: Does not consistently reverse amnesia; patient may not recall verbal instructions after procedure.
- Resedation: Occurs more frequently in patients where a large single dose or cumulative dose of a benzodiazepine has been administered along with a neuromuscular-blocking agent and multiple anesthetic agents.
- Respiratory depression: Should not rely upon to reverse respiratory depression/hypoventilation. Flumazenil is not a substitute for evaluation of oxygenation. Establishing an airway and assisting ventilation, as necessary, is always the initial step in overdose management.
- Seizures: [U.S. Boxed Warning]: Benzodiazepine reversal may result in seizures in some patients. Patients who may develop seizures include patients on benzodiazepines for long-term sedation, tricyclic antidepressant overdose patients, concurrent major sedative-hypnotic drug withdrawal, and recent therapy with repeated doses of parenteral benzodiazepines, myoclonic jerking or seizure activity prior to flumazenil administration. Use with caution in patients relying on a benzodiazepine for seizure control.

Opioids

- Classified by either chemical structure or the opioid receptor type they predominantly attach to
- Allergy to one opioid compound suggests potential allergy to all others
    - May skin or DNA test and use compound from a different group

Pharmacokinetics

- Opioids well absorbed orally
- Significant first-pass metabolism
- Transformed by glucuronidation in liver
- Metabolites are generally active
- Excreted by kidneys

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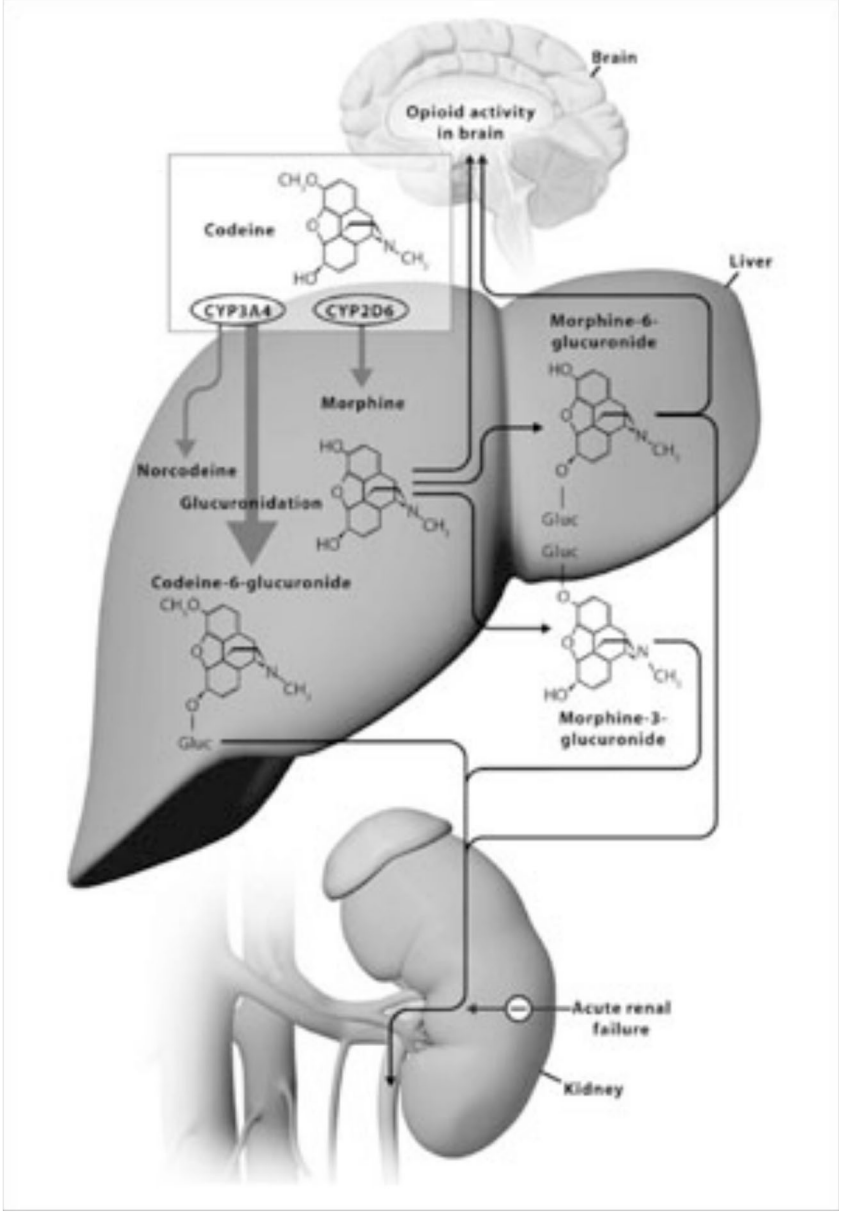

Pharmacodynamics

There are three main types of opioid receptors, mu (μ), delta (δ), and kappa (κ). The primary receptor responsible for most actions (desirable and undesirable) is the mu receptor located in the brain, spinal cord, and GI tract.

Activation of opioid receptors inhibits nerve transmission by blocking Ca^{++} influx and K^{+} outflux, interrupting nerve transmission. In the CNS this results in decreased perception of pain. Activation of mu-opioid receptors also stimulates the release of dopamine, leading to a feeling of euphoria.

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mu - has no ceiling in effects

- Increase dose= increase effect

κ- shows ceiling in effects

- Effects increase until ceiling dose then no increase in effect

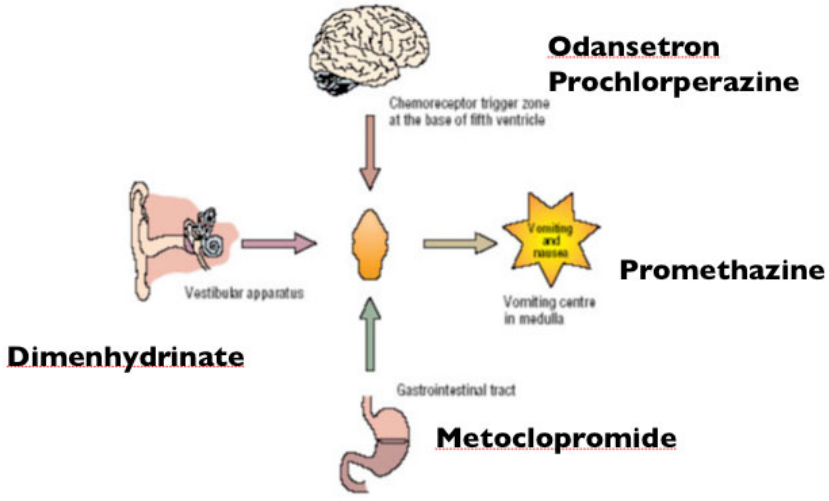
*Effects Of Opioid Receptor Stimulation**

Mu-1	**Kappa**
Supraspinal analgesia	Supraspinal analgesia
Euphoria	Spinal analgesia
Nausea/vomiting	Sedation
Miosis	Constipation
Tolerance	Miosis
Pruritus	Psychotomimetic
Mu-2	**Delta**
Spinal analgesia	Supraspinal analgesia
Sedation	Spinal analgesia
Constipation	Nausea/vomiting
Respiratory depression	Respiratory depression
	Tolerance
	Pruritus

Opioid Side Effects

Nausea and Vomiting

- ***Primarily not due to irritation of gastric mucosa!!!***
- Occurs with all opioids (more with mu and delta than kappa)
- Nausea due to direct stimulation of the vomiting center (CTZ) chemoreceptor trigger zone
- Also input from vestibular apparatus so worse if patient is ambulatory



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Pharmacologic Agents for PONV

- Promethazine (Phenergan) – Antihistamine/Anticholinergic, not as useful for opioid induced N&V, 6.25-25 mg IV, 12.5-25 mg oral
- Metoclopramide (Reglan) – Cholinomimetic. Increases gastric motility. 10-20 mg IV near end of surgery
- **Odansetron** (Zofran) – 5-HT3 antagonist. Greater anti-vomiting than anti-nausea effect. Most effective in prophylaxis when given at end of surgery 4 mg IV
- **Prochorperazine** (Compazine) – Phenothiazine. Dopamine antagonist at CTZ. 5-10 mg PO q4-6h prn

Histamine Release

- Release of histamine from basophils
- Not receptor mediated
- May be direct action
- Most often associated with morphine
- Leads to vasodilation
- Watch out for hypotension

Miosis

- Pinpoint pupils
- Effects nucleus of occulomotor nerve
- All narcotics except meperidine

Opioid Agonist Anesthetic Agents

- Fentanyl
- Alfentanil
- Sufentanil
- Remifentanil

Less used:

- Meperidine
- Morphine

Fentanyl

- Phenylpiperidine derivative
- Most widely used narcotic for ambulatory anesthesia
- Produces analgesia, drowsiness, sedation, and euphoria, but no <u>amnesia.</u>
- Dose: 25-50 mcg increments
- Onset: within 5 minutes
- Duration: 45-60 minutes
- Half Life: 2-4 hours (elimination/IV/adult)
- Side effects include; respiratory depression, bradycardia, nausea, and

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chest wall rigidity (Wooden Chest Syndrome)

Fentanyl often has limited availability. The practitioner who consistently relies on fentanyl for their sedation protocol should stock accordingly and have chosen alternatives when fentanyl is unavailable. Of the close relatives to fentanyl, **Alfentanil** shares the most common characteristics when it comes to potency (dosing), onset of action, duration of action (less), and half-life.

Alfentanil

- One of the shortest acting opioids available
- Dose: 20-50 mcg (5-15 mcg/kg)
- Onset: 1-2 minutes
- Duration: 20 minutes
- Side effects include: respiratory depression, bradycardia, rigidity involving the chest and larynx, and nausea and vomiting

Sufentanil

- Thiamyl analog of fentanyl is the most potent opioid currently in use
- Approximately 5-10 times more potent than fentanyl
- Dose: 5-20mcg (0.1- 0.5 mcg/kg) for sedation
- Onset: 2-3 minutes
- Duration: 45-50 minutes
- Side effects include: respiratory depression, bradycardia, and chest wall rigidity

Remifentanil

- Anilidopiperidine
- Similar potency to fentanyl
- Shorter half life than fentanyl
- Dose: 1 mcg/kg over 30-60 seconds
- Ultiva ™
- Onset: 1-3 minutes
- Duration: 45-60 minutes
- Side effects include hypotension, bradycardia, intraoperative awareness, respiratory depression, and chest wall rigidity

Narcotic Agonist/Antagonist

- Butorphanol (Stadol) & Nalbuphine (Nubain)
- Kappa receptor agonism, mu receptor antagonism
- Respiratory depression is minimal and is limited by ceiling effect
- Onset: 2-3 minutes
- Duration: 3-4 hours

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- Both provide analgesia similar to pure agonists
- Use is limited because of an increased incidence of nausea, vomiting, unpleasant dreams, and prolonged sedation

Naloxone

- Opioid antagonist
- Free of any intrinsic activity
- Will not produce respiratory depression
- Will not produce sedation
- Reverses: Respiratory Depression, Analgesia, Sedation
- Duration: IV- 30 minutes
- Follow IV Dose with IM if duration of narcotic longer than 30 minutes
- IM slower onset but longer duration
- Airway maintenance is primary treatment for respiratory depression
- Must be administered with great care in those who are physically dependent on narcotics
- 0.4mg/ml - 1ml ampule
- Alternative drug: none

Opioid Reversal Protocol

1. Vital signs and consciousness evaluated and recorded
2. Verify IV access patent
3. Draw naloxone 0.4mg into 1 ml syringe and administer IV
4. Evaluate vital signs and level of consciousness
5. If necessary, repeat dose 0.4mg every 2-3 minutes to 10mg
6. Evaluate vital signs and level of consciousness after each dose
7. If no response from this drug after 10mg consider another cause for symptoms
8. 911 if unresponsive

Other IV Anesthetic Agents

- Ketamine
- Etomidate
- Dexmeditomidine
- Propofol
- Droperidol

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Ketamine

Mechanism of Action

- N-methyl-D-aspartate receptor antagonist
- Dissociative Anesthesia
    - Blocks signals from the thalamus, which processes sensory input to the reticular activating system (RAS)
    - The RAS is responsible for awareness
- Other depressants inhibit the RAS
- Patient appears conscious, but unable to respond to sensory input

N-methyl-D-aspartate receptor

- Responsible for “excitation”
- Ion and voltage gated
- Ketamine binds to the glutamate site
- Antagonist
- Prevents influx of Ca^{++} ions

Pharmacokinetics

- Administer IM/IV- Peak action 10-15 min
- Lipid soluble, not significantly protein bound
- Easily crosses BBB
- Induces cerebral blood flow and cardiac output
- Rapid brain uptake, rapid redistribution (t1/2 10-15min)
- Awakening due to redistribution

Biotransformation

- Hepatic
- Several metabolites, some active
- Enzyme inducer
- Tolerance develops with repeated dosing
- Renally excreted

Effects on Organ Systems

Cardiovascular

- Unlike other agents increases arterial BP, HR, and CO
- Due to central stimulation of the sympathetic NS, and blocks reuptake of NE
- Increase pulmonary artery pressure and cardiac work
- AVOID in patients with IHD, CHF, arterial aneurysms

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Respiratory

- Ventilatory drive is minimally affected
- Potent bronchodilator
- Good agent for asthmatic patients
- May cause increased salivation
- Laryngospasm

Cerebral

- Increase cerebral blood flow, intracranial pressure and oxygen consumption
- Avoid in patients with brain lesions
- Illusions, disturbing dreams, delirium possible
- Less in children and those premedicated with BZ

Drug Interactions

- Potentiates non-depolarizing skeletal muscle blockers
- Use with theophylline may predispose patients to seizures
- Diazepam attenuates ketamine's cardiostimulatory effects and prolongs duration

Etomidate

- Depresses RAS
- Induction agent
- Binds to GABA-A receptor
- Amnesic
- Disinhibitory on motor portions of CNS
- Seizures possible
- Highly protein bound
- Rapid onset because highly lipid soluble
- Dosage: Adults 0.3 mg/kg IV
- Typical dose 20-40 mg
- Onset 30-60 seconds
- Peak effect: 1 minute
- Duration: 3-5 minutes; terminated by redistribution
- Distribution: V_d: 2-4.5 L/kg
- Protein binding: 76%
- Metabolism: Hepatic and plasma esterases
- Half-life elimination: Terminal - 2.6 hours

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Dexmedetomidine

- Marketed as Precedex
- Alpha$_2$-Adrenergic Agonist (8X more selective for the Alpha$_2$ receptor than clonidine)
- Uses:
  - Agitation associated with schizophrenia or bipolar disorder
  - Sedation of ventilated patients in the ICU
  - Veterinary Medicine
  - Procedural Sedation
- Advantages:
  - Associated with less neurocognitive dysfunction (delirium) than other anesthetic agents
  - Does not cause respiratory depression
  - Has analgesic effects
- Disadvantages:
  - Much less amnesia than benzodiazepines
  - No reversal agent:
  - Atipamezole (Antisedan), and alpha$_2$ adrenoreceptor antagonist not approved for use in humans
- Side Effects:
  - Hypotension
  - Bradycardia
  - Especially in patients with:
    - Low baseline BP
    - Elderly
    - Patients with CAD
- Dosing:
  - Loading dose of 0.5 mcg/kg over ten minutes
- Onset of action – 5-10 minutes
- Duration of action – 1-4 hours
- Half-Life – 2-3 hours

Propofol

- Facilitates GABA
- Binds the β subunit
- Oil in water emulsion
- May be painful during injection
- Good sterile technique important because formulations can promote bacterial growth

Pharmacokinetics

- IV only
- Induction and deep sedation
- Highly lipid soluble

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- Very rapid onset - “Arm-brain circulation time”
    - Time from site of injection to brain - Propofol is < 60 seconds
- Very short half life (2-8 minutes)
- Hepatic metabolism
- Inactive metabolites
- Renally cleared

Effects on Organ Systems

- Decrease in arterial blood pressure
- Drop in peripheral vascular resistance
- Decrease myocardial contractility
- Decrease preload
- Hypotension is reversed by stimulation, large doses, rapid infusion and old age
- Avoid in extremes of age and cardiovascular insufficiency
- Profound respiratory depression
- During infusion inhibits hypoxic drive and depresses normal response to hypercarbia
- Should be used by experienced personnel only!!!
- Decreases cerebral blood flow and intracranial pressure
- Has anti-pruritic properties
- Anticonvulsant properties
- Tolerance does not develop

Drug Interactions

- Potentiates neuromuscular blocking agents
- Will increase fentanyl and alfentanil concentrations.

Droperidol

Mechanism of Action

- Antagonizes the activation of dopamine receptors
- Affects CTZ
    - Antiemetic effect
- Also interferes with transmission mediated by serotonin, norepinephrine and GABA
- Tranquilizer effects

Structure

- Substituted butyrophenone
- Related to haloperidol (antipsychotic)

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Pharmacokinetics

- Usually given IV
- Rapid distribution
- Delayed sedative effects because:
    - Highly protein bound
    - High molecular weight
    - Hinders crossing of BBB
    - Prolonged duration due to tenacious receptor binding

Biotransformation

- Metabolized hepatically
- Cleared renally

Effects on Organ Systems

    - Mild alpha-adrenergic blocker
    - Decrease arterial BP because peripheral vasodilation
    - Will prolong QT interval and ST segment changes on EKG
    - Should not be given without a pre-op EKG
        - BLACK BOX in package insert

Droperidol - Properties

- No respiratory depression
- Decreases cerebral blood flow and intracranial pressure
- Potent antiemetic
- 0.05mg/kg, 2.5mg maximum
- Antidopamine effects may precipitate parkinsonian effects (avoid in those patients)
- No analgesia, amnesia
- Fallen into disfavor as anesthetic

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Agent	Use	Route[1]	Dose
Ketamine	Induction	IV	1–2 mg/kg
		IM	3–5 mg/kg
Etomidate	Induction	IV	0.2–0.5 mg/kg
Propofol	Induction	IV	1–2.5 mg/kg
	Maintenance infusion	IV	50–200 µg/kg/min
	Sedation infusion	IV	25–100 µg/kg/min
Droperidol	Premedication	IM	0.04–0.07 mg/kg
	Sedation	IV	0.02–0.07 mg/kg
	Antiemetic	IV	0.05 mg/kg[2]

[1]IV, intravenous; IM, intramuscular.
[2]Maximum adult dose without prolonging emergence is 1.25–2.5 mg.

Oral Sedative Agents

Oral sedatives in the IV sedation protocol:

1. Make the patient more relaxed for the venipuncture procedure
2. Make the venipuncture easier (due to vasodilation that accompanies CNS depression)
3. Reduce the onset of action of IV sedatives
4. Increase the duration of action of IV sedatives

Pharmacology of Four Oral Sedative Drugs

Triazolam (Halcion) – Loading dose

- Benzodiazepine
- Highly effective sedative, hypnotic
- Positive allosteric modulator of the GABA Receptor
- No active metabolites
- Plasma half-life of 2-3 hours
- Mean peak concentration of 1.3 hours
- Anticonvulsant
- Mild antiemetic
- Reversible with flumazenil
- LD50 is 5g/kg in rats
    - Equivalent to 1,400,000 0.25mg Tablets in Humans
- Supplied in 0.125mg and 0.25mg tablets

Dosage:

- Healthy Adult: 0.25mg-0.5mg
- Elderly or Debilitated Adult: 0.125mg
- Always use the lowest effective dose
- Child: Safety and efficacy not tested for patients under the age of 18

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Contraindications:

1. Pregnancy
2. Known hypersensitivity
3. Patients <18 years old
4. Acute narrow angle glaucoma
5. Severe drug interactions that affect metabolism

Warnings:

1. Other CNS depressants
2. Moderate drug interactions that affect metabolism

Diazepam (Valium) – Night Before Dose

- Benzodiazepine
- Positive allosteric modulator of the GABA Receptor
- Mild hypnotic, sedative
- Onset of action: 1 hour
- Active metabolites
- Half-Life: 20-100 hours
- Duration of action: 6-8 hours
- Mild amnesia
- Supplied in 2, 5, and 10mg tabs
- Reversible with flumazenil

Dosage:

- 2.5-10mg
- Standard night before sedation appointment dose: 5mg

Contraindications:

1. Pregnancy
2. Known Hypersensitivity
3. Pediatric pts. < 6 months
4. Severe drug interactions that affect metabolism

Warnings:

1. Elderly/debilitated patients
2. Other CNS depressants
3. Moderate drug interactions that affect metabolism

Lorazepam (Ativan) – The alternative loading dose

- Benzodiazepine
- Positive allosteric modulator of GABA receptor
- Moderate sedative, hypnotic
- Onset of action: 1 hr.

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- No active metabolites
- Half-Life: 12-14 hrs.
- Duration of action: 6-8 hrs.
- Moderate amnesia
- Supplied in 0.5, 1, or 2mg tablets

Dosage:

- 1-3mg (Elderly/Debilitated cut dose in half)

Contraindications:

- Known hypersensitivity
- Pregnancy
- Children less than 12
- Acute narrow angle glaucoma

Warnings:

- Impaired renal and hepatic function
- Elderly/Debilitated patients

Lorazepam is a useful alternative sedative/hypnotic to other benzodiazepines that have significant drug interactions with medications a patient is taking due to the impact those medications have on the CYP-450 enzyme system. The reason for this has to do with the way Lorazepam is metabolized.

Lorazepam does not undergo Phase I metabolism by CYP-450 enzymes. Instead, **Lorazepam is metabolized by a process called Glucoronidation**. Glucoronides, as well as other substances are highly polar, and can bind to non-polar foreign substances such as drugs, rendering them inactive and more easily excreted in the urine.

One straightforward way the practitioner can determine if the drug interactions between benzodiazepines and other medications is significant due to metabolism is to use a pharmacology software product like Lexi-Comp. If there is a [D] or [X] interaction with an oral benzodiazepine due to induction or inhibition of metabolism, then oral Lorazepam should be considered as an alternative.

Hydroxyzine (Atarax or Vistaril) – The Piggyback Sedative

- Class – diphenylmethane, unrelated to benzodiazepines, phenothiazines, or opiates
- Moderate sedative, hypnotic
- Antihistamine
- Antiemetic
- Onset of action: 1 hr.

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- No active metabolites (cetirizine does have anthistamine activity, but limited hypnotic and sedative action)
- Half-Life: 3-7 hrs.
- Duration of action: 3-6 hrs.
- Needs to be combined ("Piggybacked") with another sedative/hypnotic
    - No amnesia
    - No reversal agent – only be given in a single dose ≤ MRD
- Supplied in 10, 25, 50, 100mg tabs, capsules and oral suspension

Dosage:

- 25-100mg
- Best administered by mixing crushed tablet with 2-3 oz. of water to form a "slurry" and swallowed

Applications:

1. Open the nasal airway in patients with histamine-related obstruction
2. Prevent nausea and vomiting in patients prone to N&V when taking medications
3. Reduce the gag response in "gaggers"
4. Assist with the sedation of the nicotine user

Contraindications:

1. Early pregnancy
2. Known hypersensitivity
3. Children under 1 year of age
4. Acute narrow-angle glaucoma

Warnings:

1. Other CNS depressants

Nitrous Oxide – The Synergistic Drug

- Nitrous Oxide (N_2O), is an inhalation anesthetic with both analgesic and anxiolytic effects
- Safest of all inhalation anesthetics
    - MAC of 105%
- Passes rapidly though body compartments by passive gradient diffusion
- Not metabolized by the body
- Mechanism of action poorly understood, but believed to be a partial opioid agonist

Application to IV Sedation Dentistry:

- Synergistic (potentiating) actions when combined with other sedative and/or analgesic agents
- Provides short-term periods of additional sedation and analgesia

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Dosage:

- Administered in concentrations of 20% (80% O_2) to 70% (30% O_2) w/o sedation
- Administered in concentrations of 30% for no longer than 15 minutes w/IV sedation

Technique:

- 3 minutes of 100% O_2
- 3 minutes 30% N_2O
- Begin treatment (venipuncture, local anesthesia, invasive treatment)
- Discontinue with 100% O_2 after 15 minutes

Adverse Events:

- Nausea and vomiting are possible when nitrous oxide:
  - Is given at high concentrations[1]
    - [1] Mraovic, Simurina, Sonicki, Skitarelic, Gan. "The dose-response of nitrous oxide in postoperative nausea in patients undergoing gynecologic laparoscopic surgery: a preliminary study". Anesht Analg. 2008 Sep;107(3):818-23
  - For extended periods of time[2]
    - [2]Peyton, Wu. "Nitrous Oxide-related Postoperative Nausea and Vomiting Depends on Duration of Exposure". Anesthesiology 5 2014, Vol.120, 1137-1145
- Excessive CNS depression
  - If not titrated appropriately
    - When combined with other CNS depressants

Nitrous Oxide Etiquette:

- Scavenging system that redirects N_2O outside the facility
- Good ventilation
- Well-fitting mask
- Advanced armamentarium
  - Safe-Sedate
- Monitoring systems
- Reduced talking

Warnings:

- Upper respiratory infections

Contraindications:

- Pregnancy
- Malnourishment (Vit. B12 deficiency)
- Otitis Media and other obstructed cavities that are inflamed/infected

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MONITORING DURING IV SEDATION

Goals

The goal of monitoring during IV sedation is the collection of data that reflects:

1. CNS Depression
2. Physiologic Homeostasis
3. Early warning signs of adverse changes or trends before irreversible damage occurs
4. Responses to therapeutic intervention

Monitoring CNS Depression

The word “Sedation” implies that a state of CNS depression exists. In the case of IV sedation medications, as is the case with other drugs, the undesirable side effects of sedation medications is an extension of their desired effects – CNS depression. As CNS depression deepens, its impact threatens vital functions controlled by the CNS. Chief among these because of its likelihood to result in the most adverse outcome of sedation is respiratory depression.

In order to avoid the risk associated with deeper levels of CNS depression, it should be the goal of every anesthetist/practitioner to achieve the lightest level of sedation necessary for patient comfort. For nearly every outpatient procedure performed in dentistry, conscious sedation (anxiolysis, minimal or moderate sedation) is sufficiently effective for patient comfort purposes while reducing the risk associated with deeper levels of sedation.

One of the characteristics that distinguishes moderate sedation from deep sedation and general anesthesia is loss of consciousness. Another important differentiating factor is the patient’s ability to maintain their own airway (breathe on their own). This is a critical juncture in the spectrum of sedation, as ***respiratory depression significant enough to require intervention, rather than support, will most likely be preceded by loss of consciousness.*** Stated another way, as the patient’s CNS becomes increasingly depressed, monitoring *physiological parameters* become even more important, and the ability of the sedation team to intervene and assist the patient in ventilation increase in importance.

CNS depression can be monitored two different ways:

1. Observation of response to stimulation
2. EEG

Observation of response to stimulation

Consciousness can be assessed by observing a patient’s arousal and responsiveness, and can be seen as a continuum of states ranging from full alertness and comprehension, through varying degrees of mental confusion

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ranging from mild disorientation to delirium, with the eventual loss of any meaningful communication, and ending with loss of movement in response to painful stimuli. Traditionally speaking, conscious sedation (minimal or moderate) results in a patient who can respond verbally when prompted. Normal response times and articulate communication is associated with minimal sedation, while delayed, slurred responses that may require repetitive prompting are associated with moderate sedation.

Physical observations such as eyelid ptosis, posture, and response to physical stimulation are less reliable indicators when compared to verbal communication. ***It is recommended that the sedation team check for signs of verbal responsiveness every five minutes during sedation, or more often if needed whenever mild or moderate sedation is the goal.***

EEG

One of the most consistent efforts of anesthesia research has been directed at the quantitative measurement of a patient's state of anesthesia. The ability to quantitatively determine depth of anesthesia would allow for titrating anesthetic agents to a specific level, thereby allowing the anesthetist to adjust the amount of anesthetic agent to the needs of the patient, possibly resulting in a more rapid emergence from anesthesia. It is also thought that such technology would reduce the incidence of intraoperative awareness in surgeries.

Electroencephalography (EEG) is the recording of electrical activity along the scalp produced by the firing of neurons within the brain. Two commercially available monitors of anesthetic depth utilize EEG data, the Bispectral Index (BIS) monitor (Aspect Medical Systems) and the Patient State Analyzer 4000 (PSA 400 from Baxter).

The Bispectral Index is a statistically based, empirically derived complex parameter. The BIS monitor provides a single number, which ranges from 0 (equivalent to EEG silence) to 100 (equivalent to fully awake and alert). A BIS value between 40 and 60 indicates an appropriate level for general anesthesia, as recommended by the manufacturer.

BIS Number	Anesthesia Depth
95-100	Awake, Memory Intact
65-85	Sedation
45-65	General Anesthesia
<45	Burst Suppression
0	Cortical Silence

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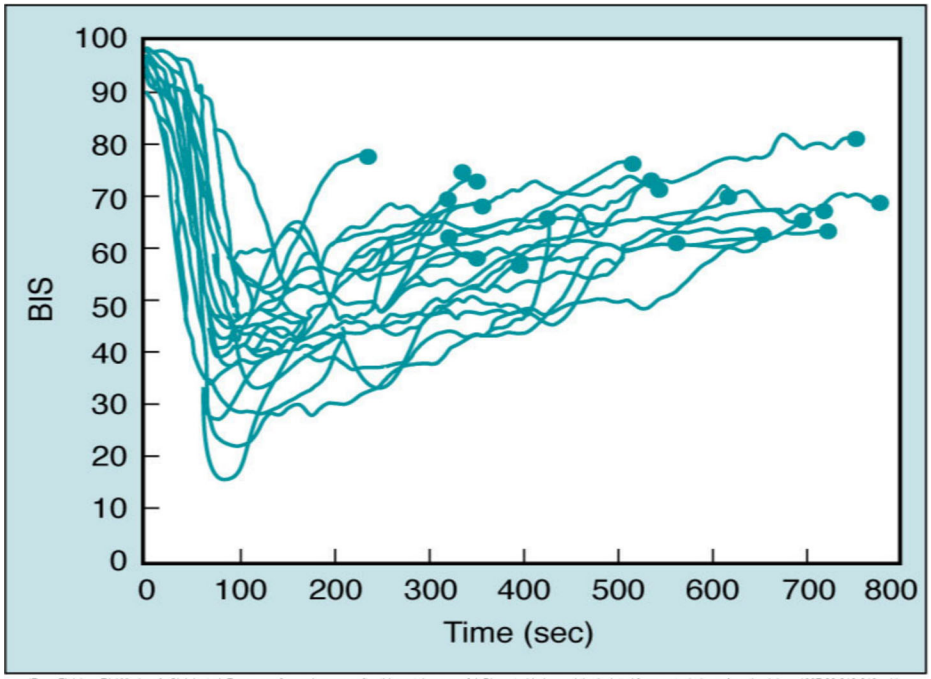

(From Flaishon RI, Windsor A, Sigl J, et al. Recovery of consciousness after thiopental or propofol: Bispectral index and the isolated forearm technique. Anesthesiology 1997;86:613-619, with permission.)

In comparing the two commercially available monitors, the BIS evaluates brain waves in one region of the brain, and the PSA 4000 evaluates brain wave activity over four parts of the brain. To date, what scientific research has been published about these monitors has failed to substantiate their usefulness (reliability and cost effectiveness).

Monitoring Physiologic Homeostasis

Krippaehne et al., compared 43 cases of morbidity and mortality (M & M) from pharmacosedation and general anesthesia in the dental office. The cases all had in common a young, healthy patient in whom multiple pharmacologic agents were used with limited monitoring and resuscitative efforts. Heart rate was not monitored in 68%, respiration in 77% blood pressure in 77%, tissue oxygen saturation in 92%, and heart rhythm in 96%. The authors concluded that ***lack of adequate monitoring is a key factor in the majority of morbid and mortal events.***

Jastak has stated, “subtle trends in vital signs are not detected because appropriate monitoring is not used, the morbid event eventually recognized by the practitioner is often the last in a series of physiologic distress signals that results in the clinician’s response being too little and too late.”

Physiologic Homeostasis describes the systems within the human organism that regulate its internal environment with the purpose of maintaining a stable, constant condition. With regard to monitoring IV sedation, the systems that deserve the most attention include: Heart Rate & Rhythm, Blood Pressure, Electrocardiography, Respiration, Oxygenation, Ventilation, & Body Temperature.

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Pre-Operative Baseline Values

Before IV sedation can be planned on any patient, vital signs should be taken and recorded as part of the routine pre-treatment patient evaluation. These measurements serve as an indication of the patient's overall physical status, with emphasis on the cardio-respiratory system. In addition to assisting the evaluator as to the patient's suitability for treatment, they serve as a "standard" for the patient. Although there are "normal" ranges within the general population for vital sign values, each individual has their own standard range within and/or around that range. This individual range will be taken into consideration as perioperative measurements are taken.

Vital signs recorded at this pretreatment visit include blood pressure, heart rate and EKG, oxygenation, height, and weight. Additional vital signs to be monitored as indicated included temperature, respiratory rate, and ventilation.

Baseline vital signs should be recorded in the patient's record at a nonthreatening time when they are likely to be more nearly normal for that patient.

Heart Rate & Rhythm

The detection of a strong, regular pulse within a normal range of values (rate) indicates that adequate perfusion of blood is permitting the exchange of oxygen and carbon dioxide between the blood and tissues. Given the availability, reliability, and affordability of modern pulse oximetry equipment, continual measurement of heart rate and rhythm during IV sedation is a straightforward and simple procedure.

The normal range of heart rate in the adult patient is between 60 and 100 beats per minute (bpm). Fluctuations above and below this range may also be acceptable given intraoperative conditions, physical status of the patient, and medications that have been administered.

When the heart rate is consistently less than 60 bpm, it is called Bradycardia. In the healthy patient in good physical condition, a slower heart rate is an expression of an efficient cardiopulmonary system that the sedation team will be made aware of during the preoperative evaluation. The sedation team should normally expect a decrease in heart rate during sedation from a generalized reduction in awareness and relaxation resulting from CNS depression.

Bradycardia can, however, be an ominous sign in patients with severely compromised cardiac function, and the sedation team must be prepared to support circulation should they encounter these ASAIV patients. Bradycardia can also be an indication of excessive CNS depression, as well as cardiotoxicity resulting from local anesthesia overdose.

Tachycardia, a heart rate consistently greater than 100 bpm, deserves more careful scrutiny during IV sedation than bradycardia. The "fast-beater's" heart is

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under greater stress not just from the extra work-load resulting from an increased rate and resulting oxygen demand, but has less time in-between beats to receive its own blood supply via coronary perfusion. If this were not enough, tachycardia results in less time for filling of the atria (preload).

There are common explanations for unexpected tachycardia during an IV sedation procedure. Inadequate anxiety-relief for instance, is an indication that more pronounced sedation is required.

In addition, discomfort will result in an accelerated heart rate, and is a signal to the anesthetist that more local anesthesia, or in some cases systemic analgesia is needed. The urge to urinate or evacuate the bowels is another forms of discomfort that can manifest as an increased pulse.

A more transient tachycardia can result from the entry of vasoconstrictor from the local anesthetic entering the general circulation. In the healthy or reasonably healthy patient, this fleeting increase in heart rate is both well-tolerated and insignificant.

A significant increase or decrease in heart rate may also be the result of improperly working monitoring equipment. In the absence of signs and symptoms of abnormal heart rate, and when no other plausible reason can explain the abnormality, the monitoring equipment should be suspected.

Finally, sustained and significant tachycardia can be a sign of a medical emergency. The sedation team should have been on alert for this occurrence based on the patient's existing physical condition and/or medications, or the tachycardia is associated with a specific incident that occurred during treatment. In any event, the importance of patient selection, advanced preparation, and decision action in the event of a medical emergency cannot be overemphasized.

Blood Pressure

Blood pressure or tension within the circulatory system is required for constant, consistent circulation of blood throughout the tissues that require the exchange of gases, nutrients, and waste products. This pressure is generated by contraction of the heart and maintained by the integrity of the circulatory system itself. The height of pressure resulting from contraction of the left ventricle is termed systolic pressure, whereas the pressure that remains within the closed system upon relaxation of the left ventricle is called diastolic pressure. During IV sedation a device must be used to continually monitor systolic and diastolic blood pressure.

The latest classifications of blood pressure and recommendations for management set forth by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure are as follows:

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JNC 7

BP Classification	Systolic BP, mmHg		Diastolic BP, mmHg	Lifestyle Modification	Drug Therapy Indicated
Normal	<120	And	<80	Encourage	None
Pre-hypertension	120-139	Or	80-89	Yes	Yes, if risk factors present
Stage 1 Hypertension	140-159	Or	90-99	Yes	Yes
Stage 2 Hypertension	≥160	Or	≥100	Yes	Yes

All other criteria considered equal, patients with normal or pre-hypertension blood baseline blood pressures, are considered candidates for outpatient IV sedation anesthesia, whereas patients with baseline Stage 1 or 2 hypertension should be evaluated medically for treatment of their hypertension prior to elective dental care. The reliability of baseline blood pressure measurements should be confirmed with multiple readings taken in as anxiety-free environment as possible.

If during the course of treatment, should any elevated blood pressure be associated with symptoms of headache, chest pain and/or tightness, or numbness in the limbs, then the procedure should be stopped immediately and emergency medical intervention begun.

If during the course of treatment consistent readings equivalent to Stage 2 hypertension recorded in the absence of symptoms, treatment should be discontinued at the next most convenient point in the procedure, and the patient dismissed to a responsible companion with instructions for medical evaluation at the earliest possible time.

As is the case with heart rate, possible reasons for an unexpected rise or fall in intraoperative blood pressure includes administration of drugs such as anesthetic agents with vasoconstrictor, interactions between the patient's medications and the sedation procedure, an uncomfortable patient, improperly functioning monitoring equipment, and finally, pathophysiology.

The **mean arterial pressure (MAP)** is a term used to describe the average blood pressure in an individual and can be calculated by:

$$\mathbf{MAP = SBP + (2 \times DBP)/3}$$

MAP is considered to be the perfusion pressure seen by organs in the body. It is believed that a MAP that is greater than 60 mmHg is enough to sustain the organs of the average person. MAP is normally between 70-110 mmHg. If the

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MAP falls significantly below this number for an appreciable time, the end organ will not get enough blood flow, and will become ischemic.

It is important that the cuff be the right size for the limb being used for the blood pressure measurement. The width of the cuff should be 20-30% of the circumference of the limb being used. If the cuff is too small, the BP will be artificially high. Conversely, if the cuff is too large, the BP will be artificially low.

The position of the cuff relative to the heart also matters. If the extremity used is above the level of the heart, the BP will be artificially low.

Electrocardiography

The ECG monitors both the heart rate and rhythm and provides warning of the development of changes in the electrical activity of the myocardium. Although 12 leads may be used, standard 3 lead waveforms - lead 1 (right arm to left arm), lead II (right arm to left leg), and lead III (left arm to left leg) - are most commonly used during anesthesia because they permit excellent detection of dysrhythmias.

Although not recommended for use in all procedures, the ECG does increase one's ability to detect possibly significant changes in the functioning of the myocardium at a time when corrective treatment may usually restore a normal rhythm.

Two common causes of dysrhythmias during sedation are (1) hypoxia, leading to myocardial ischemia, and (2) endogenous catecholamine release, secondary to inadequate pain control or too light a level of CNS depression.

In interpreting the waveform from ECG monitoring, the practitioner must understand the normal waveform in order to appreciate any deviations from normal that may be meaningful. The basic principle of ECG interpretation is to look for:

1. Rate (BPM and Consistency)
2. Normal & Consistent P-QRS-T waveform patterns
3. Presence and appearance of P wave
4. PR interval
5. Width and shape of QRS complex
6. Return to isoelectric line after QRS complex

Two of the most ominous signs that can be observed on the ECG indicate the presence of myocardial ischemia (lack of oxygen) or myocardial infarction (actual necrosis of cardiac tissue). Either is usually only seen with blockages in the coronary arteries (arteries that supply the heart muscle with blood). These conditions can be identified by looking at the part of the ECG between the QRS complex and the T wave. If this normally horizontal line is more than 1 mm below that baseline (isoelectric), myocardial ischemia is present. If the segment is 1 mm or more above the baseline, myocardial infarction is present.

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Respiration

Respiration is the exchange of air by the body with the external environment. Simply put, respiration is breathing, and may be monitored by any of the following methods:

1) Determining the respiratory rate - Most frequent disturbances are an overall rapid (Tachypnea) or unusually slow rate (Bradypnea). For adults, the respiratory rate should be between 12 and 16 breaths/minute.

2) Observing rise and fall of the chest wall – Unless accompanied by evidence of air exchange, movement of the chest wall does not guarantee air exchange (e.g. obstructive sleep apnea, FBO) just that respiratory efforts are being made.

3) Observing inflation and deflation of the reservoir bag if inhalation sedation is used. This is a valid method of determining air exchange *if* an airtight seal of the mask is maintained.

4) Holding a mirror or an ungloved hand in front of the patient's mouth or nose so that air is felt (or seen fogging the mirror) is a good method of determining exchange of air is occurring.

5) Auscultation of breath sounds. During endotracheal anesthesia, breath sounds and chest excursion must be verified after intubation and monitored continually.

Two devices used to assess respiration sounds include:

1. Precordial stethoscope
2. Pretracheal stethoscope

Two elements are considered when listening to breathing:

1) Rate of breathing
2) Presence of abnormal breathing sounds

Recognition of abnormal sound is very important. A normal unobstructed flow is relatively quiet and a smooth "whooshing" sound is heard

Abnormal Breathing Sounds

Sound Heard	Probable Cause	Management
Snoring	**Hypopharyngeal Obstruction by Tongue**	**Repeat Head tilt**
Gurgling	**Foreign Matter**	**Suction Airway**
Wheezing	**Bronchospasm**	**Administer Bronchodilator**
Crowing	**Laryngospasm**	**Suction Airway, PPO**

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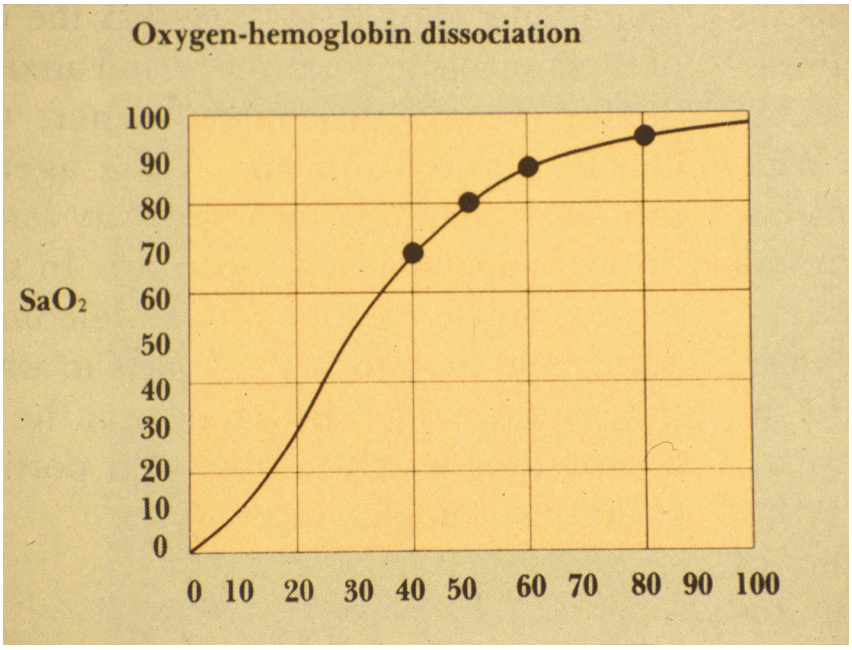
Oxygenation

Oxygenation refers to the degree of O_2 being transported in the blood and can be measured visually by the color of the skin and/or mucosa, and much more accurately (and timely) by a pulse oximeter.

Maintaining a high ratio of oxygenated hemoglobin to total hemoglobin (SaO_2) insures that the partial pressure of O_2 dissolved in the blood will be above 60 mmHg, a critical requirement for the adequate oxygenation of the patient. The relationship between the amount of oxygen dissolved in the blood and the amount attached to the hemoglobin is described by the Oxyhemoglobin Dissociation Curve.



Closer examination of the Oxyhemoglobin Dissociation Curve reveals that at high partial pressure of oxygen (60 mmHg), the curve is relatively flat and the percentage of blood oxygenation is high (>90%). Once the pO_2 dips below 60 mmHg, the corresponding amount of oxygen attached to hemoglobin drops below 90%, and as the pO_2 drops further, resulting in a precipitous decline in oxygen saturation of hemoglobin. A partial pressure of 60 mmHg is generally considered the lowest allowable oxygen level in the body before cells in vital organs begin to be starved for oxygen.

Ventilation

Ventilation should not be confused with respiration or oxygenation. Whereas respiration is the act of breathing and oxygenation reflects the saturation of blood with oxygen, ventilation is a reflection of cellular metabolism as a result of the exchange of O_2 and CO_2.

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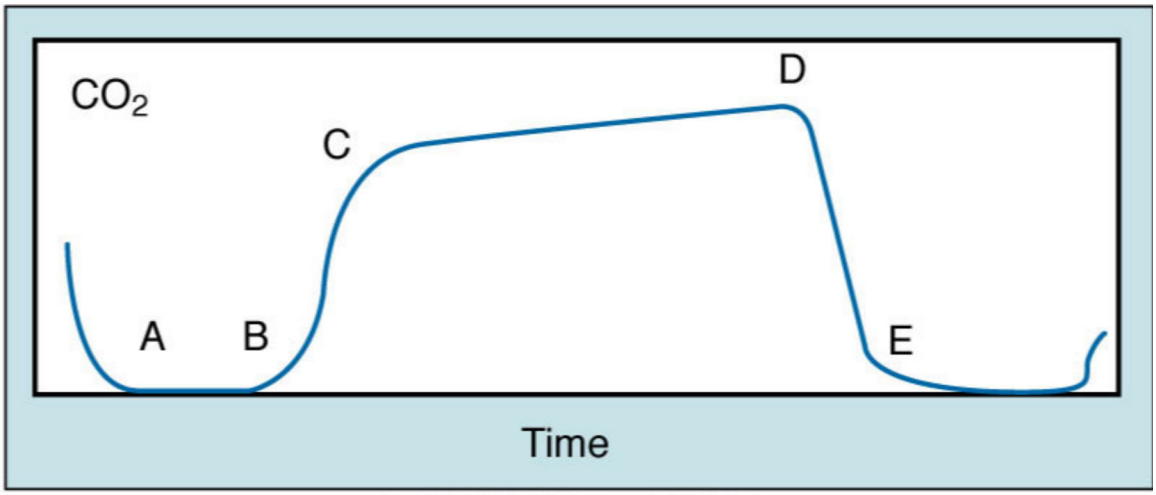
Ventilation is measured by amount of CO_2 the patient expires (End-Tidal CO_2). If the ET CO_2 = 0 mmHg, then the patient is not being ventilated, which could be an indication of upper airway obstruction or apnea.

Capnography

Refers to the comprehensive measurement & display of end-tidal CO_2, and can be displayed as the capnogram (real time CO_2 waveform)

Capnometry

- Refers to the measurement and display of CO_2 in numeric form only
- Normal $PaCO_2$ = 40 ± 5 mmHg



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Capnogram	
Phase	**Interpretation**
A-B	Exhalation of dead space (devoid of CO2)
B-C	Expiratory Up-Stroke (should be sharp up-stroke, if not – COPD)
C-D	Alveolar Emptying
D-E	Inspiratory Down-Stroke

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Body Temperature

Body temperature should be maintained at or as near to normal as possible. Certain types of anesthetic agents are more commonly associated with excessive body temperature changes. Low body temperatures, although generally less likely to develop during dental or office-type anesthesia, may cause a delay in drug metabolism and patient recovery. High Body temperatures may cause a hypermetabolic state and increase oxygen consumption.

An enteral or transcutaneous device should be readily available to monitor body temperature during or after general anesthesia

During general anesthesia, when anesthetic agents associated with malignant hyperthermia are used, monitoring body temperature continuously is encouraged

Pulse Oximetry Monitoring Equipment

The pulse oximeter in its most basic form measures the oxygenation of the patient's blood. It is designed to operate on the assumption that hemoglobin exists in two principal forms in the blood: (1) oxygenated (with O_2 molecules loosely bound) = HbO_2 and (2) reduced (with no O_2 molecules bound) = Hb. SpO2 is defined as the ratio of oxygenated hemoglobin (HbO_2) to total hemoglobin (HbO_2 + Hb).

The pulse oximeter measures the absorption of selected wavelengths of light (660 nm and 910 nm) as they pass through living tissue, such as the fingertip, toe, or earlobe. HbO_2 and Hb absorb these wavelengths of light to differing degrees. Oxygenated hemoglobin (HbO_2) absorbs light at 660 nm, whereas deoxygenated hemoglobin (Hb) absorbs light at 910 nm. The relative percentages of these two hemoglobins are calculated within the oximeter, and the SpO_2 is displayed on the screen.

In addition to measuring oxygenation, Pulse Oximeters provide a continuous measurement of the heart rate. It accomplishes this by sensing the rate at which the beam passed through the tissue (eg. nail bed) is interrupted by the flow of blood through the finger following each contraction of the heart. This interruption produces a visual and/or audio signal.

In addition, a pulse oximeter monitor may measure several other physiological parameters, depending on its intended use.

Pulse oximetry inaccuracies may be due to:

- Low O2 saturation (<70%)
- Cold extremities
- Motion artifacts
- Intense ambient light
- Nail polish
- Methemoglobin

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An ideal monitoring device for IV conscious sedation:

- Must have alarms
  - Audible
  - Visual
  - Serve as warning when parameters are outside the norm
    - If no alarms, it is simply a measuring device
- Be reliable
- Battery power capability
- Measure the most important parameters
  - Heart Rate
  - Blood Pressure
  - O_2 Saturation
  - ECG
  - Respiratory Rate
- Accurate
- Noninvasive
- Easily interpreted display
- Easy to calibrate
- Stable
- Portable
- Easily integrated with other monitoring equipment

DOCS Education endorses **Criticare 8100 EP** designed for IV Sedation practitioners.

*Ask about the DOCS Education trade in and upgrade program

Additional Features:

➢ Arrhythmia and ST Analysis detection software available as an add-on for Criticare 8100 series monitors. Software is programmed to detect and alarm

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on 14 different rhythms proving an additional level of confidence for patient monitoring

- Replacement and Back-up Blood Pressure Cuffs and Finger Sensors for Criticare and GE Monitors: Blood pressure cuffs should be replaced every 6-12 (depending on use) months as standard of care to prevent and avoid contamination. Available BP Cuff sizes include, small, medium, large and extra large. It is also a wise idea to have back-up cuffs and sensors to avoid finding out one does not work properly on the day of the appointment or with a sedated patient in the chair.

Monitoring By Qualified Personnel

Up to this point the emphasis of this chapter has been on what to monitor, how to monitor it, and when and where the measurements should be taken. Some consideration needs to be given to ***who*** should be doing the monitoring pre-operatively, intra-operatively, and post-operatively.

At this point an important term must be defined as it will be repeatedly referred to – **Qualified Personnel**.

"Qualified Personnel is any individual (s) who has met the regulatory agencies requirements for performing a particular duty or set of duties. In the absence of defined regulatory requirements, personnel are considered qualified if in the opinion of the resident doctor they are adequately trained and experienced to satisfy the standard of care for the assigned responsibilities."

Now that we have an idea as to who Qualified Personnel are, let's establish rules for what they are responsible for during outpatient IV Sedation Anesthesia:

1. Qualified personnel shall take and record vital signs pre-operatively, intra-operatively, and post-operatively
2. Qualified personnel shall monitor sedated patients at all times while on the practice premises, except when the sedated patient is in the restroom
3. Female sedated patients shall be monitored by at least one female qualified personnel
4. For minimal sedation, one qualified personnel shall monitor the sedated patient at all times, except when the sedated patient is in the restroom

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5. For moderate sedation, two qualified personnel shall monitor the sedated patient at all times, except when the sedated patient is in the restroom
6. For deep sedation and general anesthesia, three qualified personnel shall monitor the sedated patient at all times, one of which who will be a dedicated anesthetist, except when the sedated patient is in the restroom

Recommended Monitoring for Adult Patients: IV Sedation

Parameter	Pre-Op	Intra-Operative	Post-operative
Heart Rate	**Yes**	**Continuously**	**Yes**
Blood Pressure	**Yes**	**Every 5 minutes**	**Yes**
EKG	**Yes**	**Continuously**	**Yes**
Respiration	**Yes**	**Continuously**	**Yes**
SpO2	**Yes**	**Continuously**	**Yes**

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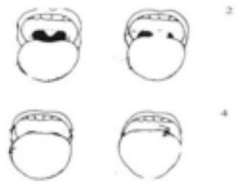
PATIENT_____ **DATE**_____

Drug Allergies:	**Previous Anesthesia History:** (IV or GA experiences)	**Pre-op vital signs:**
	_____ _____ _____	Height:_____ Wt: _____ BP:_____ Pulse:_____ Resp:_____

Rating Scales (circle patients diagram and ASA risk assessment)

Mobility Status: (check one in each category) ____Ambulatory ____Wheelchair
Condition: _____ Stable _____Unstable
Activity: (Able to move voluntarily/on command) ___4 extremities ___2 extremities ____0 extrem.
Respiration: ___Able to deep breath and cough _____Dyspnea, shallow or limited breathing
Conscious: ____ Fully awake ____Arousable _____Not responding
Color: ___Normal ___pale, other ____cyanotic



Mallampati Assessment

	ASA classification
I	No organic disease
II	Mild or moderate systemic disease without functional impairment
III	Organic disease with definite functional impairment
IV	Incapacitating systemic disease that is a constant threat to life

Comments regarding pre-assessment:	
Evaluating Dentist Signature:	**Date:**

Fluid type & total ml used:

_____

Veinpuncture attempts:____

IV Start:	**SEDATION START:**	**Monitors used:** **Person:** _____ **B/P:** _____/_____ **Pulse:** _____bpm **Oximetry:** _____SpO2 **ECG:** _____ **Other:** _____
IV Cath Size:	**PROCEDURE START:**	
IV Site:	**PROCEDURE END:**	
IV Out:	**Pt. release:**	

Rating of Sedation (Check one)

No difficulty_____, slight difficulty_____, unable to accomplish treatment_____

Explaination of Rating: _____

Anesthesia Complications: (Circle which if any)

From sedation: N/V, paradoxical effect, excessive drowsness, NONE

From venipuncture: phlebitis, infiltration, hematoma, local infxn.NONE

Disposition Upon Dismissal

[ ] Tolerated procedure and anesthetic well, alert and oriented to time and place.
[ ] Escorted to companion's vehicle by wheelchair and seat belted into departing vehicle.
[ ] Return ____ days for post-op. Appointment Date._____
[ ] Return PRN
[ ] Verbal instructions given to Patient & caregiver [ ] _____
[ ] Written post-op instructions given Name & Relation of Person Accompanying Patient

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LOCAL ANESTHETICS: A PRACTICAL REVIEW FOR SEDATION DENTISTRY

Relationship to Sedation Dentistry

Drug Interaction Potential

The most common drug that dentists administer are local anesthetics, with or without vasoconstrictor. Theoretically then, the most likely drug interaction with sedation medications will be with local anesthetics. From a practical standpoint, this is not the case. Still, the potential exists for drug interactions between local anesthetics, vasoconstrictors, and the drugs the patient presents with on board.

Increased Toxicity Potential

Given the extensive needs that the "typical" sedation presents with, as well as the desire to resolve as many of their dental needs per visit, the potential for larger doses of local anesthetic and vasoconstrictor exists. In larger amounts, these normally innocuous drugs dentists use every day can pose problems, especially in the non-healthy (≥ASA II) patient.

Special Needs Of The Sedation Patient

The sedation patient is often anxious because they are a more of a challenge to numb. Conscious sedation will allow for more profound anesthesia to be accomplished, but a better understanding of the pharmacology of local anesthetics as well as more effective techniques will result in a higher success rate of sedation.

History

Local anesthetics have a long and storied history of use in dentistry. Their action, the reversible blocking of impulse conduction along nerve axons may be the most important discovery in the history of the dental profession.

Cocaine is a naturally occurring compound indigenous to the Andes Mountains, West Indies and Java. It was the first anesthetic to be discovered and is the only naturally occurring anesthetic.

Cocaine was first isolated in 1860 by Albert Neimann. In 1884, Carl Koller introduced it for medicinal use as an ophthalmic anesthetic. Several famous clinicians experimented with its use including Sigmund Freud and William Halstead, both of which became transiently addicted to the drug.

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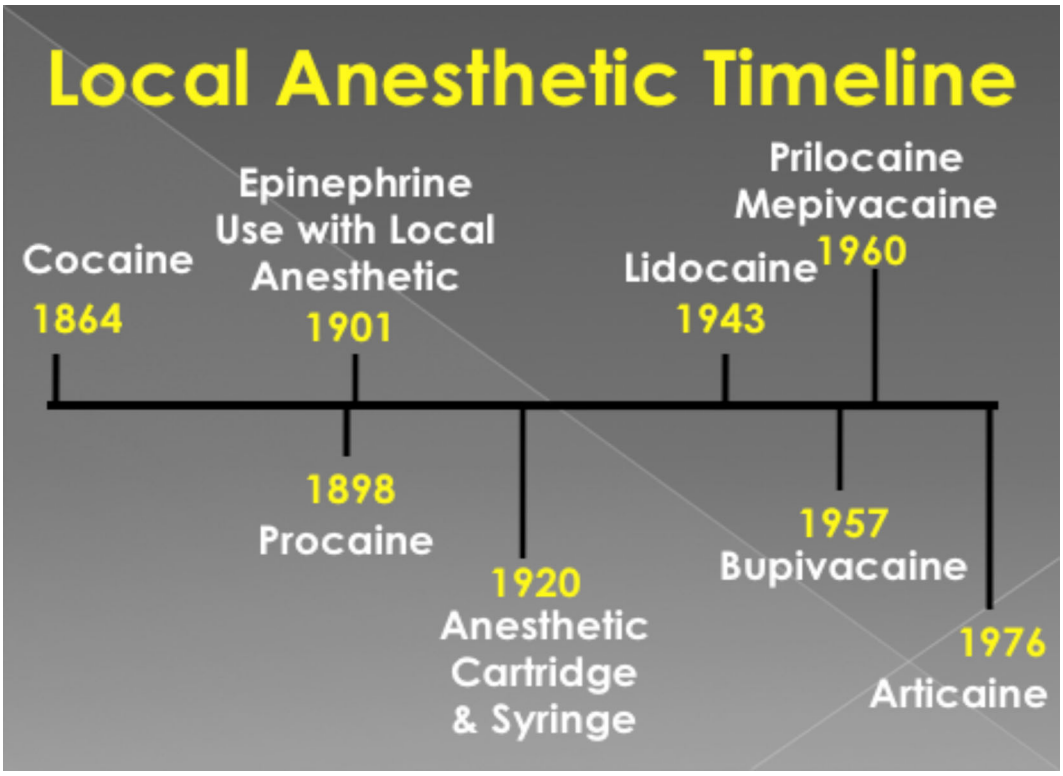
Cocaine, still the only local anesthetic with vasoconstrictor properties, remained the only anesthetic available for the next 30 years until Procaine was synthesized in 1898. Since then, several new compounds have been introduced with more desirable qualities of safety, efficacy, and duration of action.



Chemical Structure

The pharmacological qualities of local anesthetics can be traced to their chemical structure. There are two major chemical variants, the aminoamide and the aminoester.

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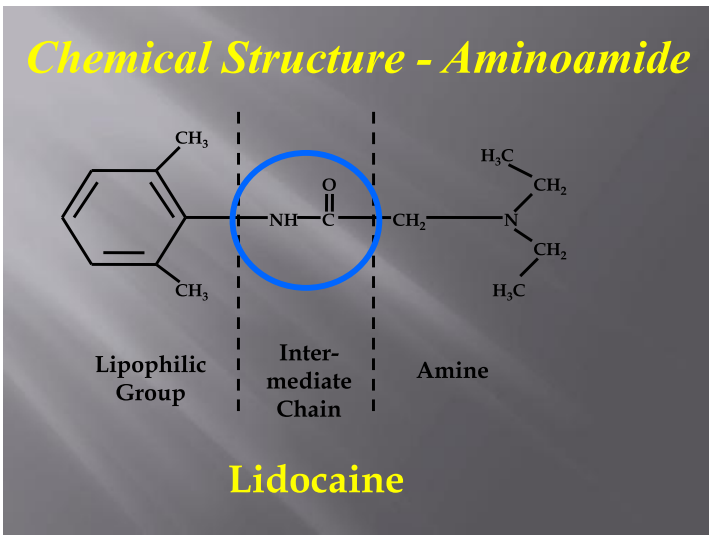

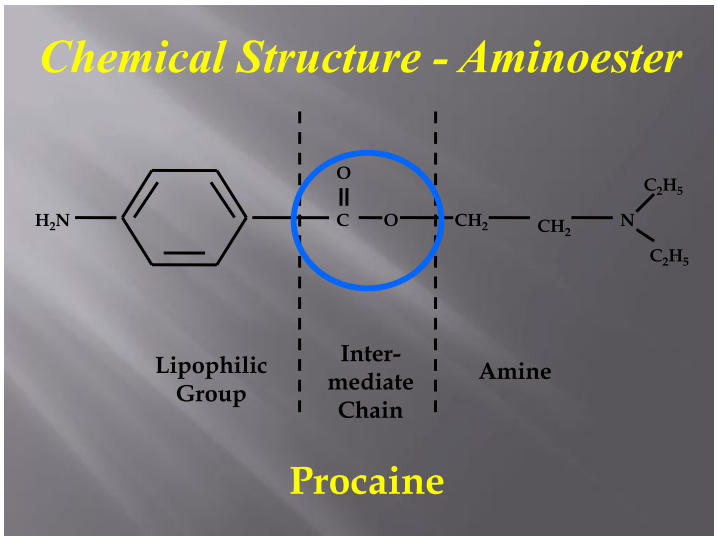

The aminoamide, of which lidocaine is a prime example contains a lipohilic group connected to an amine by an amide intermediate chain. In the case of the aminoester such as procaine, the intermediate chain is an ester. This ester portion is readily metabolized by plasma esterases, making its duration of action much shorter than the aminoamides.

Common Local Anesthetics & Their Characteristics

Lidocaine
Xylocaine®, Octocaine®
First aminoamide local anesthetic
Available in 1 & 2% concentrations
1 and 2% with 1:100,000 epinephrine
2% with 1:200,000 epinephrine

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2% with 1:50,000 epinephrine

Prilocaine
Citanest®
Less pain upon injection than other local anesthetics, possibly due to higher pH
Used in combination with lidocaine in the topical anesthetic EMLA
Rare side effect of methemoglobinemia
Available in 3 & 4% concentrations
4% without epinephrine (plain)
4% with 1:200,000 epinephrine (Citanest Forte®)
Metabolism is hepatic and renal
Half life of 10-150 minutes

Mepivacaine
Carbocaine®, Polocaine®
Available in 1, 1.5, 2, & 3% concentrations
3% without epinephrine
Surgical anesthesia of Mepivacaine without vasoconstrictor is 20-25 minutes
2% with 1:20,000 levonordephrin
Chemically related to bupivacaine
Pharmacologically related to lidocaine
Relatively low toxicity
Less pronounced vasodilator activity than lidocaine

Bupivacaine
Marcaine®, Senorcaine®, Vivacaine®
Available in 0.25, 0.5, & 0.75% concentrations
0.25, 0.5, 0.75% with 1:200,000 epinephrine
Metabolism Hepatic, Elimination Renal
Long duration of action
Half-Life 3.5 hours
Greater lipid solubility and protein binding than lidocaine
Period of analgesia that persists after return of sensation, during which time the need for strong analgesics is reduced
Greater cardiotoxicity

Articaine
Septocaine®
Contains both ester and amide groups
Acts like an aminoamide with hepatic metabolism
Ester group allows plasma metabolism via pseudocholinesterase, increasing rate of breakdown and reducing its toxicity
30 minutes half life
Available in 4% concentrations with 1:100,000 epinephrine

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Higher relative lipid solubility
Reported superior clinical properties
Long-term or transient paresthesia occurs more frequently than with lidocaine

PHARMACODYNAMICS OF LOCAL ANESTHETICS

Mechanism of Action

Local anesthetics are weak bases that exist in one of two forms, as a charged cation (RNH^+), or an uncharged base (RN). The relative percentage of these two forms affects both onset of action and efficacy, and is described by the pK_a of the agent. The pK_a of most local anesthetics is in the range of 8.0 – 9.0, meaning that at neutral pH, most of the local anesthetic exists as the charged cation form.

Many excitable membranes in the body, including nerve axons are influenced through gated sodium channels which open and close, allowing influx of sodium ions (Na^+). The resulting depolarization allows an electrical impulse to be propagated down the length of the nerve membrane. As a result of depolarization, the Na^+ channels close and the Potassium ion (K^+) channels open. Outward flow of K^+ repolarizes the membrane and returns the Na^+ channels to the rested state.

Local anesthetic agents work by binding to and inactivating sodium channels in the nerve membrane, thus blocking conduction in peripheral nerves.

Onset of Action

Being knowledgeable of the factors that determine the characteristics of the action of local anesthetics allows the practitioner to better control the outcome of local anesthesia. The onset of action is a prime example. It is determined by:
Diffusion to the site of action – Generally speaking, the closer to the nerve being anesthetized, the faster the action. This partly explains why infiltration often works faster than a block.

Nerve Morphology – The smaller in diameter the nerve, the faster anesthesia occurs. Pain fibers are relatively thin compared to motor fibers.

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More specifically, the progression of anesthesia is related to the diameter, myelination, and conduction velocity of affected nerve fibers.

Clinically, the order of loss of nerve function is as follows:

1. Pain
2. Temperature
3. Touch
4. Proprioception
5. Skeletal Muscle Tone

Lipid Solubility – Since the nerve membrane is lipid in nature, the more lipophilic (lipid loving) the agent, the more readily it penetrates the nerve sheath. This is the major determinant of potency of local anesthetics. Therefore, agents with lower solubility are generally marketed at higher concentrations.

Local Anesthetic	Lipid Solubility	Concentration (%)
Articaine	40	4
Mepivacaine	42	2-3
Prilocaine	55	4
Lidocaine	110	2
Bupivacaine	1,853	0.5

pK_a of the agent – The lower the pK_a, the higher the portion of the agent that is uncharged, which is the portion capable of penetrating the nerve membrane.

In the body, local anesthetics exist as the uncharged base or as a cation:

The relative proportions of these two forms are governed by their pKa and the pH of the body fluids.

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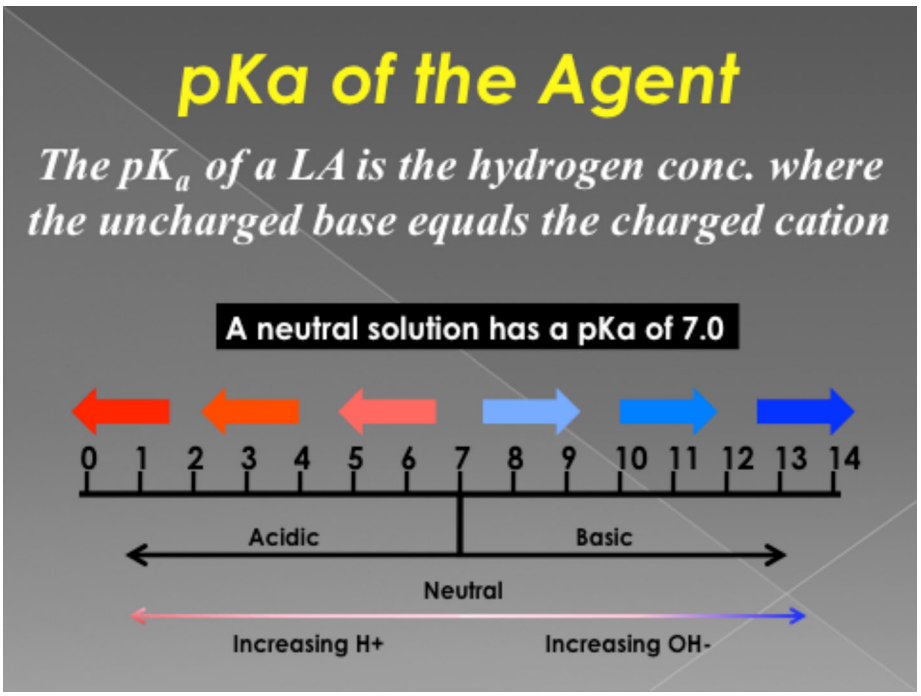

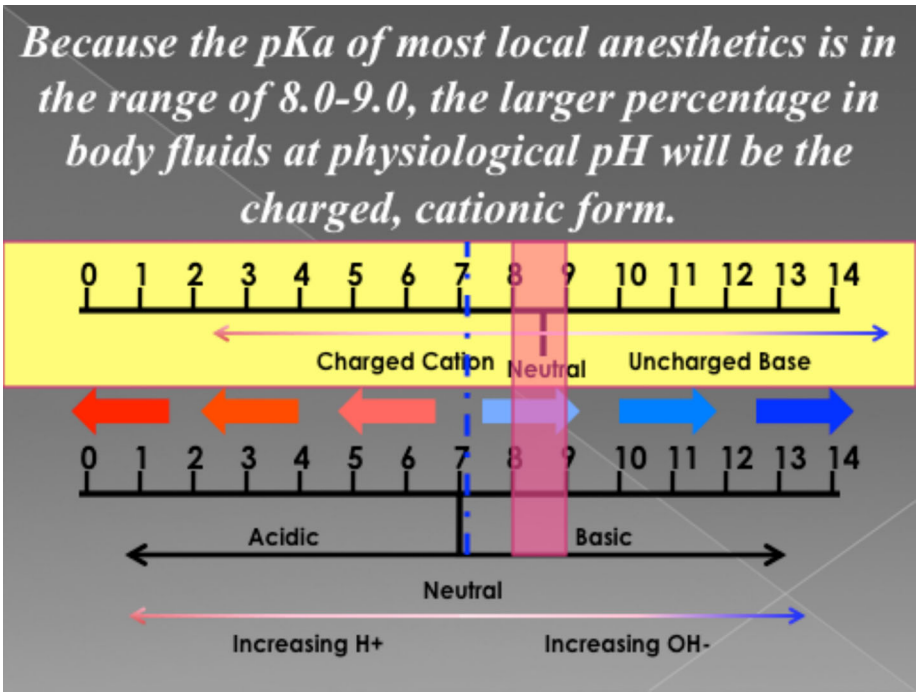

The uncharged form is more lipid soluble, therefore more readily crosses the cell membrane. This is important to producing a clinical effect, since the local anesthetic receptor is not accessible from the external side of the cell.

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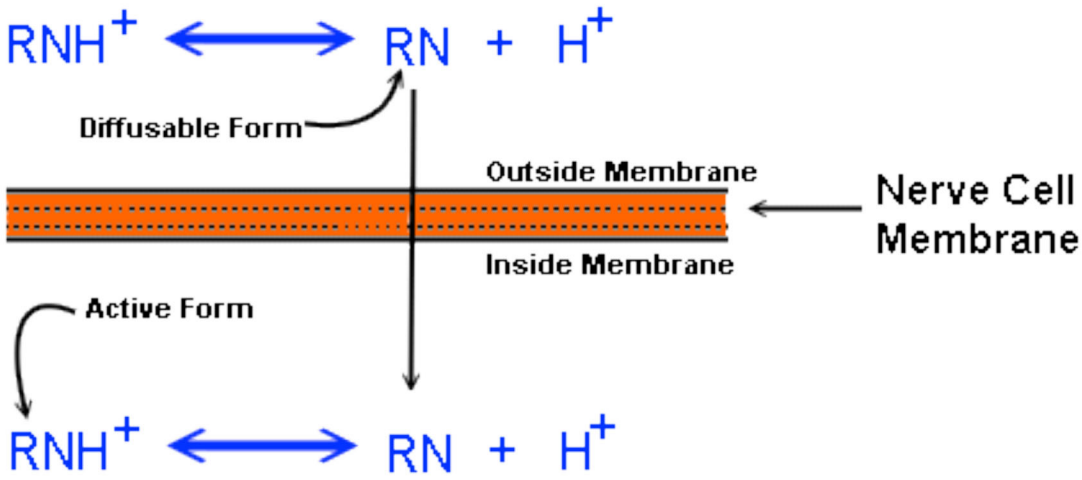
Once inside the nerve cell, equilibrium is re-established with the ionized form:



It is this ionized form that is most active at the receptor site because it is less lipid soluble and cannot exit from closed channels.

A lower pK_a means a greater fraction of the local anesthetic exists in the unionized (base) form, which more easily crosses nerve membranes leading to faster onset of action. Therefore, the onset of action of commonly used local anesthetics reflects their pK_a.

Agent	pK_a	% RN at ph 7.4	Onset in Minutes
Mepivacaine	7.6	40	2 to 4
Etidocaine	7.7	33	2 to 4
Articaine	7.8	29	2 to 4
Lidocaine	7.9	25	2 to 4
Prilocaine	7.9	25	2 to 4
Bupivacaine	8.1	18	5 to 8
Procaine	9.1	2	14 to 18

pH of the tissue – The lower the pH, the higher the portion of the agent that is charged, which is the portion least able to penetrate the nerve membrane. Inflamed tissue is more acidic, which explains why local anesthesia action in the presence of inflammation results in a slower onset of action.

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Duration of Action

The duration of action is determined by:

Volume – Doubling the dose increases duration by ~ 1 half-life.

Lipid Solubility – Cell membranes are made up of a lipid bilayer. If the local anesthetic agent dissolves easier into this bilayer, then there is less agent available to diffuse away from the cell.

Protein Binding – Higher plasma protein binding means higher binding to the receptor site in the ion channel. Therefore, increased protein binding = longer duration of action.

Agent	Approximate Protein Binding
Prilocaine	55
Lidocaine	65
Mepivacaine	75
Bupivacaine	95
Articaine	95

Diffusion away from the site – This is potentiated by vasodilation and reduced by the addition of a vasoconstrictor in the local anesthetic solution.

Other factors affecting duration of action – The type of injection (block, infiltration), and the tissue anesthetized have an effect on the duration of action of local anesthetics.

Soft tissue anesthesia outlasts pulpal anesthesia, while block anesthesia lasts longer than that obtained through infiltration alone.

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Duration of Action of Local Anesthetics (minutes)				
	Maxillary Infiltration		Inferior Alveolar Block	
Formulation	**Pulp**	**Soft Tissue**	**Pulp**	**Soft Tissue**
Articaine 4% with epi 1:100,000 or 1:200,000	60	190	90	230
Bupivacaine 0.5% with epi 1:200,000	40	340	240	440
Lidocaine 2% with epi 1:50,000 or 1:100,000	60	170	85	190
Mepivacaine 2% with 1:20,000 levonordephrin	50	130	75	185
Mepivacaine 3% plain	25	90	40	165
Prilocaine 4% with epi 1:200,000	40	140	60	220
Prilocaine 4% plain	20	105	55	190

While the duration of action is more relevant to the quality of the anesthesia, the agent's half-life describes the length of time the agent is in the patient's system, capable of causing adverse reactions including interactions with other drugs. Despite the differences between duration of action and half-life, the longer acting local anesthetics also have longer elimination half-lives.

Elimination Half-Lives of Local Anesthetics	
Local Anesthetic	**Elimination Half-Life (hrs.)**
Lidocaine	1.6
Mepivacaine	1.9
Prilocaine	1.6
Bupivacaine	3.5
Articaine	1.8

CALCULATING LOCAL ANESTHETIC DOSAGE ADMINISTERED

When calculating the amount (mg) of local anesthetic administered, the first step is to determine the mg/ml given the concentration of the

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agent. This is accomplished by moving the decimal point of the concentration one (1) place to the right.

For example:
Bupivacaine 0.5% - Moving the decimal point one place to the right = 5. Therefore, Bupivacaine 0.5% contains 5 mg/ml.

Other examples:
Lidocaine 2% = 20 mg/ml
Prilocaine 4% = 40 mg/ml
Articaine 4% = 40 mg/ml

Once the amount of mg/ml is determined then this amount is multiplied by the number of ml (cc) administered. For example:

If one 1.8 ml cartridge of 2% lidocaine is administered then,
1.8 ml X 20 mg/ml = 36 mg of lidocaine administered

CALCULATING VASOCONSTRICTOR DOSAGE ADMINISTERED

As when calculating the amount (mg) of local anesthetic administered, the first step in calculating the amount (mg) of vasoconstrictor administered is to determine the mg/ml given the concentration of the agent. This is accomplished by moving the decimal point of the concentration one (1) place to the right.

From the dilution:
1:1000 = 0.1% or 1mg/ml
1:10,000 = 0.01% or .1mg/ml
1:20,000 = 0.005% or .05mg/ml
1:50,000 = 0.002% or .02mg/ml
1:100,000 = 0.001% or .01mg/ml
1:200,000 = 0.0005% or .005 mg/ml

If administering 1.8 ml of solution containing 1:100,000 epinephrine, then multiply 1.8 X 0.01 = 0.018mg

LOCAL ANESTHETIC ADVERSE REACTIONS

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Local anesthetics, like any drugs carry with them the potential for adverse reactions. These include the following:

- Psychogenic
- Allergy
- Toxicity
- Local Reaction
- Methemoglobinemia
- Paresthesia
- Drug Interaction with Vasoconstrictors in the Local Anesthetic

Of these, the adverse reactions of most significance to the sedation dentists are local anesthesia toxicity, paresthesia, and vasoconstrictor toxicity and drug interactions.

Toxicity – Local anesthetics can block conduction in other tissues (eg. Heart, Brain) in addition to its action on peripheral nerves. However, this is unusual during our applications because it requires larger amounts of circulating local anesthetic atypical in a dental setting. Still, these larger than normal amounts can occur if excessive amounts of local anesthetic are administered, local anesthetic is injected directly into a blood vessel, or local anesthetic is administered in large amounts in the absence of a vasoconstrictor.

Signs of Local Anesthetic Overdose include:
Low/Moderate Overdose Levels

- Confusion
- Apprehension
- Restlessness
- Slurred Speech
- Muscular twitching/tremor of the face
- Elevated heart rate, blood pressure, respiratory rate

Moderate/Severe Overdose Levels

- Seizures
- Generalized CNS Depression
- Depressed heart rate, blood pressure, respiratory rate

Treatment of Local Anesthetic Overdose:

- Stop dental treatment

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- Oxygen via a controlled airway
- Monitor vital signs
- Reassure patient, allow time for drug to distribute and be metabolized
- If patient becomes unstable, activate EMS and emphasize PABCDs

Prevention of Local Anesthetic Overdose*:
Use good technique

- Use judiciously
- When appropriate, use vasoconstrictor
- Avoid intravascular injections

*Local Anesthetic overdose can occur more easily in children, the elderly, and medically complex patients
Remember the maximum recommended doses:

Drug	Maximum Dose	Max. # of Cartridges
Articaine 4% with epi	7 mg/kg in adults (up to 500mg) 5mg/kg in children	7
Bupivacaine 0.5% with epi	2 mg/kg (up to 200mg)	10
Lidocaine 2% with epi	7 mg/kg (up to 500mg)	13
Mepivacaine 2% with levonordephrin	6.6mg/kg (up to 400mg)	11
Mepivacaine 3% plain	6.6mg/kg (up to 400mg)	7
Prilocaine 4% plain or with epi	8mg/kg (up to 500mg)	8

Paresthesia – Most likely to occur with the inferior alveolar or lingual nerves, and rarely lasts longer than eight weeks. Some literature

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supports the conclusion that it is most likely to occur with certain local anesthetics (prilocaine and articaine).

Articaine Controversies

Efficacy

- In a 2000 report, a majority of 94 dentists stated that articaine anesthesia was more profound than “routinely used anesthetics”.
- In addition, the onset of effects was faster and the anesthesia achieved greater success among difficult-to-anesthetize patients.
- According to Dudkiewicz et al., articaine was able to diffuse through bone onto the lingual side of teeth.
- Kanaa et al. found mandibular buccal infiltration to be more effective with septocaine than with lidocaine Vree and Gielen found that articaine diffuses better through the soft tissue and bone than other local anesthetics.
- Mounting body of evidence that says articaine does not outperform the old standard lidocaine.
- Prospective, randomized, double-blind study by Mikesell et al.,. It was concluded that for pulpal anesthesia, articaine was similar to lidocaine.
- Studies by Berlin et al., as well as Claffey et al., resulting in similar conclusions

Toxicity

- Haas and Lennon, in a 21 year retrospective study, reported that paresthesia occurred most often following the injection of articaine and prilocaine.
- Hillerup and Jensen examined 54 injection injuries in 52 patients caused by mandibular block analgesia affecting the lingual and/or inferior alveolar nerve. The results indicated neurotoxicity as a central etiological factor.

Recommendations

- Use good technique
- If you use articaine for block anesthesia, use it judiciously.
- For maximum risk elimination of dental injection paresthesia, avoid articaine during block anesthesia.

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The Mandibular Cocktail Block Technique

- A technique for profound anesthesia, maximum comfort, and minimum risk of local anesthesia and vasoconstrictor toxicity for the sedation patient with full mouth dentistry in one appointment.
- The purpose is to provide profound pulpal anesthesia in the lower arch for the duration of the sedation appointment, preventing discomfort later in the appointment and the need for re-administration of local anesthetic.
- Utilizes four different local anesthetics to exploit the advantages that each has to offer:
  1. Prilocaine's higher pH – more comfortable injection
  2. Articaine's efficacy and diffusion qualities
  3. Lidocaine's efficacy and safety – reduces the amount of articaine needed
  4. Bupivacaine's extended anesthesia with block anesthesia
- Technique:
  1. Topical anesthesia on dry tissue for one minute
  2. ¼ Cartridge of 4% Prilocaine Plain
  3. ½ Cartridge of 2% Lidocaine with 1:100,000 epinephrine
  4. ½ Cartridge of 4% Articaine with 1:100,000 epinephrine
  5. 1 Cartridge of 0.5% Bupivacaine with 1:200,000

Vasoconstrictor Toxicity related to medical conditions – The addition of vasoconstrictors to local anesthetics serves four functions:

1. Prolong duration of action of the local anesthetic
2. Enhance the depth of anesthesia
3. Enhance hemostasis
4. Reduce systemic local anesthetic toxicity

In many randomized studies, pain control is significantly impaired in those patients receiving local anesthetics without vasoconstrictors. Inadequate pain control increases endogenous production of norepinephrine, which increases heart rate, cardiac workload and cardiac stress.

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Most of the adverse reactions associated with vasoconstrictor toxicity have to do with its affect on the cardiovascular system, and should be avoided in the following situations:

Cardiovascular Diseases

- Resting blood pressure >200mm Hg systolic or >115mm Hg diastolic
- Myocardial infarction within the past six months
- Cerebrovascular accident (stroke) within the past six months
- Unstable angina (angina at rest or not well controlled with medication)
- Coronary artery bypass graft within the past six months
- Uncontrolled arrhythmias
- Uncontrolled congestive heart failure

The maximum recommended dose of vasoconstrictor in cardiovascular patients is 0.04mg.

- 1 cartridge of 2% lidocaine with 1:50,000 epinephrine
- 2 cartridges of 2% lidocaine with 1:100,000 epinephrine
- 4 cartridges of 2% lidocaine with 1:200,000 epinephrine
- 2 cartridges of local anesthetic with 1:20,000 levonordephrin (1/5 potency of epinephrine)

Treatment modifications for patients at risk with vasoconstrictors:
Monitor blood pressure and heart rate preoperatively
Consider limiting epinephrine to 0.04mg or levonordephrin to 0.02mg
Monitor blood pressure and heart rate 5 minutes post-injection
May re-administer epinephrine or levonordephrin if blood pressure and heart rate are stable
Avoid 1:50,000 epinephrine
Never use epinephrine-impregnated retraction cord

Other medical conditions also warrant caution when considering the use of a vasoconstrictor with a local anesthetic:

- Uncontrolled hyperthyroidism
- Pheochromocytoma
- Sulfite-sensitive asthma
- True sulfite allergy

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Vasoconstrictor Toxicity related to drug interactions – In order to better understand the interaction between some drugs and vasoconstrictors, it is helpful to review the different receptors affected by vasoconstrictors and the resulting action.

Alpha 1 receptors exist in blood vessels and when affected by vasoconstrictors, result in vasoconstriction. With regard to the use of vasoconstrictors in dentistry, this is a desired effect.

Beta 1 receptors exist in the heart and activation of these receptors results in tachycardia and hypertension, clearly undesirable side effects. Norepinephrine actually has higher beta-1 activity than either epinephrine or levonordephrin and would cause increase in heart rate and increase in cardiac work-load. This has actually led to cessation of use of norepinephrine as a vasoconstrictor in local anesthetics.

There are also beta-2 receptors in the vessel walls, causing slight vasodilation, balancing somewhat the alpha 1 effect. This effect is not desirable. Under normal circumstances, the alpha 1 effect of vasoconstrictors dominates over the beta effects. The net result is increased blood pressure and increased heart rate.

In the choice of vasoconstrictors, one would like to optimize the alpha effect and minimize the beta-1 and beta-2 effects.

Anti-hypertensive drugs that fall in the class of **alpha blockers** reduce blood pressure by blocking the alpha 1 vasoconstriction activity, allowing the beta 2 effect to go unchecked. Examples of alpha blockers are:

Blood Pressure Medications
- Aldomet
- Labetolol
- Phentolamine
- Hytrin
- Cardura

Antipsychotics
- Phenothiazines

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The significance to dentistry is that in the presences of an alpha blocking agent, epinephrine actually causes vasodilation, shorter durations of anesthesia and more bleeding. This phenomenon is called **Epinephrine Reversal**.

To avoid epinephrine reversal, use levonordephrin in place of epinephrine:

Vasoconstrictor	**Beta 1 Selectivity %**	**Beta 2 Selectivity %**	**Relative Alpha Potency%**
Epinephrine	50	50	100
Levonordephrin	75	25	15

Interestingly, this alpha adrenergic blocking effect is the proposed mechanism of the soon to be released "local anesthetic reversal agent" **Phentolamine Mesylate** (formerly known as NV-101). This drug will be sold under the brand name, **Oraverse®,** and has been shown to effectively reduce the time from soft tissue local anesthesia to normal sensation by 56%. Although its exact mechanism is unknown, it is thought to result from a predominance of beta-2 vasodilation in the area of local anesthesia administration, therefore resulting in more of the local anesthetic being absorbed into the blood vessels.

Non-Selective beta blocking agents act by blocking both the beta 1 effect (resulting in decreased heart rate) and the beta 2 effect (resulting in less vasodilation of peripheral blood vessels). Non-selective beta blockers are widely used in patients with hypertension, angina, congestive heart failure and arrhythmia. Examples of non-selective beta blockers are:

Nadolol (Corgard)
Propranolol (Inderal)

Selective beta blocking agents block only the beta 1 effect (resulting in decreased heart rate), and not the vasodilation which somewhat counteracts the alpha 1 effect. Examples of selective beta blockers are:

Atenolol (Tenormin)
Bisoprolol (Zebeta)

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Metoprolol (Lopressor, Toprol XL)

When administered a vasoconstrictor, patients on cardio-selective beta blockers exhibit the “typical” increase in blood pressure without the “typical” increase in heart rate.

However, when administered a vasoconstrictor, patients on non-selective beta blockers often notice a marked increase in blood pressure. The goal with these patients is to dose local anesthetics with vasoconstrictors “low and slow”.
Patients taking **tricyclic antidepressants** (TCAs) have been shown to have an increased pressor response in combination with epinephrine and levornordephrin. Examples of the TCAs include:

Imipramine (Tofranil)
Amitriptyline (Elavil)
Desipramine (Norpramin)
Nortriptyline (Aventyl)
Doxepin (Sinequan)
Protriptyline (Vivactil)

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INCREMENTAL ORAL SEDATION
FOR IV SEDATION PREMEDICATION

Definition

The administration of small doses of an oral sedative at appropriate intervals to achieve and maintain sedation.

Rationale

The Time/Concentration Curve for Incremental Oral Administration differs from that of the Single-Dose Protocols in that additional sedative medication doses are given incrementally ("Dosing To Effect" – Not Titration*) to keep the patient at a "steady state" of sedation. This allows for better control of depth and length of sedation while maintaining the lowest total dose of a medication. Remember to use the lowest effective dose of any sedative.

Along with the ability to readily extend the depth and length of the sedation, incremental dosing affords the clinician the opportunity to administer smaller doses initially as well as throughout the appointment). This allows for an extra degree of safety not always possible with bolus dosing. In addition, the ability to administer additional doses allows for fewer sedation failures due to the high degree of individual variability.

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*Titration is not a meaningful term when used to describe the actions of oral medications – in this case the desired action of sedation in the dental setting. As stated above, "Incremental Oral Administration is defined as the administration of small doses of an oral sedative at appropriate intervals to achieve and maintain conscious sedation". In this definition, "small doses" refer to dosage amounts that would normally not produce a moderate level of sedation (conscious sedation) by itself. "Appropriate intervals" in this definition refers to two aspects of the incremental approach. The first is the technique of timing the assessment of an individual's response susceptibly to a previous dose, not the mean peak concentration, so that appropriate additional medications may be administered if necessary (with the understanding that additional sedative action will continue to occur from the previous dose). The second aspect that "appropriate intervals" refers to is the timing of additional small doses during an extended visit to maintain a comfort level for the patient. "Achieving…conscious sedation" refers to a minimally depressed level of consciousness whereby the patient retains their protective reflexes, can maintain there own airway, and responds to physical and verbal stimuli. "Maintaining conscious sedation" refers to the re-administration of small doses at appropriate intervals (usually tied to the half-

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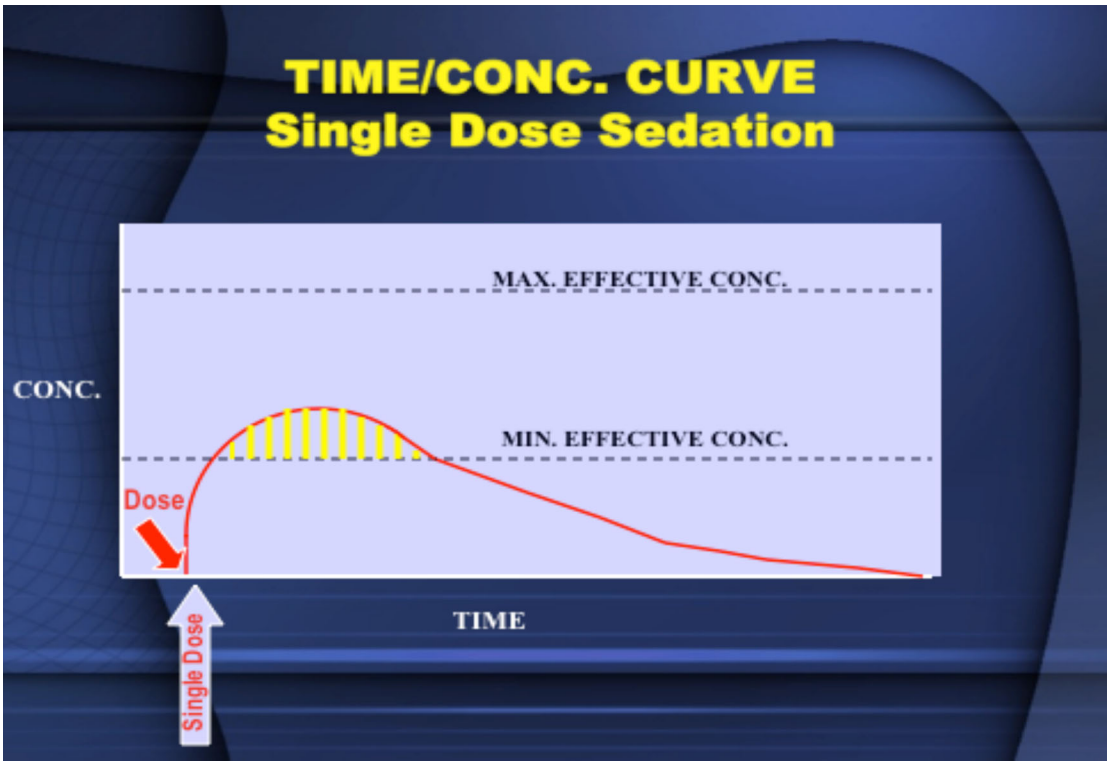
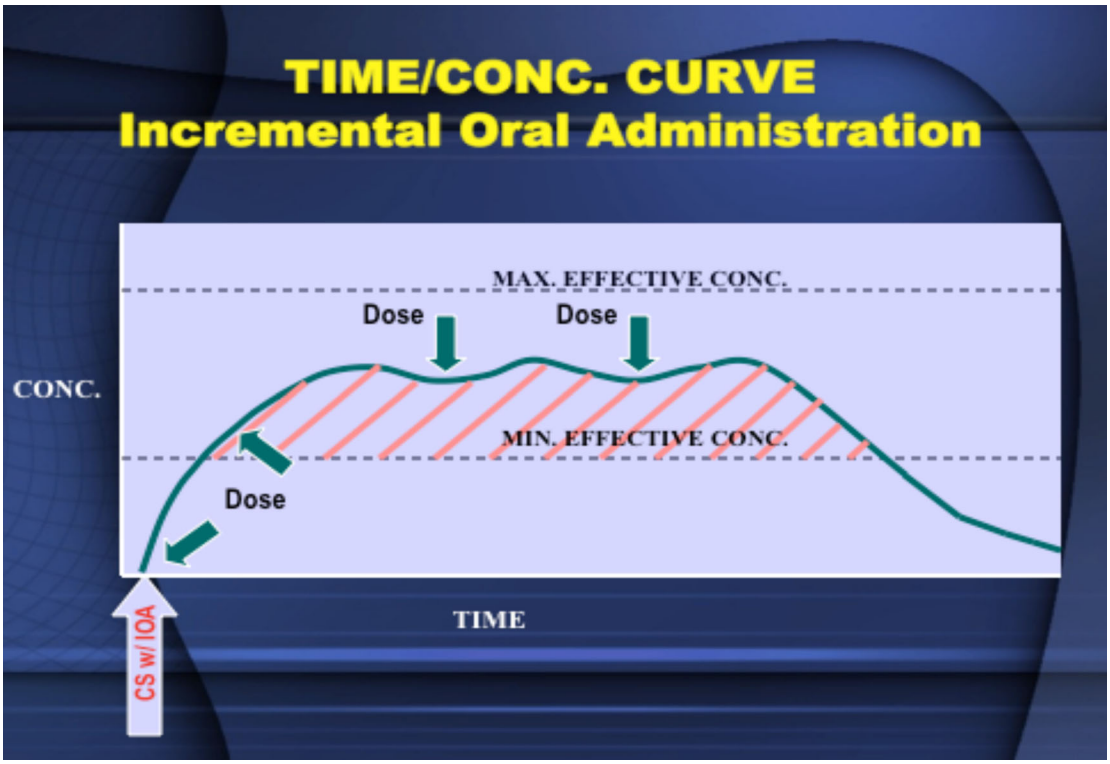
life of the medication) to continue the comfort of the patient with the understanding that the appropriate amount of time be allotted after the last dose for the patient to recover and be dismissed safely.





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Indications

- Moderate to high fear patients
- Longer Appointments (2-9 hrs.)

Drug List for Protocols

The drugs recommended for minimal sedation are the same drugs recommended for anxiolysis. These drugs all have a long track record of performance and safety for minimal sedation in dentistry. The clinical difference between their use for single dose and incremental oral minimal sedation involves the total dosage given and the time of administration. The other important difference between the use of these drugs for anxiolysis and conscious sedation lies in your intent as a practitioner for its use.

Drug (Generic Name)	Drug (Brand Name)
Diazepam	Valium
Triazolam	Halcion
Hydroxyzine	Atarax or Vistaril
Lorazepam	Ativan
Nitrous Oxide	Laughing Gas

Protocols

As with the anxiolysis and single-dose protocols, the incremental dose protocol utilizes an appropriate dose of a long-acting benzodiazepine, diazepam, the night before the appointment at bedtime. This dose serves two primary purposes:

1. The highly anxious patient is allowed to rest better the night before the appointment making for a more pleasant pre-operative experience.

2. The highly anxious patient has a sedative/hypnotic agent on board when they present the next day for their appointment making them not only more relaxed, but more susceptible to lower doses of pre-operative sedation prior to their appointment. This occurs through a synergistic effect between the two benzodiazepines, as well as obtunding the patient's anxiety resulting in better absorption from the gastrointestinal tract.

There are three categories of drug administration during the day of the appointment for the incremental dose protocol:

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1. **<u>One hour prior to the appointment</u>** – The medication of choice is taken by the patient one hour before the appointment. The medication is taken at the patient's home or while on their way with a companion driving them to the office.

2. **<u>At initial assessment</u>** – When the patient presents to the office, having taken the pre-operative dose as instructed, they are seated in the operatory. Physiologic monitoring begins at this time and is constant throughout the appointment. At the appropriate time interval, based on half-life kinetics, the patient's level of sedation is assessed. Based on this determination, additional medication may be administered in appropriate doses to maintain sedation. No additional medication is required at this point if the patient is sedated properly. Remember, the patient is NEVER left unattended.

3. **<u>At subsequent assessments during the appointment</u>** – Depending on susceptibility to a medication, the patient may require additional medication prior to or during treatment. After initial assessment, if additional medication is administered, the patient should be reassessed in 30-60 minutes. If the patient is at the proper level of sedation (a comfortable patient that fulfills the definition of minimal sedation), then dental procedures can be initiated as your schedule permits. If at this assessment it is determined that the patient is not yet properly sedated, additional doses of medication should be considered. It should be noted that the peak clinical effect of the original dose may not have occurred yet. Therefore, the dose of any additional medication should be considered judiciously. Absorption time may differ from individual to individual. This is why the protocol suggests NPO for 6 hours to improve absorption predictability.

One may question the 30-60minute assessment interval when considering the pharmacological characteristics of the drugs involved. How can a proper assessment be obtained when the peak concentration of an orally administered drug does not occur for more than an hour?

The 30-60 minute assessment interval is explained through the inactivation, redistribution, and elimination that are constantly taking place at receptor sites in the central nervous system. Even though the peak concentration has not occurred, a realistic assessment of the patient's susceptibility to that dose can be determined especially when administered sublingually.

Once dentistry has begun, reassessment should occur every two to three hours until the appointment is completed. It should be emphasized that this

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two to three hour interval is an average for triazolam and lorazepam respectively, and each patient should be evaluated individually. Some patients will not need additional medication after dentistry has begun. Others will show signs of diminished sedation before the average two hour interval requiring more frequent assessment, and if appropriate, more frequent dosing. In all cases, additional medication should not be administered 2-4 hours prior to dismissal to allow for drug elimination.

Incremental Protocol #1
ASA I & II Adults 18 y.o. and older

- **Diazepam –** Night before at bedtime, 0 - 10 mg, swallowed
- **Triazolam –** 1 hr. prior to appointment 0.125 – 0.25mg, swallowed
- **Triazolam –** At initial assessment and reassessment, 0 – 0.5mg, sublingual
- **Nitrous –** in office, during delivery of local, titrate to effect with appropriate protocol

At Bedtime night before appointment – Diazepam

Amount of Night Before Dose of Diazepam Relative to Patient Characteristics

Factor \ Dose	0 mg	2.5 mg	5 mg	10 mg
Age	←	≥ 65	NO	NO
BMI	←	←	←	≥ 30
History	←	Hyper-response	Normoresponse	Hyporesponse
Existing Medications	≥ 3 CNS Drugs &/or Insomnia Drug, or [D] or [X] interaction between DZP & existing medication	2 CNS Drugs	0-1 CNS Drug ←	
Other	Patient drives &/or works on the day of prior to the sedation appointment	NO	NO	NO

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Day of the appointment

	Initial Dose (1 hr. prior to appoint-ment)	Initial assessment (1 hr. after initial dose)	Reassessment (q0.5hr until dentistry begun, and q2h thereafter)
Elderly/Debilitated/ CNS depressants*	**0.125 mg Triazolam**	**0 - 0.25 mg Triazolam[1]**	**None to 0.25 mg Triazolam[1]**
All others*	**0.25 mg Triazolam**	**0 - 0.5 mg Triazolam[1]**	**None to 0.5mg Triazolam[1]**

***Nitrous Oxide titrated to effect during administration of local anesthesia**

[1] See Assessment and Reassessment section of this chapter.

THE "GOLDEN STANDARD" PROTOCOL

(For an 8 a.m. appointment where dentistry is planned to begin at 9 a.m.)

1. **The patient (Adult ASA I or II) has received prior to their appointment a single dose of 0.25 mg of triazolam (for elderly, debilitated, or patients with a history of hyperreactivity, 0.125mg would be appropriate). They have also received a single dose of 5 mg of diazepam unless they are characteristics that would cause them to hyporespond (10 mg), or hyper-respond (0 – 2.5 mg).**

2. **Night Before Appointment at Bedtime: The patient swallows a single dose of diazepam (if appropriate).**

3. **7 AM: The patient takes a single dose of triazolam on an empty stomach (6 hours) one hour before appointed time at the office. A responsible companion escorts patient.**

4. **8 AM: The patient arrives at the office with a companion, (one hour before dental appointment time). The patient's compliance with medications is obtained and they are then escorted to the bathroom.**

5. **8:03 AM: The patient is seated in the operatory and has wristwatch and glasses removed and given to their companion.**

6. **8:04 AM: Continuous monitoring of vital signs (BP every 5 minutes, Heart Rate, and SaO_2) via pulse oximeter is initiated. Remember that the patients level of consciousness is assessed at each BP reading.**

7. **8:05 AM: The patient is assessed for additional medications. This assessment is a measure of patient susceptibility, not maximum**

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clinical effect. If necessary, additional medication is provided sublingually.

8. **8:35 AM: After 30-60 minutes the patient is reassessed as to their sedation state. If additional medication is deemed necessary, it should be delivered sublingually. The purpose of the incremental system is to achieve the lowest appropriate dose for a comfortable visit.**

9. **8:54 AM: Six minutes prior to dental appointment time, oxygen is introduced with the appropriate protocol!**

10. **8:57 AM: Nitrous oxide is introduced with appropriate protocol.**

11. **9:00 AM: Local anesthesia is administered.**

12. **Nitrous oxide is removed with the appropriate protocol.**

13. **Begin dentistry.**

14. **Continuous monitoring and checking patient every 5 minutes for appropriate verbal responses.**

15. **When temporary patient cooperation is needed (bite adjustments, bathroom breaks or x-rays) 2 oz. of a clear juice drink with a straw is provided. This should improve cooperation for approximately 5 minutes. The mechanism here is not pharmacological reversal of sedation but physiological stimulation through minor hypoglycemic reversal (the patient has been npo for >8hours.)**

16. **When dentistry is completed, the patient consumes at least 10 ounces of a clear sugar drink. Gatorade type drinks work well.**

17. **The patient will arouse and must be completely conscious to be able to make the journey home safely with their responsible companion.**

18. **Ask the patient to tell you where they are? What day is it? What is their name? This will give you a psychological assessment of orientation X3. Make sure the patient is responding appropriately and is ambulatory.**

19. **Record all appropriate information in the chart… "Patient tolerated treatment well and was ambulatory and oriented X3 when they left".**

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20. **The patient and their companion are given written postoperative instructions. Remember to have the companion sign a copy of these instructions.**

21. **Call all patients that night (remind them to hydrate!).**

Incremental Protocol #1 is the “golden standard”. Although there is limited scientific research at present to support its use, the protocol has an extensive clinical record. It has been effective for 75-90% of all patients requiring minimal sedation.

Additional protocols (Protocols #2-4) are provided for situations where protocol #1 is less effective or not indicated. These protocols are not supported by any scientific research at present, however based upon the pharmacological characteristics of the medications, and clinical experience, they are viable alternatives. These additional protocols are indicated in the following situations: sedation resistant patients, smokers, or instances where triazolam is contraindicated (ie complex medical conditions or potential drug reactions). Lorazepam has a metabolic pathway that is more resistant to the effects of cirrhosis and hepatic failure. As a result Lorazepam may be preferable to triazolam in a patient with liver failure. The metabolism of lorazepam differs from that of triazolam. This characteristic may make lorazepam an acceptable alternative to triazolam when contraindications to triazolam exist.

Modifications to the Golden Protocol for the afternoon appointment:

Occasionally, a patient will need to be scheduled in the afternoon to accommodate the needs of the office or the patient. When this occurs, the following needs to be kept in mind:

1. The patient still needs to be NPO for six hours prior to the appointment. If their appointment time is 1 p.m., for instance, then they must be NPO since 7 a.m.. To deprive a patient of any nourishment since the night before for such an appointment increases the likelihood of hypoglycemia. All other preoperative instructions remain the same, except for #2 below.
2. The preoperative dose of valium the night before should be eliminated and taken instead on the morning of the appointment. If taken at the time of their morning meal, consideration should be given to a larger dose. Once the valium is taken, the patient cannot work or drive, or participate in any other activity forbidden on the day of a sedation appointment.

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Incremental Protocol #2

ASA I & II Adults 18 y.o. and older

- **Diazepam –** Night before at bedtime, 0-10 mg, swallowed
- **Triazolam –** 1 hr. prior to appointment 0.125 – 0.25 mg, swallowed
- **Hydroxyzine –** At initial assessment 0 – 100 mg, slurry drink swallowed
- **Triazolam –** At initial assessment and reassessment 0 – 0.5 mg sublingual
- **Nitrous –** in office, during delivery of local, titrate to effect with appropriate protocol

At Bedtime night before appointment – Diazepam

Amount of Night Before Dose of Diazepam Relative to Patient Characteristics

Factor \ Dose	0 mg	2.5 mg	5 mg	10 mg
Age	↔	≥ 65	NO	NO
BMI	↔	↔	↔	≥ 30
History	↔	Hyper-response	Normoresponse	Hyporesponse
Existing Medications	≥ 3 CNS Drugs &/or Insomnia Drug, or [D] or [X] interaction between DZP & existing medication	2 CNS Drugs	0-1 CNS Drug ↔	
Other	Patient drives &/or works on the day of prior to the sedation appointment	NO	NO	NO

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Day of the appointment

	Initial Dose (1 hr. prior to appoint-ment)	Initial assessment (1 hr. after initial dose)	Reassess-ment (q0.5hr until dentistry begun, and q2h thereafter)
Elderly/Debilitated/ CNS depressants*	**0.125 mg Triazolam**	**0 - 0.25 mg Triazolam[1] 25 mg – 50 mg Hydroxyzine**	**0 - 0.25 mg Triazolam[1]**
All others*	**0.25 mg Triazolam**	**0 - 0.5 mg Triazolam[1] 50 mg – 100 mg Hydroxyzine**	**0 - 0.5 mg Triazolam[1]**

***Nitrous Oxide titrated to effect during administration of local anesthesia**

[1] See Assessment and Reassessment section of this chapter

Incremental Protocol #3

ASA I & II 12 y.o. and older (check state regulations)

- **Diazepam –** Night before at bedtime, 0-10 mg, swallowed
- **Lorazepam –** 1 hr. prior to appointment 1 -2 mg, swallowed
- **Lorazepam –** At initial assessment and reassessment 0 – 3 mg, sublingual
- **Nitrous –** in office, during delivery of local, titrate to effect with appropriate protocol

At Bedtime night before appointment – Diazepam

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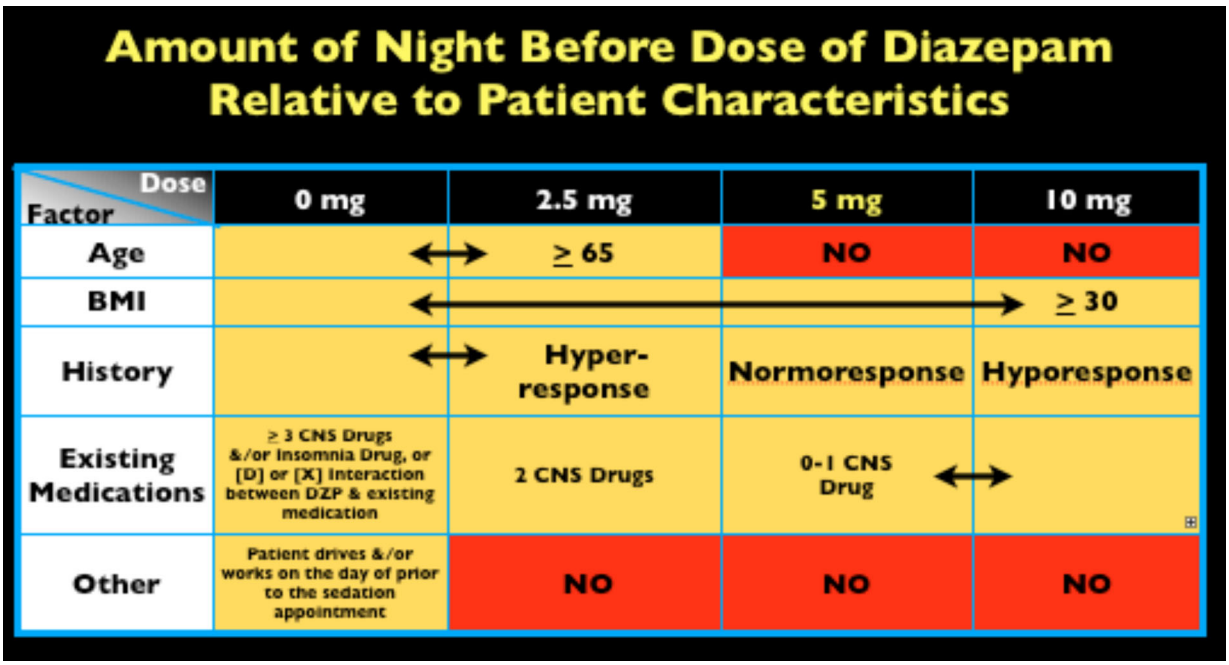

Day of the appointment

	Initial Dose (1 hr. prior to appoint-ment)	Initial assessment (1 hr. after initial dose)	Reassess-ment (q0.5hr until dentistry begun)
Elderly/Debilitated/ CNS depressants*	**1 mg Lorazepam**	**None to 2 mg Lorazepam[1]**	**None to 2 mg Lorazepam[1]**
All others*	**2 mg Lorazepam**	**None to 3 mg Lorazepam[1]**	**None to 3 mg Lorazepam[1]**

***Nitrous Oxide titrated to effect during administration of local anesthesia**

[1] See Assessment and Reassessment section of this chapter

Incremental Protocol #4

ASA I & II 12 y.o. and older (check state regulations)

- **Diazepam –** Night before at bedtime, 0-10 mg, swallowed
- **Lorazepam –** 1 hr. prior to appointment 1 – 2 mg, swallowed
- **Hydroxyzine –** At initial assessment 0 – 100 mg, slurry drink swallowed

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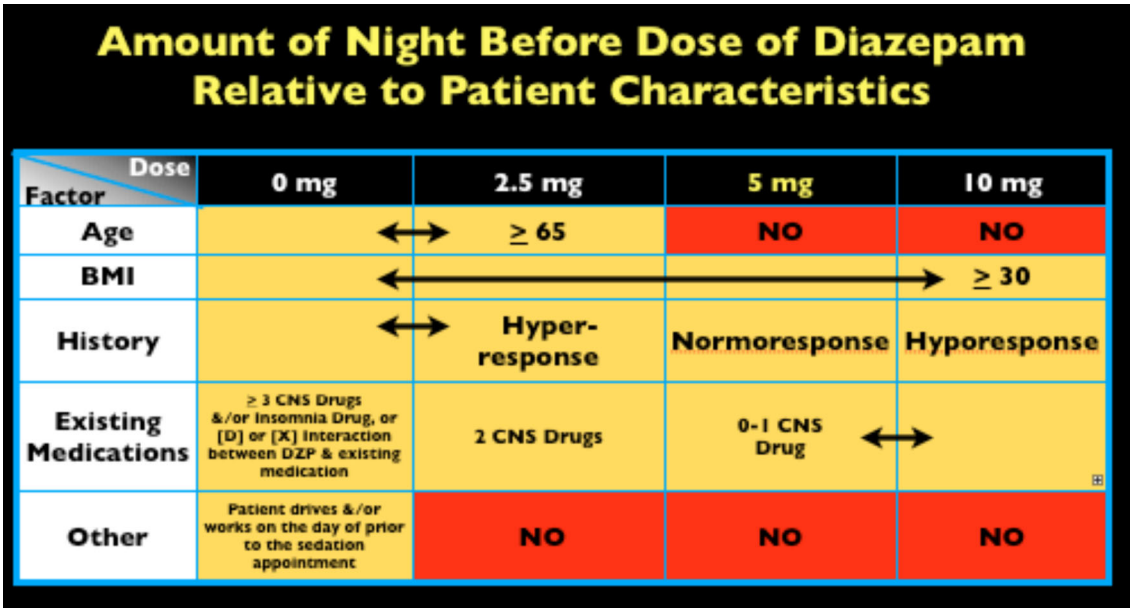
- **Lorazepam –** At initial assessment and reassessment 0 – 3 mg sublingual
- **Nitrous –** in office, during delivery of local, titrate to effect with appropriate protocol

At Bedtime night before appointment – Diazepam



Day of the appointment

	Initial Dose (1 hr. prior to appointment)	Initial assessment (1 hr. after initial dose)	Reassess-ment (q0.5hr until dentistry begun)
Elderly/Debilitated/ CNS depressants*	**1 mg Lorazepam**	**0 - 2 mg Lorazepam[1] 25 mg – 50 mg Hydroxyzine**	**0 - 2 mg Lorazepam[1]**
All others*	**2 mg Lorazepam**	**0 - 3 mg Lorazepam[1] 50 mg – 100 mg Hydroxyzine**	**0 - 3 mg Lorazepam[1]**

***Nitrous Oxide titrated to effect during administration of local anesthesia**

[1] See Assessment and Reassessment section of this chapter

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ASSESSMENT FOR ADDITIONAL MEDICATION (See Protocol 8:05AM)

1. **Sit at eye level with the patient.**
2. **Ask the patient to rate their level of sedation.** "Mary, on a scale from 1 to 10, where 1 is that you are totally relaxed (like don't bother me please!) and 10 is your totally awake (like you are ready to go Christmas shopping), where are you on that scale?" Using the individual patient's social history, construct the above questions to best relate examples of a relaxed or excited state.
3. **Observe the following:**
    - **a.** Patient's ability to make eye contact. Good eye contact indicates a low level of sedation. Poor eye contact is indicative of a deeper level of sedation.
    - **b.** Patient's speed of answering. Appropriate response time indicates less sedation; a delay in response indicates a deeper level of sedation.
    - **c.** Patient's language
        - i. Slurred speech. Clear annunciation indicates less sedation; slurred speech, a deeper level of sedation.
        - ii. Use of "ums" and pauses. Clearly timed speech indicates less sedation; ums and pauses indicate a deeper level of sedation.
        - iii. Volume of speech – is it hard to hear them? Appropriate volume indicates less sedation; decreased volume indicates a deeper level of sedation.
    - **d.** Did the patient give you a number in response to the "scale" question? You cannot rely on the number given as the primary indication of the level of sedation. You must take into consideration all the listed observation factors before making a determination. If you must insist on a numbered response from the patient, this also indicates a deeper level of sedation.
    - **e.** Did the patient ask you to repeat the scale? This indicates confusion and a deeper level of sedation.
    - **f.** Patient's physical posture. If the patient is sitting upright and erect this indicates a lighter level of sedation. If the patient leaning or seeking a supine position, this indicates a deeper level of sedation.
4. **Based on your observations of the patient, determine if additional medication is required.**
5. **Before administering additional medication, ask the patient if they would like to be more sedated.** If the patient says "no", do not deliver additional medication. The patient may be comfortable at this lighter level. As always, we must ensure the use of the lowest dosage possible.

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6. **If you have determined that additional medication is required (and the patient said "yes"), then you must determine the appropriate dose according to the following:**
    - **a.** 0.5 mg Triazolam or 3 mg Lorazepam would be appropriate when the patient responded clearly, quickly and appropriately to the "scale" question. Eye contact was good and posture is erect. There is no evidence of confusion (substitute 3 mg Lorazepam for protocols #3 & 4 and add 50mg Hydroxyzine for protocols #2 & 4).
    - **b.** 0.25 mg Triazolam or 2 mg Lorazepam would be appropriate when the patient's response is clear but slightly delayed. They have moderate eye contact and their volume may be slightly decreased and may respond to the "scale" question without using numbers. The patient's posture is relaxed but self-supporting (substitute 2 mg Lorazepam for protocols #3& 4 and add 50mg Hydroxyzine for protocols #2 & 4).
    - **c.** 0.125 mg Triazolam or 1 mg Lorazepam would be appropriate when the patient's response is slightly slurred and delayed. They may ask you to repeat the "scale" question. Eye contact may be inconsistent and the patient may begin to lean (substitute 1 mg Lorazepam for protocols #3& 4 and add 50mg Hydroxyzine for protocols #2 & 4).
    - **d.** No additional medication would be appropriate when the patient's response is slurred and delayed. The patient may be very quiet and use "ums". Confusion is evident. No eye contact is made and the patient leans and seeks a supine position.

REASSESSMENT <u>AFTER</u> ADDITIONAL MEDICATION (See Protocol 8:35AM)

1. **Sit at eye level with the patient.**
2. **Ask the patient to rate their level of sedation *<u>by turning the scale upside down to test for confusion</u>*.** "Mary, on a scale from 1 to 10, where 1 is your totally awake (like you are ready to go Christmas shopping), and 10 is that you are totally relaxed (like don't bother me please!), where are you on that scale?" Using the individual patient's social history, construct the above questions to best relate examples of a relaxed or excited state.
3. **Observe the following:**
    - **a.** Patient's ability to make eye contact. Good eye contact indicates a low level of sedation. Poor eye contact is indicative of a deeper level of sedation.
    - **b.** Patient's speed of answering. Appropriate response time indicates less sedation; a delay in response indicates a deeper level of sedation.
    - **c.** Patient's language:

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I. Slurred speech. Clear annunciation indicates less sedation; slurred speech, a deeper level of sedation.
II. Use of “ums” and pauses. Clearly timed speech indicates less sedation; ums and pauses indicate a deeper level of sedation.
III. Volume of speech – is it hard to hear them? Appropriate volume indicates less sedation; decreased volume indicates deeper level of sedation.

d. Did the patient give you a number in response to the “scale”? question? You cannot rely on the number given as the primary indication of the level of sedation. You must take into consideration all the observation factors before making a determination. If you must insist on a numbered response from the patient, or they did not catch the scale being switch around, this also indicates a deeper level of sedation.

e. Did the patient ask you to repeat the scale? This indicates confusion and a deeper level of sedation.

f. Patient’s physical posture. If the patient is sitting upright and erect this indicates a lighter level of sedation. If the patient leaning or seeking a supine position, this indicates a deeper level of sedation.

4. **Based on your observations of the patient, determine if additional medication is required.**
5. **Before administering additional medication, ask the patient if they would like to be more sedated.** If the patient says “no”, do not deliver additional medication. The patient may be comfortable at this lighter level. As always, we must ensure the use of the lowest dosage possible.
6. **If you have determined that additional medication is required, you must determine the appropriate dose.**
    a. 0.5 mg would be appropriate when the patient responded clearly, quickly and appropriately to the “scale” question with a 9 or 10. Eye contact was good and posture is erect. There is no evidence of confusion (substitute 3 mg Lorazepam for protocols #3 & 4).
    b. 0.25 mg would be appropriate when the patient’s response is clear but slightly delayed. They have moderate eye contact and their volume may be slightly decreased and may respond to the “scale” question without using numbers. The patient’s posture is relaxed but self-supporting (substitute 2 mg Lorazepam for protocols #3 & 4).
    c. 0.125 mg would be appropriate when the patient’s response is slightly slurred and delayed. They may ask you to repeat the “scale” question. Eye contact may be inconsistent and the

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patient may begin to lean (substitute 1 mg Lorazepam for protocols #3 & 4).

d. No additional medication would be appropriate when the patient's response is slurred and delayed. The patient may be very quiet and use "ums". Confusion is evident. No eye contact is made and the patient leans and seeks a supine position.

Patient Dismissal

Important note: all patients should be fully conscious, ambulatory and psychologically oriented before considering dismissing them in the care of a responsible companion.

- Make sure they are oriented X3
- Do not be afraid to use Flumazenil (Romazicon or Anexate) if you are having trouble getting them to respond to verbal commands or the constant physiological monitoring indicates a trend towards non-manageable oxygen desaturation.
- Review postoperative instructions with the responsible companion (see New Patient Packet). Remember to have the companion sign a copy of these instructions.
- Review dental procedures with the patient (when they are completely recovered)
- Have the companion walk with their hand on the patient's elbow for balance
- Remind them to rehydrate
- Have companion drive patient directly home and contact the office as soon as they arrive
- Always be encouraging
- Schedule hygiene instructions appointment the next day

Do Not Dismiss When:

- Patient is sleepy and not ambulatory
- Patient is unable to answer correctly when asked: where they are, what day it is and what is their name (ambulatory x 3)
- Patient is nauseous or vomiting
- Patient is experiencing excessive bleeding
- Patient's vital signs are not stable
- No responsible companion is available to take patient home and attend to their needs.

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Nitrous Oxide Sedation/Analgesia

BENEFITS OF NITROUS OXIDE SEDATION

Nitrous Oxide/Oxygen sedation has many advantages over other forms of anxiety/pain control, while the disadvantages are few in number.

1. **Safety** - Nitrous oxide (N_2O) analgesia/sedation in dentistry and medicine enjoys an impeccable safety record, which has withstood the test of time longer than any other drug.
    a. Gardner Quincy Colton (1864-1897) was the first to study the safety of nitrous oxide, and documented 193,000 cases with no adverse reactions.
    b. Ruben, a Danish researcher, reported 3 million cases in which $N_20/0_2$ was used in the dental office with no adverse reactions.
    c. Niels Bjorn Jorgensen cited 4 million episodes without complications.
    d. Today, more than 24 million surgical procedures are performed each year in the United States, and nitrous oxide/oxygen continues to provide safe, effective analgesia/sedation.

    When used properly, the patient is mildly sedated and responds to verbal commands. Protective defenses such as the cough and gag reflexes remain intact.

    N_2O/O_2 sedation may be used safely with other forms of sedation including oral premedication. The only drug interactions between nitrous oxide and other drugs have to do with accentuation of the side effects of nitrous oxide (CNS depression, nausea and vomiting).

2. **Ease of use** – The equipment used in nitrous oxide delivery is straightforward, as is the administration technique.

    For the clinician, the drug is easily titrated to the level of sedation/analgesia required for the procedure. Titration is the process of precisely administering a drug incrementally to a specific level or endpoint of action. Because titration is easily accomplished with N_2O/O_2, nitrous oxide sedation and analgesia is more efficient and less likely to result in over-sedation.

    Due to its ease of use and wide margin of safety, minimal training for nitrous oxide delivery is required.

3. **Effectiveness** – In addition to being used to induce general anesthesia in combination with other anesthetic agents, many health disciplines use N_2O/O_2 sedation alone as an effective means of alleviating patient anxiety and mild discomfort during ambulatory and outpatient procedures.

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- **Pain Relief** – While the level of pain control afforded by N_2O can vary from patient to patient, the analgesic properties of N_2O are well documented. Multiple studies report that a mixture of 20%-50% N_2O has the same analgesic efficacy as 15 mg of morphine. These analgesic properties are useful in alleviated the severe pain associated with myocardial infarction.
- **Anxiety Relief –** N_2O/O_2 sedation can significantly impact fear and anxiety by producing sedation and/or a sense of well-being. Sedation enables the patient to become calm, relaxed, and able to tolerate the situation better and with less difficulty. This relaxed feeling has a positive effect on the patient's pain threshold.

Nitrous oxide is especially effective when combined with other forms of pain and anxiety control. Such an example is N_2O/O_2 and local anesthesia. Local anesthesia and N_2O/O_2 combined offer a superior pain and anxiety management alternative compared to either alone.

4. **Quick onset of action and recovery –** N_2O/O_2 sedation has a rapid onset of action, with clinical effects often beginning within 30 seconds, and peak effects usually occurring in less than 5 minutes. Upon termination, N_2O/O_2 is quickly and easily reversed. It is virtually completely eliminated from the body with 100% pure oxygen for a minimum of 5 minutes after termination of the drug. Upon conclusion of recovery, cognitive function returns completely back to normal.

5. **Amnesic properties –** After N_2O/O_2 sedation/analgesia, patients often remark how quickly time passed during a long procedure, nor can they recall the severity of their pain or anxiety or its duration. The passage of time tends to become unclear or compressed with nitrous oxide use.

6. **Acceptance –** Patient acceptance of N_2O/O_2 use is well documented. In general, patients view its use as pleasant and effective.

7. **Few side effects –** Nitrous oxide has very few side effects, all of which are mild when used properly. These include CNS depression and nausea and vomiting. In addition, N_2O/O_2 sedation analgesia may be advantageous when other sedative/analgesic drugs are contraindicated (i.e. allergy).

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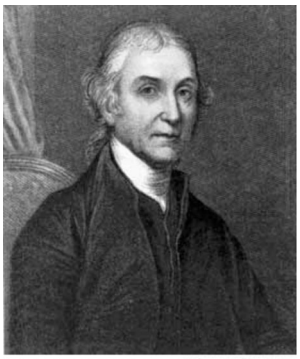
HISTORY OF NITROUS OXIDE SEDATION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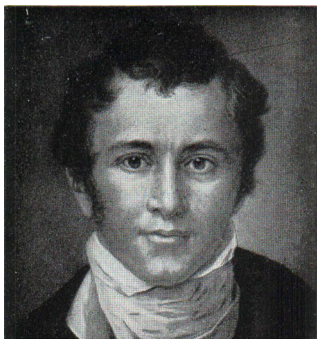
Sir Joseph Priestley is credited with the discovery of N_2O, somewhere between 1771 and 1777. During his experiments with nitrous oxide, he became aware of another gas, which he called "good air", and he found it "fit for respiration". This gas was to be known as oxygen.

In 1798, Sir Humphrey Davy became the first to chronically inhale pure N_2O. At the time N_2O gas was thought to be the cause of dangerous diseases. Davy found the experience very pleasurable however, and felt like laughing. Davy published the results of his experiments in 1800.



Sometime later, Davy experienced diminished pain from a toothache while using N_2O and began to believe it could affect pain sensations. He had no idea of the significance of this experience.

For the next forty years, experimentation with N_2O continued, but mostly as a novelty at exhibitions for entertainment purposes. N_2O use became a trendy activity at social events and in university settings. The future firearm magnate Samuel Colt, posing as a physician at age nineteen, gave street corner sideshows featuring N_2O. Such shows were advertised as evening entertainment in which participants could "laugh, sing, speak, or fight."

Meanwhile the medical community was hungry for pain relief for commonly performed operations like amputations, tooth extractions, and draining abscesses. Mercifully, these surgeries were often completed in less than 90 seconds, but for more lengthy procedures, patients would endure tormenting pain, and would commonly pass out due to exhaustion and/or shock.

Available forms of anesthesia at that time were time-consuming, unpredictable, and at best mildly effective. These consisted of alcohol, opium, tourniquets or ice. Rather than endure necessary yet excruciatingly painful surgery, patients sometimes committed suicide.

On December 10 1844, Horace Wells, a dentist, attended an exhibition in Hartford, Connecticut, given by "Professor" Gardner Colton, to demonstrate

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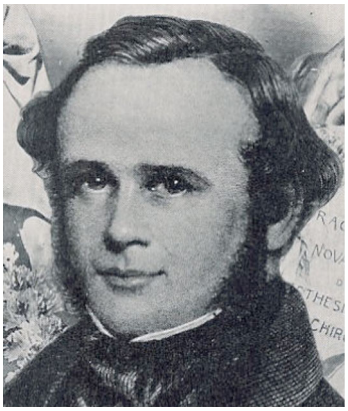
the exhilarating effects of inhaling N_2O. Although Dr. Wells volunteered to try the gas on stage himself, it was another participant, Samuel Cooley, who would inspire Dr. Wells. While under the influence of the gas, Cooley injured his leg, causing a deep, bloody laceration. He was unaware of the extent of the injury and professed that he did not feel any pain.



Wells was intrigued by the lack of pain felt by Cooley and wondered aloud to Colton whether a tooth could be removed under the influence of N_2O. Colton agreed to bring a supply of gas and meet Wells at his office the next day. There Wells breathed the gas himself while a colleague extracted one of his teeth. Wells exclaimed it "the greatest discovery every made," and "a new era in tooth pulling".

Wells used N_2O anesthesia for extractions on several patients with great success. Anxious to demonstrate the use of N_2O anesthesia to the medical community, Wells was given such an opportunity in front of several physicians and students in Boston in 1845. It was during that exhibition that the patient Wells was extracting a tooth on jumped in the chair, leading observers to believe he was in pain (some time later the patient indicated he did not experience any pain).

As a result of the seemingly failed demonstration, Wells was labeled a "charlatan" and a "fake". Obsessed with proving the legitimacy of his techniques, Wells alluded to in his writings to being severely depressed over the debacle in Boston.

Present in the audience during the demonstration in Boston was William Morton; a dental student of Wells who would later became a physician. The experience would inspire Morton to search for a more potent anesthetic gas, eventually culminating in his work with ether.

In 1846, Morton performed a surgical demonstration with ether in the Massachusetts General Hospital, later called the "ether dome". Morton's experiment was similar to Wells' in terms of success; the patient stated that he felt the procedure as it was performed. However, the demonstration was well received by the audience primarily because of Morton's status as a physician rather than a dentist.

As a result, surgical procedures using ether grew in popularity. Other individuals claiming to be the first to discover anesthesia with ether led Morton to fight for recognition. Tragically, Morton would die in 1848 without being officially recognized for his accomplishments.

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In 1847, English obstetrician James Simpson began to use chloroform rather than ether as an analgesic for labor pains (due to the odor and tendency for nausea and vomiting produced with ether), despite others' claims of negative consequences. Chloroform continued to be a major anesthetic agent into the 1860s and became the standard issue to soldiers who, when injured in battle, could self-administer it.

Meanwhile, Wells continued to seek credibility and be recognized for his accomplishments. Although addiction was poorly understood at that time, Wells had become a chloroform addict. Inhaling chloroform contributed to his mental decline and, while in a chloroform-induced stupor, Wells threw acid on a prostitute in New York City and was imprisoned in 1848. He asked to retrieve some personal items from his home and returned to prison with a razor and some chloroform. He wrote his last words to his wife and committed suicide by cutting his femoral artery while under the influence of the drug. His suicide on May 30, 1848, ended his life at the age of 33. Wells was unaware of the recognition that would soon be credited to him naming him the "Father of Anesthesia".

The same Gardner Quincy Colton who supplied Wells with N_2O in 1844 was responsible for its resurgence in the early 1860s. He documented 193,000 cases with no fatalities.

In 1868, Dr. Edmund Andrews suggested that when 100% N_2O is used, the blood is not appropriately oxygenated, and that one fifth of the volume should be O_2. In that same year, Paul Bert developed equipment to deliver both O_2 and N_2O simultaneously to a patient.

Dentistry continued to be the primary health discipline to use N_2O into the 20th century. However, its use was sporadic due to the limitations of equipment, as well as lack of knowledge of the technique.

Other anesthetic agents would later become the drugs of choice for general anesthesia such as cyclopropane (discovered in 1929). IV agents didn't become popular as general anesthetics until the introduction of sodium thiopental in 1935.

Curare was used for light levels of sedation in the 1940s in combination with nitrous oxide in high-risk patients to provide muscle relaxation without undue risk to the cardiovascular system.

The discovery of lidocaine in the 1940s temporarily diminished the use of nitrous oxide until a later time when N_2O use resurfaced to assist with the management of anxiety.

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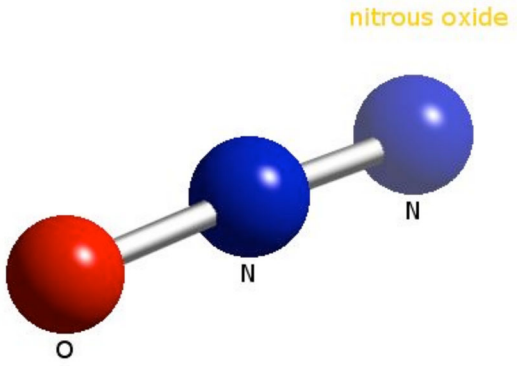
A study by Goff in 2003 indicates that approximately ¾ of general dentists provide nitrous oxide analgesia/sedation.

In medicine, nitrous oxide remains functional and commonly used for general anesthesia as a way to provide rapid induction of other, more potent agents.

PHYSICAL & CHEMICAL PROPERTIES

Nitrous Oxide (Dinitrogen Monoxide - N_2O) is a sweet smelling, colorless gas. When compressed into a cylinder, it becomes a liquid. Its specific gravity is 1.53, which indicates that it is heavier than air (specific gravity of 1). Because of this, the greatest conentration of unscavenged gas will be near the floor.



Although nonflammable itself, N_2O *supports* combustion. If the gas were to come in contact with a combustible source at a high temperature and elevated pressure, such as inside a cylinder, an explosion could occur.

PHARMACOKINETICS

Pharmacokinetics describes the passage of drugs from the external environment through the body and the various processes that the body exerts on the drugs. Generally speaking, pharmacokinetics involves absorption, distribution, metabolism, and excretion of drug molecules.

The concentration of a drug within a compartment of the body (such as the plasma) where the drug exerts its action correlates with its onset and duration of action. The drug's **mean peak concentration** is usually associated with the height of its action, and its **half-life** affects how long the drug remains in the body or a body compartment. All of these terms, plus more, can be depicted over time in the ***Time-Concentration Curve*** (See Figure 1 next page).

Onset time – The time from initial drug administration until the MEC is reached. In other words, how long it takes for the drug to work (also called onset of action).

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C_{max} & t_{max} – C_{max} is the maximum concentration from a dose of a drug in a compartment. The t_{max} is the time it takes to reach that maximum concentration. Where these two numbers intersect on the Time-Concentration Curve is the **Mean Peak Concentration**, and usually correlates to the height of the effect of a drug.

MEC (Minimum Effective Concentration) - The concentration of a drug in a compartment at which therapeutic effects first appear.

MTC (Maximum Therapeutic Concentration) – The concentration at which therapeutic effects stop increasing with increasing concentration.

Therepeutic Range – The concentration range of a drug in a compartment between the MEC and the MTC.

Duration of action – The range of time after a dose of a drug is administered where the concentration of the drug in a compartment is sufficient enough to cause therapeutic effects. On the Time-Concentration Curve, it corresponds to the time the drug concentration exceeds the MEC.

AUC (Area Under the Curve) – Represents the time and concentration of the drug in a compartment. This is the shaded area outlined by the Time-Concentration Curve.

Half-Life - The time at which half of a drug has been eliminated from the body compartment being considered. Drugs are eliminated from compartments through re-distribution to other compartments, metabolism, and elimination. Drugs most often are not eliminated from a body compartment in a linear fashion (zero-order kinetics), but rather eliminated relative to the remaining concentration (first-order kinetics). This explains that while 50% of a drug is eliminated in one half-life, it often takes four half-lives for 90% of a drug to be eliminated.

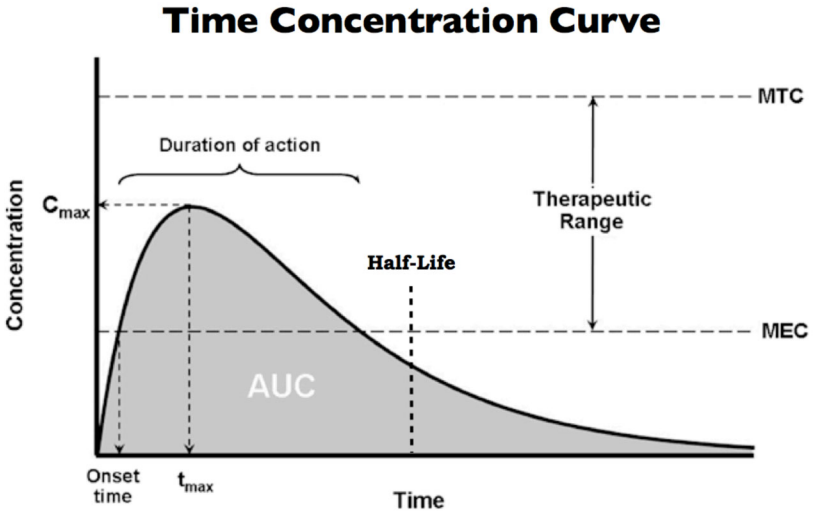

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Figure 1. Time Concentration Curve.

The effect of a drug relative to its dose can be illustrated by the ***Dose-Response Curve*** (See Figure 2 below). In this curve, the "no-effect range" is analogous to the onset time and MEC of the Time-Concentrative Curve. As the dose increases, so does the effect, up until the maximum effect (aka ceiling effect) is reached. At this point, the therapeutic effects no longer increase with increasing dose. However, undesirable side effects may continue.

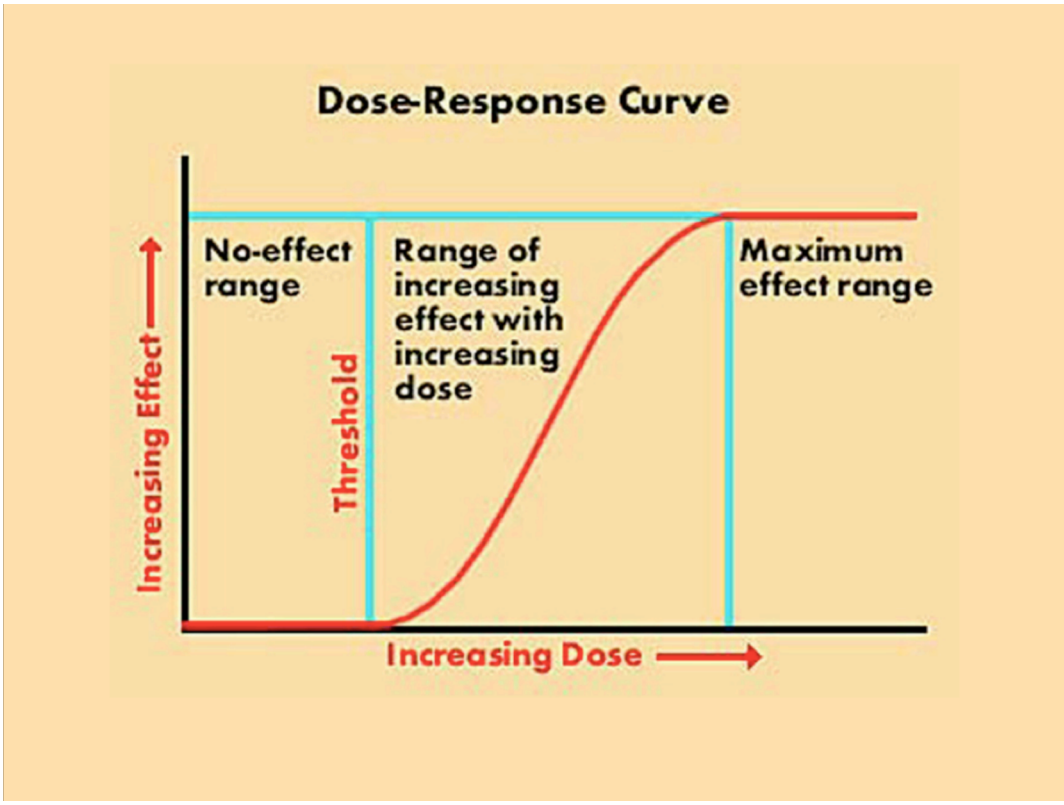

Figure 2. Dose-Response Curve

Absorption & Distribution

Inhaled gases such as N_2O enter and move around the body through partial pressure gradients (See Figure 3, next page). There are two dimensions to these gradient differences – degree and direction. Gases move in a higher to lower pressure direction. The degree refers to the magnitude of the pressure difference – the greater the difference, the more efficient the transfer. This same mechanism accounts for the passage of gases necessary for normal respiration.

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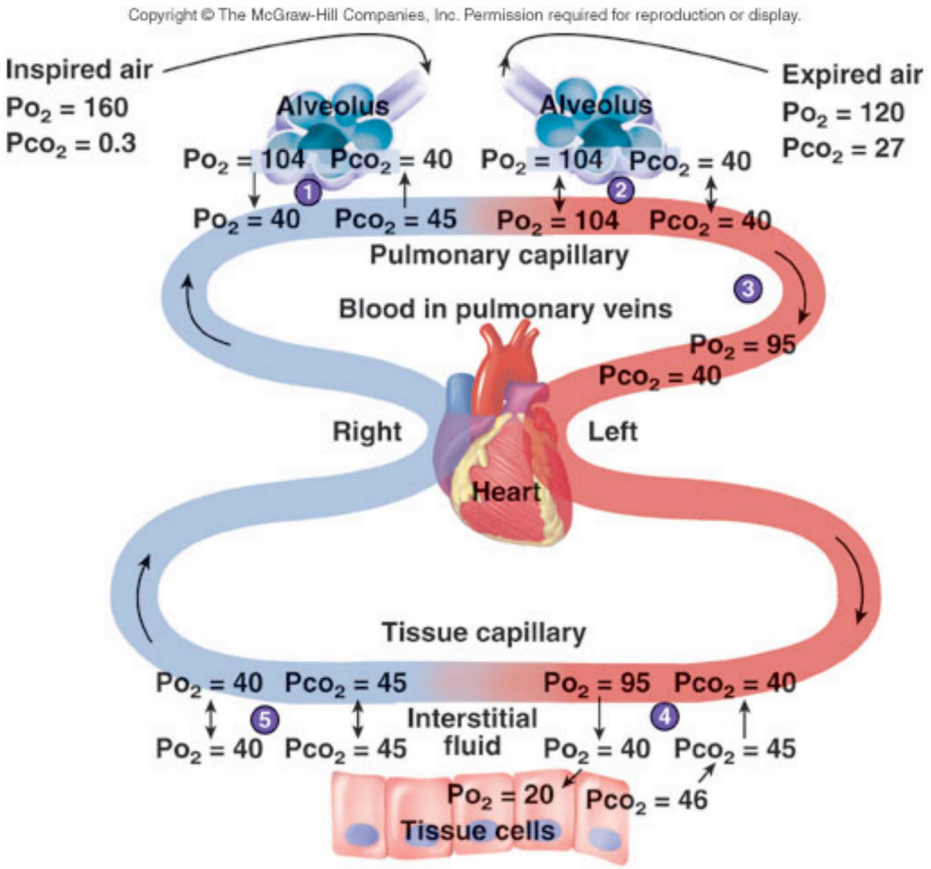

Figure 3. Gas Movement Related To Partial Pressures In The Body.

The difference between the partial pressures of N_2O in the lungs and the pulmonary blood circulation dictate how quickly the nitrous oxide crosses the pulmonary membrane and enters the bloodstream. N_2O crosses the alveolar membrane easily. The solubility of the drug then determines how quickly equilibration occurs, and clinical action begins.

If a drug is highly insoluble, it will reach equilibrium rapidly, cross the blood-brain barrier quicker, and reach the brain faster. Therefore, the more insoluble a drug is, the quicker it works. Consider the following example:

Imagine two cups of warm water: Into one cup you put a spoon of sugar and into the other a spoon of sand. Which will be in higher concentration in the bottom of the cup? The sand, of course, since it is insoluble in water. The sugar dissolves, so very little reaches the bottom. Eventually, the water will become saturated with sugar, and sugar will collect in the bottom of the cup. For the purposes of the subject of this discussion, bottom of cup, read brain.

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The interaction between a drug, the brain, and other tissues until equilibrium is reached is expressed in values called partition coefficients. These values indicate the efficiency of drug transfer to various body compartments.

The Blood-Gas Partition Coefficient (BGPC) is calculated by the amount of gas needed to saturate the blood divided by the amount of gas (See Table 4 below). If the blood-gas partition coefficient is high, more drug and/or more time will be necessary to achieve equilibration or the level necessary for movement of the agent to the brain. In the example cited above, the partition coefficient of sand is low compared to sugar.

Drug	BGPC	Onset (Mins)
Desflurane	0.42	1-2
Nitrous Oxide	0.47	1-2
Sevoflurane	0.69	2-3
Isoflurane	1.4	3-5
Ether	12	15-20

Table 4. Blood-Gas Partition Coefficients Of Some Inhalation Anesthetics.

N_2O is an example of a relatively insoluble drug. Its blood-gas partition coefficient is 0.47. It remains unchanged in the blood and does not combine with any blood elements. Uptake by the body is limited, resulting in equilibrium being achieved quickly. This explains why the peak clinical effects of nitrous oxide may be seen within 3-5 minutes after administration.

Metabolism & Elimination

Nitrous oxide is not metabolized in the liver as many drugs are. Ninety-nine percent of it is eliminated through the lungs without biotransformation. A very small (0.004%) amount of N_2O is metabolized in the gastrointestinal tract by anaerobic bacteria, resulting in potentially toxic free radicals. This minute amount does not pose any significant threat to the body.

The partition coefficients of N_2O between tissues, muscles, and fat are low. Equilibration occurs quickly in these tissues due to the inability of the tissues to hold N_2O. Because of this, N_2O is not stored in the body to any extent resulting in rapid and complete elimination at the end of administration.

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PHARMACODYNAMICS

Pharmacodynamics describes the action of a drug - the mechanisms of chemical and physical transformation at the effector site within the organism. The complete mechanism of the pharmacodynamics of nitrous oxide is yet to be determined.

Findings to date indicate that N_2O induces opioid peptide release in the brain stem leading to the activation of descending noradrenergic neurons, which results in modulation of the nociceptive process in the spinal cord. In addition, several receptor-effector mechanisms including dopamine receptors, alpha-2 adrenoreceptors, benzodiazepine receptors (GABA) and N-methyl-d-aspartate (NMDA) receptors have been implicated in the pharmacodynamic action of N_2O, although the relationship of one with the other is not known.

The potency of an anesthetic agent reflects the relative amount of drug necessary to achieve a defined action. For gaseous agents, drug potency is measured by its minimum alveolar concentration (MAC). MAC is defined as the amount of drug necessary to prevent movement in 50% of subjects responding to a surgical incision.

Nitrous oxide is the least potent (weakest) of all inhalation anesthetics (See Table 5 below). The MAC for N_2O is 103%. This value indicates that at normal atmospheric pressure N_2O *alone* is not able to produce profound surgical anesthesia.

Drug	MAC (%)
Nitrous Oxide	103
Desflurane	7.3
Sevoflurane	2.1
Ether	1.9
Isoflurane	1.2

Table 5. Minimum Alveolar Concentrations Of Some Inhalation Anesthetics

An inhalation anesthetic's MAC, may or may not be affected by various factors:

Factors that will not affect the MAC:

1. Gender
2. Time (MAC after 10 hours is the same as during the first hour)
3. pH or pCO_2
4. Elevations or mild drops in BP
5. Hypothyroidism
6. Chronic Alcoholism
7. Anemia (unless severe)

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Factors that will affect the MAC:

1. Significant hypoxia decreases MAC
2. Increasing age decreases MAC
3. Circadian rhythms alter MAC +/- 10%
4. Benzodiazepines and opioids reduce the MAC
5. Pregnancy decreases the MAC
6. MACs are additive (i.e. 50% N_2O + 50% MAC Desflurane = 1 MAC of anesthetic.)

From a practical standpoint, the desired action from N_2O administration is to decrease anxiety and increase the pain threshold, while remaining within the level of minimum sedation. At this level of CNS depression, the patient responds to verbal commands. The term MAC_{awake} refers to the minimum alveolar concentration at which patients still respond to commands. For N_2O, the MAC_{awake} of N_2O = 0.2-0.6 MAC or 20-60% N_2O.

Nitrous Oxide & Other Gases

The partial pressure of N_2O is approximately 31 times greater than that of nitrogen (N_2). Because of this, N_2O rapidly replaces the N_2 occupying any body space, including the blood. Not only does N_2O rapidly replace N_2, but it also increases the volume and/or pressure of the space formerly occupied by nitrogen.

Higher concentrations of N_2O promote a more rapid uptake of the gas, and there is a simultaneous effect occurring with the other gases being administered. The rapid uptake of N_2O allows the second gas to be drawn in much faster than it would normally if it were being administered alone. This is because the rapid uptake of N_2O results in an increased concentration of the second gas in the alveoli. This phenomenon, called the "second-gas effect", allows minimal amounts of a more potent anesthetic to be administered simultaneously with N_2O.

Nitrous Oxide & Altitude

Extreme altitudes above 10,000 feet do affect the use of N_2O (See Figure 6). Because of the barometric pressure change, those administering N_2O/O_2 sedation at significant elevations must be aware of the need for an increase in N_2O concentration to obtain the same effects as at sea level. In Denver, Colorado, where the elevation is 5,280 ft. above seal level, a 5% increase in N2O may be necessary relative to llocations at seal level.

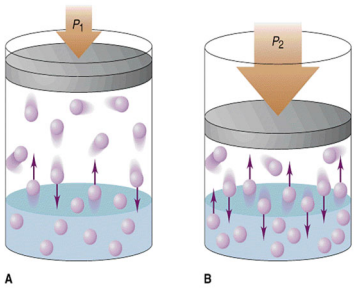

Figure 6. Differences In Altitude Above 10,000 (Sea Level) Cause Different Densities Despite Similar Concentrations.

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MANUFACTURE & DISTRIBUTION OF NITROUS OXIDE

The raw ingredient in the process of manufacturing nitrous oxide is ammonium nitrate (NH_4NO_3), the same ingredient commonly used for fertilizer and a primary ingredient in explosives.

The manufacturing process involves heating ammonium nitrate to approximately 250 degrees Celsius, at which temperature it separates into N_2O, water in the form of steam, and some contaminants. This mixture is allowed to cool and the water is extracted. The contaminants are removed, the gas is compressed, then dried to remove any remaining water. The resulting product is stored as a liquefied, compressed gas at 300 psig (pound-force per square inch gauge) at 4 degrees Fahrenheit in insulated and refrigerated storage tanks. The product is kept refrigerated until insulated tanker trucks directly transfer it to storage facilities or to other gas distributors.

Approximately 90% of the N_2O produced is used in health settings. Dental ambulatory clinics use ~ 10% of the N_2O produced. The food industry uses approximately 7% as a propellant for dairy products such as whipped cream. Nitrous oxide produces a whipped cream that is four times the volume of the liquid. Nitrous oxide, being lipophilic, also inhibits the degradation of the fatty cream, whereas if pressurized air were used, the oxygen would cause the cream to become rancid.

The remaining users of nitrous oxide include the chemical industry, in which nitrous oxide is used in the production of sodium azide, the explosive agent that inflates an automobile air bag.

Nitrous oxide is also used to increase engine performance in the racing industry.

The major manufacturers of N_2O in North America are: Airgas Puritan Medical in Radnor, PA; Air Liquide America, Houston, TX; and AGA, Mexico City, Mexico.

Regulation & Control Of Nitrous Oxide

The N_2O industry is regulated by the Food and Drug Administration (FDA), which has established practices and requirements with which companies who produce and package gases must comply. The U.S. Department of Transportation (DOT) oversees the transport of the gas.

Nitrous oxide is contained in storage cylinders of varying sizes, each imprinted with information including the origin and age of the cylinder and inspection compliance. How long a storage cylinder remains in circulation depends on its physical condition. It is not uncommon to see cylinders that are 70 years old in distribution. These cylinders are color coded for easy identification, however the most accurate description of cylinder contents is the cylinder label. The following table

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demonstrates the designated colors for nitrous oxide and oxygen cylinders in eleven different countries.

Country	Cylinder Color for N_2O	Cylinder Color for O_2
Argentina	Blue	White with green cross
Australia	Blue	White
Canada	**Blue**	**White**
China	Gray	Blue
India	Blue	Black with white shoulders
Germany	Gray	Blue
Japan	Black	White
South Africa	Blue	Black with white shoulders
Sweden	Blue	White
United Kingdom	Blue	White
United States	**Blue**	**Green**

It is important to remember that under no circumstances should grease, oil, or any other lubricating substance come in contact with the gas or gas delivery equipment. If the hydrocarbons in such materials were to catch fire and then contact the pressurized N_2O, an explosion could occur.

There are specific requirements regarding where cylinders can be stored, as well as ventilation and fireproofing of the storage area. In addition, cylinders should be secured in a place that is not readily accessible or visible to others.

A full N_2O cylinder contains approximately 95% liquid and 5% vapor. Liquid N_2O in the tank is vaporized by room air temperature outside the tank as the gas is used. Because of this heat transfer process, the tank becomes cool to the touch. Frost may be seen on the tank surface during prolonged and continuous use.

A full cylinder of N_2O will have a pressure gauge reading of approximately 750 psi at 70 degrees Fahrenheit. At lower temperatures, the cylinder pressure will be proportionately less.

Because the liquid is vaporized as the gas is used, this reading is not proportional to the actual amount of gas available in the cylinder. The amount of liquid remaining in the tank is enough to maintain a pressure of 750 psi until the liquid volume reaches 20% of the tank. Therefore, the gauge will not show a pressure decrease until the tank contains approximately 20% N_2O. The following diagram compares the difference in pressure readings versus cylinder volume between nitrous oxide and oxygen:

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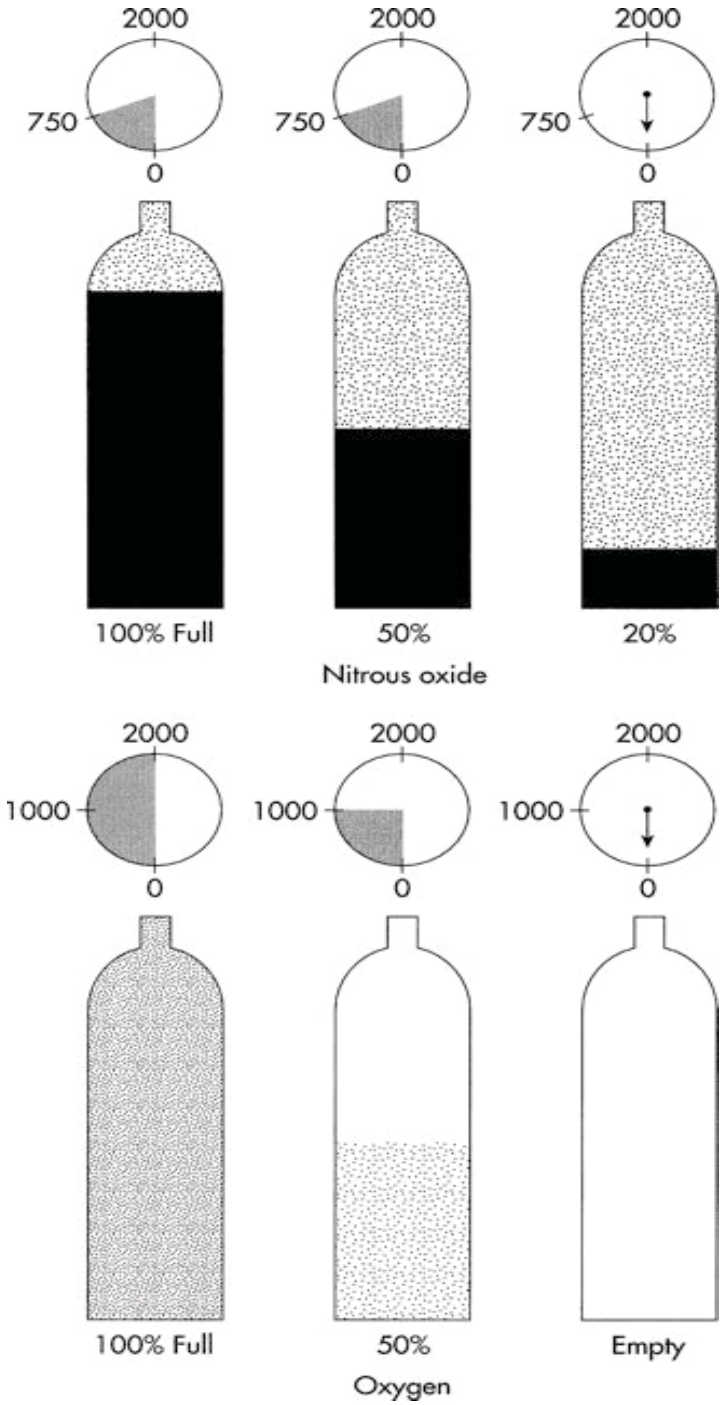

When the O_2 cylinder is full, the amount of pressure is approximately 2000 psi. The pressure gauge for O_2 will accurately indicate the amount of gas present in the cylinder at all times until the cylinder is exhausted. Because the N_2O/O_2 sedation machine is driven by O_2 flow, it is important to monitor the amount of O_2 left in the cylinder while it is in use. If the O_2 tank becomes empty during a procedure, there will be no flow of N_2O, and the sedation experience will be interrupted. Therefore, it is important to have additional O_2 cylinders available to prevent this occurrence.

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Estimating Time Left In Tanks

The formula for estimating the length of time remaining from a tank at a constant flow of liters/minutes is:

(PSI X F)/Liters per minute = Time remaining in minutes

Where PSI is the remaining tank pressure, "F" is a constant depending on the size of the tank (0.3 for E cylinders and 3.0 for H cylinders", and Liters/minute is the flow rate for the gas in question

Example: If you have a flow of 10 liters/minute with 1000 PSI remaining in an E cylinder:

(1000 X 0.3)/10 = 30 minutes remaining

A Rule of Thumb for estimating time per full tank is, given the fact that an H tank contains ~ 6900 liters of O2, and an E tank contains ~ 680 liters of O2:

At 6 L/min flow rate, H tank lasts 19.2 hours, while an E tank lasts 1.9 hours

This calculation will work for estimating the remaining time left for a tank of oxygen regardless of how much oxygen is left in the tank. However, it is only practical for estimating the remaining time left in a nitrous oxide tank when the tank is full.

Nitrous Oxide Delivery Equipment

N_2O/O_2 sedation equipment has evolved dramatically since its discovery.

- Sir Frederick Hewitt in 1887 was the first to incorporate oxygen with N_2O into a machine that delivered both gases.
- Early machines were designed as intermittent (on demand) flow.
- A pivotal point in the progression in the technology of N_2O/O_2 delivery was the addition of the fail-safe O_2 mechanism in the mid-1970s. This device prevented the delivery of 100% N_2O and ensured that no less than 21% O_2 would ever be delivered to the patient. Today the minimum percentage of O_2 that can be delivered is 30%.

A central N_2O/O_2 supply system can be used when a large quantity of gas is supplying several units. Providers may prefer this system because of the frequency of N_2O/O_2 use, cost savings, convenience, and space saving.

The central system manifold connects several large cylinders of gas together and allows for the transfer of gas supply from one tank to another when the previous tank is emptied. The newest manifolds have several safety features built into the system, including pressure-relief valves to exhaust any gas higher than 75 psi, and

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alarm systems that alert the staff when the pressure of the gas falls below 45 psi or becomes greater than 60 psi.

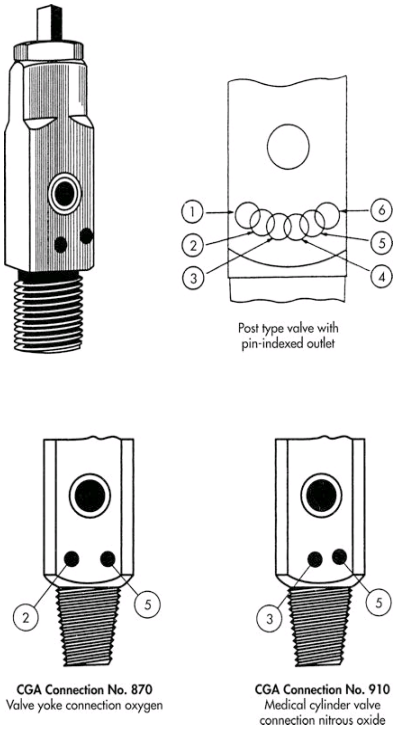
All gas from the manifold must pass through pre-cleaned, degreased copper tubing, and all connections must be silver-soldered at 1000 degrees Fahrenheit. The piping system must be pressure tested with air for 24 hours at no less than 150 psi before patient use. The copper tubing for the delivery of O_2 is 1/2-inch while 3/8-inch tubing for N_2O is the standard. The difference in tubing size prohibits the inadvertent crossing of lines.

For the gas to be dispensed safely there must be a means of reducing the amount of gas pressure from the cylinder. A pressure-reducing valve or regulator is necessary to ensure safe delivery of gas through the equipment and to the patient. The high pressures of 750 psi in the N_2O tank and 2000 psi in the O_2 tank are reduced to lower pressures of approximately 50 psi.

A portable gas delivery system can also be used when N_2O/O_2 sedation is not used frequently. The unit houses smaller tanks and may be moved easily from room to room.

The yoke stand of the portable machine is the backbone and supporting structure on which the equipment rests. Some stands hold two tanks, whereas others hold four.

Metal pins, specifically arranged, protrude from the yoke onto which the cylinder is attached. This precise method of attachment is designed to prevent the incorrect cylinder attachment to the yoke and is known as the *pin index safety system or Pindex Safety System*.



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Regulators on the portable systems are similar to those on central delivery systems. Hoses connect the regulators to the flow meter, and are color coded for the gases and differ in size and threaded-end connections to prevent improper gas flow to the machine.

The flow meter is a calibrated device that sits on top of the yoke assembly, mounted to the wall, or located within the cabinetry. The flow meter indicates the amount of gas being delivered to the patient.

One of the most important safety features of the sedation unit is the flow meter's fail-safe mechanism. A valve opens to allow N_2O flow only when there is flow of O_2 to the system. Any time the O_2 flow is less than 30%, N_2O stops flowing.

The reservoir bag serves four purposes: Its primary purpose is to provide a source of additional gas should the patient inspire more gas than is being supplied through the hoses. Typically a reservoir bag holds approximately 3 liters of gas.

In addition, the reservoir bag provides a mechanism for monitoring the patient's respiration.

It also provides a means for assessing the proper flow rate for each patient.

The reservoir bag also functions in an emergency as a method of providing positive-pressure O_2 to the patient. The bag is gently squeezed to empty its contents into the pulmonary tree; the action is similar to a manual-resuscitator bag.

Gas is delivered through tubing or hose stemming from the unit and attached to the breathing apparatus.

Gas is administered to the patient through a full-face mask or nasal hood. Nitrous oxide and oxygen in the dental outpatient setting is typically delivered via a nasal hood. All currently manufactured nasal hoods are latex free, whereas older products may not be.

The nasal hood is designed to fit snugly over the patient's nose so that gas does not leak out the sides. They are available in various sizes and scents. Many current models of nasal hoods are designed for single patient use, and are not sterilizable. Other reusable versions of the hoods are also available but must be sterilized between patients.

All nasal hoods should have scavenging capabilities. Scavenging nasal hoods are designed to provide fresh gas to the hood for the patient through one or two hoses while one or two additional hoses evacuate gas being exhaled by the patient. These hoses are connected to a vacuum system that exhausts the gases out of the facility.

Manufacturers recommend servicing sedation units periodically. Each company has specific time frames for maintenance of its units. Servicing a unit includes procedures such as recalibration, pressure testing, internal component checking and replacement, as well as other factory testing procedures.

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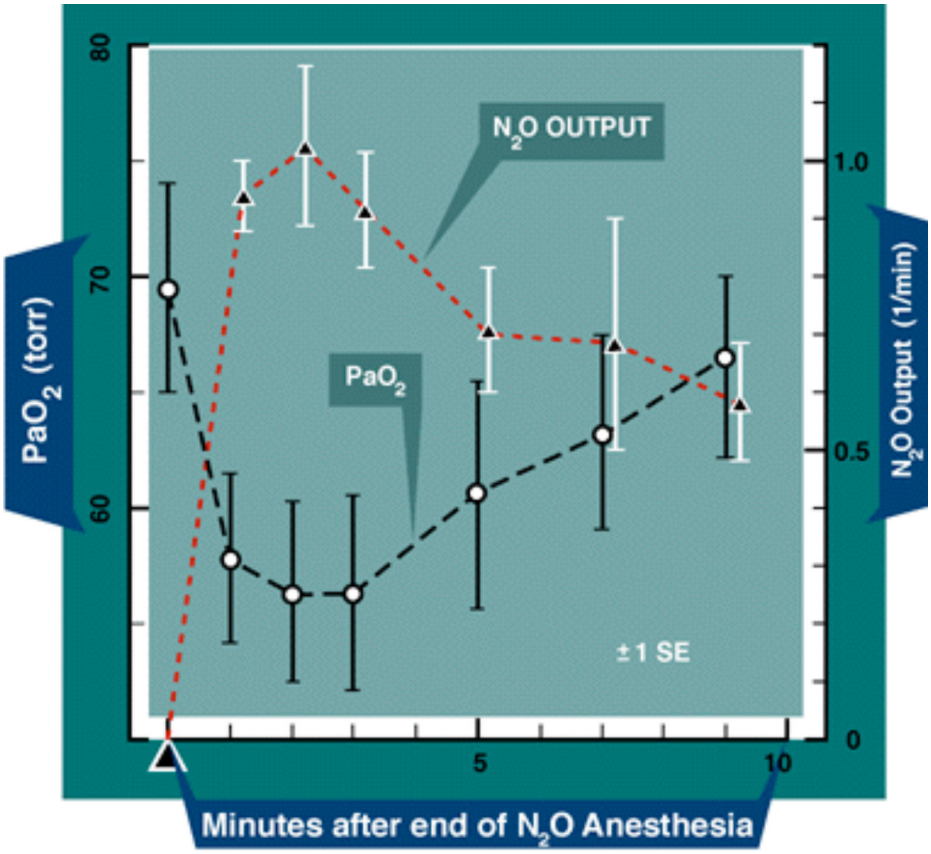
Diffusion Hypoxia

It has been hypothesized that headache, lethargy, and nausea can occur because of decreased O_2 saturation levels in the blood caused by the rapid exit of N_2O on its termination. The application of 100% pure O_2 for the first 3-5 minutes after N_2O termination has been traditionally advocated to prevent O_2 desaturation of blood.



However, there is research that questions the clinical significance of diffusion hypoxia and whether these symptoms are even associated. Indeed, several researchers have reported no diffusion hypoxia when breathing room air instead of 100% O_2. Despite the doubts raised as to the validity of diffusion hypoxia, it can be argued that it is prudent to administer 100% O_2 postoperatively for a minimum of 5 minutes.

THE EFFECT OF NITROUS OXIDE ON BODY SYSTEMS

When administered with oxygen, nitrous oxide is compatible with human physiology, actually improving a patient's perfusion while decreasing his or her anxiety.

Cardiovascular System

N_2O does not negatively affect the cardiovascular system to produce any significant physiologic changes. The interaction between N_2O and contractility, cardiac output,

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stroke volume, heart rate, and rhythm is minimal, and in the case of cardiac disease, insignificant.

It has been shown that N_2O with O_2 has a positive effect on myocardial ischemia by providing supplemental O_2 and can be very helpful during and right after a myocardial infarction.

Respiratory System

Any condition such as an upper respiratory tract infection that affects air exchange through the nose can result in insufficient amounts of N_2O/O_2 entering the respiratory system and causing its desired effect. In addition, the drying effect of the gas administration may result in mucous plugs that interfere with air exchange, and increased pressure in infected sinus cavities may result in additional discomfort.

Because vomiting during N_2O administration is possible, the potential for aspiration, although unlikely, exists. If the patient is adequately oxygenated, prevented from entering deeper levels of sedation, nitrous oxide is used judiciously (% & time), and follows the pre-sedation guidelines, the risk of aspiration is extremely small.

Nitrous oxide use is not contraindicated in asthmatic patients, because it is nonirritating to mucous membranes. In fact, its sedative nature can reduce the likelihood of an asthmatic event caused by anxiety.

For patients with debilitating respiratory conditions (eg. COPD), the use of N_2O/O_2 may be a relative contraindication. These individuals may be on a hypoxic drive, therefore the richer concentration of oxygen delivered with nitrous oxide may impair their stimulus to breathe. It is prudent to seek a medical consultation with the physician treating their respiratory condition.

Patients with cystic fibrosis or emphysema may present with bullae on the lungs that contain air and fluid. N_2O may irritate these conditions due to the expansive nature of the gas.

Central Nervous System

N_2O is a CNS depressant, although its exact mechanism is unknown. It is known that its effects on cerebral blood flow, velocity, and perfusion pressure are minimal. Its effects on intracranial pressure is less significant than with other inhaled anesthetics, and when used appropriately is clinically insignificant.

Due to the rapid replacement of N_2 with N_2O in air spaces, notable intracranial pressure increases have been found in cases of pneumoencephalography (medical procedure where cerebrospinal fluid is drained and replaced with air, oxygen, or helium to allow investigators to better see the brain on a radiograph). N_2O should not be used for one week after this procedure.

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Chronic exposure to N_2O as in the case of its abuse has shown evidence of CNS injury. This CNS damage manifests itself in the forms of numbness and weakness in extremities as well as ataxic gait.

Hematopoietic System

Megaloblastic and pernicious anemia have been found in patients who have been exposed to high concentrations of N_2O for an extended period. N_2O has been implicated in the interference of the enzyme methionine synthase, an enzyme necessary for DNA synthesis and Vitamin B12 metabolism. Pernicious anemia and megaloblastic anemia are conditions that are associated with this vitamin deficiency.

Gastrointestinal System

Because of its expansive nature and its propensity for infiltrating air spaces within the body, N_2O could adversely affect the non-rigid air space contained in the bowel in the case of bowel obstruction. As a result, it is less desirable to use N_2O/O_2 sedation in such a patient in order to avoid increasing expansion, pressure, and discomfort.

Genitourinary And Reproductive Systems

N_2O/O_2 sedation does not pose any negative effect on the genitourinary system itself, however there are concerns with nitrous oxide administration and pregnancy.

It should be pointed out that the N_2O/O_2 combination has been a commonly used pharmacologic agent in obstetrics. Research confirms its safety with pregnant women during labor and delivery.

Regarding pregnancy and the delivery of dental care, the following considerations regarding N_2O/O_2 sedation should be considered:

Most pharmacologic agents cross the placental barrier; N_2O is no exception. Low levels of oxygen can affect organogenesis during the first trimester.

Although N_2O, when delivered appropriately, should not physiologically threaten the fetus, it can, as in the case of radiation, be blamed should fetal anomalies occur.

Therefore, it is prudent, if for no other reasons, the peace of mind of the patient and to minimize potential liability to the practitioner, to forego the use of nitrous oxide during pregnancy.

If for emergency purposes, the risks are less than the need for N_2O, then N_2O use should be avoided during the first trimester, and appropriate medical consultation should be obtained and documented before any use in the second or third trimesters.

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The chronic exposure of nitrous oxide and its effect on the genitourinary and reproductive systems of dental personnel will be considered later in this workbook.

Neuromuscular System

N_2O/O_2 does not directly affect muscle action, but given in high enough concentrations can cause muscle relaxation secondary to CNS depression.

Cancer

N_2O has never been shown to cause or affect cancerous cell growth. However, the incidence of pulmonary fibrosis and other pulmonary diseases can increase in patients taking bleomycin sulfate, an antineoplastic agent used typically for the treatment of lymphomas, testicular tumors, and squamous cell carcinomas. This occurs indirectly if the O_2 administered with N_2O is administered in concentrations greater than 30%.

Allergies

There have been no known reported allergies to N_2O. Individuals sensitive to latex may experience contact dermatitis from the administration armamentarium. Latex-free nasal hoods, conduction tubing and reservoir bags are available to avoid this occurrence.

Malignant Hyperthermia

Malignant hyperthermia is a genetic condition consisting of extreme muscle hyperactivity, leading to a potentially fatal increase in body temperature after exposure to certain volatile anesthetic agents. Patients who know of familial tendencies and history can be tested to avoid this problem. N_2O/O_2 sedation is not considered a trigger for malignant hyperthermia and can be safely administered to susceptible individuals.

Psychological and other Mental Disorder Conditions

Nitrous oxide itself is a substance that produces euphoria. If the patient is suffering or recovering from addiction or mental illness, the relaxing euphoric sensations from nitrous oxide may exacerbate or trigger undesirable tendencies or encourage addictive behaviors. Discretion in such cases is warranted.

For patients with a mental deficiency such as with Down's Syndrome, it is necessary to determine the ability of the individual to understand the sedation procedure. If that level of understanding is not present or the practitioner is unable to determine it, nitrous oxide/oxygen should not be used. The same rational is appropriate for persons with Alzheimer's disease or autism. N_2O/O_2 sedation can be used as long as the level of understanding is present and there are no other conditions present that would be relative contraindications.

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N_2O/O_2 use should be avoided in patients under the influence of drugs or alcohol. Although alcohol is initially a stimulant, it and other classes of drugs such as barbiturates, opiates, and benzodiazepines are potent CNS depressants. They should never be present when a healthcare provider is administering N_2O/O_2 unless prescribed for a legitimate medical purpose.

Patients under psychiatric and/or psychological care should be carefully assessed before N_2O/O_2 use. Many patients are treated with antidepressants or other psychotropic drugs. It is important to understand the pharmacology of theses drugs and be aware of any synergistic effects. Medical consultation should be considered before N_2O/O_2 administration.

N_2O may enhance drugs that are used directly to induce sleep or that have drowsiness as a side effect. Again, seek medical consultation to determine the extent of the underlying condition. Ensure that the patient has not just recently taken these drugs before N_2O/O_2 administration.

Severely phobic individuals may not benefit from N_2O/O_2 administration, as N_2O/O_2 may not be able to provide adequate relief to accomplish the intended procedure. Deeper levels of sedation that can be accomplished with N_2O/O_2 alone, may be the procedure of choice in these situations.

In some cases, patients with claustrophobic tendencies may feel uncomfortable with the use of a nasal hood. Usually, these patients relax enough during N_2O/O_2 sedation that this is overcome.

Middle Ear Disturbances

Because N_2O infiltrates the rigid, noncompliant cavity of the middle ear, increased pressure and resulting discomfort results. In some cases, significant damage such as tympanic membrane rupture, graft displacement, and other complications have been observed. Also, the negative pressure that results from the rapid departure of N_2O after general anesthesia can cause other side effects, especially after recent ear, nose, and throat complications utilizing higher concentrations than used for minimal sedation.

Eye Surgery

Patients should be questioned about recent ophthalmic surgery before N_2O/O_2 sedation is used. In patients undergoing vitreo-retinal procedures, a gas bubble is typically placed in the eye to assist with the healing process. This gas bubble could expand with N_2O use and complicate healing or promote injury.

Other

N_2O should not be used on patients who have been scuba diving in the last 24 hours to avoid decompression sickness.

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Titration of N_2O/O_2 Gases

Titration is a method of administering a drug in incremental amounts until a desired endpoint is reached. For N_2O/O_2 sedation, N_2O is given in incremental doses until a patient has reached a comfortable, relaxed state of sedation. The ability to titrate N_2O is a significant advantage because it allows the specific amount of drug that is required by the patient to be delivered. If titration is done properly, the patient does not receive any more of the drug than is necessary. Occasionally, patients have had negative experiences with N_2O/O_2 sedation. In such cases, the operator most likely did not titrate properly. Therefore, the concept of titration is the single most important skill to be learned by the sedation clinician to be a competent, safe administrator of any drug by any route, including N_2O (See Figure VI-1).

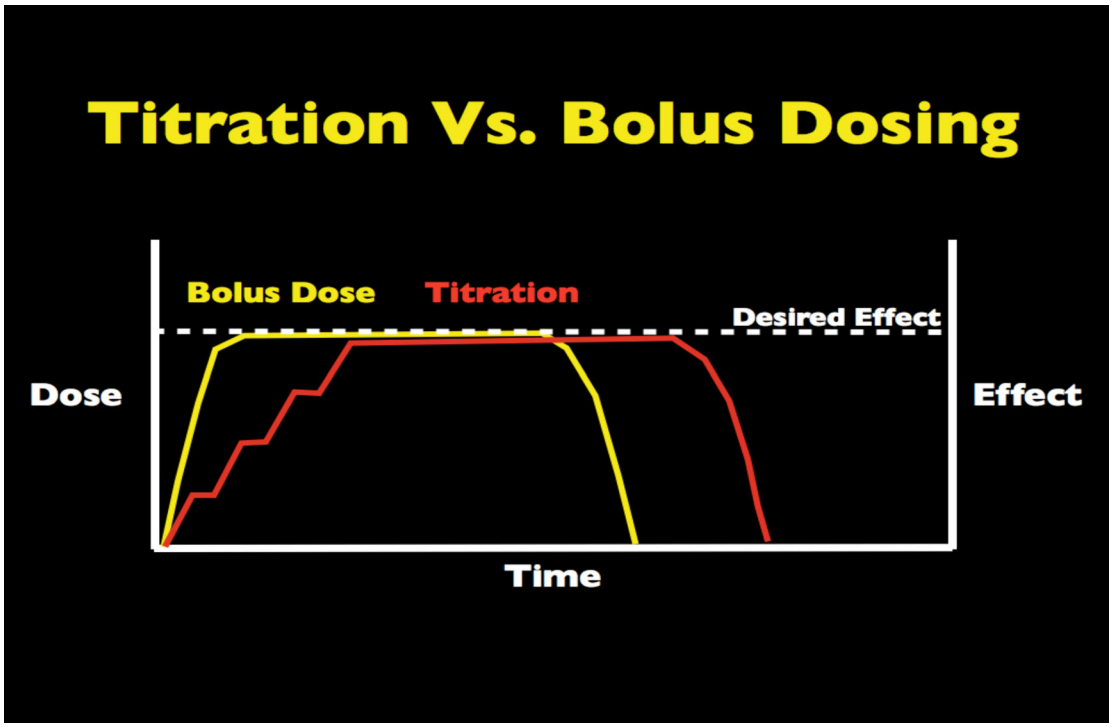

Figure VI-1. Titration Vs. Bolus Dosing.

The "Rapid Induction Technique" for N_2O/O_2 administration has been advocated by some clinicians. This technique involves administering a large quantity of N_2O (up to 50%) initially to patients, particularly children. Advocates of this technique maintain it is necessary to quickly calm an intensely fearful or uncooperative child. This technique in skilled hands can produce favorable results. The rapid induction technique is **not** recommended for adults.

Titration allows for individual variability. It is essential to treat each patient as an individual, taking into account the concept of individual bioavailability. What works for one will not necessarily work for another, nor will an individual respond in the same way on different days.

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While most patients will fall within a typical dose range (see Fig. IV-2 below), there will always be a small percentage who will react as “hyperresponders” or “hyporesponders”.

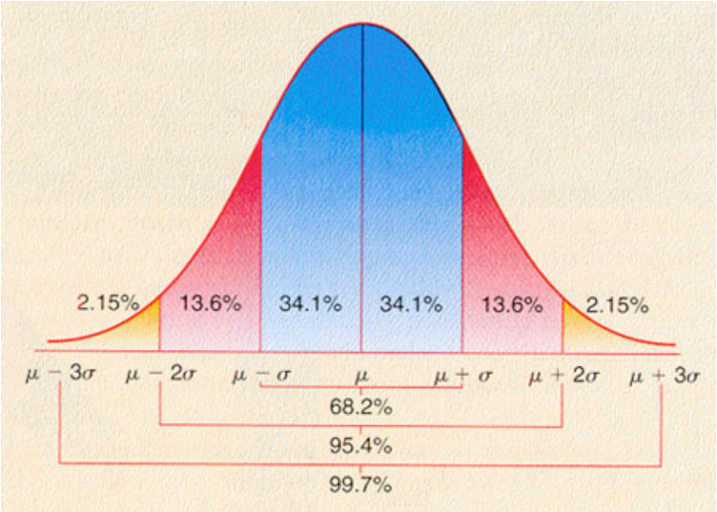

Figure IV-2. Expected General Population Dose Response Curve.

Titration minimizes the chances of a severe, possibly life-threatening reaction should the patient be allergic to the drug. Since the discovery of N_2O, no allergic reaction to the drug has ever been reported.

In addition, idiosyncratic (unpredictable) reactions may be encountered that are untoward and inexplicable (e.g., excitement after receiving a sedative drug). Titrating the drug uncovers such responses and minimizes unexpected reactions.

Regarding titration within the technique of N_2O/O_2 sedation, it is critical that once signs and symptoms of sedation begin to appear, the patients’ responses are closely monitored and time is given for doses to reach their peak effect.

Signs And Symptoms Of N_2O/O_2 Sedation

There is a wide range of responses to nitrous oxide sedation, with individual variability causing some to experience several symptoms, while others only a few. The degree of symptoms from similar doses can be different as well, with some experiencing very intense levels while others may only notice subtle changes. Knowledge of the physiological and psychological changes gives the provider valuable information as to how to titrate the administration of nitrous oxide to the desired level of sedation.

Psychological Signs - The patient has less fear and anxiety. Their demeanor can be described as happy, content, or even ambivalent.

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Body Movement - Drooping shoulders, legs uncrossing, and arms laying looser on arm rests are common signs. Patients may take deeper respirations when they are relaxed.

Eyes - The appearance of the eyes give valuable clues as to the patient's level of sedation. The apprehensive patient's eyes will be active, alert, and wide open. With the advent of sedation, the eyes will no longer focus on the activities of others. The blink rate will be slower, the eyes will appear "glazed" or "glassy", and the patient may comment that they feel heavy.

Facial Expression - The sedated patient exhibits less tension in their face. This is especially evident in the muscles of the forehead and brow. In addition, the patient will smile easily, and may mention that they can't help but smile. This may be followed by a giggle or short laugh.

Awareness - When appropriately sedated, patients are fully conscious and aware of their surroundings. With relaxation, patients' surroundings are no longer threatening, and patients will have a sense of well-being.

Patients will be able to respond rationally and coherently to the operator's inquiry or directions. They can maintain conversation. With N_2O/O_2 sedation, protective cough and gag reflexes remain intact. It is important to stress that patients refrain from talking to receive the drug's effects and minimize contamination of the environment.

Other signs and symptoms - Some patients may experience tingling in the fingers, toes, cheeks, lips, tongue, head, or chest area. It is common for a patient to experience circumoral numbness.

There may be a noted heaviness in the patient's thighs and/or legs. Conversely, some patients experience a lighter feeling in their extremities.

Because of the drug's effect on the tympanic membrane, the patient's voice may resonate or carry a hypernasal tone. In addition, the patient may sense that the operator's voice is farther away and therefore harder to hear.

Patients may notice they feel warm. Their cheeks and/or neck may flush as a result of vasodilation.

Signs And Symptoms Of N_2O/O_2 Oversedation

The oversedated patient may notice discomfort, but often does not remark of the unpleasant feelings until afterwards. The patient may experience dreams and/or hallucinations, including sexual fantasizing.

The signs of relaxation disappear, and they may become restless and fidgety. On occasion, they may become combative. They may also become very sluggish with their motions or unable to move their extremities.

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The eyes of an oversedated patient may become fixed and nonresponsive. They may want to sleep and have difficulty keeping their eyes open. Occasionally they may comment that their field of vision narrows, losing some peripheral vision.

Fits of uncontrolled laughter may occur. When this happens, mouth breathing causes the level of sedation to become milder. When nose breathing resumes, the oversedation resumes. This roller coaster sedation effect may result in nausea.

Patients may no longer feel that they are in the same surroundings. Disassociation or out-of-body experiences may occur.

The patients' perception of their hearing may change, and the perception of normal sounds may be very intense.

The pleasant warmth felt in earlier states might intensify and become uncomfortable. The patient may express various signs of being too warm or even hot.

The patient may become very drowsy, which is not to be confused with being relaxed. Words may be slurred or repeated, and sentences may be incoherent. The patient may not be able to respond to verbal commands. The patient can actually become unconscious.

Dizziness, light-headedness, and spinning sensations are common during oversedation. This can produce transient nausea for some patients. The tingling sensations felt in their extremities may now be felt throughout their body. This may feel similar to a shaking sensation.

Vomiting may occur with oversedation, and is more common in children. Silent regurgitation is a type of action in which vomitus reaches the level of the epiglottis and can lead to aspiration into the lungs. This situation demands immediate advanced medical attention.

Patients who have had negative experiences with N_2O will, most often, not want to repeat the experience. Conscientious, well-trained and knowledgeable practitioners will not allow oversedation to destroy the capability of N_2O/O_2 to remain a viable option for pain and anxiety management.

Fundamentals Of Nitrous Oxide Administration

- The key to the technique of nitrous oxide administration is careful titration and observation of the patient's response.
- It is important that the operator be confident and positive about the nitrous oxide experience. At the same time, the patient's expectations should be managed. It is best to under-promise and over-deliver rather than vice versa.
- Informed consent must be obtained from each patient before each N_2O/O_2 administration.

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- The amount of N_2O required by a patient on any given day or time varies. Beware of the fixed-dose philosophy.
- The procedure begins and ends with 100% pure O_2.
- The patient should never be left alone. The patient must be constantly monitored by a professional trained in N_2O/O_2 sedation.
- Accurate documentation of all procedures must be maintained in the patient's treatment record.
- When possible, the patient should be advised to avoid eating a big meal that includes fatty, fried foods before the appointment when N_2O/O_2 will be administered.
- **Nitrous oxide should only be administered in the presence of a properly functioning scavenging system. Such a system includes an accurate flowmeter, scavenging masks, and a vacuum system able to eliminate gases at a rate of at least 45 L per minute. These gases must be vented to the outside environment.**

POTENTIAL BIOHAZARDS FOR HEALTH PERSONNEL ASSOCIATED WITH CHRONIC EXPOSURE TO NITROUS OXIDE

Nitrous oxide, when used properly, is a safe inhalation anesthetic agent. A long track record of effective sedation and analgesia testify to this. However, chronic exposure to health personnel involved in its administration deserve special consideration.

Background

In 1967, Vaisman reported that both male and female anesthesiologists in Russia experienced reproductive problems at a significantly higher rate than the general population. In addition, he concluded that these problems were caused by the occupational hazard of being chronically exposed to anesthetic gases. Because N_2O was a common denominator in most anesthetic applications, it was implicated as the causative agent.

Cohen published articles in the 1970s and 80s dealing with anesthetic health hazards. One 1980 published study surveyed more than 50,000 dentists and dental assistants who were exposed to trace anesthetics. The results suggested that long-term exposure to anesthetic gases could be associated with an increase in general health problems and reproductive difficulty.

Since these reports were published, the literature has continued to document and describe potential adverse effects of chronic exposure to N_2O. However, this literature has been primarily retrospective; therefore, inherent biases in study design were inevitable. Because of these flaws, a causal relationship between the exposure of an individual to N_2O/O_2 and reproductive sequelae cannot be definitively established.

During the 1970s, Bruce investigated the possibility of N_2O affecting perceptual cognition and psychomotor skills of personnel exposed to varying concentrations of the gas. Their results were widely noted, because they reported audiovisual

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impairment in just hours of exposure to as little as 50 parts per million (ppm). Multiple attempts to reproduce the research results of Bruce have failed. Subsequently, these researchers have retracted their conclusions.

The National Institute for Occupational Safety and Health (NIOSH) became interested in these and other results being published, and in 1977 evaluated the scavenging potential of the equipment used in both the operating room and the outpatient setting. It was determined that 25 ppm was achievable in the operating room but not attainable in a setting such as a dental operatory. Therefore, NIOSH chose 50 ppm to be the maximum exposure limit for personnel in a dental setting. Although this limit has not been strictly enforced by the Occupational Safety and Health Administration (OSHA), it remains the recommended standard to date.

Despite the negative publicity N_2O has received, other studies in the literature claimed no deleterious health effects associated with chronic exposure to N_2O, especially in low concentrations.

In 1995, a worldwide literature search on the topic of biohazards associated with N_2O use was conducted at the University of Colorado. The conclusions drawn from this literature review clearly indicated that there was no scientific basis for the previously established threshold levels for the hospital operating room or the dental setting. This became the basis for a meeting of interested parties representing dentistry, government, and manufacturing. The result of this meeting was a formal position statement that a maximum N_2O exposure limit in parts per million has not been determined.

There has been evidence that chronic exposure to high levels of N_2O does have an effect on reproduction. However, to date no evidence that a direct causal relationship exists between reproductive health and scavenged low levels of N_2O has been found.

Sweeney *et al* showed that the first sign of any biologic effect from N_2O was at a chronic exposure level of 1800 ppm. Experts in the field concur that the level of 400 ppm is a reasonable exposure level that is both attainable and significantly below the biologic threshold established by Sweeney et al.

N_2O interferes with the enzyme methionine synthase, which is responsible for recycling homocysteine back to methionine. Methionine synthase plays a vital role in the metabolic pathways of vitamin B12 and folic acid, as well as the production of DNA. Megaloblastic hematopoiesis and leucopenia result after prolonged exposure to N_2O.

Because of this interference, it was postulated that fetal development might be impaired because of exposure to N_2O. Animal studies that used approximately 60% N_2O for 24 hours on pregnant rats produced miscarriage and other fetal abnormalities.

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Neurologic disorders associated with chronic N_2O exposure appear as myeloneuropathy. Symptoms such as sensory and proprioception impairment may be permanent but are usually temporary with slow recovery.

Recommendations

Because of the significant demands for folic acid during organogenesis (first trimester), and the reported link between high levels of homocysteine and birth defects, postponement of N_2O sedation during pregnancy is recommended. For a pregnant female employed in a setting that uses N_2O, it is important to know the exposure levels of N_2O and to use all recommended trace gas scavenging methods. Depending on the individual and the situation, that employee should determine whether she should avoid the office setting and any N_2O exposure for the first trimester.

Discussions continue as to the toxicity of nitrous oxide, and it continues to seem that nitrous oxide is safe when administered in low therapeutic doses for short periods of time. Nitrous oxide levels in the dental office can be maintained at a safe level through:

- Use of a scavenging system that redirects N_2O outside the office is the most effective means.
    - The scavenging mask system has become standard on all product lines in the United States and Canada. When expired N_2O is exhaled through the nose, vacuum suction ports transport this gas through the central suction to the outside atmosphere. This evacuation flow rate has been established by NIOSH as optimal at 45 L/min.
- Good ventilation in the operatory dilutes the concentration of gas in one area of the office.
- Well-fitting masks reduce leakage - One of the newest advances in nitrous oxide administration is the ***Safe-Sedate*** Nasal Hood. This apparatus provides more efficient delivery of gases into the nasal nares, while reducing extraneous nitrous oxide into the operatory. The ***Safe-Sedate*** Nasal Hood also is latex-free and disposable, preventing cross-contamination.

- Reduced talking by the patient decreases exhalation of nitrous oxide into the operatory environment.
- Gas can leak at any place of connection on the equipment. This includes the manifold and wall-mounted connections and any hoses associate with each. Conducting tubing and the reservoir bag are also areas of concern. Because of the breakdown of these materials over time, it is possible for these items to crack and subsequently leak around pleats and/or seams.
    - The soap and water method for determining leaks is recommended for

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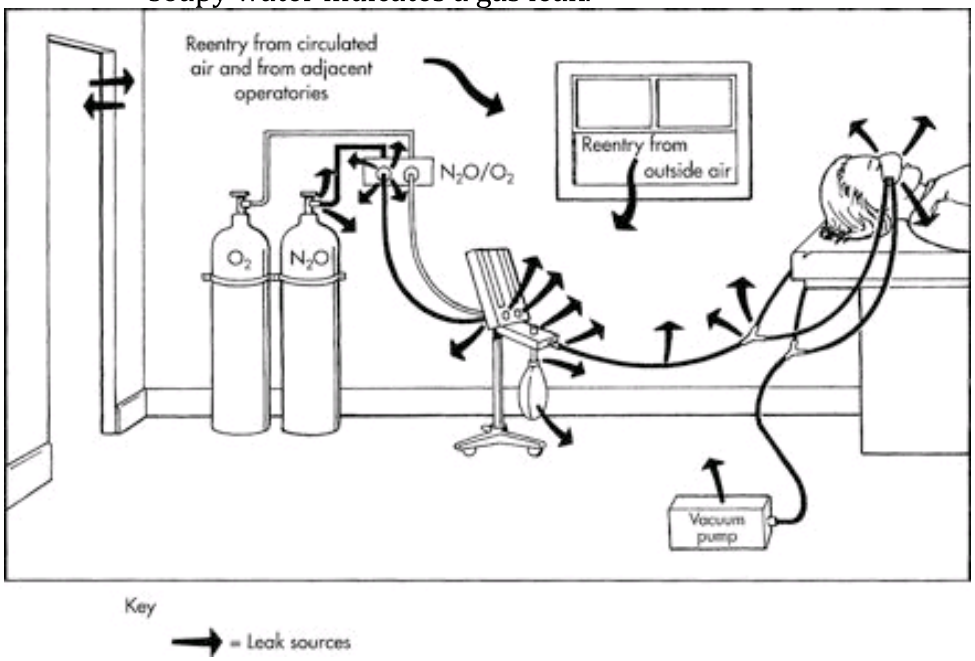
inspecting these areas. The presence of bubbles after application of the soapy water indicates a gas leak.



- There are several commercially available monitoring systems to alert office personnel of potential unsafe levels of trace N_2O. These include Infrared (IR) spectrophotometry, time-weighted average (TWA) dosimetry, and hand-held monitoring devices**.**
    - **IR spectrophotometry** uses electromagnetic energy to detect levels of N_2O in the atmosphere. This technology can detect levels lower than 1 ppm. An advantage of IR spectrophotometry is the ability to detect minute levels of gas immediately in the ambient air. The instrument can be rented for a reasonable fee, and is the recommended method of establishing a baseline reference of N_2O exposure levels.
    - **Time-weighted average dosimetry** allows for the determination of N_2O exposure of an individual over a specific time period. The badge or vial is worn for the recommended period and then returned to the company for analysis. An active material in the badge absorbs the N_2O. The amount is read by an IR spectrophotometer to determine the parts per million of exposure. Theses systems are relatively inexpensive and easy to use.
    - **Hand-held monitoring devices** are small, lightweight, portable N_2O gas monitors that give a continuous measure of N_2O in the ambient air. It fits in the palm of your hand and has both ppm and TWA measurement capabilities that are activated with the touch of a button. This monitor can be used to detect leaks around equipment. Another beneficial feature is its ability to store data for record keeping and monitoring purposes.
- It is strongly recommended that a dental practice that administers N_2O/O_2 institute an education program for its team members that describes the hazards of N_2O and defines the preventive measures for reducing or eliminating trace gas contamination.

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Summary

To date, no direct evidence exists of any causal relationship between chronic low-level exposure to N_2O and potential biologic effects. The maximum safe concentration of N_2O has not been determined; however, every attempt should be made to reduce the level of trace N_2O to exposed healthcare personnel. Practitioners cannot be too cautious when their health and that of their coworkers is at stake.

Continuing education should be required of healthcare professionals who use N_2O/O_2 sedation, as should evidence of proper training with state-of-the-art equipment and administration techniques.

NITROUS OXIDE ABUSE ISSUES

Identifying The Problem

N_2O has been used for recreational purposes since its discovery in the late eighteenth century. Inhaling the gas was popular at social gatherings at that time and still occurs today. N_2O abuse is an issue about which everyone with access to it should be aware. When chronically abused, N_2O can have serious legal and medical ramifications.

All drugs that produce euphoria have the potential to be abused, whether they are injected, swallowed, or inhaled. The term inhalant in this instance applies to a group of substances that produce psychoactive reactions and are defined solely by their route of administration. Typical modes of administration are nasal inhalation called sniffing or snorting, inhaling contents from a bag or bagging, holding a saturated rag in the mouth and inhaling or huffing, and spraying the substance directly into the mouth.

Inhalants were second behind marijuana as the most commonly used illicit drugs by teenagers in the 1990s. A 1993 report from the National Institute on Drug Abuse (NIDA) indicated that approximately 17% of U.S. adolescents said they had sniffed inhalants. For youth and others who may be disadvantaged financially, inhalants present a way of "getting high" that is cheap and easily obtained. Another alarming statistic involves pregnant women. The National Institute on Drug Abuse reports that 12,000 pregnant women use inhalants each year.

McGarvey *et al* surveyed 285 adolescents and found that the five most frequently inhaled substances were gasoline, refrigerant (Freon), butane, glue, and nitrous oxide.

Nitrous oxide is relatively inexpensive, readily accessible, and legal to purchase, and produces a rapid, euphoric high. Users recommend its use to others claiming that it produces feelings of floating or flying; vivid, visual colorful images; and loss of all inhibitions, making the user feel invincible.

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N_2O abusers tend to be somewhat older than those inhaling other volatile agents, and is popular today at college social activities such as parties and concerts. Healthcare personnel who have ready access to the gas are also noted recreational users. In a 1977 anonymous survey of medical and dental students, 20% admitted to the use of N_2O socially.

Besides its availability in medicine and dentistry, nitrous oxide is approved by the Food and Drug Administration (FDA) as a food ingredient. The dairy industry uses it as a whipping cream propellant. When used for these purposes, N_2O is found in 3-inch-long containers called whippets. These containers hold approximately 4-5 L of N_2O, and have been misused for recreational purposes.

Nitrous Oxide Hardware
Photos by Astro Blue & Erowid, © 2001 Erowid.org

Similarly, approximately 3 L of N_2O is found in aerosol cans of whipping cream. The "grocery store high" is the name given to abusers getting a "hit" out of whipping cream containers without buying the product. Restaurant owners have reported the necessity of locking the supply of whipping cream cans to prevent restaurant workers from sampling the N_2O while on the job.

Since nitrous oxide is minimally metabolized in the body, N_2O is rarely detected in routine urine tests. Therefore, drug abusers may substitute N_2O for other, more detectable drugs.

Nitrous oxide is not a very addictive drug. Evidence does suggest that N_2O is involved with the endogenous opioid system directly at a receptor site and/or indiretly by activating opioid neurotansmitters. It is thought that N_2O acts as a partial opioid agonist, making it less addictive than a full agonist such as morphine. Tolerance to the effects of N_2O does develop.

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The Damage

For most healthy surgical or ambulatory patients undergoing N_2O/O_2 sedation, the effects of inactivated methionine synthase are temporary and insignificant. Pregnant women are at greater risk, as are those with imparied immunity, and exisitng vitamin B_{12} deficiencies.

Studies on laboratory animals have shown that enzyme inactivation occurs rapidly during N_2O overexposure and that recovery is slow. Animal studies have also shown evidence of fetal toxicity, resulting in skeltal abnormalities and miscarriages.

Several studies with humans revealed significant hematopoietic changes as well.

Those persons who are vitamin B12 deficient may not be aware of the deficiency until the N_2O exposure produces neurologic changes.

Neurological symptoms have been reported primarily by individuals who have admitted to the use of N_2O recreationally; however, in some cases, affected individuals were exposed to significant amounts of unscavenged N_2O in hospitals or during dental surgery. Symptoms relating to peripheral neuropathy included tingling and numbness in the extremitites, weakness and incoordination, lack of strength and dexterity in the hands, slowed gait, and positive Lhermitte's sign (electric shock feeling on flexion of the neck).

Often the neurological symptoms do not occure immediately, appearing several months into the overexposure period. Generally, neurologic function slowly returns to normal after termination of overexposure to N_2O. In some cases, it may not be complete.

The cases of overexposure reported in the literature involving dentists or other health professionals who deliver N_2O to patients occur because of improper scavenging equipment. Other cases are young adults recreationally inhaling N_2O from whipping cream chargers. Most of the overexposure incidents reported were periods of 1-2 hours per day, at least three times a week, and for periods of several months to years. In some cases, individuals reported falling asleep and incurring up to 4 hours of exposure; one individual admitted self-administraion of 2-4 hours per day and another admitted 6-7 hours per day.

There have also been reported cases of frostbite from nitrous oxide abusers. The temperature of the N_2O gas exiting a cylinder may be as low as -55 degrees Celcius.

The literature contains several reports of asphyxial death from N_2O, all from recreatonal use. Several victims have been healthcare workers, others worked in restaurants, and some were users outside the workplace. Other reports describe victims being discovered with plastic bags over their heads and belts or similar devices aroung their necks to keep the bags closed. In these situations, chargers are frequently used and discharged inside the plastic bag.

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Sexual Phenomena Associated With N_2O

Euphoric effects have long been associated with N_2O since it was first introduced. These include aberrations of a sexual nature.

In addition, allegatons of sexual impropriety have been made against healthcare professionals after patients were sedated with N_2O/O_2. The specific cases reported have been primarily against male practitioners by female patients. The cases involve claims such as the patient falling asleep and being frightened by a vision, pressure sensations in the genital region, becoming sexually stimulated, feeling naked, dreaming, and fantasizing. In nearly every instance, concentrations of N_2O higher than 50% were used. In addition, no other personnel accompanied the dentist during the sedation experience.

Lambert suggested that hypnosis is potentiated by N_2O. He suggested that a person may experience a hypnotic state spontaneously or by the suggestive language on the part of the professional while N_2O is being administered. He also recommended that practitioners consider their statements carefully without lending comments that could be misinterpreted. It is also prudent and recommended that a third party be present at all times and that N_2O concentrations greater than 50% be avoided.

Legislation And Regulation Issues

Currently, there are few regulations governing the sale of N_2O. Distributors are ethically obligated to confirm that their customers are purchasing the drug for appropriate use. However, there are no control measures to ensure that this is happening. Those selling N_2O for purposes other than appropriate use may be held responsible for their actions. In March 2001, "delivering a misbranded drug into interstate commerce" was the charge resulting in an Arizona man being sentenced 15 months in prison and a fine of $40,000. His sale of N_2O in whipped cream chargers led to the death of a college student.

It is the responsibility of the practitioner to ensure that tanks are properly stored and locked securely so that the likelihood of others stealing a tanks or abusing N_2O is minimized.

ETHICAL AND LEGAL CONSIDERATIONS REGARDING N_2O ADMINISTRATION

Healthcare professionals are legally responsible for abiding by the laws and rules as determined by the governing agencies that regulate the practice of their discipline. It is imperative to follow the ASA practice *Guidelines for Sedation and Analgesia by Non-anesthesiologists*, because these will be the standards to which your actions will be compared should legal and/or ethical concerns be questioned.

Widespread differences exist among healthcare disciplines as to who may administer and/or monitor the N_2O/O_2 sedation procedure. Depending on the discipline, a provider may be legally allowed to monitor a patient under N_2O/O_2 sedation and not be allowed to administer the drug. This may be the case for certian

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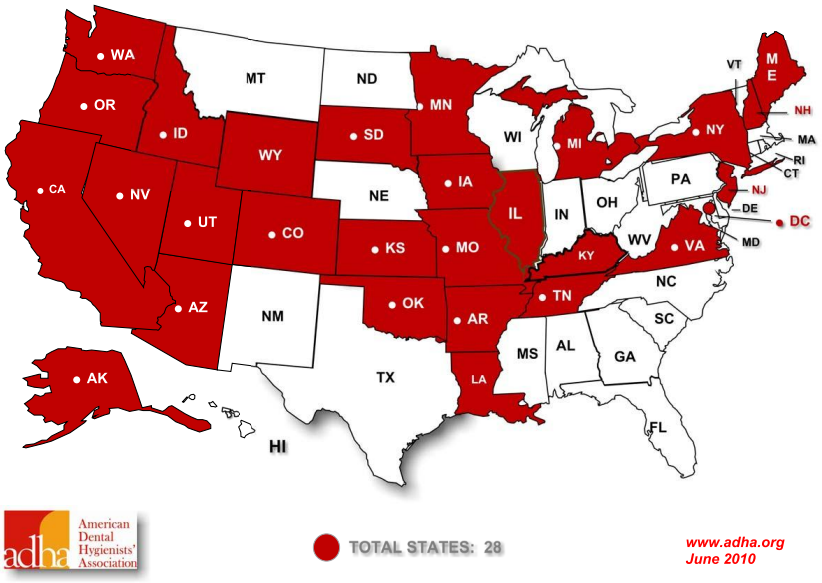
dental auxiliaries. In addition, the regulations determine the level of supervision necessary for N_2O administration.

States Where Dental Hygienists Can Administer Nitrous Oxide



It is also considered standard of care that informed consent be obtained from the patient or parent or guardian before the administration of N_2O/O_2 sedation. In some states, written consent is required. However, even if not mandated, obtaining a patient's or parent/guardian's signature is advised.

Governing boards and licensing agencies may require a specified course of training in the area of N_2O/O_2 sedation before practitioners can incorporate this procedure into their clinical practices. Approved courses usually consist of both didactic and clinical components, totaling a specified minimum number of hours. After successful completion of such a course of study and accompanying fee payment, the practitioner may be granted a permit or license to administer N_2O/O_2 sedation. Commonly, students completing their professional curriculum are exempt from additional coursework in this area unless this information was not part of the professional program.

It is advisable for a previously educated provider to participate in an N_2O/O_2 sedation refresher course. Updated material, new equipment, or a review of the appropriate administration technique is usually included, which could improve the success of this procedure in clinical practice.

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Airway Maintenance & Emergency Airway Management in the Sedated Dental Patient

Airway Maintenance – Maintenance deals specifically with protecting the airway from compromise.

Rationale: The practice of dentistry involves treatment in and around the oral cavity, the initial segment of the anatomical airway. Given the **necessity of a patent airway to normal ventilation and sustaining life processes**, the critical importance of protecting the airway at all times is obvious. **we often recline our patients to a supine position, have impaired access and visibility** due to the inherent nature of the oral cavity, **reduce sensory innervation with local anesthetics, and work with and generate small foreign bodies,** it is even more apparent that airway maintenance is our number one safety concern in dentistry**. A sedated patient, with the accompanying depression of the central nervous system, is at even more risk due to decreased conscious awareness and obtunded reflexes.** Therefore, the primary concern of the sedation team should be at all times the maintenance and protection of a patent airway.

Protecting the Airway of the Sedated Patient

Proper Position: The upper airway is surrounded by soft tissue that has the potential to result in partial or total obstruction. In the absence of swelling of the soft tissue lining the oral cavity, oropharynx, and laryngopharynx, **the anatomical structure most likely to cause obstruction is the tongue**. Fortunately, the tongue is connected to the mandible, so that during times of normal muscle tone, the forward position of the mandible will prevent the tongue from occluding the oropharynx. However, when our patients are reclined and sedated, muscle tone is relaxed, and the tongue is capable of airway obstruction (see Figure 1).

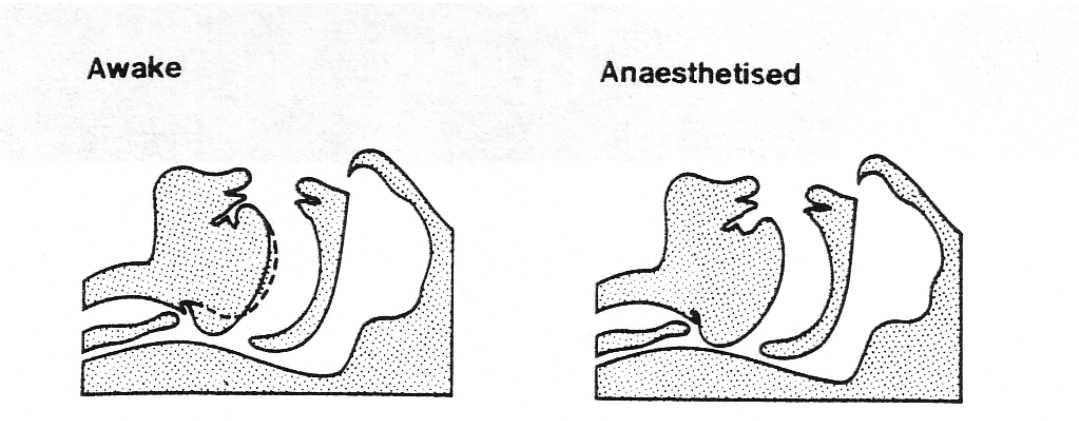

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Figure 1.

Therefore, **it is essential that the mandible be positioned forward (chin is raised).** This is most easily accomplished by the use of a chair pad that allows the patient's head to fall back and be cradled into a notch located at the head of the pad (see Figure 2).

Figure 2. A chair pad with separate head rest is shown here. The head rest shown is not used to facilitate proper head position.

Vigilant Monitoring: The sedation team must continually observe the position of the patient's airway, as well as looking and listening for:

- **The rise and fall of the chest (depth and rate of respiration) – normal respiratory rate in adults is 12-16/min., in children 18-22/min.**
- **The expansion and contraction of a reservoir bag** if being used – as long as the mask or tube creates a good seal, the rise and fall of the reservoir bag is a good indication of respiration.
- Breathing sounds (with or without a stethoscope) – **normal breath sounds are a smooth, barely audible "whoosh". Abnormal sounds include snoring (partial obstruction of the upper airway), gurgling (water in the upper airway), wheezing (bronchospasm), or crowing (larngospasm).**
- **The patient's skin and mucosa color** – hypoxia gives skin and mucosa colors a bluish tint. This is usually a very late sign in the oxygen desaturation process.

In addition, the sedation team must be trained in the use and interpretation of monitoring equipment designed to measure the oxygenation of the blood (oxygenation) as well as the expiration of carbon dioxide (end-tidal CO2, capnography, and capnometry).

The saturation of available hemoglobin sites with oxygen to a level of 90% as measured by the pulse oximter is an indication of at least adequate oxygen

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saturation. The significance of the 90% level is explained by the Oxyhemoglobin Dissociation Curve.

End-tidal CO2 reflects actual ventilation, and is a more immediate indication of the delivery and use of oxygen for cellular metabolism, and is usually reserved for deeper levels of central nervous system depression (deep sedation and general anesthesia).

Avoiding Over-Sedation: The central nervous system has complete or partial control of most physiological functions, including respiration. Sedative drugs can alter the fine balance that normally insures the respiratory system responds appropriately to oxygen needs of the body. As has already been pointed out**,** sedative drugs also impact muscle tone, resulting in greater likelihood of airway obstruction by soft tissues. Therefore, the risk of airway compromise and respiratory depression increases proportionately with the depth of sedation (See Figure 3).

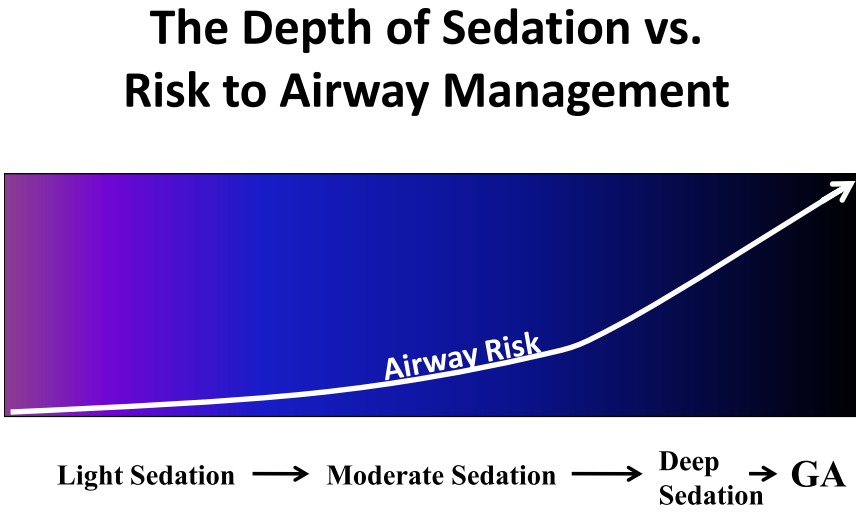

Figure 3.

If the patient can respond readily to verbal and physical stimulation, in other words they subscribe the level of consciousness defined by light or moderate levels, then they should be able to independently and continuously maintain their own airway. Despite this, all precautions such as proper positioning and monitoring are still adhered to.

Patient Selection: Risk to the airway is also dependent on individual patient characteristics. **Patients who are: obese;** have **chronic obstructive pulmonary disease (COPD); smoke**; and or have **asthma or obstructive sleep apnea (OSA),** are at greater danger of developing an airway emergency.

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Obese and OSA patients have airway obstruction issues. Patients who smoke or have COPD have compromised gas exchange mechanisms. Patients with asthma are more likely to experience bronchospasm.

Even in the absence of these medical conditions, the anatomy of the upper airway can certainly clue the practitioner to the possibility of airway compromise during sedation as well as make emergency airway management more challenging. Examination of the **position and quantity of the soft palate and uvula when the patient opens wide and sticks out their tongue without phonating is the basis for the Mallampatti classification** used by anesthesiologists to grade the difficulty of intubation (See Figure 4). This same classification can be utilized by those not intending to use intubation for anesthesia to evaluate the degree of potential soft tissue impingement of the airway.

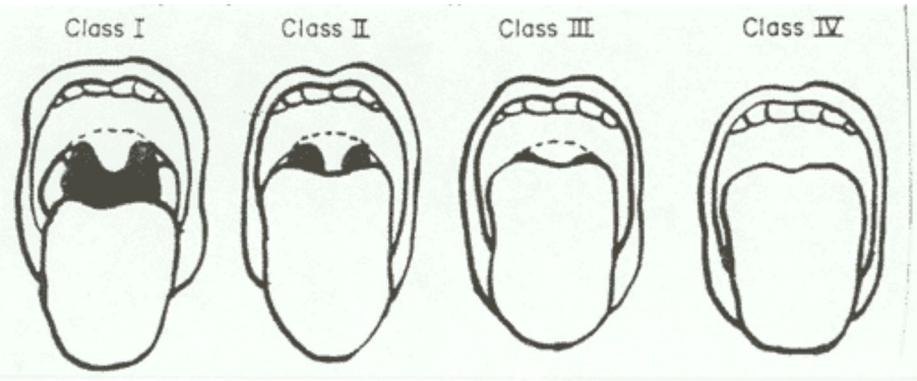

Figure 4. Mallampatti Classification.

The age of the patient also is a factor in the risk to airway maintenance and management. **Generally speaking, because of the anatomy of the less mature airway, the likelihood of both airway obstruction as well as complicated emergency airway management is greater in the child than in the adult.**

Children have relatively large heads, short necks, large tongues and soft tissue, and narrow nasal passages, which are readily blocked by secretions or edema. Many of the structures of the upper airway exhibit differences in location and texture. In the lower airway, the angles of the right and left mainstem bronchi with the trachea are equal. Pediatric patients have fewer lung alveoli. The 20 million present at birth increase to 300 million by age 8.

A higher metabolic rate in infants and children results in a proportionally greater alveolar ventilation (100 to 150 ml/kg/min) than in adults (60 ml/kg/min). Since the metabolic demand for oxygen is 60% greater than in the adult, hypoxemia develops rapidly in the pediatric patient (see Figure 5).

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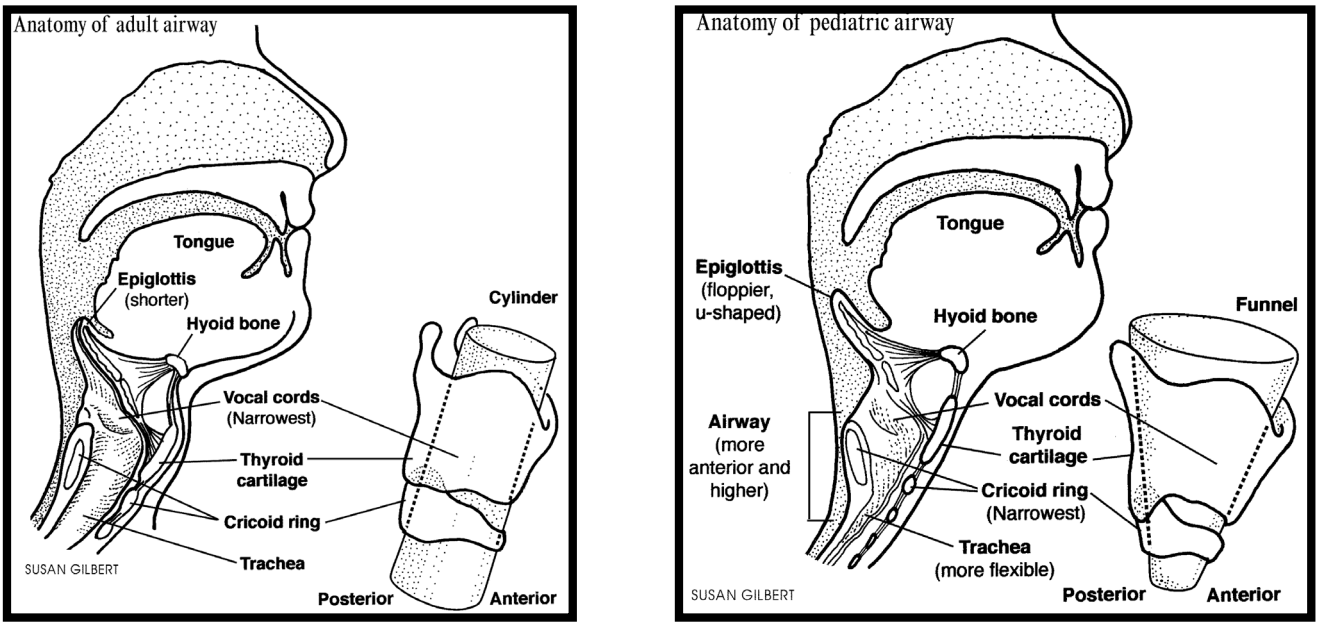

Figure 5. Adult vs. Pediatric Upper Airway Anatomy

Physical Barriers: Protection of the airway from foreign objects is not only accomplished through careful handling and skillful suctioning, but through the **use of physical barriers between the oral cavity and the oropharynx.** When possible, the use of rubber dams, the Iso-Lite®, or gauze packing should be used to prevent unintended entry of foreign bodies into the airway.

Securing Loose Objects: When possible, **small objects (eg. Rubber dam clamps, implant wrenches) should be secured by dental floss when used in the oral cavity.** Crowns and veneers can be delivered with devices designed to use suction or tacky materials to prevent dislodgement into the airway.

Airway emergencies, like all emergencies can be prevented with careful attention to detail. This involves training of the doctor and all auxiliary personnel, patient selection, proper patient positioning, monitoring, avoiding deeper than intended levels of sedation, and using physical barriers and securing loose objects to prevent foreign body obstruction. When the airway emergency occurs, emergency airway management must be initiated promptly and skillfully.

Emergency Airway Management

Skillful airway management is the first step in the successful resuscitation of a compromised patient. When faced with an unsecured airway, the clinician has little margin for error. Neurologic damage caused by hypoxia occurs within minutes.

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Airway Basics

Anatomy – A thorough understanding of airway anatomy is necessary to understand both the origin of the airway emergency as well as the steps to control it. The airway can be divided into two basic categories, the upper and lower portion. The upper portion is the area most likely to be involved with dental procedures and is the source of emergency intervention (see Figure 6).

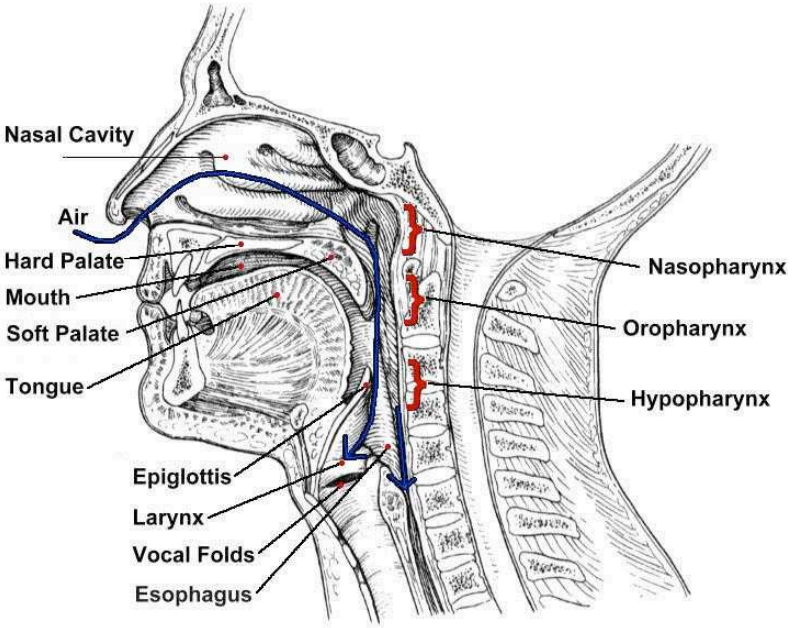

Figure 6. Anatomy of the Upper Airway

As indicated in Figure 6, the normal path of air travels into the nasal cavity, through the nasopharynx, past the oropharynx and hypopharynx, and through the larynx and vocal cords and into the trachea. All along this path the airway is lined by soft tissue which is normally covered and bathed in body secretions (mucus, saliva, etc.). During normal physiologic function, the body maintains airway patency throughout this passageway by both autonomic and somatic control. However, during an airway crisis, some or all of this control is blocked or lost, and the practitioner must be prepared to take over for these functions so that oxygen containing air can continue to reach vital organs and the cells that constitute them.

Airway Management Common To All Emergencies

Common management to all airway emergencies is the P – A – B – C – D protocol where:

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P = Positioning
A = Airway
B = Breathing
C = Circulation
D = Definitive Care (Including Drugs, Defibrillation, and Delivery to Advanced Care Personnel)

Positioning
The significance of airway positioning has already been covered relative to airway maintenance. Likewise, when managing the unconscious patient, the airway must be supported by lifting the mandible.
This can be accomplished by several methods including the following:
.

Head Tilt / Chin Lift (see Figure 7)

- Most effective for opening the airway
- No evidence of trauma
- Dentures do not need to be removed,
  they help maintain normal contour of the mouth
- Good for rescue breathing

Technique

- Place one hand on the forehead of the patient and tilt the head backward
- Place fingers of the other hand under the patient's jaw and tilt upward

Figure 7. Head Tilt/Chin Lift Maneuver.

Jaw Thrust Technique (see Figure 8)

- Grip the angles of the mandible with both hands
- Pull forward, while at the same time tilting the patient's head backward

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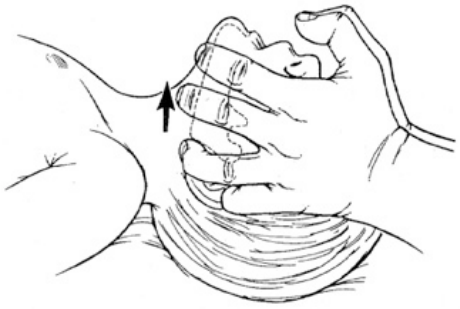

Figure 8. Jaw Thrust Technique.

Modified Jaw Thrust

- Method of choice when cervical spine injury is suspected
- No extension of the neck
- Technique is to put forward traction on the mandible without heal tilt

Triple Airway Maneuver (see Figure 9)

- Place fingertips of each hand on the angles of lower jaw
- Displace the jaw forward
- Use thumbs to retract patient's lower lip

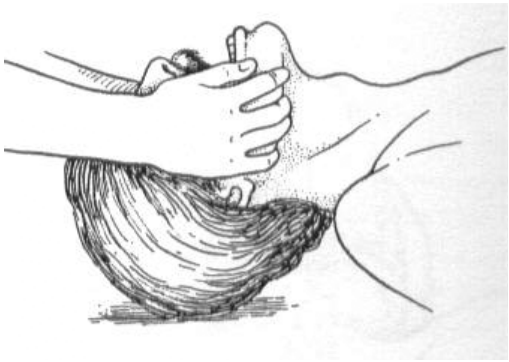

Figure 9. Triple Airway Maneuver.

Mandibular Displacement (see Figure 10)

- Effective with spontaneous breathing edentulous patients
- Dangerous for the rescuer if the patient has teeth

Technique –

- Steady the head
- Place thumb in patient's mouth
- Fingers underneath the chin
- Pull upward

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Figure 10. Mandibular Displacement

Airway
During an airway emergency, positioning alone may not be enough to result in spontaneous respiration. In such cases, an airway support device is useful to open the airway and/or allow supplemental or positive pressure oxygen (at 21% - 100%) to be delivered to the lungs. There are numerous devices available for these purposes. The choice of device will be dependent on consideration of the advantages/disadvantages inherent in each option, as well as the training and personal preference of the practitioner. Five such devices will be considered here:

Oropharyngeal Airway
Nasopharyngeal Airway
Laryngeal Mask Airway
Combi-Tube
King L-TD Airway

Oropharyngeal Airway
The oropharyngeal airway (see Figure 11) may be used to maintain airway patency after proper positioning is achieved. It may relieve soft tissue obstruction of the airway by displacing the tongue anteriorly, providing a patent airway. Placement of an oral airway may precipitate vomiting, laryngospasm, or aspiration if placed in a semiconscious patient.

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Figure 11. Oropharyngeal Airways of different sizes.

Of all the airway devices, the oropharyngeal airway (OPA) is perhaps the easiest device for a dentist with limited training and experience to insert.

Insertion Technique

1. Insert airway with the concave side toward roof of the mouth (see Figure 12).
2. When the tip of the airway reaches the back of the tongue (past the soft palate), rotate the airway 180° so the tip end of the airway is pointing down toward his throat. If the airway is difficult to insert or rotate, grasp the patient's tongue with the fingertips of the hand not holding the airway and gently pull the tongue forward. Advance the airway until the flange rests against the patient's lips (see Figure 12).
3. The airway must be positioned posterior to the tongue displacing the tongue away from the posterior pharyngeal wall (see Figure 13). To test placement, the tip of the tongue may be pulled anteriorly to confirm correct placement of airway.

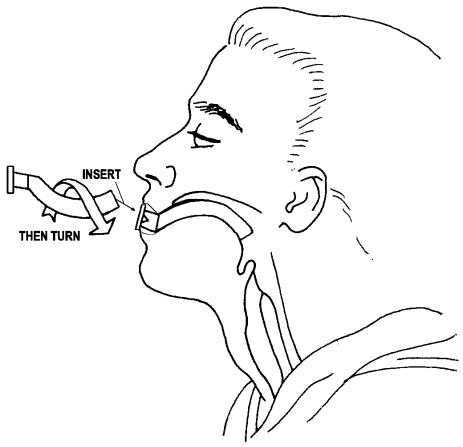

Figure 12. Oropharyngeal Airway Insertion.

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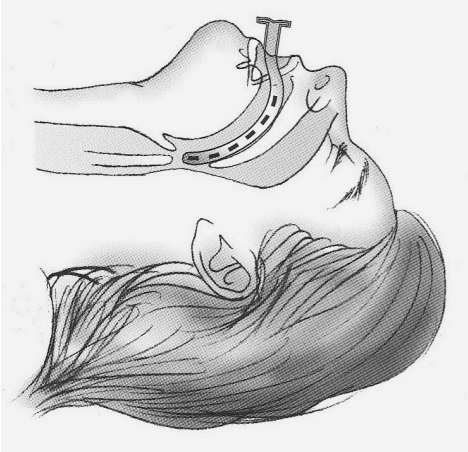

Figure 13. Correct positioning of the Oropharyngeal Airway.

Indications
- Unconscious patients without a gag reflex
- Breathing or nonbreathing patients
- Used as a bite block in seizures and with endotracheal tube in place

Contraindications
- Presence of a gag reflex
- Severe maxillofacial injuries

Oropharyngeal Airway Advantages
- Allows air to pass around and through the device
- Helps prevent obstruction by teeth and lips
- Helps manage unconscious patients who are breathing spontaneously or need mechanical ventilation
- Makes suctioning of the pharynx easier
- Bite block during seizures and ET protection

Oropharyngeal Airway Disadvantages
- Does not isolate the trachea
- Cannot be inserted when teeth are clenched
- May obstruct the airway if not inserted properly
- Can be dislodged easily
- Should never be inserted in a conscious or semiconscious patient with a gag reflex
- May precipitate vomiting and laryngospasm

Nasopharyngeal Airway

A nasopharyngeal airway is similar in basic design to the oropharyngeal airway and may be placed through a nostril into the posterior oropharynx. In this position it may relieve soft tissue obstruction of the posterior pharynx

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(see Figure 14). Nasal airways tend to be less stimulating than in oral airways in an awake patient, but may cause epistaxis. Nasopharyngeal airways are contraindicated in patients with known or suspected basilar skull fractures and thus probably should be avoided in the head-injured patient.

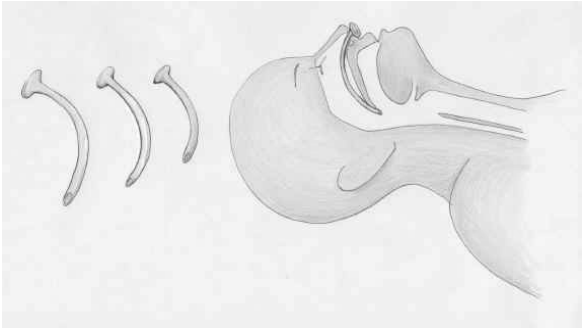

Figure 14. Nasopharyngeal Airway.

Contraindications

- Nasal obstructions
- Patients prone to nosebleeds
- Head injuries (basilar skull fractures)

Advantages

- Can be easily and rapidly inserted
- It bypasses the tongue
- May be used when a gag reflex is present

Disadvantages

- Does not isolate the trachea
- Smaller than the oropharyngeal airway
- Difficult to suction through
- Can cause severe nosebleeds
- Can cause pressure necrosis of nasal mucosa
- Difficult to insert if nasal damage is present

Laryngeal Mask Airway - Laryngeal mask airways are used in anesthesia and in emergency medicine for airway management. They comprise a tube with an inflatable cuff that is inserted into the pharynx (see Figure 15). They cause less pain and coughing than an endotracheal tube, and are much easier to insert. However, a standard laryngeal mask airway does not protect the lungs from aspiration, making them unsuitable for anybody at risk of this complication.

The device is useful in situations where a patient is trapped in a sitting position, suspected of trauma to the cervical spine (where tilting the head to maintain an open airway is contraindicated), or when intubation is unsuccessful.

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The device sits tightly over the top of the larynx. It avoids tracheal intubation and can be used with spontaneous respiration or artificial ventilation. However, it may not protect the airway from the aspiration of regurgitated material. Patients who have been treated with the laryngeal mask airway claim it does not irritate the throat as intubation typically does.

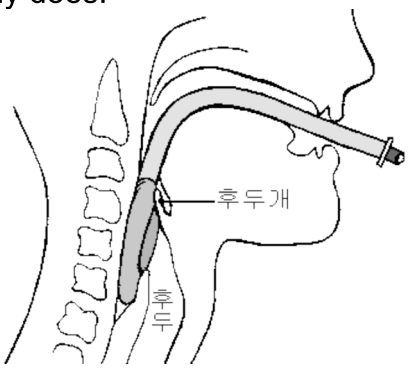

Figure 15. Laryngeal Mask Airway (LMA) inserted.

Combi-Tube - The Esophageal Tracheal Combitube was developed in the early eighties with the intent to allow for a quick and easy method securing the patient's airway and adequately ventilating the lungs (see Figure 16).

The invention of the Combitube aimed to enter either the esophagus or the trachea and allowing ventilation and oxygenation in both positions. This aim was achieved by designing a double lumen tube, one lumen resembling a tracheal tube, the other an esophageal obturator type tube with a distal blocked end and perforations at the pharyngeal level.

Indications:

- Ventilation in normal and abnormal airways
- Failed intubations
- Airway management in trapped patients

Contraindications:

- Patients with intact gag reflexes
- Patients with known esophageal pathology
- Use in patients under 5 feet with standard Combitube, under 4 feet with Combitube SA (small adult)

Advantages:

- Requires minimal training
- May be more useful in non-fasted patients
- Successful passage and ventilation in many patients via esophageal route
- Portable, useful in remote setting
- Functions in either the trachea or esophagus

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Disadvantages:

- Mostly adult sizes
- Potential for esophageal trauma
- Problems maintaining seal in some patients

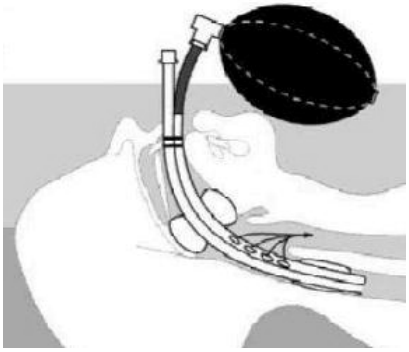

Figure 16. Combitube Placement.

King LT-D Airway - The KING LT-D is a disposable supraglottic airway created as an alternative to tracheal intubation or mask ventilation (see Figure 17). The KING LT-D is designed for positive pressure ventilation as well as for spontaneously breathing patients, thereby allowing maximum versatility as an airway management tool.

It has quickly gained favor among emergency medical personnel because it is easy to insert and results in minimal airway trauma. It is considered by many to be the Safest and most reliable disposable supraglottic airway tool for emergency ventilation when direct laryngoscopy is not feasible. The KING LT-D is 100% latex free and is provided sterile for single patient use.

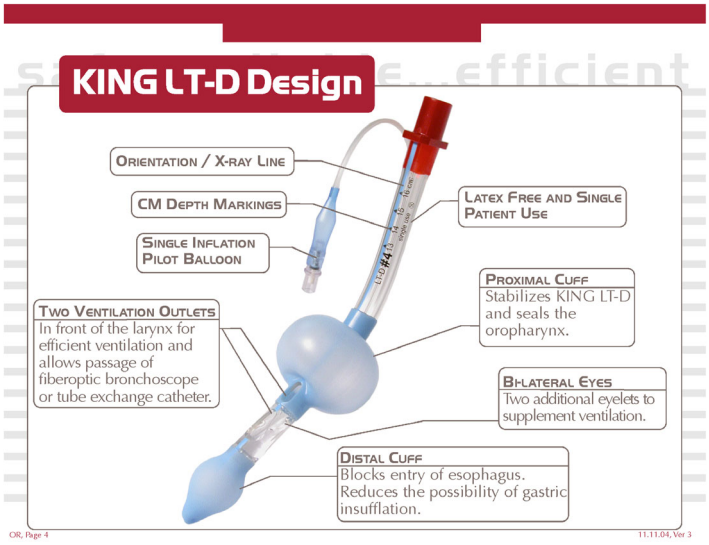

Figure 17. King LT-D Airway.

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Positive Pressure Oxygen with a Bag-Valve Mask Assembly

After placement of an airway device, check again for adequacy of ventilation. If needed, assist with positive pressure ventilation with a Bag-Valve-Mask Assembly (see Figure 18). This should be done with a source of high-flow oxygen attached to a bag-valve device consisting of a self-inflating bag, non-rebreathing valve, and no pressure release valve. This bag-valve device has standard 15 mm/22 mm fittings that allow it to be connected to a mask or an endotracheal tube. Experience and practice in maintaining proper mask fit will increase efficiency of air movement.

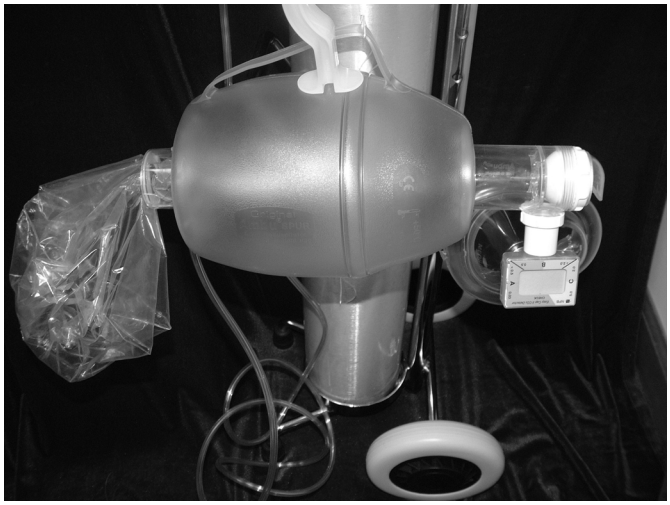

Figure 18. Bag-Valve-Mask Assembly for delivery of positive pressure oxygen.

Advantages

- Delivers oxygen or room air to a patient who cannot breath independently
- Hyperexpands lungs, improving alveolar ventilation
- Prevents hypoxia
- Contains self-inflating bag, one way valves, reservoir, and transparent face mask
- Available in various sizes and each practitioner should have ALL the sizes

The office portable emergency oxygen supply must be ready at all times for immediate use. In order to insure this, someone on the clinical team should be accountable for verifying a full supply of oxygen, and that all accessories are attached to the apparatus.

Bag-Valve Concentrations

- Without oxygen - 21%
- With oxygen, no reservoir - 60%
- **With oxygen and reservoir - 90 to 95%**
- With demand valve attachment - 100%

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Carbon Dioxide Detector

These simple to use and read devices fit between the mask and bag valve assembly (see Figure 19). They detect end tidal CO2 (the most accurate determination that ventilation is taking place) through a color change scale. The pale lavender color indicates carbon dioxide levels below 0.03%, whereas the pale yellow color is an indication of favorable CO2 expiration levels of up to 5.0%.

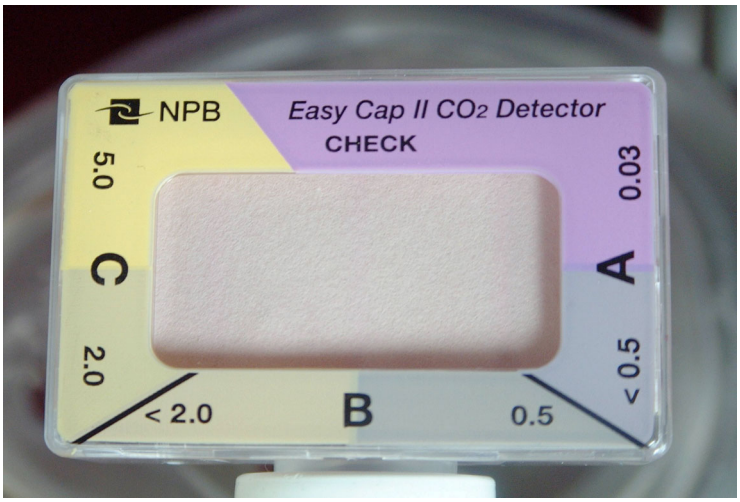

Figure 19. Carbon Dioxide Detector

Demand Valve

Another commonly available alternative to the use of the manual Ambu Bag for sufflation of air is the Demand Valve (see Figure 20):

Figure 20. Demand Valve

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Demand Valve Advantages

- High-pressure tubing connected to oxygen supply and activated by push button or lever
- Delivers 100% oxygen
- Easy to operate, manual trigger button
- Often equipped with inspiratory release valve which allows patient to breath

Demand Valve Disadvantages

- Does not provide a feel for chest compliance
- Barotrauma and gastric distention can be caused by overventilation
- Oxygen tanks quickly drain
- Should not be used with intubated patients

Circulation

If the emergency patient has a pulse, then circulation support in the form of external cardiac compressions or defibrillation need not be provided. However, it is important to have the ability to provide both in the patient with an airway emergency as cardiovascular events often accompany airway emergencies.

Airway Emergencies Most Likely To Be Encountered

Soft Tissue Obstruction (Including Laryngospasm and Anaphylaxis) – Soft Tissue Obstruction has already been covered in detail. The practitioner must appreciate that this is the most common cause of an obstructed airway, and is also the easiest to correct with proper positioning.

However, occasionally the soft tissue obstruction is not amenable to simple positioning alone. Such as is the case with edema of the soft tissue surrounding the airway as can occur with anaphylaxis, as well as the spasm of the vocal cords, termed laryngospasm.

Anaphylaxis is an acute systemic (multi-system) and severe Type I Hypersensitivity allergic reaction. Anaphylaxis occurs when a person is exposed to an allergen, to which they have already become sensitized. This can be a life-threatening event because of rapid constriction of the airway, often within minutes of onset, which can lead to respiratory failure and respiratory arrest. Brain and organ damage rapidly occurs if the patient cannot breathe.

Treatment involves administration of epinephrine IM (or IV infusion in severe cases), Benadryl IM, steroids such as Decadron, IV Fluid administration and in severe cases, pressor agents (which cause the heart to increase its contraction strength) such as Dopamine for hypotension, administration of

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oxygen and intubation during transport to advanced medical care. In severe situations with profuse laryngeal edema (swelling of the airway), a cricothyroidotomy may be required to maintain oxygenation.

Laryngospasm is an uncontrolled/involuntary muscular contraction spasm of the laryngeal cords. The condition typically lasts less than 30 or 60 seconds, and causes a partial blocking of breathing in, while breathing out remains easier. It may be triggered when the voice box or the area of the windpipe below the voicebox detects the entry of water or other substance. It is characterized by a high pitched scream-like sound, and might be frightening for some to witness. Some people suffer from frequent laryngospasms, whether awake or asleep.

Laryngospasm is treated by hyperextending the patient's head and administering mechanical ventilations with 100% oxygen. In more serious cases it may require intubation. If orotracheal intubation is not possible a cricothyroidotomy is done to create an airway.

Foreign Body Obstruction - Foreign bodies may cause acute airway obstruction. Recognition of the obstruction is the first step. An appropriate verbal response to questions indicates a patent airway in the awake patient. If the patient is unconscious, determine if there is air movement with respiratory effort, snoring, gurgling, or stridorous noises indicative of obstruction. If ***foreign body obstruction*** is the problem, techniques such as the ***Heimlich maneuver*** or suctioning of debris from the airway may be needed.

Physical Findings

- Patient cannot speak or breathe
- Patient makes the universal sign for choking
- Marked agitation
- May become unconscious (within 1 minute) unless the airway is re-established
- Death may result within 4-6 minutes without oxygenation

Treatment

- Positioning:
  - Conscious – sitting upright, or turned to the right side
  - Unconscious - supine
- Attempt Heimlich Maneuver
- Encourage patient to cough to dislodge obstruction

If unconscious:

- Use abdominal thrusts
- Activate EMS

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General Considerations

- Minimize risks of swallowing objects by tying dental floss:
  - Rubber dam clamps
  - Bite blocks
  - Gauze, cotton rolls
  - Small removable appliances and instruments

If a patient is only partially obstructed, and the patient is coughing or wheezing, and actively trying to dislodge the object DO NOT INTERVENE!

- Sit them up and encourage them to cough!
- Use the Heimlich Maneuver to attempt to dislodge the obstruction if coughing does not improve the situation
- If this is unsuccessful and the patient has lost consciousness, a cricothyrotomy may be a procedure of last resort to save the patient

If a patient is unconscious and the chest is moving, do not assume the obstruction has been removed - REPOSITION via chin-lift, head-tilt to alleviate tongue and soft-palate obstruction.

CNS Depression – The natural rhythmic breathing pattern can be disrupted by a depression of the ventilatory drive normally controlled centrally within the medulla. This depression can be the result of sedative drugs, trauma, or other phenomenon such as a cardiovascular or cerebrovascular event.

The fundamental airway management of these patients is no different from most emergency events in that the P – A – B – C – D protocol is followed. The one important difference is that when the possibility of respiratory depression from the administration of sedative drugs such as those from the opiate or benzodiazepine class exists, then the use of the reversal agents, naloxone and flumazenil respectively, is indicated.

Bronchospasm – Bronchospasm is a temporary narrowing of the bronchi (airways into the lungs) caused by contraction of the muscles in the lung walls, by inflammation of the lung lining, or by a combination of both. This contraction and relaxation is controlled by the autonomic nervous system. Contraction may also be caused by the release of substances during an allergic reaction.

The most common cause of bronchospasm is asthma, though other causes include respiratory infection, chronic lung disease (including emphysema and chronic bronchitis), anaphylactic shock, or an allergic reaction to chemicals.

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Treatment involves the administration of Beta2-agonists such as albuterol, which relax airway smooth muscle and may modulate mediator release from mast cells and basophils. Beta-agonist inhalers (bronchodilators) act to ease symptoms of asthma by relaxing muscles surrounding the walls of the bronchial tubes. In severe cases, epinephrine may be administered IM or IV as well.

Definitive Airway Management

Although the above methods have their uses, a definitive airway will be required in an emergency situation. This is usually accomplished by Emergency Medical Technicians (EMTs). Definitive **airway management** enables the caregiver to control ventilation, administer high levels of oxygen, and to some degree protect against, aspiration. There are three major methods of obtaining a definitive airway: (1) orotracheal intubation, (2) nasotracheal intubation, and (3) a surgical airway. In choosing between the nasal and oral routes of intubation, the clinician's skills and experience and the status of the patient should be taken into account.

Orotracheal Intubation

This method of establishing a definitive airway uses a laryngoscope to place an endotracheal tube directly into the trachea. Once in place, a bulb is inflated near the distal tip of the tube to help secure it in place and protect the airway from blood, vomit, and secretions (see Figure 21).

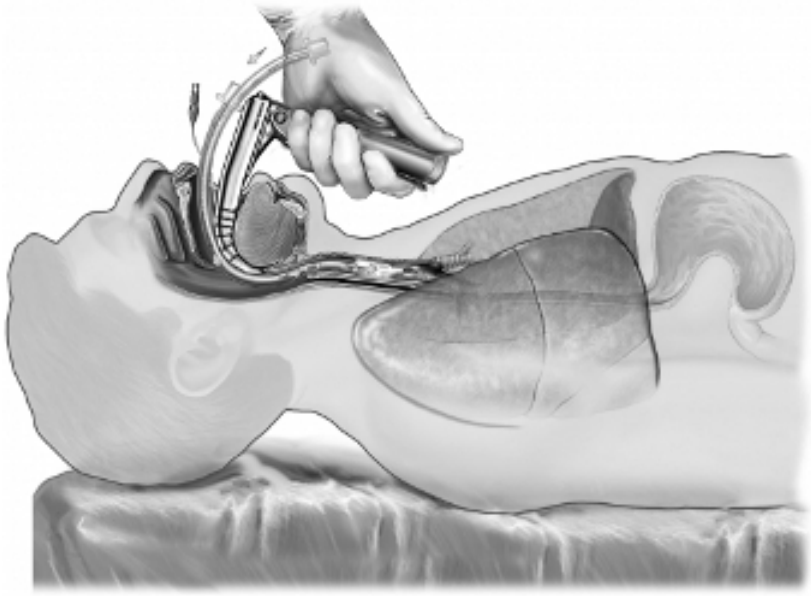

Figure 21. Orotracheal Intubation.

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Nasotracheal Intubation
An alternative to orotracheal intubation, nasotracheal intubation is similar except the endotracheal tube is inserted through the nose and nasopharynx instead (see Figure 22).

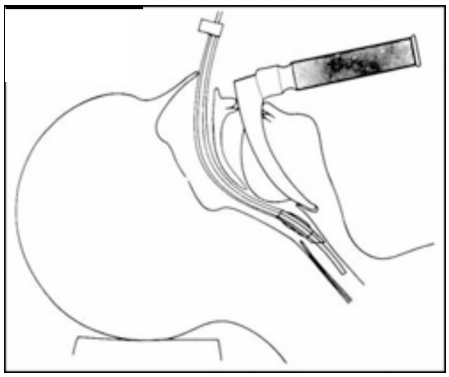

Figure 22. Nasotracheal Intubation.

Surgical Airway
Given the higher likelihood of foreign body obstruction in the dental sedation patient versus the general population, as well as the possibility that the foreign body will be lodged below the position of the vocal cords, and given the critical element of time in which a patent airway must be established, every practitioner should have a working knowledge of the surgical airway technique. The surgical technique easiest to perform with less accompanying post-surgical complications is the cricothyroidotomy (see Figure 23).

There are two basic types of cricothyroidotomy: Needle Cricothyroidotomy and Surgical Cricothyroidotomy. In a needle cricothyroidotomy, a syringe with a needle attached is used to make a puncture hole through the cricothyroid membrane that overlies the trachea. After the needle has reached the trachea, a catheter is passed over the needle into the windpipe and attached to a bag-valve device. In a surgical cricothyroidotomy, the doctor or other emergency worker makes an incision through the cricothyroid membrane into the trachea in order to insert a piece of tubing for ventilating the patient.

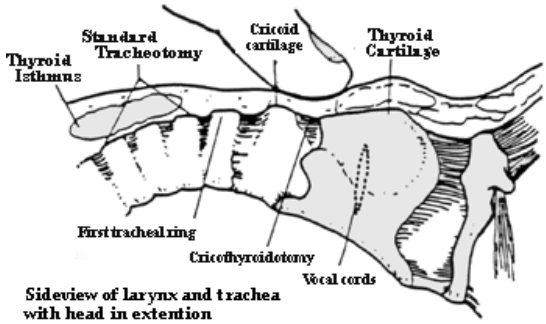

Figure 23. Location for the Cricothyroidotomy Technique.

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Positive Pressure Oxygen with Bag-Valve Mask Assembly Operating Instructions for 2 people

- Use only on unconscious patient demonstrating signs of respiratory distress
- Clear mouth and airway of visible foreign bodies
- Have assistant connect the oxygen reservoir system to 100% O2 and adjust flow to 15 liters per minute
- Connect the CO2 detector to the Bag-Valve Mask
- Position head for opening the airway
- Apply the mask firmly to the patient's face, placing the rounded cushion between the lower lip and chin, and the narrow cushion as high on the ridge of the nose as possible
- Hold the mask firmly against the face with the thumb and index finger, while keeping the chin and head tilted back with the other three fingers
- Have assistant slide his/her hand under the support strap.
- Squeeze the bag with both hands at a rate of 12 -15 times per minute for adults
- During insufflations observe the rise of the patient's chest.
- Release the bag abruptly and observe the lowering of the chest and the change in color of the CO2 detector
- If continued resistance to insufflations is encountered, check for airway obstruction or correct the backward held tilt
- If no resistance, but no chest movement or CO2 detector change, the reposition the mask

Airway Practical

Two Person Sequence

1. Confirm patient is unresponsive and not breathing
2. Activate EMS (911)
3. Request emergency O2, AED, and Emergency Kit
4. PABCD, D = Oxygen
5. If no pulse – AED*

*For the purposes of this exercise we are assuming no breathing, but the patient has a pulse

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DENTAL ORGANIZATION FOR CONSCIOUS SEDATION
RESPIRATORY ARREST & EMERGENCY OXYGEN DELIVERY

Supply List

All of the following items should be attached and/or connected to cart system within a supply bag

- Complete set of oropharyngeal airways
- CO_2 Detector (in foil pouch protected from exposure to air)
- Bag-Valve Assembly:
  - Tubing
  - Clear mask
  - Ambubag
  - One-way value
  - Reservoir bag

Equipment List

- Portable Positive Pressure Oxygen Unit:
  - E-Tank of Oxygen Unit
  - Wrench to open Oxygen Valve
  - Regulator
- Pulse Oximeter

Two-Person Emergency Oxygen Rescue Sequence

1) Confirm patient is unresponsive and not breathing
2) Activate EMS by calling 911
3) Begin Basic Life Support (BLS) following the PABCD protocol
   - Positioning
   - Airway
   - Breathing
   - Circulation
   - Definitive Care including defibrillation and emergency drugs (in this case oxygen)

4) If no pulse, then request AED and emergency kit. If there is a pulse, but no breathing, begin rescue breathing according to the following sequence:

   GREEN is for "Airway Manager"
   BLUE is for "Ventilator"

   A. Open the airway.
   B. Request Emergency Kit.
   C. Determine the proper size Oropharyngeal Airway by measuring from the corner of the mouth to the front of the ear lobe.
   D. Place the Oropharyngeal Airway by inserting it upside down, then rotating downwards after it reaches the soft palate.

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E. Open CO_2 Detector Pouch and place CO_2 Detector between Mask and Bag Assembly (press pieces together very firmly).
F. Hand bag-valve-mask assembly to Airway Manager.
G. Position mask over nose and mouth to create a seal while raising the chin.
H. Turn on Emergency Oxygen Valve and adjust Oxygen Flow to 15 Liters.
I. Remind “Ventilator” of the proper rate of once every four seconds.
J. Squeeze the Bag completely once every four seconds while observing both the rise and fall of the chest as well as the color change of the CO2 Detector.
K. Monitor the CO_2 detector to assure ventilation is occurring.
L. If the chest is not rising and falling with each manual ventilation and/or the CO_2 detector is not registering adequate ventilation via appropriate color change, then ask the airway manager to check mask seal, position of oropharyngeal airway, and raise the chin.
M. Reposition the mask to maintain a good seal.
N. Check for consciousness and spontaneous breathing after one-minute and every minute thereafter.

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TWO-PERSON POSITIVE PRESSURE OXYGEN DELIVERY FOR RESPIRATORY ARREST PRACTICAL

Seminar Location: _____ Date: _____

Name: _____

"Airway Manager"

	Position the patient and airway
	Place oropharyngeal airway
	Position mask over nose and mouth and create seal
	Remind "ventilator" of proper rate
	Reposition mask to maintain seal if necessary
	Monitor CO_2 detector to assure ventilation
	Halt insufflation after each one-minute cycle, monitor vital signs and check for consciousness

Notes:

"Ventilator"

	Select appropriate oropharyngeal airway and hand to "Airway Manager"
	Attach CO_2 detector to bag-valve-mask assembly
	Hand bag/valve/CO_2 mask assembly to airway
	Turn oxygen tank on and check pressure level of oxygen supply
	Adjust flowmeter to 25 liters/min.
	Squeeze bag once every 4 sec. w/ both hands w/ quick complete release
	Monitor rise and fall of chest

Notes:

_____ _____

DOCS FACULTY Date

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IV Initiation and Access

Rationale for IV Access During Sedation

1. Administration of medications
    a. Anesthetic agents
    b. Adjunctive agents
    c. Emergency drugs
2. Partial replacement of fluid lost to deficit

Catheters and Needles

1. Hollow metal needle
    a. Detached from syringe and left in vessel with IV line attached
    b. May perforate posterior wall of vessel
    c. Not routinely used unless only option available in an emergency
2. Winged needle (scalp vein needle)
    a. “Butterfly” 21g and 23g most often used in dentistry
    b. Wings taped down to stabilize needle
    c. May perforate posterior wall of vessel
3. Indwelling Catheter
    a. Synthetic catheter over needle
    b. Flexible so virtually no risk of perforation upon movement of patient

Safety needles now available that retrace into protective sheath

Needle Gauge	Color
14	Orange
16	Grey
18	Green
20	Pink
22	Blue
24	Yellow

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Catheter	Flow Rate (ml/min)
14 gauge 1.75 inch	330
16 gauge 1.16 inch	220
18 gauge 1.16 inch	105
20 gauge 1.00 inch	65
22 gauge 1.00 inch	35
24 gauge 0.75 inch	20

Which catheter size?

- Purpose of IV
  - Fluid resuscitation
    - 18 gauge or larger x2
  - Sedation
    - Usually 20 gauge or smaller
  - Maintenance line
    - Usually 18 gauge or smaller
- Age of patient
  - Elderly debilitated vs healthy adult

Which IV Solution?

For IV sedation we use an isotonic solution

- Maintains fluid homeostasis
- Deficit rule of thumb: Volume Loss in ml = (Body Weight [kg] + 40) X Time (hrs.) NPO

Hypotonic solution will cause cells to draw fluid

Hypertonic solution will draw fluid from cells

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SOLUTION	CONSTITUENTS	TONICITY
Normal Saline (NS)	Sodium- 154 mEq/l Chloride- 154 mEq/l	Isotonic
1/2 Normal Saline (1/2NS)	Sodium- 77 mEq/l Chloride- 77 mEq/l	Hypotonic
Dextrose 5% (D5W)	Dextrose- 278 mmol/l	Isortonic (become hypotonic as dextrose is metabolized)
Dextrose 5% in 1/2 NS	Sodium- 77 mEq/l Chloride- 77 mEq/l Dextrose- 278 mmol/l	Hypertonic
Lactated Ringers	Sodium- 130 mEq/l Chloride- 109 mEq/l Lactate- 28 mEq/l Potassium- 4 mEq/l Calcium- 3 mEq/l	Isotonic

Armamentarium

1. Drip set
    a. Surgical “macrodrip”
        i. 15 drops/ml
    b. Microdrip
        i. 60 drops/ml
        ii. Use for medication drips for precise dose
2. Gloves
3. Alcohol prep
4. Tourniquet
5. Dressing (such as Tegaderm)
6. Tape
7. Gauze
8. IV fluid

Mechanisms to prevent inadvertent administration of drugs:

- Different size syringes
- Color-Coded Labels

Anatomy of a vein

- •Thin wall as compared to an artery
- •May be difficult to locate if hypovolemic

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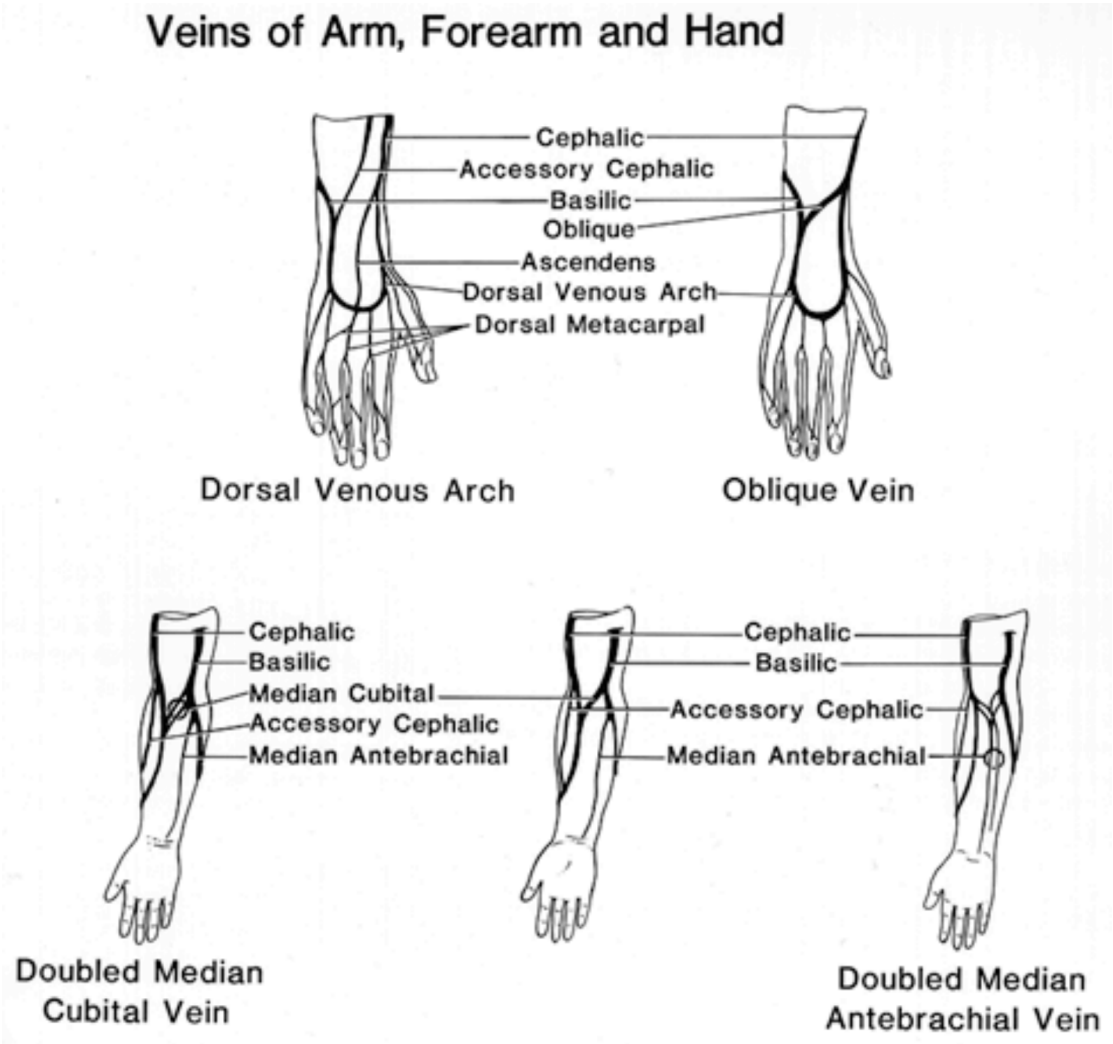
Anatomy Of Venipuncture



Selection of venipuncture site

- Condition of superficial veins
  - Tortuous, straight, hardened by age, scarred from previous use
- Relationship of vein to other anatomic structures
  - Nerves, arteries
- Duration of venipuncture
  - If greater than 2 hours want a site that allows freedom of movement
- Clinical status of the patient
  - Injury or disease present at site of venipuncture
  - History of phlebitis
- Age and size of the patient
  - Obese patients may have vessels that are difficult to locate
  - Young patients may have vessels that are difficult to locate
- Type of IV procedure
  - Irritating drugs

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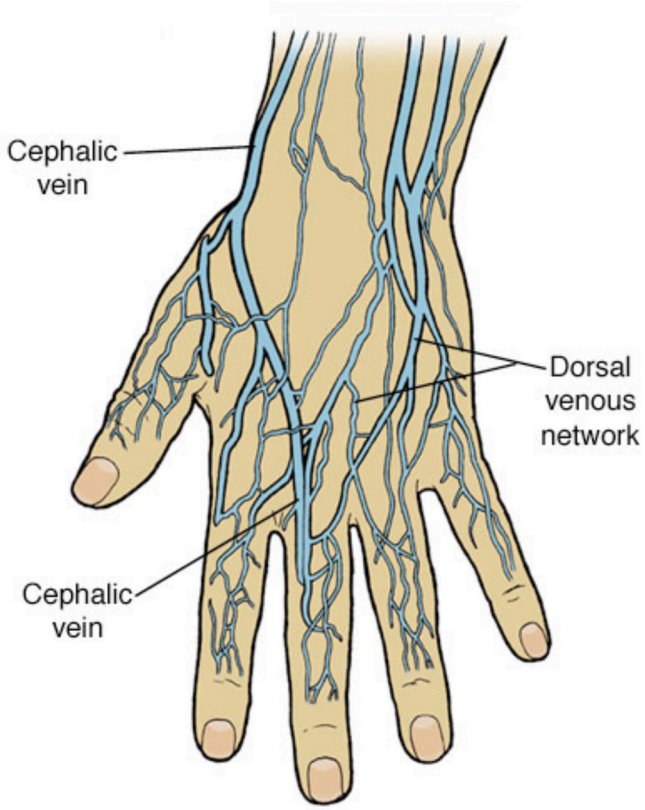
Comparison of Venipuncture Sites				
Preference	Site	Close to Important anatomy	Roll or vein Movement	Joint Immobilization
1	Dorsum of hand	No	Yes	No
2	Ventral forearm	No	No	No
3	Lateral antecubital	No	No	Yes
4	Dorsal wrist	No	Yes	Yes
5	Medial antecubital	Yes	No	yes

Dorsal Hand

- Usually 20 gauge or smaller
- Must find straight vein that is long enough to accept entire length of catheter
- Look for “anchored veins” that are less likely to roll
- Cephalic or Radial Vein= “intern’s vein”
    - Advantages – Superficial, visible, and of good size
    - Disadvantages – Rolls easily (mobile), Difficult to immobilize



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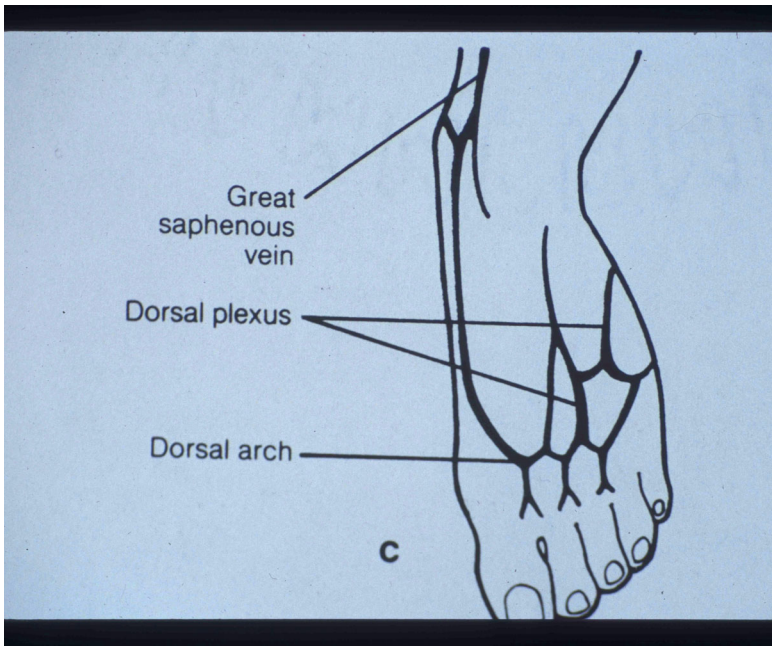
Dorsal Plexus or Great Saphenous Vein



- Advantages: Superficial, relatively large, anatomically safe, no need to immobilize
- Disadvantages: Accessibility limited, veins tend to roll
- Contraindicated: Diabetics, patients with poor circulation

Patient positioning

- Seated and/or reclined
- Immobilize extremity if necessary for comfort and safety
- Keep extremity in full extension to make the vein taut and keep low to engorge with blood
- Keep all armamentarium in close proximity

Site Preparation

First: Make yourself a better target:

- Apply tourniquet with knot away from IV site
- Hold arm, hand, or foot below heart level
- If in arm, have patient open/close hand

If you still don't see veins…

- Try "pooling" – Move tourniquet from a/c to forearm, then wrist
- Light slapping or rubbing of the skin ("bad vein"!)
- Anything to produce heat: warm, moist towel
- Use nitrous oxide – causes peripheral vasodilation

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Prior to insertion of catheter

- Clean site with alcohol wipe
- Start at site and use circular motion
- Let dry completely before puncture

Insertion of catheter

1. Apply distal traction to vein using non-dominant thumb
2. Insert needle bevel up at 15-30 degrees
3. Penetrate skin then vessel and look for blood return (a sudden lack of resistance is felt as the catheter enters the vein)
4. Lower angle of catheter and advance needle as close to parallel as possible

This is the “Indirect Method” – First pierce the skin, then reduce angle, and advance the catheter into the vein

The alternative is the “Direct Method” – Hold catheter with bevel up at 15-20 degree angle and thrust the catheter through the skin and into the vein in one thrust. This method is preferred for large veins and indirect method for smaller veins.

5. Ensure catheter is in vessel
6. Hold hub and retract needle while advancing catheter with index finger
7. The hub should abut skin when finished
8. Apply pressure above hub to halt flow of blood through catheter

Inability to advance catheter:

•Try to pull the vein a bit tauter and advance the catheter.

•If unsuccessful, traction can be reduced (or even released) and further attempts at advancement can be pursued.

•"Float" the catheter in.

> •If the catheter can be partially advanced but meets resistance before insertion is completed, infusing saline through the line during advancement may facilitate passage.
>
> •Excessive pressure should not be used in order to prevent infiltration.
>
> •Secure the catheter while it is partially inserted.

•If a long catheter is used (1" or longer) it may be possible to have a patent line with only part of the catheter in the vein.

> •This is not ideal and should not be relied upon unless other access is not available.

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•If this method is chosen, free flow of fluids and absence of infiltration must be assured, and extra care should be taken in securing the line.

Two points deserve special consideration:

1.First, it is the plastic catheter, NOT THE NEEDLE, that should be advanced into the vein.

•If the sharp tip of the needle is the leading point, vessel injury and resultant infiltration is likely.

2.Secondly, the catheter should never be forced into the vein.

•If resistance is met, do not advance the catheter

“Finishing the Job”

Attach IV line, remove tourniquet, assure free flow of fluid without infiltration, and secure catheter in place with tape or dressing.

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IV Sedation Protocols For The Outpatient Dental Setting

Factors to consider when choosing an IV sedation protocol:

- Sedation/Analgesia
- Appointment Length
- Drug Interactions

Sedation/Analgesia Requirements

IV sedation allows for both sedation (to address anxiety), and analgesia (to address existing and/or anticipated discomfort) objectives to be accomplished. Depending on the patient's needs, one or both objectives may be required. Since accomplishing both objectives may require multiple medications, and it is always safest for the patient to limit the number of medications to only those that are necessary, it is best to determine ahead of time what the anticipated need of the patient will be.

Sedation may be accomplished through the administration of several different medications within multiple classes, including barbiturates, opioids, and benzodiazepines, among others. Benzodiazepines, like midazolam, combine efficacy, high therapeutic index, minimal side effects, reversibility, quick onset of action, and desirable half-lives. For these reasons, the preferred class of medications for outpatient intravenous conscious sedation to accomplish sedation are the benzodiazepines (eg. midazolam and lorazepam).

Analgesia is often accompanied by sedation. For example, opioids such as fentanyl, have sedative qualities, although in doses used in the outpatient setting, their primary attribute is analgesia. The benzodiazepines, in conscious sedation doses, have no appreciable analgesic effects.

Appointment Length

Among the benefits of IV sedation compared to other methods of administration, is quick onset of action. Drugs that enter the general circulation quickly are also susceptible to rapid metabolism and elimination. This shorter duration of action can be a desirable quality in that the recovery period is shortened.

By the same token, the shorter duration of action when the provider of treatment is also the anesthesia provider, means more frequent dosing, which can be an inconvenience to the provider resulting from frequent interruptions to the delivery of care. This inconvenience becomes more significant the greater the length of the appointment.

In addition, more frequent re-dosing of shorter acting drugs means a greater amount of drug will be administered over the course of a longer appointment. Given this consideration, the published or generally accepted maximum amount of that drug may be reached before the planned treatment is completed. In such cases, longer acting drugs can be utilized to avoid this potential complication.

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In addition to using longer acting intravenous agents, the anesthesia provider also has the option of combining different modes of administration to lengthen the anesthesia. For instance, oral (swallowed and sublingual) administration results in longer duration of action. Combining both the oral and IV routes of administration has the advantage of reducing the frequency of IV dosing, among others.

Drug Interactions

Interactions of a significant degree should cause the anesthesia provider to pause and consider the ramifications of administering a drug that will increase the potential for side effects. The degree of significance can be determined via the vast array of resources now available to the healthcare professional and includes pharmacology software.

It should be noted that many adverse drug interactions are lessened with IV administration due to the ability to titrate intravenous solutions.

Other Considerations

The Hypo-responder

- CYP 450 induction
- History of drug and alcohol abuse

Adjunctive Sedation Methods

- Use of Nitrous Oxide during IV initiation
- Use of oral triazolam pretreatment
- Sedative hypnotic taken the night before the procedure

Nitrous Oxide

- Beneficial effects during IV initiation
- Sedation
- Vasodilation
  - Via a second messenger - “Possible mechanism behind the vasodilating effect of nitrous oxide in the human brain”, The Internet Journal of Anesthesiology™ ISSN: 1092-406X
  - Results and conclusion: N_2O inhalation elevated the plasma concentration of cGMP. (A smooth muscle relaxant)
  - This suggests that N_2O mediates at least part of its vasodilating effect through an increase in cGMP.
- MAC- minimum alveolar concentration 107%
- When given in combination with benzodiazepines IV or PO the MAC is lowered
- The patient may easily progress to deep sedation or general anesthesia

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Benefits of Oral Sedation With Benzodiazepines Prior To IV Sedation

"Oral Triazolam Pretreatment for Intravenous Sedation" Stopperich, et. al. 1993

- 22 third molar extraction cases
- 0.25 mg triazolam po or placebo 45-60 minutes before venipuncture
- Prior to venipuncture anxiety was quantified using three common scales

Results

- All patient has similar baseline anxiety levels
- Triazolam patients:
  - Had significantly decreased anxiety at the time of venipuncture.
  - Dose requirements for IV sedation medications were also decreased
  - The venipuncture experience was reported to be significantly less unpleasant
  - Global ratings of the surgical experience also favored triazolam

Protocols

Objectives

1. Step wise procedure to provide safety, efficacy, and patient comfort
2. Separate protocols for:
   a. Sedation
   b. Sedation and Analgesia
   c. Extended Sedation (Avoids CYP Inhibition/Induction)
   d. Extended Sedation and Analgesia
   e. Combination protocols with IV, Oral agents, and Nitrous Oxide/Oxygen

Case #1

- 35 year old female
- Has not had a dental visit for 10 years.
- Has as deep carious lesion on #12 with obvious pulpal involvement. She is quite anxious about any treatment and would like to be "very relaxed" for the procedure
- No medications
- Vital signs normal and no significant medical history
- You expect the procedure (RCT #12, P & C, Crown Coverage) to last 2 hours

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Protocol #1 – Objective - Sedation For Non-Invasive Procedures Four Hours Or Less

Night Before Dose

Diazepam at bedtime 0-10 mg
Based on patient age, BMI, history and existing medications

Age & Night Before Diazepam

- 65 years of age
- All things considered equal, cut dose in half
- For elderly patients will be no greater than 2.5 mg

BMI & Night Before Diazepam

- Weight does not tell the whole story
- % Body Fat is the issue
- 30 BMI = Obesity
- For most obese patients will be 10 mg

WEIGHT lbs	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215
kgs	45.5	47.7	50.0	52.3	54.5	56.8	59.1	61.4	63.6	65.9	68.2	70.5	72.7	75.0	77.3	79.5	81.8	84.1	86.4	88.6	90.9	93.2	95.5	97.7
HEIGHT in/cm	Underweight						Healthy					Overweight					Obese							Extremely obese
5'0" - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
5'1" - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	36	37	38	39	40
5'2" - 157.4	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	33	34	35	36	37	38	39
5'3" - 160.0	17	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	32	32	33	34	35	36	37	38
5'4" - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37
5'5" - 165.1	16	17	18	19	20	20	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35	35
5'6" - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	25	26	27	28	29	29	30	31	32	33	34	34
5'7" - 170.1	15	16	17	18	18	19	20	21	22	22	23	24	25	25	26	27	28	29	29	30	31	32	33	33
5'8" - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	32	32
5'9" - 175.2	14	15	16	17	17	18	19	20	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	31
5'10" - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	23	24	25	25	26	27	28	28	29	30	30
5'11" - 180.3	14	14	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	25	26	27	28	28	29	30
6'0" - 182.8	13	14	14	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29
6'1" - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28
6'2" - 187.9	12	13	14	14	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27
6'3" - 190.5	12	13	13	14	15	15	16	16	17	18	18	19	20	20	21	21	22	23	23	24	25	25	26	26
6'4" - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	22	22	23	23	24	25	25	26

History & Night Before Diazepam

- Prior documented hyporesponse experience
- For most hyporesponders will be 10 mg
- For most hyper-responders will be 2.5 mg

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Medications & Night Before Diazepam

- 3 or more CNS depressants/day – Forego night before diazepam
- Insomnia drug at bedtime – Forego night before diazepam
- [D] or [X] Interaction between diazepam & existing medications – Forego night before diazepam
- 2 CNS depressants/day – use 2.5 mg diazepam

1 CNS depressant/day – use 5 mg diazepam

Other Considerations & Night Before Diazepam

- Patient needs to drive to the sedation appointment – Forego night before diazepam
- Patient needs to work prior to sedation appointment – Forego night before diazepam

Amount of Night Before Dose of Diazepam Relative to Patient Characteristics

Factor \ Dose	0 mg	2.5 mg	5 mg	10 mg
Age	↔	≥ 65	NO	NO
BMI	←			≥ 30
History	↔	Hyper-response	Normoresponse	Hyporesponse
Existing Medications	≥ 3 CNS Drugs &/or Insomnia Drug, or [D] or [X] Interaction between DZP & existing medication	2 CNS Drugs	0-1 CNS Drug ↔	
Other	Patient drives &/or works on the day of prior to the sedation appointment	NO	NO	NO

In Office Day Of The Appointment

1. Assessment upon arrival – verify compliance with pre-operative instructions including NPO
2. Initiate O_2 via nasal hood
3. N_2O/O_2 administration via appropriate method. Discontinue after IV access
4. Establish IV line with D5W, normal saline, or lactated ringers solution
5. Administer 1 mg of Midazolam IV
6. Evaluate every 3 minutes, and titrate 1-2 mg of Midazolam IV to effect, constantly monitoring patient
7. Administer local anesthesia

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Case #2

- 48 year old male patient
- Vital signs normal
- Meds: HCTZ 25 mg daily
- NKA
- No other significant history
- Recurrent decay under 4 unit bridge 18-21
- Non restorable abutments require extraction (18,20,21)
- Requests IV sedation

Protocol #2 – Objective - Sedation For Invasive Procedures Four Hours Or Less

- Two agents
- Midazolam and Fentanyl
- Useful for surgical cases or when pain and inflammation expected
- Up to 4 hours duration
- Extra caution with multiple agents
- Analgesia AND sedation from Fentanyl

Night Before - Diazepam at bedtime 0-10 mg, based on patient age, BMI, history, and other medications

In-Office Day Of The Appointment

1. Assessment upon arrival – verify compliance with pre-operative instructions including NPO
2. Initiate O_2 via nasal hood
3. N_2O/O_2 administration via appropriate method. Discontinue after IV access
4. Establish IV line with D5W, normal saline, or lactated ringers solution
5. Administer 1 mg of Midazolam IV
6. Evaluate every 3 minutes, and titrate 1-2 mg of Midazolam IV to effect, constantly monitoring patient
7. Administer local anesthesia
8. Prior to invasive procedure, administer Fentanyl 25-50 mcg
9. Evaluate every 3 minutes, and titrate 25-50 mcg to effect, constantly monitoring patient

Why do we use an opioid if…?

- We are using local anesthetic, which is quite effective
- We are achieving adequate sedation with midazolam
- The analgesic duration of fentanyl is limited (~1 hour)
- Opioids are devoid of amnesic effects

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Case #3

- 65 year old female
- Type IV periodontal disease
- Takes Synthroid, Prilosec, Daily Vitamins
- ASA II
- Requires 4 quadrants of periodontal surgery, extraction of 2 partially bony impacted 3rd molars
- Requests sedation

Protocol #3 – Objective – Extended Sedation For Procedures 3 Hours Or Longer

- Useful when prolonged procedures are planned that may extend beyond three hours.
- Useful with hyporesponders who require more frequent dosing
- Useful with patients taking medications that have significant interaction with protocol #1 and/or protocol #2 meds
- Utilizes IV Lorazepam – longer onset of action and half-life compared to Midazolam

Night Before - Diazepam at bedtime 0-10 mg, based on patient age, weight, history, and other medications

In Office Day Of The Appointment

1. Assessment upon arrival – verify compliance with pre-operative instructions, including NPO
2. Initiate O_2 via nasal hood
3. N_2O/O_2 administration via appropriate method. Discontinue after IV access
4. Establish IV line with D5W, normal saline, or lactated ringers solution
5. Administer 2 mg of Lorazepam IV
6. Evaluate every 5 minutes, and titrate 2-3 mg of Lorazepam IV to effect, constantly monitoring patient
7. Administer local anesthesia

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Case #4

- 38 y.o. female
- Fibromyalgia
- Takes Lyrica
- Wants sedation for extraction of remaining 6 teeth and placement of 10 implants
- ASA III

Protocol #4 – Objective – Extended Sedation For Invasive Procedures 3 Hours Or Longer

- Useful when prolonged procedures that include invasive treatment are planned that may extend beyond three hours.
- Useful with hyporesponders who require more frequent dosing
- Useful with patients taking medications that have significant interaction with Midazolam
- Utilizes IV Lorazepam – longer onset of action and half-life compared to Midazolam
- Two agents
- Lorazepam and Fentanyl
- Extra caution with multiple agents
- Analgesia AND sedation from Fentanyl

Night Before - Diazepam at bedtime 0-10 mg, based on patient age, weight, history, and other medications

In-Office Day Of The Appointment

1. Assessment upon arrival – verify compliance with pre-operative instructions including NPO
2. Initiate O_2 via nasal hood
3. N_2O/O_2 administration via appropriate method. Discontinue after IV access
4. Establish IV line with D5W, normal saline, or lactated ringers solution
5. Administer 2 mg of Lorazepam IV
6. Evaluate every 5 minutes, and titrate 2-3 mg of Lorazepam IV to effect, constantly monitoring patient
7. Administer local anesthesia
8. Prior to invasive procedure, administer Fentanyl 25-50 mcg
9. Evaluate every 3 minutes, and titrate 25-50 mcg to effect, constantly monitoring patient

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Combination Oral/IV Protocols On Day Of Appointment

There are advantages to combining oral sedation with intravenous sedation that include:

1. Reducing the amount and frequency of intravenous agent administered
2. Prolonging the duration of the sedation action intra-operatively and post-operatively
3. Reducing stress on the patient who has a needle phobia
4. Reducing stress on the IV administrator
5. Making IV access easier

When oral and intravenous sedation is combined, the IV protocols listed earlier in this section remain the same except they often involve lower doses of the IV agent (and oral compared to oral only), and less frequent dosing. From a practical standpoint, intravenous access is delayed relating to IV only protocols, as the slower onset of action of oral sedatives is accounted for.

The oral sedation agents include Triazolam (Halcion), Lorazepam (Ativan), and Hydroxyzine (Atarax or Vistaril). The decision to use one or more of these agents is derived from their pharmacotherapeutic properties.

Triazolam – The benzodiazepine, triazolam, has a long track record of safe and effective sedation in dentistry. It is a short-acting, potent, highly efficacious sedative/anxiolytic agent with a high degree of amnesia and therapeutic index. Its “on-label” application is for night-time sleep and preoperative sedation in adults 18 years and older. When combined with IV sedative/analgesia, it is administered to the patient who is NPO for six hours prior to the appointment in doses of 0.125 – 0.25 mg an hour prior to arrival at the office, and in doses of 0 – 0.25 mg every 20 minutes until the desired level of sedation is achieved – usually anxiolysis. This is followed by IV access and IV drug administration.

Lorazepam – Lorazepam, like triazolam and midazolam, comes from the benzodiazepine family. Unlike triazolam, lorazepam is a mid-acting sedative, with slightly less efficacy and amnesia. Other than its longer duration of action, compared to triazolam, it has been studied for safety and efficacy by the FDA for children aged 12 and older. The other major difference between lorazepam and benzodiazepines like triazolam and midazolam, is the different metabolic pathway. Lorazepam is not metabolized via oxidative metabolism by cytochrome enzymes, but rather is conjugated with glucuronic acids to more polar compounds that are then excreted. This quality of lorazepam makes it useful to administer in patients who take medications that induce or inhibit CYP-450 enzymes.

Lorazepam is administered like triazolam in these combination oral/IV protocols, except the dosage amount is different. The patient should take 1-2 mg one hour prior to the appointment, and upon arrival is assessed for 0-2 mg every 20

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minutes until the desired level of sedation Is achieved – usually anxiolysis. This is followed by IV access and IV drug administration.

Hydroxyzine – Chemically, hydroxyzine is a diphenylmethane. Pharmacologically, it is an antihistamine – specifically, a H_1-Receptor Antagonist. While having short-acting sedative/anxiolytic characteristics, hydroxyzine also has strong anti-emetic properties. Unlike triazolam and lorazepam, hydroxyzine has no amnesic properties, and there is no reversal agent for it. Because of this, its use is restricted in these protocols to a single dose in the range of 25-100 mg. Its usefulness is in patients who have trouble breathing through their nose, patients who commonly experience nausea and vomiting with medication, severe gaggers, and the nicotine addict.

Labeling the Combination Oral/IV Protocols

The IV protocols are labeled 1-4 based on the objective of the anesthesia. When combined with oral administration the numbered protocols are followed by the "O" letter. For example, if IV sedation protocol #1 were combined with an oral sedation protocol that includes any of the following combinations of oral sedatives – Triazolam, Lorazepam, Triazolam/Hydroxyzine, Lorazepam/Hydroxyzine, then the protocol would be labeled "IV sedation protocol 1-O". Where as IV sedation protocol #3 combined with oral sedation would be labeled "IV sedation protocol 3-O".

Important point to remember about the protocols

- It is easier to administer additional increments to the patient than reverse what has already been given – When in doubt about dosing, Dose Low & Slow

Which IV Solution?

- Blood tonicity is 280-303 milliosmoles/l
- Fluid in the range of 240-340 mOsm/l are considered to be isotonic
- >340 mOsm/l hypertonic
- <240 mOsm/l hypotonic
- We use isotonic solutions for IV Sedation

Characteristics of IV Fluids

- Isotonic- stay in vasculature
- Hypertonic- pull fluid from the cells
- Hypotonic- flow into the cells

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Characteristics Of Intravenous Solutions			
Solution	NS	Lactated Ringers	D5W
mOsm/l	308	272	280
Na^+	154 mEq	130 mEq	0
Cl^-	154 mEq	109 mEq	0
K^+	0	4 mEq	0
Ca^{++}	0	3 mEq	0
Mg^{++}	0	0	0
Lactate	0	29 mEq	0
Dextrose	0	0	50 g/l

Relative Maximum Doses of Drugs

When sedation drugs are administered intravenously, they can be titrated to effect, allowing for the considerable variability within the general population, and permitting the anesthetist to better achieve the intended level of sedation. The overall amount of drug necessary to achieve this level of sedation as well as maintain it over time will vary considerably. It is with this knowledge of wide variability between individuals and within individuals depending on length of sedation required that the following Relative Maximum Doses of Drugs are given. The amounts are meant to be suggested standard limitations and not meant to be interpreted as absolute allowable maximums.

Drug in IV Protocols 1-4	Maximum Dose	Maximum Total
Diazepam (oral)	10 mg	20 mg
Fentanyl (IV)	50 mcg	100 mcg
Hydroxyzine (oral)	100 mg	100 mg
Lorazepam (IV)	2 mg	8 mg
Lorazepam (oral)	2 mg	4 mg
Midazolam (IV)	2 mg	15 mg
Nitrous Oxide	30%	30%
Triazolam (oral)	0.25 mg	0.75 mg

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All Patients Are Told Prior To IV Sedation:

- NPO 6h, except for water with meds
- No OTC sedatives 24 hours before/after sedation appointment
- No chance of pregnancy
- Must have a responsible person take them home
- Must have a responsible person both bring and take them home if sedative pre-medication is taken
- No contact lenses
- No driving for 24 hours
- Nor operating hazardous devices for 24 hours
- No heavy lifting for 24 hours
- No stairs for 24 hours
- No important decisions for 24 hours

The Diabetic Patient & IV Sedation

- HbA1c should be <7%, HbA1c > 8.5% are poor risk for surgery
- Patient should be NPO 6h, except for water with meds
- Patient should withhold insulin and other diabetic medications
- Use D5W
- Check blood glucose baseline, prior to, during (q1h), and after appointment
- Keep appointment 3 hours or less

Adjuncts To IV Sedation Procedures

- Rubber Dams and other forms of isolation are standard of care to protect the IV sedation patient's airway from foreign bodies
- Mouth Props are required to hold the patient's mouth open, while allowing the patient to rest their jaw without the fatigue that would accompany a long appointment
- Molt Mouth Gag instruments can be useful in assisting the sedation patient in opening wider
- Cheek retractors can be used to retract soft tissue and serve as a reminder to open wide enough when rubber dams or Iso-Lites are impractical, such as during a full-arch impression
- Chair Pads offer comfort during sedation and assistance in keeping the patient's head and airway properly positioned
- The Companion Chair (wheel chair) provides a means to safely transport the sedation patient to the departing vehicle following the sedation procedure
- Pill Crushers provide a means to improve absorption by increasing the surface area exposed to the mucosa

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IV Sedation Complications

LOCAL COMPLICATIONS

Non-Running IV Solution

- IV fluids not running downhill enough
- Bevel of needle against wall of vein
- Tourniquet left on arm

Hematoma

- Most common complication of venipuncture
- Extravasation of blood from punctured vessel into surrounding tissue
- More common in the elderly with more friable vessels
- Unable to seal after initial puncture (blood thinning agents)
- Occurs after removal if inadequate pressure applied
- Prevention & Treatment:
    - Remove tourniquet to decrease venous pressure
    - Remove needle and apply pressure with gauze

Venospasm

- Spasmodic constriction of a vein
- Protective mechanism
- As needle nears vein, it appears to disappear or collapse
- May feel a burning sensation
- Resolves without treatment
- Do not withdraw needle if not in vessel, attempt to re-locate vessel

Infiltration

- Similar to hematoma but IV fluid instead of blood
- Cause is perforation of vein wall
- Painless colorless swelling
- Occurs after IV drip turned on
- IV fluids we use are isotonic so less irritation
- Place gauze at site and hold pressure

Prevention:

- Make sure needle/catheter is in vein
- Press rubber flash bulb to see blood return prior to turning drip on

Extravascular Drug Administration

- Actual drug injected into subcutaneous tissues
- Pain

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- Drug essentially administered SQ
- Delayed absorption of drug
    - Delayed onset
    - Prolonged sedation
- Tissue Damage
    - Localized to area of injection
    - Most serious tissue damage with diazepam, promethazine and pentobarbital
    - If drug causes vasoconstriction could get tissue sloughing

Causes of Extravascular Drug Administration

- Needle not in vein
    - Needle slips out of vein
    - Needle pierces vein and exits other side
- Vessel leaks with rapid hematoma formation

Prevention:

- Squeeze flashbulb to make sure in vein
- Run constant drip – as long as there is a drip, needle is in vein
- Observation - will see tissue swelling if outside of vessel

Recognition:

- Patient complains of burning and intense pain that does not migrate down arm
- Swelling at injection site

Management of Extravascular Drug Administration

- If small amount of drug injected remove needle and apply pressure
    - Stops bleeding and disperses drug
    - If less than 2ml this is all that needs to be done
- If large volumes administered (very unlikely) consider ER consult and administration of local anesthetic without vasoconstrictor and topical nitroglycerin paste
- Monitor depth of sedation in patient

Intra-Arterial Injection

- Most significant localized complication
- Very infrequent occurrence - Difficult to enter an artery

Prevention

- An artery has a pulse
- Tourniquet can mask this pulse
- Careful in medial antecubital area where artery may be superficial

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Signs & Symptoms

- **MOST IMPORTANT SIGN: Upon removal of tourniquet, the blood will pulsate into the IV tubing because of greater pressure**
- Arteries have more spastic activity than veins and more intense burning of penetration by needle
- If artery penetrated more intense inflow of blood AND different color!

Treatment

- Immediate treatment necessary
- Irritation of arterial wall occurs
- Spasm causes decrease in blood supply
- Goal is to prevent tissue damage and gangrene
- If no drug is administered, no damage done
- Remove needle and apply pressure for 10 minutes.

If drug administered LEAVE NEEDLE IN PLACE

- Will be used to administer treatment

Recognition of IA Injection

- Severe pain radiating from injection site to tips of fingers
- Check radial pulse
- If absent, immediate management necessary
- Compare skin color of affected hand with opposite hand
- Compare temperature of limbs
- Major cause of injury is chemical endarteritis resulting in thrombosis and ischemia
- Small areas of gangrene or loss of fingers or limb

Management of IA Injection

- Leave needle in place
- Administer procaine

  1%, 2-10ml into artery

  - Anesthetic/Vasodilator/Diluent
  - Breaks spasm
  - Mandatory Hospitalization!!
  - Accompany patient to hospital

Thrombophlebitis

- Inflammation of vein wall and thrombus formation

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Clinical Features of Thrombophlebitis:

- Edema
- Inflammation
- Tenderness
- Delayed onset
    - 24-48 hours afterwards but may be up to a week later

Thrombophlebitis Causes

- pH of infusion liquid
- Components of preparation
- Duration of IV infusion
    - Concern in hospital
- Mechanical factors
    - Bevel and dull needle
- Venipuncture technique
    - Improper fixation of needle
- Size of needle in relation to vein caliber
- Type of needle
    - Metal vs catheter
- Presence of infection or disease
- Age of patient
- Site of venipuncture
    - Leg has higher incidence

Prevention

- Use sharp sterile needles
- Follow atraumatic, sterile venipuncture technique
- Secure the needle firmly in position
- Dilute drugs when possible
- Use larger veins

Management

- Patient returns to office for evaluation
- Limit activity of the limb (sling)
- Elevate limb
- Moist heat 3 to 4 times daily
- NSAIDs
- Antibiotics and anticoagulant usually NOT part of therapy unless condition worsens
- Refer to vascular surgeon
- Usually resolves in 3-4 weeks

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SYSTEMIC COMPLICATIONS

Air embolism

- Extremely unlikely
- Eliminate air bubbles from syringes and IV lines
- Monitor IV bag and do not allow to become empty
- Small bubbles absorbed by blood
- Patient can tolerate 1ml/kg of air in venous circulation

In unlikely event place patient on left side to avoid lungs and head down to avoid brain.

Over-hydration

- Unlikely as well
- Too much solution
- Morel likely in the very young and the patient with CHF

Rule of thumb:

- Dose should not exceed 1.5x time NPO (hrs) X pt. wt. in kg
- Example: 50kg NPO 6 hours -1.5 x 6 x 50 = 375 ml

Nausea and Vomiting

- Most likely with opioids
  - Dosage dependent
  - Ambulatory greater than non-ambulatory
- Frequent changes in position
- Hypoxia
- Swallowing blood
- Main risk is aspiration of vomitus into tracheobronchial tree

Vomiting - Prevention

- Patient should be NPO
- During procedure protect airway
- At home patient warned not to be supine to avoid aspiration

VOMIT – Acronym for causes of nausea and vomiting

- **V**estibular
- **O**bstruction of bowel
- Dys**M**otility of upper GI
- **I**nfection and Inflammation
- **T**oxins stimulating the CTZ

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PONV secondary to Vestibular input

- Promethazine (Phenergan) **-** Dosage 6.25 – 25 mg IV, 12.5-25 mg oral
- Diphenhydramine (Benadryl) **-** Dosage 25-50 mg IM/IV
- Scopolamine Patch (Transderm Scop) – Apply one patch night before

PONV secondary to Obstruction of bowel and constipation

- Dulcolax - 5-15 mg PO single dose, or 10 mg suppository single dose

PONV secondary to DysMobility of upper gut

- Metoclopramide (Reglan) – 20 mg IM/IV at end of procedure

PONV secondary to Infection and Inflammation

- Promethazine (Phenergan) **-** Dosage 6.25-25 mg IV, 12.5-25 mg oral
  or,
- Diphenhydramine (Benadryl) **-** Dosage 25-50 mg IM/IV

PONV secondary to Toxins stimulating the CTZ

- Prochlorperazine (Compazine) – 5-10 mg PO q4-6h as needed
- Droperidol – 0.625-2.5 mg IV at end of procedure
- Ondansetron (Zofran) – 4 mg IV at end of procedure

Which of the following agents would be most helpful in treating nausea associated with fentanyl administration?

A. Promethazine (phenergan)

B. Ondansetron (Zofran)

C. Prochlorperazine (Compazine)

D. Metoclopramide (Reglan)

E. Both B and C above

Which of the following agents would be most helpful in treating nausea associated with airsickness?

A. Promethazine (Phenergan)

B. Scopolamine Transdermal Patch (Transderm-Scop)

C. Prochlorperazine (Compazine)

D. Metoclopramide (Reglan)

E. Both A and B above

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A patient presents with nausea and reports that she feels "full" after eating only a few spoonfuls of her lunch. Which agent is most appropriate for treating this patient?

A. Promethazine (Phenergan)

B .Scopolamine Transdermal Patch (Transderm Scop)

C. Prochlorperazine (Compazine)

D. Metoclopramide (Reglan)

E. None of the above

Localized Allergy

- Narcotics release histamine as a normal pharmacological response.
  - This resolves within a few minutes and is not a true allergic reaction
- Tape allergy also has been reported.
  - Localized to area around tape
  - Should use hypoallergenic tape

Drug allergies should be managed vigorously

- Epinephrine 0.3-0.5 mg SQ or
- Epinephrine 1:10,000 3-5 ml IV
- Prevention is of utmost importance

Respiratory Depression

- Discussed in emergency lectures
- PABCD
  - BLS protocol
- Reversal agents

Emergence Delirium

- As patient emerges from sedation experiences:
- Delirium, hallucination, anxiety, or rage
- May be associated with recall of an unpleasant life experience
- Rapid emergence and disinhibition

Drugs most often implicated:

- Benzodiazepines
- Scopolamine- most often
  - Avoid in patients younger than 6 and older than 65
  - Not part of our protocol
- Ketamine

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Treatment:

- If suspect disinhibition consider reversal with flumazenil
- If rapid emergence consider opioid or benzodiazepine

Laryngospasm

- Protective reflex seen mainly in deeply sedated patients
- In moderate sedation, protective reflexes intact so normally do not see this
- May be provoked by stimulation of larynx by fluids or foreign substances
- COMPLETE RESTRICTION OF AIR FLOW

Management

- Removal of any offending foreign material
- Use large suction - This may break spasm
- Positive pressure O2 - Force O2 past vocal folds
- As level of CO2 increases consciousness decreases and laryngospasm will break
- Skeletal muscle relaxant
    - Not recommended for the untrained
    - Not recommended for our purposes

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Challenges to IV Sedation

Given the wide variation in patient population, the IV sedation clinician will face patients that present with challenges to the **primary requirements of IV sedation**, which are:

1. **Safety**
2. **Efficacy**

These two requirements can be closely related. Making the sedation procedure safer can decrease efficacy. Conversely, increasing efficacy can decrease safety.

Being prepared for **challenges to either safety or efficacy** in advance will allow the clinician a better chance to address and overcome them. While this is not an exhaustive compilation of potential challenges, it does address some of the most common ones**, such as advanced age, extra high fear, obesity, social habits, high daily intake of sugar, medical conditions, drug interactions, and genetic variations.**

Advanced Age

The elderly patient (≥ 65 years of age) has **compromised drug clearance** as a result of lessened ability to metabolize and bind medications. The net result is **increased potency of the sedation drug**, making **careful titration of smaller doses essential to prevent toxic levels of CNS depressants**.

Advanced age impacts the **quality of the airway and** the **protective reflexes** designed to protect it. There is reduced alveolar gas exchange, vital capacity (inhalation and exhalation capability) and increased residual volume (trapped air). This results in **lower partial pressures of oxygen, and higher levels of carbon dioxide in the blood**. To compound these effects, **the elderly patient's response to hypoxia and hypercarbia is thwarted**. As a result, the sedation team should **carefully monitor airway, oxygenation, ventilation, and consider using 100% supplemental oxygen throughout the procedure**.

As a whole, the elderly population presents with **more medical conditions**, and these conditions are **less likely to be well controlled** than in their younger counterparts. The body compartment impacted by the medical condition is more likely to be compromised, making the condition more severe, limiting normal activity, thus **affecting the elderly patient's ASA status, and increasing the risk of anesthesia.** This should prompt the sedation team to **seek information from the patient's treating physician, as well as more closely monitor the elderly patient. Any patient between 50-60 years of age should have had a medical evaluation with lab work within the last year prior to the sedation appointment. Any patient 60 years and older, should have had a medical**

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evaluation with lab work within the last six months prior to the sedation appointment.

Given the elderly patient's **increased risk for compromises in their health, and less potential to respond to those compromises when under physical or emotional stress, the sedation team should consider shorter appointments than with their younger counterparts.**

As a result of the increased incidence of medical conditions, the elderly patient will be taking **more medications**, along with a greater likelihood of side effects and drug interactions, both between existing medications and with sedation medications. The sedation team must **evaluate these medications for their impact on the sedation procedure, and make adjustments as necessary to avoid significant drug interactions.**

Finally, **venipuncture is more challenging** with the elderly patient **due to smaller, more fragile blood vessels**. Not only is accessing such vessels more difficult, there is an **increased risk of complications from venipuncture** including hematoma, and thrombic phlebitis. Therefore, **careful attention must be paid to venipuncture technique**, including utilizing a site where blood vessels tend to be larger, such as the forearm.

Extra High Fear

The patient with higher than normal levels of fear than the "typical" sedation patient require **special care in both behavioral as well as pharmacologic management** on the part of the sedation team.

The extra high fear patient needs more behavioral management to calm their high levels of anxiety. Often **a demonstrated level of extra patience and care mean the difference between success and failure**. While they need reassurance that their comfort is the sedation team's primary concern and they will be comfortable for any treatment, **managing their expectations** of the experience in order to avoid any perception that they cannot trust the sedation team is paramount.

These patients have a **higher minimum effective concentration** needed to effect comfortable sedation. This not only **increases the onset of action, the amount of medication required, but also results in a narrower therapeutic range** for the medication(s) being used, increasing the risk of oversedation. The sedation clinician needs to exercise **discipline in titrating the sedation/analgesic medications** for these patients, resisting any temptation to administer too much CNS depressant, too fast.

The sedation team needs to entertain the idea that **conscious sedation may not be enough to address this patient's high level of fear and anxiety.** Informed consent covers this possibility.

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Obesity

For the sake of this discussion, the obese patient will be defined as having a **BMI ≥ than 30**, but the sedation team needs to appreciate that **obesity presents in differing degrees of severity**. The patient with a BMI of 45 is many more times at risk to the challenges obesity presents to safe, effective sedation than the patient who's BMI is 30. **Consideration should be given to sedating the morbidly obese patient in a different setting** than the typical outpatient environment **and/or using a dedicated anesthesiologist** to handle the sedation and monitoring part of the procedure.

The primary challenge the obese patient presents the sedation team with **is maintenance of a patent airway.** Increased soft tissue around the upper airway together with any CNS depressant with muscle-relaxation capability is a potentially dangerous combination. The goal **when sedating the obese patient** is too **sedate to the lightest possible level of CNS depression needed to accomplish comfort, pay special attention to the patient's airway position, and using 100% supplemental oxygen throughout the appointment.**

Given the lipophilic nature of the medications used in the sedation procedure plus the larger amount of fat tissue in the obese patient, a hypo-response is expected. This **hypo-response will manifest in an increased onset of action, and a greater amount of medication required** to reach the desired level of comfort.

This higher affinity for the sedation medication coupled with the higher body fat in obese patient, also accounts for an **extended duration of action**. The larger amounts of medication needed to effect comfort are released from fat stores slowly, making the recovery longer. This extended recovery time, together with the increased airway risk make **the obese patient's recovery outside the supervised monitoring of the dental office a greater concern for the patient's safety. Using shorter-acting CNS depressants, along with careful monitoring is needed** to reduce this risk to a manageable level.

Venipuncture can be challenging in the obese patient due to the extra soft tissue surrounding the surface veins. While these veins may not roll as much, they are often difficult to visualize or palpate, requiring methods designed to cause both vasodilation and pooling of blood in the arm/hand. Alternate sites like feet are sometimes used when blood vessels in the arms are unavailable for venipuncture.

Social Habits

The **patient who smokes or uses nicotine** in other forms is a challenge to sedate. Their **hypo-response is due to the stimulant effect of the nicotine (counteracting the depressant effects of the sedative/analgesics), and the liver enzyme induction of cigarette smoke**. **When their normal schedule of nicotine**

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intake is interrupted by the sedation appointment, the withdrawal-like craving is nearly impossible to sedate over.

With this in mind, it is helpful with the nicotine user employ **a pre-sedation routine that balances the need to reduce the hypo-responding effects of nicotine by using less prior to the sedation appointment with the hypo-response of nicotine craving by using some amount of nicotine**.

In order to accomplish this, the **recommended pre-sedation routine for the smoker's use of nicotine is based on a sliding scale relative to the frequency of their smoking habit:**

- For the patient who smokes **< ½ packet of cigarettes/day – withhold smoking 12 hours prior to the appointment**
- For the patient who smokes **½-1 packet of cigarettes/day – withhold smoking 8 hours prior to the appointment**
- For the patient who smokes **1-1.5 packs of cigarettes/day – withhold smoking 4 hours prior to the appointment**
- For the patient who smokes **> 1.5 packs of cigarettes/day – the patient should just prior to the sedation appointment**

Other forms of nicotine use have very similar factors at play, therefore requiring the same concepts of pre-sedation routine, but with differing methods of administration.

As a result of all the issues surrounding nicotine use and addiction, **the nicotine user will require larger amounts of medication** to effect relatively similar levels of sedation compared to the non-nicotine user, but as always, careful attention should be paid to the principle of titrating to effect with appropriate doses at appropriate intervals.

Marijuana is a commonly used drug for both medicinal and recreational purposes, made more common by the expanding number of governing bodies that legalize its use. Marijuana is **a CNS depressant with addictive properties, capable of both additive CNS depression when combined with other CNS depressants (like sedation medications), as well as tolerance to all CNS depressants**. This combination of effects potentiates the risk of reaching toxic levels of sedation medications in the patient.

The sedation team should inquire about the use of marijuana specifically, and recommend a pre- and post-sedation routine that minimizes the risk to the sedation patient. This **pre-sedation routine includes withholding the marijuana 24 hours prior to sedation, and 24 hours after the sedation appointment**. Whenever possible, short-acting sedation medications should be used to reduce the overlap of the half-life of the sedation drug from overlapping with the patient's marijuana use.

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While the recreational use of drugs and subsequent addiction is listed here in the social habit section, addiction, which may have begun as a social habit, is in fact a medical condition, and will be referenced as such later in this chapter.

Recreational drug misuse and abuse is a worldwide problem of epidemic proportions. The current opioid crisis is the direct or indirect cause of an alarmingly high amount of death and destruction. Given its widespread use, the risk to the sedation patient must be assessed and minimized.

Opioids are CNS depressants, and **additive CNS depression when combined with sedation medications should be expected**. More often, these patients have built **up substantial tolerance to CNS depressants that the problem is not a hyper-response, but extreme hypo-response. This leads to a narrower therapeutic range of the sedating drug, making oversedation more likely**.

Obviously**, sedating a patient who is under the influence of a non-prescribed opioid is contraindicated**. But sometimes, the signs and symptoms of "under the influence" are not as apparent as one might hope for. The answer to this dilemma lies in careful pre-sedation screening.

First and foremost, **users of non-prescribed opioids not in recovery are not candidates for sedation** in the outpatient setting. Any sedation candidate who admits to prior recreational opioid use should be considered a current user until a trusted medical provider who has evaluated them psychologically and with lab work has verified otherwise.

Anyone whom the sedation dentist or team has **doubts about regarding their forthrightness regarding the use/misuse of addictive drugs are not candidates for sedation**.

When sedating the patient with a past or current use of opioids and other addictive drugs, yet are still deemed appropriate candidates for sedation, **extra-careful attention should be given to the patient's level of consciousness as well as their vital signs. Naloxone and/or flumazenil should be available** and when appropriate, administered by the appropriate protocol.

High Daily Intake of Sugar

The **patient** who indicates a **high daily intake of sugar** presents a challenge in that their **body physiology has adjusted for their sugar intake schedule. When that schedule is interrupted, as with the NPO regimen prior to a sedation appointment, a craving for sugar, as well as a risk for hypoglycemia may ensue**. The former makes comfort and cooperation harder to achieve, while the latter carries the risk for a medical emergency.

Hypoglycemia may cause unusual behavior, confusion, and when severe, can be dangerous, resulting in seizures, loss of consciousness, or death. The

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sedation team should **monitor the high daily intake sugar patient carefully, taking the patient's blood sugar with a glucometer every hour during the sedation appointment. The recommended range without diabetes is 70 to 130 mg/dL**.

The sedation team should also look for other **signs/symptoms of hypoglycemia, which include: shakiness, dizziness, sweating, hunger, irritability or moodiness, anxiety or nervousness, and headache**.

Using an **IV fluid that contains sugar, such as D5W**, will make low blood sugar less likely. Another way to prevent and address both the craving and the hypoglycemia crisis is to **administer additional dextrose through the IV sedation line**. If there isn't a line in place, **the conscious patient may be allowed to eat a couple tablespoons of yogurt or applesauce**. Either of these measures not be performed prophylactically, but used at the onset of signs/symptoms of sugar craving and/or hypoglycemia.

Medical Conditions

The list of medical conditions that could present a challenge to either the safety and/or efficacy of IV sedation is long. This section will focus on those conditions most often seen and/or are likely to have the biggest potential impact on the sedation process

1. **Respiratory Disease is arguably the biggest red flag** of any medical condition the sedation team can be faced with. The **second-most negative side effect to the CNS depression is a decreased ventilatory drive** resulting in low PO_2, and a high PCO_2. The **most serious being total loss of the ventilatory drive**. Fortunately, the likelihood of this happening is extremely small in the healthy, or relatively healthy patient. The **patients at greatest risk are those with chronic respiratory disease, namely, chronic obstructive pulmonary disease (COPD).**

   Patients with COPD have either emphysema (disease of the alveoli), or chronic bronchitis (disease of the bronchioles). If they have either condition long enough and severe enough, they are likely to develop the other as well. Presently, there is no cure for COPD, and the best outcome occurs when the disease is diagnosed early, managed medically, and accompanied by lifestyle changes.

   The risk to the sedation patient who has COPD is a greater likelihood of desaturation (low levels of oxygen), and a compromised response to measures to correct the desaturation (desensitized hypercarbic drive). While low PO_2 can stimulate the hypoxic drive to breathe (backup to the CO2 drive), the oxygen drive mechanism is often compromised as well, and when given oxygen to address the low PO_2, respiration can be further obtunded. When

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chronic low levels of PO_2 exist, cardiac tissue can be irritated, leading to an adverse cardiac arrhythmia event.

The bottom line is that **COPD patients are much more likely to be ASAIV category patients, and not suitable candidates for IV sedation in the outpatient setting.**

Patients with asthma can be acceptable candidates for IV conscious sedation as long as their condition is considered mild-moderate, and under good control. If so, the main concern with these patients is if they experience the acute manifestation of their disease – bronchospasm.

Bronchospasm is the reversible constriction of bronchioles by inflammation and/or smooth muscle contraction. There may also be excess mucous production that also makes the airway passages smaller, resulting in labored breathing by the patient. This acute version of the chronic asthmatic condition is caused by allergens, physical or emotional stress, or other irritants such as cold air or cigarette smoke. The **event is often transient and responsive to a short-acting beta 2-adrenergic receptor agent, such as albuterol**. If the patient responds favorably, the sedation appointment can be continued as soon as the patient is comfortable enough to do so.

Sedating a patient in the acute phase of their asthmatic condition is contraindicated, and should bronchospasm occur during sedation, the patient is allowed to use their inhaler, or in the event their inhaler is insufficient, the inhaler in the office emergency kit.

A medical consult should be obtained if it cannot be determined whether the asthma is mild-moderate, or under good control. The sedation team should monitor this patient carefully. The treating physician may advise prophylactic administration of the patient's inhaler.

2. **Liver Disease**, as with respiratory disease, can make sedation riskier, given the liver's important role in biotransformation and synthesis of plasma proteins. The bottom line is that impaired liver function **can make sedation medications more potent (hyper-response), potentially resulting in toxic levels**.

   The **two most likely causes of impaired liver function are hepatitis** (liver infection), **and cirrhosis** (damage to the liver by toxin – most commonly alcohol or drugs). If the patient indicates either of these conditions, **then a medical consultation is necessary to determine the risk the sedation procedure poses to the patient. If that risk is deemed acceptable, then careful titration of medications least likely to be affected by compromised biotransformation is recommended**.

3. Occasionally, patients who have **sensitivity to the medications** used during sedation will be encountered. The majority of the medications used fall into one

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of two categories (Benzodiazepines, Opioids). Allergic reactions to benzodiazepines are extremely rare, and any untoward reaction to them is usually an idiosyncratic reaction that manifests as unusual behavior or nausea and vomiting.

Sensitivity to opioids is much more common. Members of the opioid class fall into multiple chemical subclasses, so sensitivity to a member of one subclass may not mean sensitivity to another subclass. Still, unless sensitivity testing is done by a specialist to verify that, the sedation team should assume sensitivity to all opioids and avoid their use altogether.

If an **allergic reaction should be encountered during sedation**, then the severity of the reaction should be ascertained and treated accordingly. If the **reaction is mild, then Benadryl can be administered IV, or IM. If the reaction is anaphylactic in nature, then epinephrine should be administered IV, SC, or IM**.

Nausea and vomiting may occur during IV sedation. The **ramifications** of which **range from an unpleasant experience for patient and team, to a potentially life-threatening event** should the patient aspirate vomitus.

Often these patients know in advance they are prone to nausea and vomiting when administered medication. This gives the sedation team a heads-up to **monitor carefully for signs and symptoms**. The patients can also be administered prophylactic **anti-emetic drugs like ondansetron at the beginning of the appointment, as well as at the end.**

4. **Diabetic patients** present a challenge to the sedation process directly by the **impact sedation can have on circulating blood glucose**. Hypoglycemia (low blood sugar) is much more of a concern than hyperglycemia, both because of the NPO regimen, and because hypoglycemia is more likely to be a severe acute event, while hyperglycemia is more of a chronic condition. For this reason, **we would rather our sedated diabetic patients run "sweet" rather than "sour".**

   Careful assessment of the diabetic patient prior to sedation identifies the poorly controlled condition. **Those patients with an HgA1c greater than 8.5% are at greater risk of experiencing adverse events during and after the sedation appointment. A baseline blood sugar should be obtained prior to the appointment, and on the day of the appointment blood sugar levels should be measured upon arrival and every hour thereafter during the appointment**. If hypoglycemia is encountered, the sedation team acts as outlined in the previous hypoglycemia section of this chapter.

5. **Chronic Pain Patients** present a challenge to the sedation process partly because of their condition, which can make **them more difficult to make comfortable**, but mostly because of the medical management of their chronic pain. This **medical management often involves multiple analgesics, many of**

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which are CNS depressants themselves, resulting in tolerance to other CNS depressants like the ones used in the sedation appointment. As a result, **these patients almost always hyporespond**, and while the titration process can overcome a hyporesponse, the result is **slower onset of action, greater amount of drug administered, shorter duration of action, and narrowed therapeutic range (greater potential for oversedation).**

6. **Psychiatric Conditions such as generalized anxiety, depression, bipolar condition, and schizophrenia can present their own challenges to sedation in the form of behavior that is not always overcome by conscious sedation**. The altered outcome can range from a hypo-response to behavior that can be uncooperative and combative. **These patients are also likely to be on multiple CNS depressants, making additive CNS depression and drug interactions more likely.**

   The sedation team needs to entertain the idea that **conscious sedation may not be enough to address these patient's needs.** Informed consent covers this possibility.

7. **Restless Leg Syndrome (RLS)** deserves special mention due to the challenge to the sedation team in managing its manifestations during sedation. RLS affects up to 15% of the population, and generally happens when the patient is at rest, **making relaxation harder to achieve**.

   The condition has many risk factors including genetic tendencies, CNS depressants, and diabetes. Medications include levodopa, a dopamine agonist such as pramipexole, and gabapentin. **While sedative/hypnotic agents in the benzodiazepine class are not considered triggering agents, it is often tempting to think of the patient exhibiting RLS signs as needing more sedation, when in fact, this does not help within the realm of conscious sedation, and only increases the potential for oversedation.**

Drug Interactions

For the most part, **the medications in the IV sedation protocols have little interaction capability other than additional CNS depression when combined with existing CNS depressants the patient is taking**. This severity of this interaction can vary, with the impact usually described by pharmacologic software like Lexi-Comp as additional CNS depression (hyper-response) with an associated warning to use less sedation medication. In reality, patients taking CNS depressants build up tolerance to those CNS depressants as well as others like our sedation medications, making for a hypo-response. **Given the wide range of possibilities, it is best to follow the sedation mantra of titrating low and slow**.

Other severe drug interactions (listed as [D] or [X] interactions as determined by Lexi-Comp) have to do with inhibition or induction of metabolizing

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enzymes by the medications the patient is taking to control a medical condition. These interactions affect the AUC (area under the curve on the Time/Concentration graph) of the sedation drug, resulting in a hyper-response (inhibition), or a hypo-response (induction).

The inhibition and induction of sedation medication metabolism can be addressed in one of two ways during IV sedation. One, careful titration prevents over or under-sedation by administering small amounts of medication and waiting until the full response of the previous dose has had time to take effect, then evaluating that response. The second method involves getting around the drug interaction altogether by using a sedation medication not metabolized by the enzymes in question. Such a drug is the benzodiazepine, Lorazepam.

Thorough assessment of all potential drug interactions, their impact on the sedation procedure, and any needed alterations to the sedation protocol are an essential part of safe sedation.

Genetic Variations

Polymorphism refers to the **variety of ways genetic manifestations can affect** physiologic processes within members of different populations. When applied to **sedation pharmacology and physiology**, **the impact is usually seen in how the patient responds to the sedation medication – as a hypo-responder, or a hyper-responder.**

If it is the former, then the patient often has an abundance of a particular metabolizing enzyme responsible for the biotransformation of the sedation medication. If the latter, then a deficiency of the enzyme.

A unique example of polymorphism is the challenge of the patient with red hair. This population appears to be a distinct phenotype linked to larger anesthetic requirements – in other words, a hypo-response.

Some genetic variations negatively affect physiology of the sedation patient in other ways. For instance, Down's Syndrome patients have more potential for a compromised airway due to larger amounts of soft tissue around the oral cavity and neck.

Careful analysis of the available information about a patient before treatment can clue the sedation team into the expected reaction of the patient to sedation medications. When such information is unavailable, then careful titration and monitoring will reduce the negative potential of these genetic variations.

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Other

"Fidgeters" are restless resters. They don't have RLS, but still move quite a bit when resting, and therefore **will move during sedation**. **As with the RLS patient, more sedation is not the answer.** An opiate, like Fentanyl, is reportedly helpful to calm these movements.

The frequent urge to urinate can have an impact on the sedation experience for both patient and provider. The sedation team is clued into the likelihood of this prior to the sedation appointment by the patient's answers to screening questions regarding frequency of their bathroom routine. **The sedation team should expect the sedated patient to need to go during sedation as often as they usually do during a typical day.**

Confirming that the flow of IV fluid is not excessive may help, but the best advice regarding managing these patients frequent need to go is just to go with the "flow". The sedation team should be confident the sedated patient will be safe in the bathroom by themselves before leaving them alone. The use of a handicap bar beside the toilet, not allowing the sedated patient to lock the door, and assisting the patient as needed to satisfy their urge are all important steps to deal with this challenge.

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UPDATE ON NEW DRUGS WITH SIGNIFICANT DENTAL IMPLICATION

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There are new drugs introduced into clinical use almost monthly. However, a number of drugs is beginning to gain market share and will have a significant impact on how we manage the patients through dental procedures. We would like to continue to keep you abreast of such development by bringing you updates on these new drugs.

Case 1 55 year old gentleman with atrial fibrillation and hypertension comes in for consideration of sedation dentistry.

He is on

Losartan 100 mg qd
Hydrochlorothiazide 25 mg qd
Eliquis 5 BID
Diltiazem CD 300 qd

He will need about 3 hours of dentistry

Cleaning
Bleaching
Periodontal surgery

He has significant gingival inflammation and it is anticipated that he will have some bleeding.

What should you do with the Eliquis?

1. Stop the Eliquis the day before
2. Hold the Eliquis the morning of
3. Resume Eliquis the evening of
4. All of the above
5. None of the above

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NEW ORAL ANTICOAGULANTS

Oral anticoagulation is important in the prevention of embolic strokes in patients with atrial fibrillation. Coumadin is effective and can decrease the incidence of strokes by 2/3. However, coumadin is cumbersome to use, discontinuation by patients and physicians are high and many patients have inadequate control. The drug also has numerous drug interactions. Most cumbersome is the fact that the dose of coumadin has to be carefully titrated to an INR between 2-3.

PRADAXA

Pradaxa (Dabigatran) is a new anticoagulant that is competing with coumadin for the oral anticoagulation market.

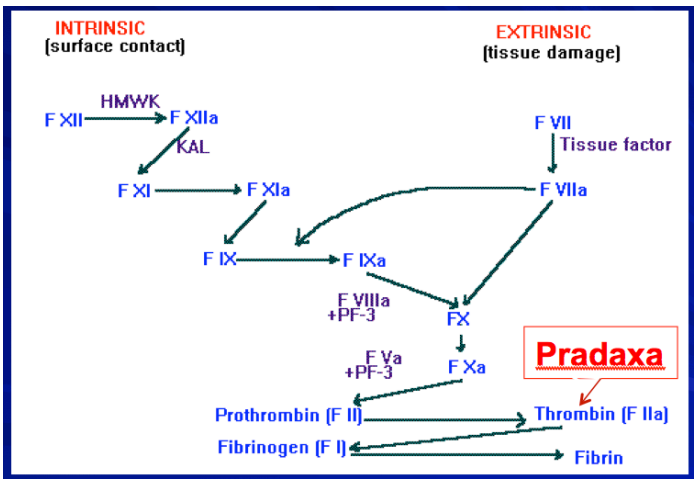
It is a ***direct thrombin inhibitor*** which has been approved for use in

1. Non-valvular atrial fibrillation
2. Prevention of embolic cerebrovascular accidents
3. Post-operative prophylaxis after knee surgery
4. Treatment of DVT
5. Treatment of pulmonary embolism

It is prescribed as a BID drug in range of 75 to 150 mg BID.

Pradaxa, on the other hand, is an oral pro-drug, has predictable bioavailability, is 80% excreted by the kidneys and has a half-life of 12-17 hours. It does not need regular monitoring.

In dentistry, you will encounter increasing number of patients coming into your practice on Pradaxa. Because of its short half-life, you can reverse anticoagulation relatively easily and resume anticoagulation relatively rapidly.



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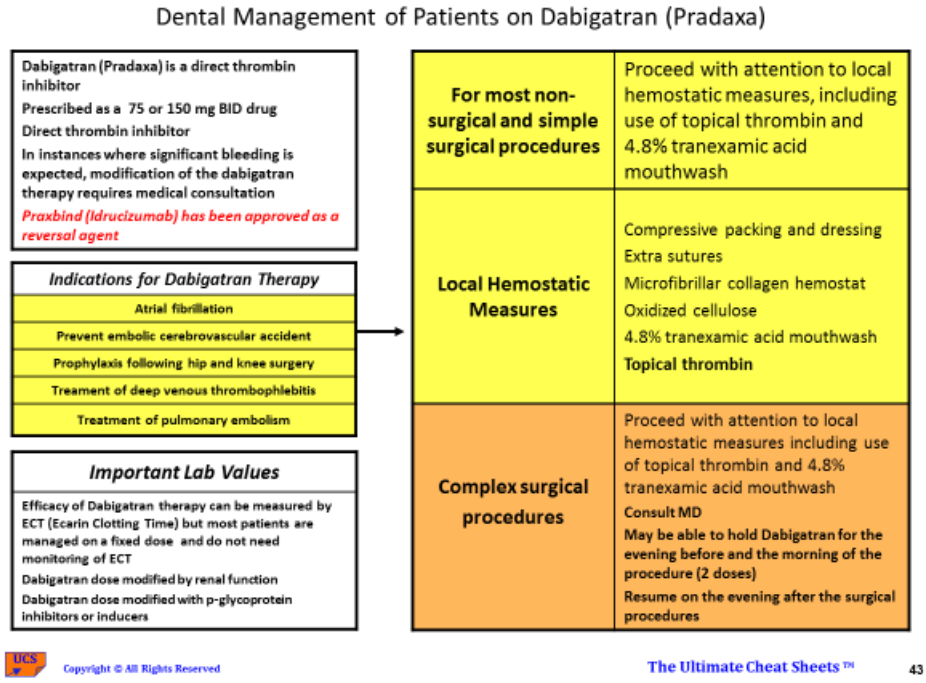
Depending upon your expectation of bleeding risks, you can hold Dabigatran from one to three doses in the 24 hours before the planned procedure and resumed on the night of the procedure.

An important development is the FDA approval of Praxbind, that can bind and inactivate Pradaxa, therefore providing a reversal agent for Pradaxa.



Dabigatran has drug drug interactions with drugs such as amiodarone, quinidine and verapamil. In patients on Pradaxa, one should avoid ASA, NSAIDs Celecoxib, prednisone, solumedrol and p-glycoprotein inhibitors or inducers, such as ketoconazole, dronedarone and rifampin.

It should be used cautiously in the elderly and in patients with decreased renal function.

XARELTO

On the heels of Pradaxa, Xarelto (rivaroxaban) has been approved as a new oral anticoagulant. It has approval to treat atrial fibrillation, prevention of embolic cerebrovascular accident, prophylaxis following hip and knee surgery and for treatment of DVT or pulmonary embolism.

Xarelto inhibits Factor Xa, one of the final steps in the coagulation cascade. The drug is given once a day, usually in the evening.

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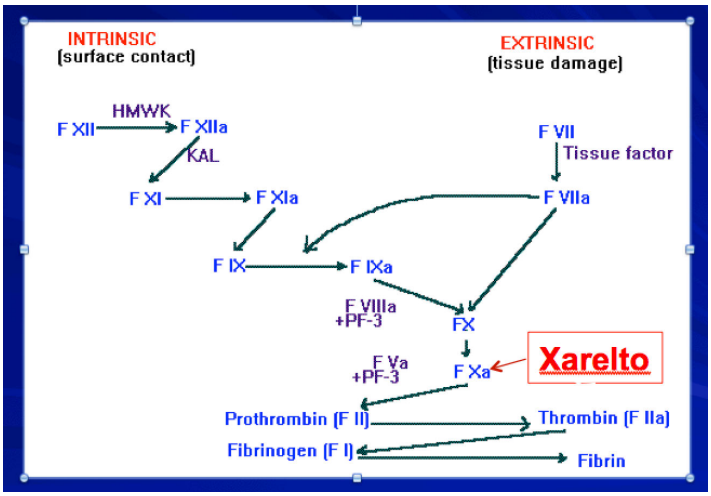
For patients undergoing complex procedures with significant bleeding risk, Rivaroxaban can be held the night before the procedure and resume the night of the procedure.



It has multiple pathways of elimination: via the liver and via the kidneys and its dose is affected to a small extent by CYP3A4 and p-glycoprotein inhibitors and inducers.

Drugs that one should avoid include ASA, NSAIDs, Celecoxib, all macrolides, all erythromycin derivatives, prednisone and solumedrol.

ELIQUIS

Eliquis (Apixaban) is an inibitor of Factor Xa, similar to Xarelto. The drug has been approved for use in non-valvular atrial fibrillation, for post-operative prophylaxis after hip and knee surgery and for treatment of DVT or pulmonary embolism. It is prescribed as a 2.5 or 5 mg bid drug.

Because of the mechanism of metabolism, there may be drug-drug interaction with drugs metabolized by CYP3A4 and P-glycoprotein inhibitors. Drugs to avoid include ASA, NSAIDs, Celexocib, prednisone and solumedrol. CYP3A4 and P-glycoprotien inhibitors such as ketoconazole, itraconazole, protease inhibitors, clarithromycin and rifampin should also be avoided.

SAVAYSA

Savaysa (Edoxaban) is an inibitor of Factor Xa. The drug has been approved for use in non-valvular atrial fibrillation, for post-operative prophylaxis after hip and knee surgery and for treatment of DVT or pulmonary embolism. It is prescribed as a 60 mg qd drug.

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Because of the mechanism of metabolism, there may be drug-drug interaction with drugs metabolized by P-glycoprotein inhibitors. Drugs to avoid include ASA, NSAIDs, Celexocib, prednisone and solumedrol. P-glycoprotien inhibitors such as ketoconazole, itraconazole,,and rifampin should also be avoided.

NOACS

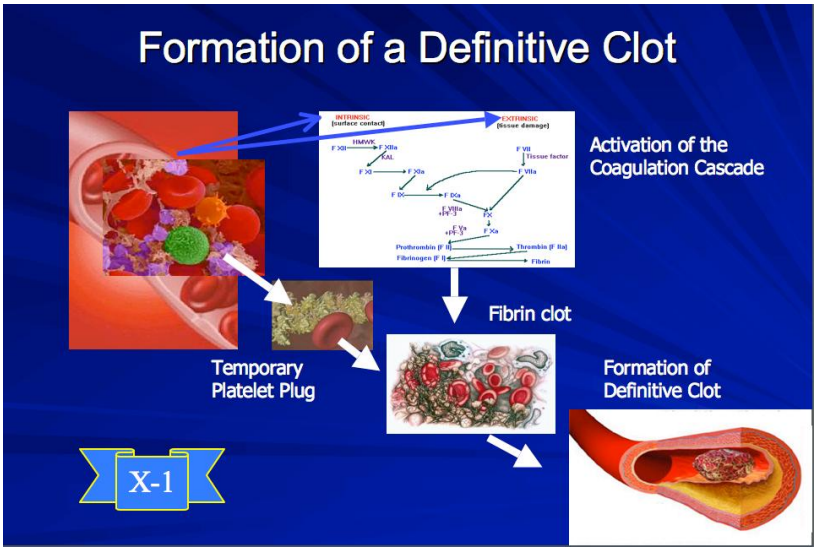
NOACS are increasingly the mainstay of anticoagulation and are regarded to be as efficacious as Coumadin with less bleeding complications. They are now the first line drugs for management of DVT and PE.

Pradaxa, Xarelto, Eliquis and Savaysa are NOT panacea. There are concerns over the finding in post-marketing studies of bleeding, particularly in the elderly and in patients with renal dysfunction.

However, the development of reversal agents are important for the safety of NOACS:
Praxbind is a reversal agent for Pradaxa.
A recent advance is the development of a **reversal agent for the other three agents: Andexanet alpha** is a FXa molecule that lacks catalytic and binding activity. This **Decoy** looks like FXa but has not of its activity and is an antidote for Xarelto, Eliquis and Savaysa.

NEW ANTIPLATELET DRUGS

Platelets are responsible for the formation of a temporary plug.



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Antiplatelet drugs are of importance in preventing a second event in patients who have had either a myocardial infarction or stroke. They are also widely used for patients who have stents placed in their coronary or peripheral circulation.

There are reversible antiplatelet medications (NSAIDs, Persantine) but the important antiplatelet drugs inhibit platelet function irreversibly and would paralyze the platelets for their lifetime (12 days) (Aspirin, Plavix, Effient, Brilinta)

ASPIRIN

ASA irreversibly bind to cyclooxygenase and is a NSAID. Aspirin would inhibit platelet function for the lifetime of the platelets (12 days). Risk benefit analysis indicate that ASA has small benefit in prevention of primary event and should be used judiciously except in high risk individuals. There is also some indication that daily or every other day ASA may prevent cancer (Lancet April 2016), particularly colorectal cancer.

If ASA is to be stopped for a procedure, it should be held for 7 days.

PLAVIX (CLOPIDOGEL) 75 mg

Plavix prevents platelet adhesion and formation of a temporary plug by irreversibly binding to the P2Y12 receptor on platelets. It is often used in combination with aspirin, particularly in patients with stents. It is now generally accepted that Plavix should be continued for 12 months after stent placement.

If Plavix is to be stopped for a procedure, it should be held for 7 days.

EFFIENT (PRASUGREL) 5 or 10 mg

Effient is similar to Plavix and irreversibly inhibits platelet function via the ADP path. It is initially prescribed for patients allergic to Plavix but is slowly gaining market share on its own.

If Effient is to be stopped for a procedure, it should be held for 7 days.

BRILINTA (TICAGRELOR) 60 or 90 mg

Brilinta also works via the P2Y12 receptors and is an irreversible antiplatelet agent. There is some data to support better outcome than Plavix in patients with acute coronary syndrome or myocardial infarction after stent placement treated with ticagrelor.

If Brilinta is to be stopped for a procedure, it should be held for 7 days.

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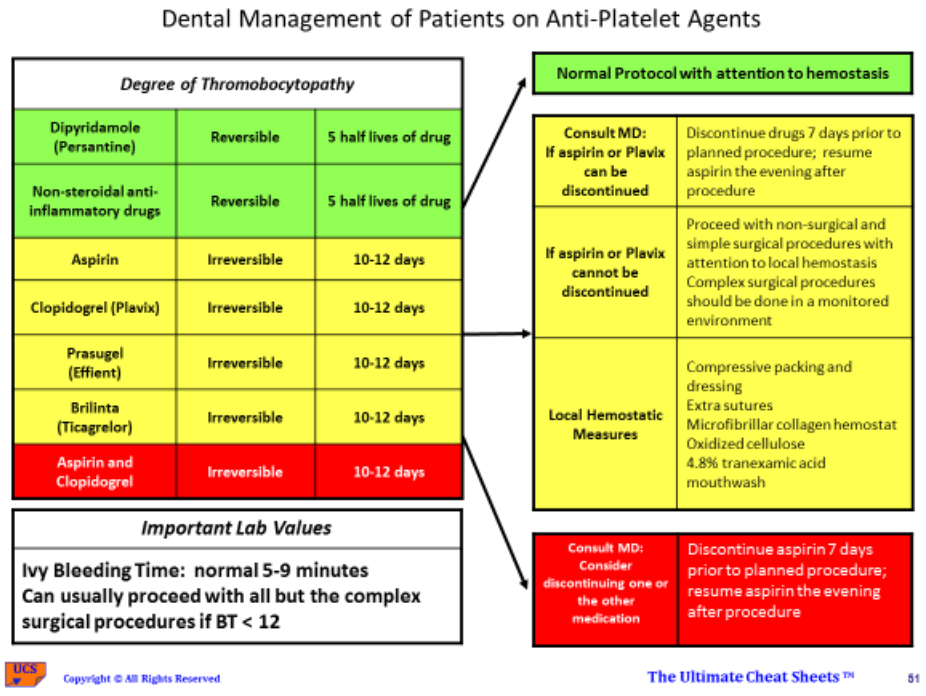
STOPPING ANTI-PLATELET DRUGS

In general, dentistry can be safely performed on patients on single antiplatelet inhibition. However, significant bleeding can occur in patients on double antiplatelet therapy (DAPT).

It is important NOT to do any elective surgical procedures within 6 months after a stent placement.

Patients with double antiplatelet therapy are at significant risk of bleeding. In situations where hemostasis is a major concern, it would be prudent to **consult the physician** and see if ASA can be held for a week, to be resumed on the evening after the procedure.



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Drugs for Patients on Anti-Platelet Agents

Drugs to Avoid	
Analgesics	**Aspirin**
	All non-steroidal anti-inflammatory drugs

Drugs that are Safe	
Antibiotics	No contraindication to commonly prescribed antibiotics
Analgesics	Acetaminophen
	Celecoxib (Celebrex)
Narcotics	No contraindication to commonly used narcotics
Sedatives	No contraindication to commonly used sedative

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PATIENT ASSESSMENT for INTRAVENOUS SEDATION

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All patients entering a dental practice should have filled in a medical questionnaire. This document is usually very brief but it should be the basis for the dentist to have a better feel for problems that may arise. Depending upon the nature of the dental practice, you would want to custom-prepare your own questionnaire in order to reflect the type of procedures you routinely do on your patients. Your questionnaire needs to be adjusted to accommodate the specific issues arising from sedation.

It is important to try and build in some guidelines when you review the medical history and try to have some ways of being able to raise a **Red Flag** when things are likely to pose a problem in the dental management of the patient.

The **health history form** should be used to:
1. Screen for medical problems,
2. Allow for risk assessment and stratification,
3. Allow for assessing and evaluating the need for modifications to dental care, and
4. Allow you to ask specific questions relevant to sedation.

Patients are getting older and are retaining their dentition longer. Their medical condition is also getting progressively more complicated. One out of three adults in this country has hypertension, one out of seven has hyperlipidemia and one out of eight adults has diabetes. It is therefore not unusual to have to carefully evaluate the medical needs of the patient prior to planned sedation.

AGE OF THE PATIENT

The age of the patient would be the first thing that can raise a Red Flag. **Patients greater than the age of 65** should raise a **Red Flag**. They are more likely to have hypertension, hyperlipidemia, diabetes, heart disease and vascular disease.

They have compromised upper airway reflexes, making respiratory compromise much more of a concern

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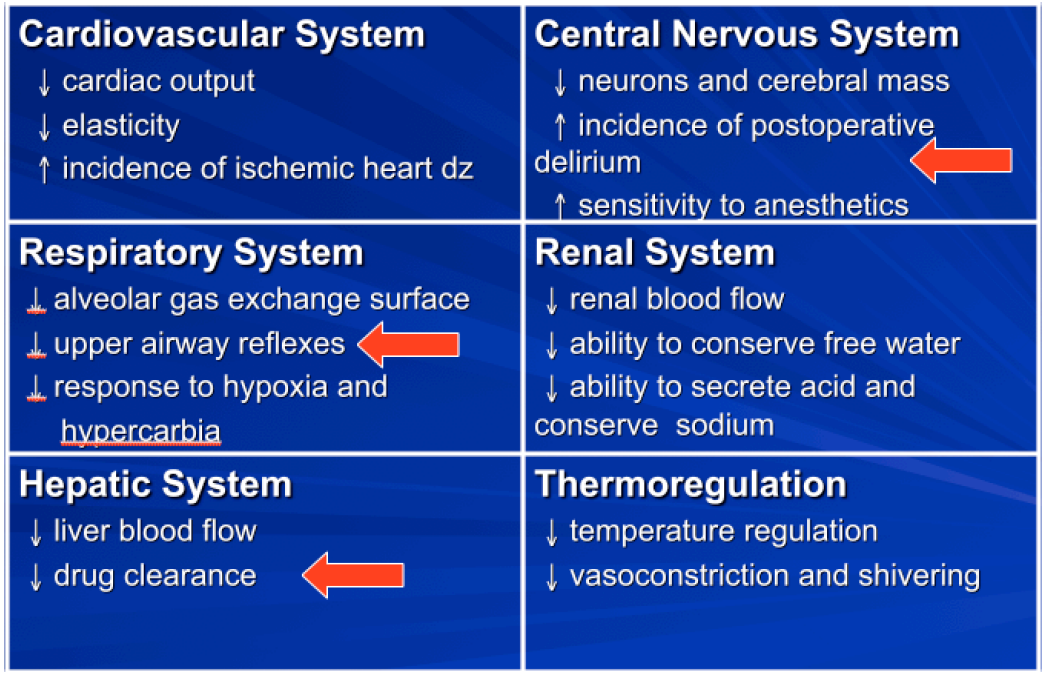
They metabolize and excrete drugs much more slowly and are more likely to develop adverse drug reactions. This is particularly important in sedation dentistry since the dose of benzodiazepines has to be reduced in these patients: in general, we would recommend half the normal dose for sedation.

They are also more prone to having increase in incidence of postoperative delirium with increased sensitivity to anesthetics. Changes that we see with aging includes:



In patients above the age of 65, you need to reduce sedation medication by one half.

It is important to stress that not everyone over the age of 65 will be problematic but it is clear that they are more likely to have multiple medical co-morbidities that would complicate dental treatment plans.

DATE OF LAST MEDICAL EXAMINATION

It is important to pay attention to the last time a patient has had a medical evaluation. A patient who has never seen a physician would have no known medical problems at all!

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Frequency of medical evaluation is dependent both upon the age of the patient and the complexity of the medical issues.

In general, young and healthy patients should have had a medical evaluation within the past two years.

Patients above the age of 50 should have had an evaluation within the past year.

Patients age 51 to 60 should have had an evaluation within the past 6 months

Patients above the age of 60 should have had a physical examination, a blood screen and an electrocardiogram done within the past 3 months.

Obviously, the frequency of medical follow-up is dependent upon the complexity of the medical issues the patient has and more frequent follow-up is necessary for the more complicated medical patient.

Any patient **above the age of 60** and any patient with **complex medical issues** who has not had a medical evaluation within the past 6 months should raise a **Red Flag**.

Complicated patients with kidney disease, liver disease, congestive heart failure and immunosuppression are much less likely to be lax in their medical follow-up. Depending upon the complexity of their illnesses, these patients are usually followed on at least a biannual basis. These patients should be referred back to their physician for a comprehensive medical evaluation if they have not been seen within the past 6 months.

MEDICATIONS THE PATIENT IS TAKING

Pay attention to the medication list. It often tells you more than your patient tells you.

You should request for a record of:

-all prescribed medications
-all over the counter medications
-all nutraceuticals and supplements

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COMMON MEDICAL CONDITIONS

Hypertension, heart disease and diabetes mellitus are the most common conditions where patients may seek sedation. Some patients seeking dentistry may have a coronary stent.

In most of these conditions, the patients are generally believed to be safer sedated than not sedated because of their underlying medical conditions.

HYPERTENSION

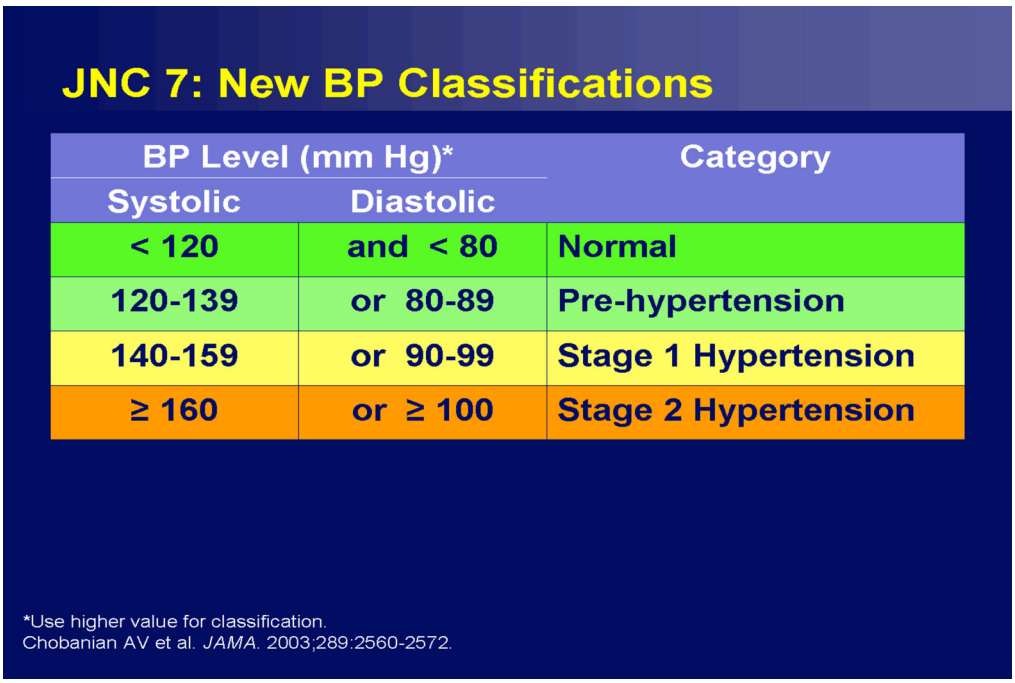
Hypertension affects 73 million patients in the Unites States (1 in 3 adults). It is further estimated that that another 30% of the population has pre-hypertension. It is therefore a common medical condition that all dentists would encounter.

Hypertension can result in a number of target organ damages including:

- Transient ischemic attacks and strokes
- Left ventricular hypertrophy, coronary heart disease, and heart failure
- Retinopathy
- Renal failure
- Peripheral vascular disease

NEW JNC 8 GUIDELINES (2014)

JNC 7 in 2003 and JNC 8 in 2014 classified different categories of blood pressure as below:



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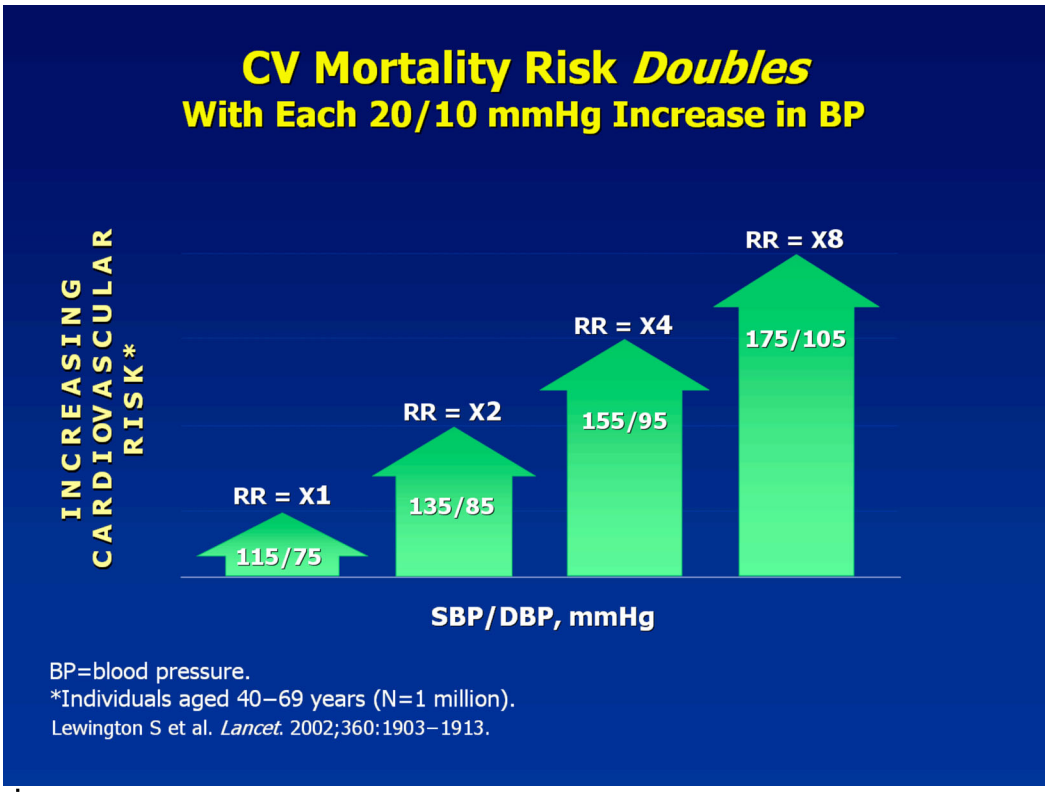
If we define 120/80 as the normal blood pressure, for every 10 mm increase in diastolic blood pressure or 20 mm increase in systolic blood pressure, the risk for cardiac mortality doubles:

A patient with pre-hyertension has 2X the relative risk
A patient with Stage 1 hypertension has 4X the relative risk and
A patient with Stage 2 hypertension has 8X the relative risk.



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In JNC 8, Evidence Based Medicine indicated that while there is data to support aggressive treatment of Stage 1 and 2 Hypertension, there is no evidence that treating Pre-Hypertension changes the outcome.

There is a stepwise increase in prevalence of hypertension (as defined by BP of >140/90) with age:

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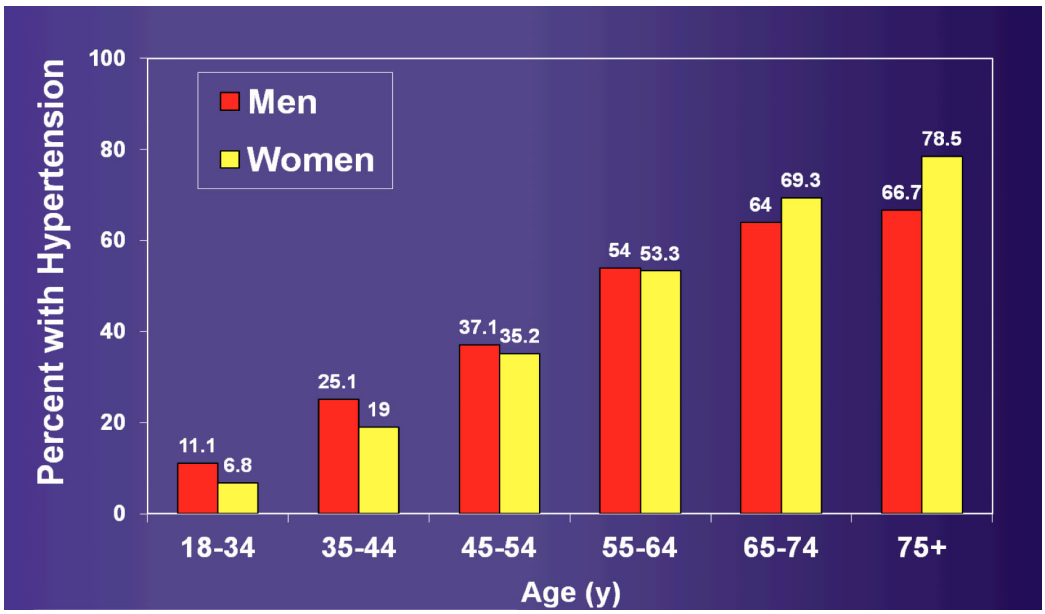
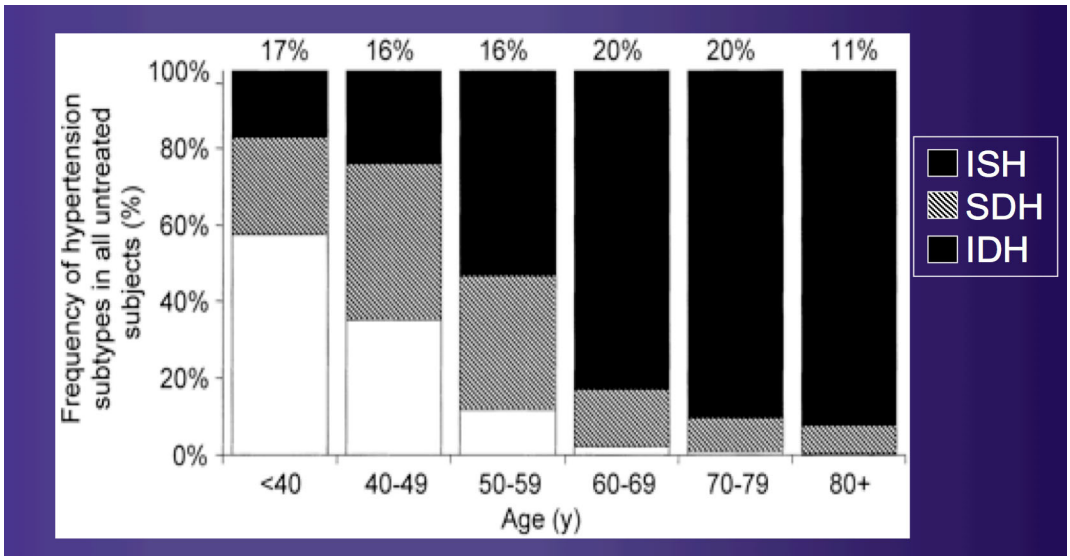


ISOLATED SYSTOLIC HYPERTENSION

It is also increasingly obvious that Isolated Systolic Hypertension causes as much morbidity as Diastolic Hypertension. Isolated Systolic Hypertension (ISH) is particularly common in patients above age 60.



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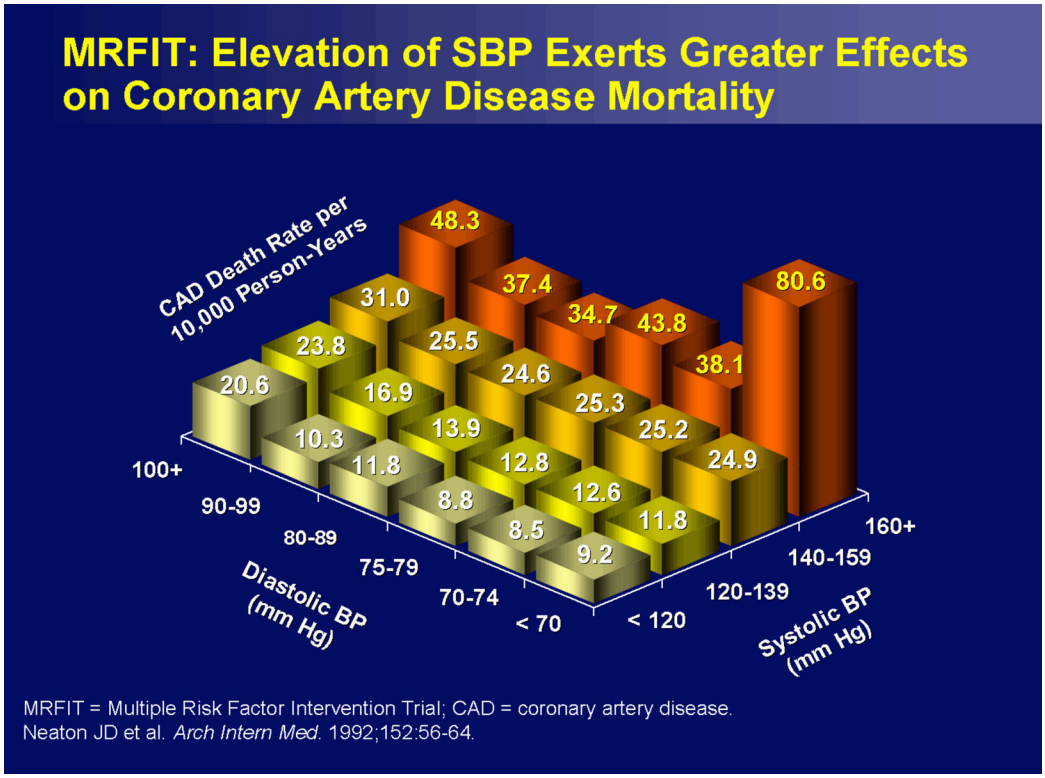

On the basis of the wealth of currently available evidence, JCN 8 recommends a major paradigm shift in urging that systolic BP become the major criterion for the diagnosis, staging and therapeutic management of hypertension, particularly in middle-aged and older Americans.

JNC 8 TREATMENT RECOMMENDATIONS

JNC 8 did not make adjustments to the risk categorization but did make adjustments in treatment algorithms. These recommendations are based upon evidence-based studies.

Lifestyle changes including salt restriction, exercise and weight control continues to be important for all patients. Treatment can begin with diuretics, ACE inhibitors, angiotensin receptor blockers or calcium channel blockers. Medical regimen should be steadily escalated until the patient is at goal.

In patients above age 60, the systolic goal has been relaxed to 150. JCN 8 also has specific recommendations for treatment of specific populations:

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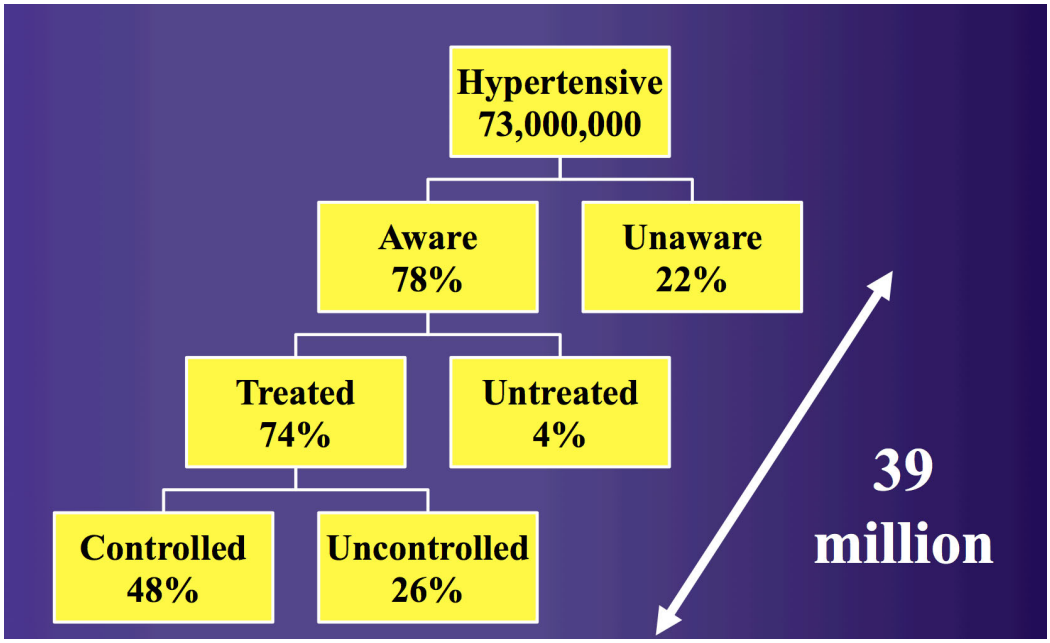
Patient Demographics	TREATMENT GOALS
> Age 60	Treat to <150 systolic Treat to < 90 diastolic
< Age 60	Treat to < 140 systolic Treat to < 90 diasolic
Diabetics	Treat with ACEI or ARB
Chronic Kidney Disease	Treat with ACEI or ARB
Non-blacks	Treat with ACEI, ARB, calcium channel blocker or diuretics
Blacks	Begin treatment with diuretics or calcium channel blocker

HOW WELL IS BP UNDER CONTROL IN THE GENERAL POPULATION?

It should be understood that in population studies, only 48% of hypertensive patients have blood pressure under control (<140/90). it is therefore not uncommon to encounter blood pressures that are not ideally controlled in the dental office, particularly in fearful patients.



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SEDATION OF THE HYPERTENSIVE PATIENT

If the first blood pressure is >140/90, reassure the patient and repeat the blood pressure in 5 minutes.

Sedation in Stage 1 patient is safe: blood pressure often comes down with sedation.

Patients who should raise a **RED FLAG** include:

1. Patients with Stage 2 hypertension:
    a. Systolic blood pressure greater than 160 mm Hg or
    b. Diastolic blood pressure greater than 100 mm Hg
2. Patients with complicated hypertension:
    Patients on 4 or more drugs for management of hypertension

The good news is that there is good evidence that hypertension can be controlled, but it will take an intense and sustained effort.

Understanding the blood pressure guidelines and their implications would allow the clinician to assess risks and allow for an ASA categorization

These patients are at significantly elevated risk for adverse cardiovascular outcome.

These algorithms can be used to guide dental management. This is the rationale for the development of the **Ultimate Cheat Sheets, The Practical Guide for Dentists.** This is designed as a Chair-side Compendium for daily use by the dental professionals.

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Dental Management of Patients with Hypertension

Blood Pressure (mm Hg) Systolic	Diastolic	Category
<120	and <80	Normal
120-139	or 80-89	Pre-hypertension
140-159	or 90-99	Stage 1 Hypertension
>160	or >100	Stage 2 Hypertension

Dental examination only
Palliative care under monitored environment
Stress reduction is of paramount importance
Complex prosthetics and surgical procedures should not be undertaken
Vasoconstrictors absolutely contraindicated

Normal Protocol	
Stress Reduction	Patient rapport Short appointments
Sedation Protocol	Consider oral conscious sedation Consider use of nitrous oxide

Stress Reduction	Patient rapport Short appointments
Sedation Protocol	Consider oral conscious sedation Consider use of nitrous oxide
Limit use of vasoconstrictors	Less than 2 carpules of local anesthetics containing 1:100,000 epinephrine or 1:20,000 levonordefrin
All non-surgical and simple surgical procedures	Normal protocol
Complex prosthetic and surgical procedures	Strongly consider sedation protocols

HYPERLIPIDEMIA

Hyperlipidemia is an extremely common medical condition with significant impact on cardiovascular risk.

With better understanding of the importance of lipid profile, we now have an exceedingly aggressive goal for optimization of the lipid profile.

In general, when a lipid panel is ordered, we would have values generated for total cholesterol, LDL (bad cholesterol), HDL (good cholesterol) and triglycerides (also carries LDL).

The current goal is to have total cholesterol below 180 mg/dL, LDL below 70, HDL greater than 45 and triglyceride below 150.

Lowering LDL is particularly important since it plays a critical role in cardiovascular morbidity and mortality.

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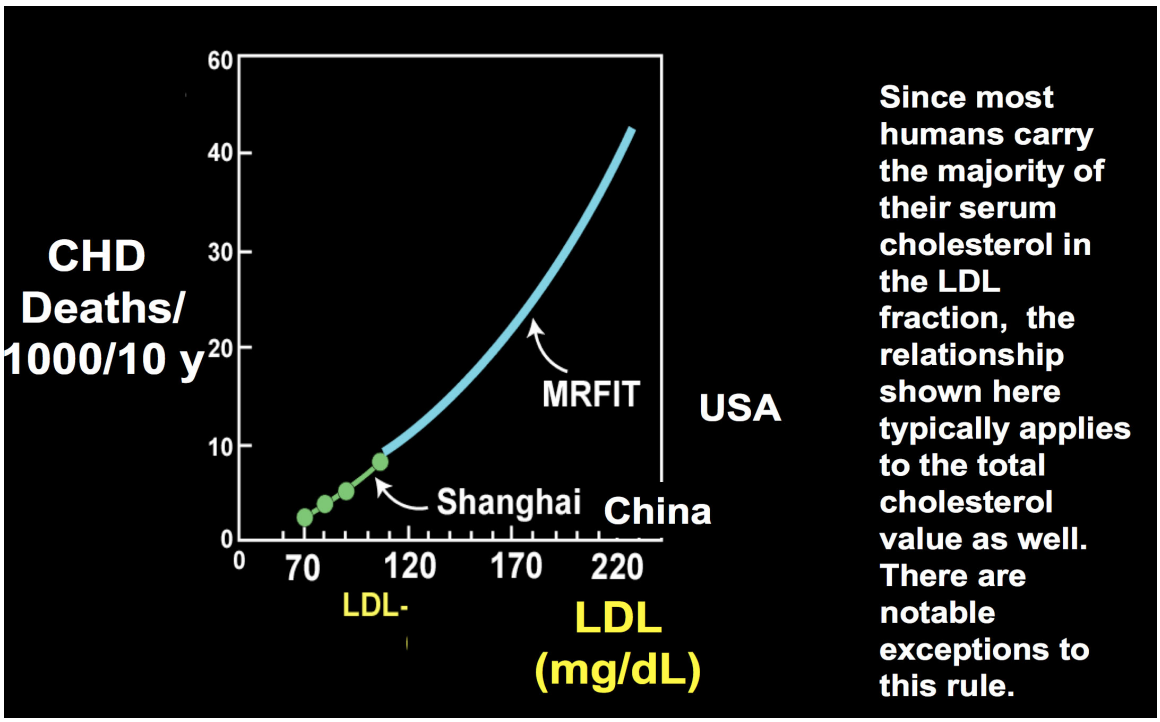
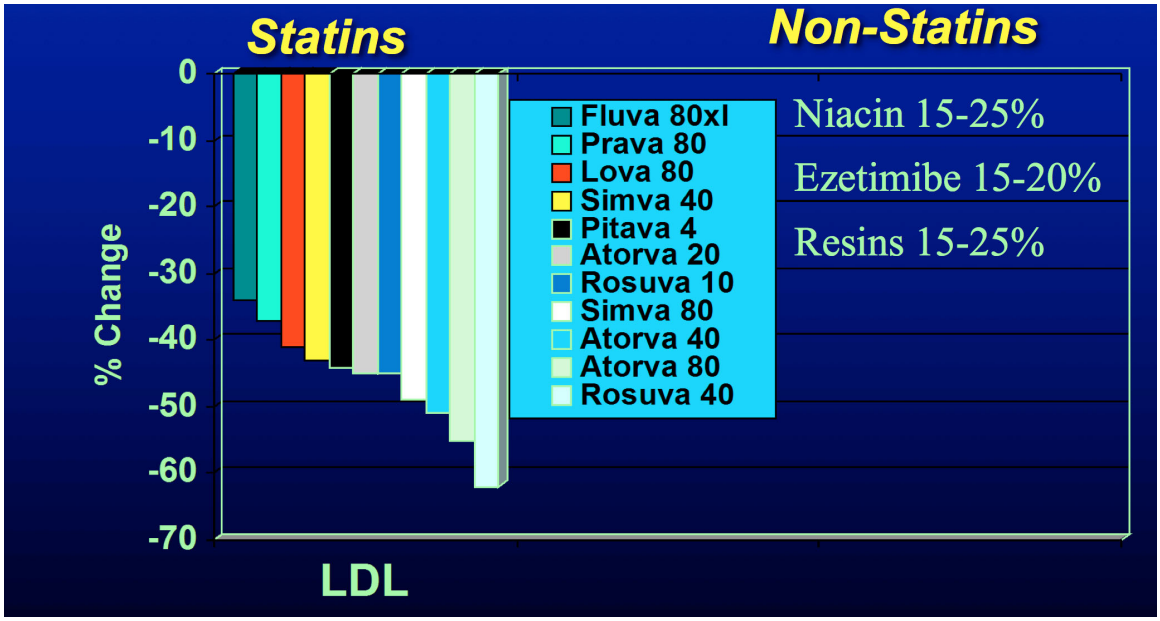


A large number of drugs are now on the market for LDL lowering:



HDL is called the good cholesterol because of very convincing epidemiologic association between elevated HDL having a protective effect on cardiovascular morbidity and mortality.

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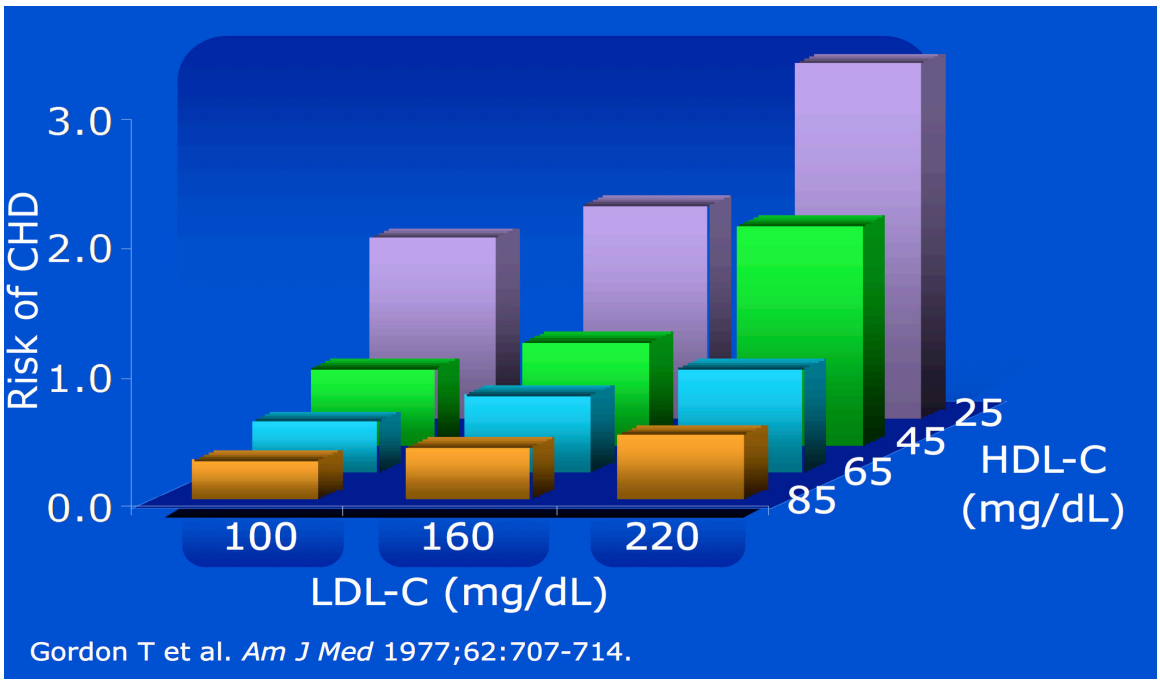

Exercise is known to raise HDL.

While the epidemiologic relationship between HDL and cardiovascular risk is clear, there is no evidence that HDL has a causal relationship to cardiovascular risks. None of the agents designed to raise HDL have demonstrated cardiovascular benefit.

The only effective way to lower triglyceride is through diet, exercise and weight loss. None of the TG lowering agents has demonstrated cardiovascular benefits beyond their role in LDL reduction.

DIABETES

Diabetes mellitus now affects **one out of 8** Americans and is a rapidly growing problem. Diabetes mellitus now affects 190 million people worldwide and is estimated to affect 330 million by 2025. In a typical practice of 1,000 patients, a dentist would expect 110-120 patients with diabetes mellitus. The total aggregate cost of diabetes mellitus in the United States is estimated to be $245 billion per year.

Diabetes is classified according to the etiology of the disease:

Patient with Type 1 diabetes mellitus has diabetes as the result of insulin deficiency.

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Patient with Type 2 diabetes mellitus has insulin resistance and is treated with.

Patient with Type 1 diabetes mellitus deserves more scrutiny since they tend to have more of the metabolic and medical complications of the disease. They should raise a **Red Flag** in view of the complexity of managing a patient with Type 1 diabetes mellitus.

In the United States 1.5 million patients have Type 1 diabetes mellitus and 24.5 million patients have Type 2 diabetes. Patients with Type 1 diabetes mellitus are generally more complicated because of the duration of the disease and its associated complications.

Perhaps more worrisome is the estimate that there are 74 million patients in the US with pre-diabetes.

Patients with Type 1 diabetes mellitus are treated with insulin. Insulin pump and Continuous Glucose Monitoring are increasingly a part of management of these patients.

Patients with Type 2 diabetes are started on life style modification, diet, exercise with the goal to lose weight. In many instances, they would need oral hypoglycemic agents to get blood sugar into reasonable range. In some instances, control cannot be established without the addition of insulin.

Diabetic patients are also much more prone to hypertension, hyperlipidemia, coronary artery disease, peripheral vascular disease, renal disease, peripheral neuropathy and retinopathy. They are also more prone to infections, including oral infections. Complications are particularly common in patients with Type 1 diabetes who may have extensive complications even at an early age. Patients with **diabetic triopathy** (renal disease, eye disease and peripheral neuropathy) should raise a **Red Flag** since they are much more likely to have problems with their dental management.

Diabetic complications have made diabetes mellitus the sixth major reason for death in the United States. Diabetes is the leading cause of renal insufficiency, blindness, non-traumatic amputations and is a major risk factor for development of vascular events.

Diabetics are particularly prone to oral infection. This can lead to poor glucose control. In general, it is important to aggressively control oral infection and this may result in more liberal use of antibiotics in the diabetic patient.

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ADEQUACY OF DIABETIC CONTROL: HEMOGLOBIN A1C

Patients whose diabetes is not under adequate control should raise a Red Flag since they are much more prone to having infection that is difficult to control. We now monitor adequacy of control by the determination of HbA1c, which provides an assessment of the average blood sugar level over a 90 day period.

All diabetics should have an HbA1c value from within the last 3-6 months. All **patients with a HbA1c of greater than 8.5%** should raise a **Red Flag**:

Hemoglobin A1C	Level of Control
< 6.5% (BS 135)	Good
6.5-8.5% (BS 150-200)	Fair
> 8.5% (BS >210)	**Poor (Red Flag)**

Ideal HbA1c is <6%. For every 1% decrease in HbA1c, there is a 21% decrease in mortality, 21% decrease in diabetic complications, 14% decrease in myocardial infarction and 37% decrease in microvascular complications.

In the diabetic patient with poor control (HgA1c > 8.5), there will be problems with tissue healing, a higher likelihood of infection and a much higher likelihood of hardware failure.

The adequacy of control and the presence of diabetic complication can then be used to guide dental management, including significant changes in treatment protocols. These guidelines form the basis for recommendations in the **Ultimate Cheat Sheets.**

SEDATION PROTOCOL FOR THE DIABETIC PATIENT

1. NPO after midnight
2. First morning appointment
3. Take half the normal doses of insulin or hypoglycemic agents
4. IV with D5 ½ NS at 75 cc/hour
5. Maximum 3-hour appointment
6. Make sure that the patient can resume oral intake afterwards

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SMOKING AS A RISK FACTOR

Smoking markedly accelerates vascular disease with resultant increased incidence of cardiovascular, cerebrovascular and peripheral vascular disease.

There are also special sedation issues that will be addressed elsewhere in the curriculum.

Most importantly, smoking is an important risk factor to address in the dental evaluation of the patient as a candidate for sedation

PUTTING ALL OF THESE RISK FACTORS TOGETHER: RISK CLUSTERING

It is increasingly clear that patients with one risk factor are more prone to having other risk factors that cluster.

Some common clustering:
1. Hypertension
2. Hyperlipidemia
3. Diabetes mellitus
4. Smoking history

If we are considering a patient with hypertension, it is clear that the likelihood that the patient will have other associated risks are high

Importance of Risk Cluster in Sedation Dentistry

The likelihood that a patient may have more than one of these common risk factor is therefore quite high. It is important to understand the each of the risk factor contributes to the overall risk of sedation.

While hypertension alone may constitute a certain risk, the presence of hypertension in a diabetic patient with hyperlipidemia and a smoking history would escalate the risk considerably.

In the instances where there is risk clustering, we should adjust the ASA accordingly: presence of more than 2 of these common risk factor should increase the ASA category by one grade. In essence, the diabetic patient with hypertension and hyperlipidemia would be an ASA III patient despite the fact that all of the conditions are under adequate control.

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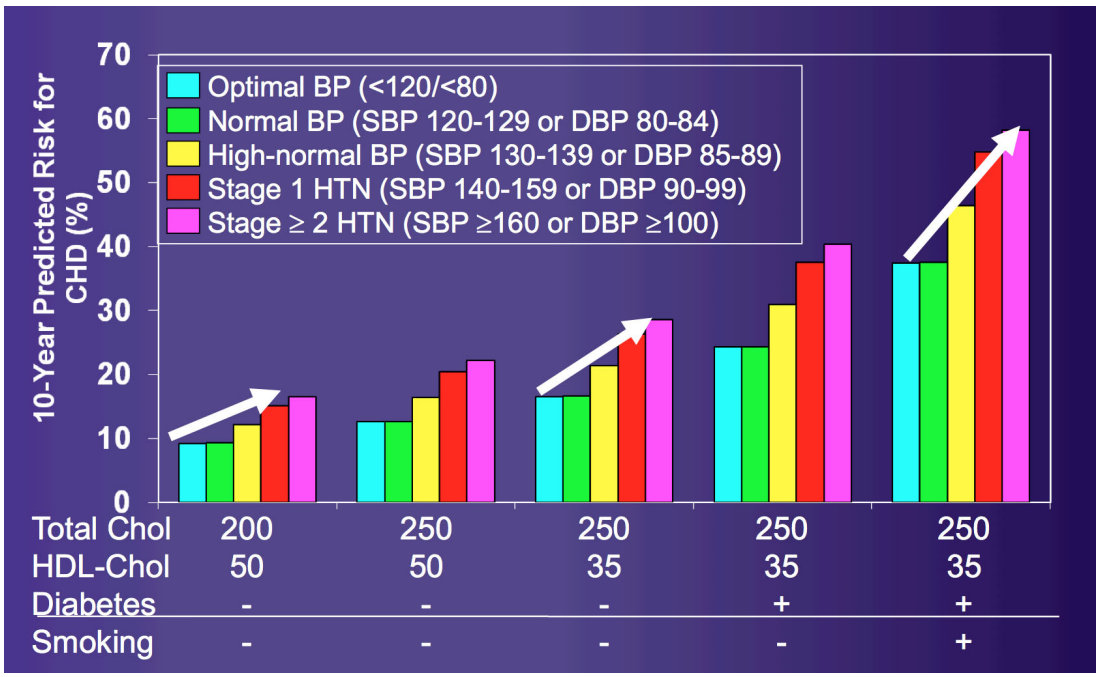
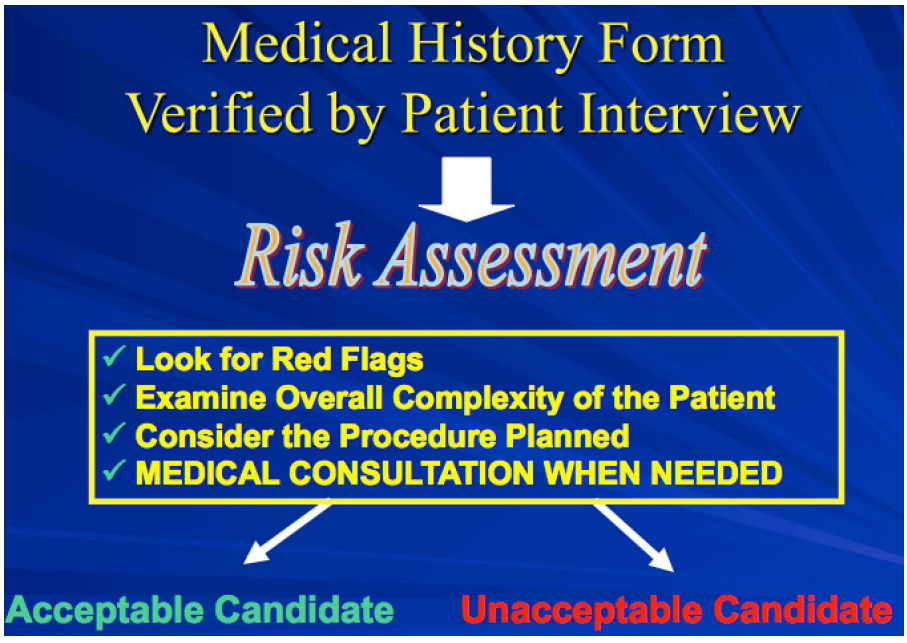


In conclusion, based on the medical history form, confirmed by patient interview, we would be able to look for red flags, examine the overall complexity of the patient, consider the procedure planned, consult medicine when appropriate, we would be able to determine the appropriate candidate for sedation.



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ANGINA

Angina is chest pain as a result of myocardial demand outstripping the oxygen supply to the heart.

Symptoms of angina arise from cardiac ischemia secondary to imbalance between cardiac demand and oxygen supply. Patients who have angina have significant coronary artery disease and are at increased risk for angina, arrhythmia and myocardial infarction.

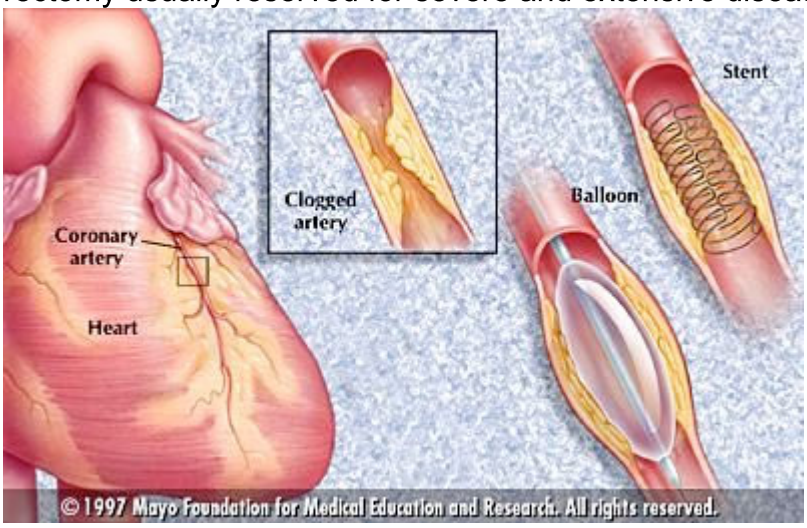
In 2012, most patients with symptomatic coronary artery disease undergo cardiac catheterization and have percutaneous interventions when appropriate. Patients with unstable angina, impending or evolving myocardial infarctions and patients with markedly positive stress tests are now treated with balloon angioplasty with the placement of stents.
These percutaneous coronary interventions (PCI) include:

Coronary angioplasty

Placement of stents
- Bare metal stents
- Drug eluting stents

Atherectomy

The choice of the procedure depends upon the severity of the disease with atherectomy usually reserved for severe and extensive disease.



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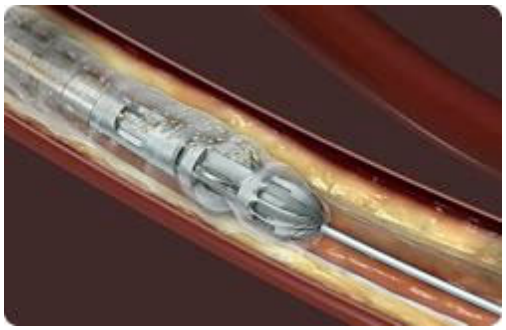

Many patients with coronary stents are on ASA and **Plavix** for 6 or more months after the placement of the stent.

Antibiotic premedication is not necessary for coronary stents. Because stents are not infrequently placed in patients with a myocardial infarction, it is prudent to avoid dental procedures within 6 months of a stent placement.

It is important not to interrupt Plavix therapy since premature discontinuation has been associated with acute thrombosis of the stent, particularly the newer drug-eluting stents. Patients on Plavix therapy would have platelet function abnormalities and special care should be placed on local hemostasis.

For every patient who identifies angina as a medical issue, you should ask the following questions:

1. How frequently do you have angina?
2. Has the pattern of your angina changed?
3. Have you had angina at rest?
4. Have you had recent changes in your medications for angina?

ANY positive answer to the above question is indicative of unstable angina. Patients with unstable angina would have increase in frequency or severity of angina. They may develop angina at rest and they may have breakthrough angina even on a maximal medical regimen.

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Since **unstable angina** is the prodrome to myocardial infarction, it should raise a **Red Flag**. Segregation of stable vs. unstable angina would provide a basis for changing dental management in the patient with coronary artery disease.

It is important to be careful with use of vasoconstrictors in any patient with angina.

Dental Management of Patients with Angina

Angina: chest pain as a result of myocardial demand outstripping the oxygen supply to the heart	
Stability of Angina	
Stable angina	Infrequent episodes Precipitated by stress and/ or exercise Relieved by rest Relieved by nitroglycerin Stable pattern
Unstable angina	Increasing frequency of episodes Increasing severity Angina with minimal exertion or stress Angina at rest

Emergency Management of Angina
Stop dental procedure
Administer nitroglycerin sublingually
Administer oxygen
Monitor blood pressure and pulse
If pain is not relieved after 5 minutes, give a second nitroglycerin and transfer patient to ER

Stress Reduction	Patient rapport; short appointments
Limit use of vasoconstrictors	2 carpules of local anesthetics containing 1:100,000 epinephrine or 1:20,000 levonordefrin
All non-surgical and simple surgical procedures	Normal protocol
Complex prosthetic and oral surgical procedures	Consider sedation protocol
Sedation Protocol	Consider oral conscious sedation Consider use of nitrous oxide

Dental examination only; refer back to MD for management
Palliative care under monitored environment
Complex prosthetic and surgical procedures should not be undertaken
Stress reduction is of paramount importance
Use of vasoconstrictors absolutely contraindicated

PROTOCOL FOR INTRAVENOUS SEDATION FOR PATIENTS WITH ANGINA:

- **Exclude all patients with unstable angina**
- First morning case
- Case limited to three hours
- NPO after midnight
- Morning anti-anginal medications with minimal amount of water
- Limit use of vasoconstrictors
- D_5 ½ NS at 75 cc per hour

MYOCARDIAL INFARCTION

Myocardial infarction is the result of irreversible ischemia culminating in damage of myocardial issue. This is usually a result of occlusive coronary artery disease secondary to plaque rupture. Patients who have had a

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myocardial infarction or have had the placement of a coronary stent within the past 6 months should raise a **Red Flag**. These patients are much more prone to developing rhythm disturbances, recurrent myocardial infarction and congestive heart failure.

Dental Management of Patients with Prior Myocardial Infarction

Myocardial Infarction: result of injury incurred as a result of prolonged ischemia to the heart

History of Myocardial Infarction	
Beyond 12 months	At slightly increase risk
6 to 12 months	Modest increase in risk
Within past 6 months	At significant risk for arrhythmia, congestive heart failure and recurrent myocardial infarction

Emergency Management of MI
Stop dental procedure
Administer nitroglycerin sublingually
Administer oxygen
Administer nitrous oxide, if available
Monitor blood pressure and pulse
Ask patient to chew an aspirin 325 mg
If pain is not relieved after 5 minutes, give a second nitroglycerin and transfer patient to ER

Stress Reduction	Patient rapport; short appointments
All non-surgical and simple surgical procedures	Normal protocol
Complex prosthetic and oral surgical procedures	Consider sedation protocol
Sedation Protocol	Consider oral conscious sedation Consider use of nitrous oxide
Limit use of vasoconstrictors	2 carpules of local anesthetics containing 1:100,000 epinephrine or 1:20,000 levonordefrin

Dental examination only; refer back to MD
Palliative care under monitored environment
Complex prosthetic and surgical procedures should not be undertaken
Stress reduction is of paramount importance
Use of vasoconstrictors absolutely contraindicated

Medications with Dental Implications	
Calcium channel blockers	Gingival hyperplasia
Non-selective beta blockers	Epinephrine may accentuate hypertension; levonordefrin is the preferred vasoconstrictor

PROTOCOL FOR INTRAVENOUS SEDATION FOR PATIENTS WITH A PRIOR MYOCARDIAL INFARCTION:

- **Exclude all patients with MI within the past 6 months**
- First morning case
- Case limited to three hours
- NPO after midnight
- Morning cardiac medications with minimal amount of water
- Limit use of vasoconstrictors
- D_5 ½ NS at 75 cc per hour

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Patients with chronic obstructive pulmonary disease pose a special problem for sedation. These patients have low baseline oxygen saturation and some may have a history of **carbon dioxide retention.**

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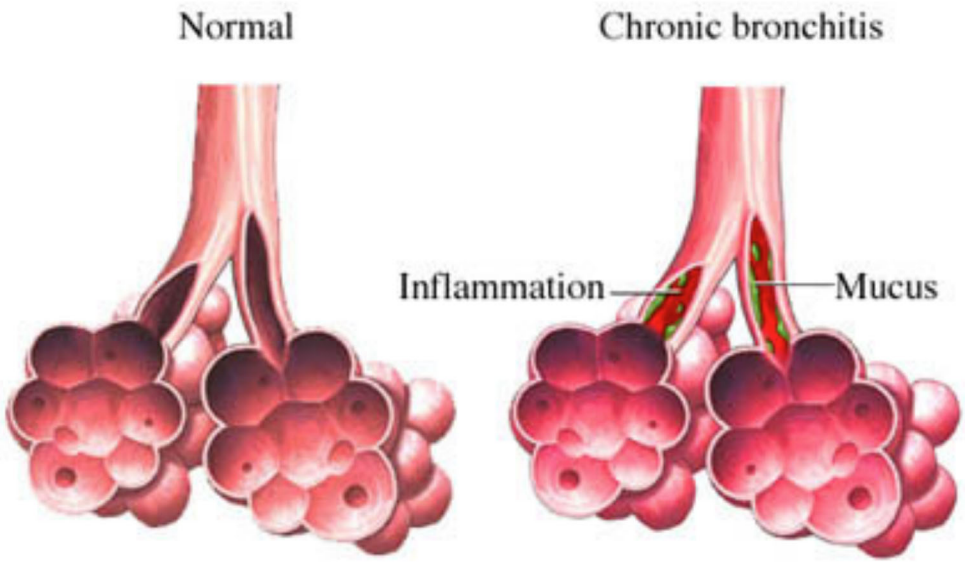
It is critical to consult the physician about the nature of the pulmonary disease and the pulmonary reserve. One should specifically ask if the patient has a history of carbon dioxide retention.

There are two types of COPD:

1. Chronic Bronchitis



Patients with chronic bronchitis have inflammation in the bronchi. The over-secretion of mucous from the glands of the bronchial tree would result in cough and sputum production The mucous produced can also cause obstruction in the form of mucous plugs. This can create problem with ventilation, hypoxemia and, in severe cases, carbon dioxide retention.

2. Emphysema

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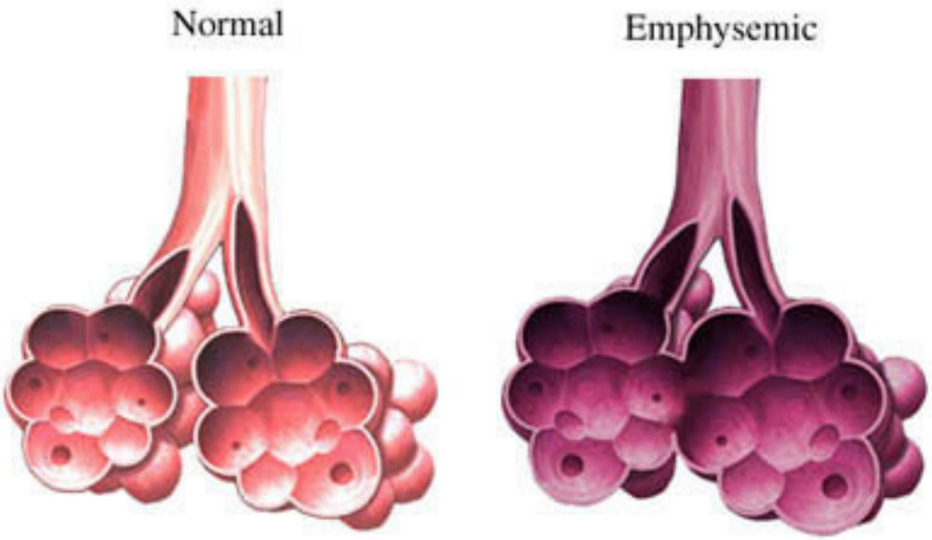

Patients with emphysema have obliteration of the septae between their air sacs. This markedly reduces the area available for gas exchange. Patients with emphysema can have problems with ventilation, hypoxemia and, in severe cases, carbon dioxide retention.

The most important history to obtain in patients with COPD is the adequacy of control of their respiratory symptoms. Patients with frequent flares or patients with carbon dioxide retention should raise a **RED FLAG.**

Patients with a history of carbon dioxide retention have severe COPD and sedation is absolutely contraindicated.

PROTOCOL FOR INTRAVENOUS SEDATION FOR PATIENTS WITH COPD

- **Exclude all patients with history of carbon dioxide retention**
- First morning case
- Case limited to three hours
- NPO after midnight
- Morning COPD medications with minimal amount of water
- Instruct patient to bring his/her own bronchodilator
- Limit use of vasoconstrictors
- Oxygen supplementation
- D_5 ½ NS at 75 cc per hour

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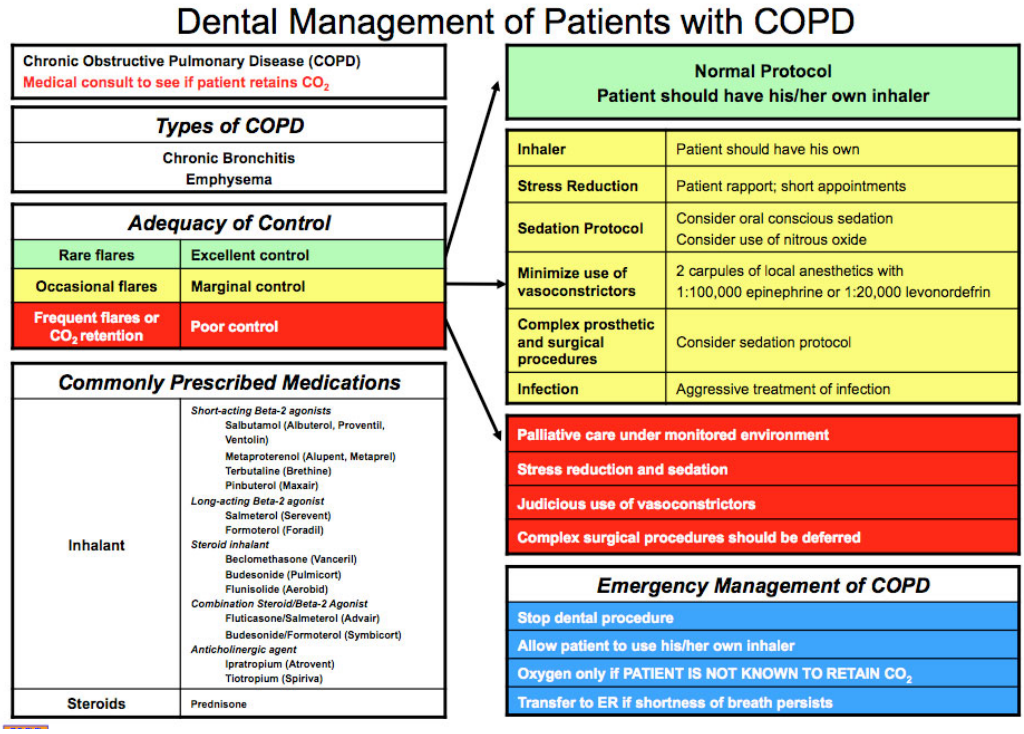

ASTHMA

Asthma is a conditioned characterized by episodic reversible narrowing of the airways. This would result in acute episodes of shortness of breath and wheezing. The disease can begin at any age but about half the patients develop asthma before the age of 10.

Asthma affects about 2% of the population and is responsible for 50,000 deaths per year in the United States. Asthma is responsible for 2 million visits to the emergency room annually.

The most common cause of asthma is an inherited immunologic disorder that allows inhaled antigens to trigger a hypersensitivity response mediated by immunoglobulin E (IgE) and thus produce reversible bronchial narrowing. The trigger can be inhaled allergens, cold or exercise.

A detailed history is crucial in the dental evaluation of patient with asthma. The severity of the patient's asthma should be noted. Patients with prior history of respiratory insufficiency are obviously problematic and should be managed cautiously.

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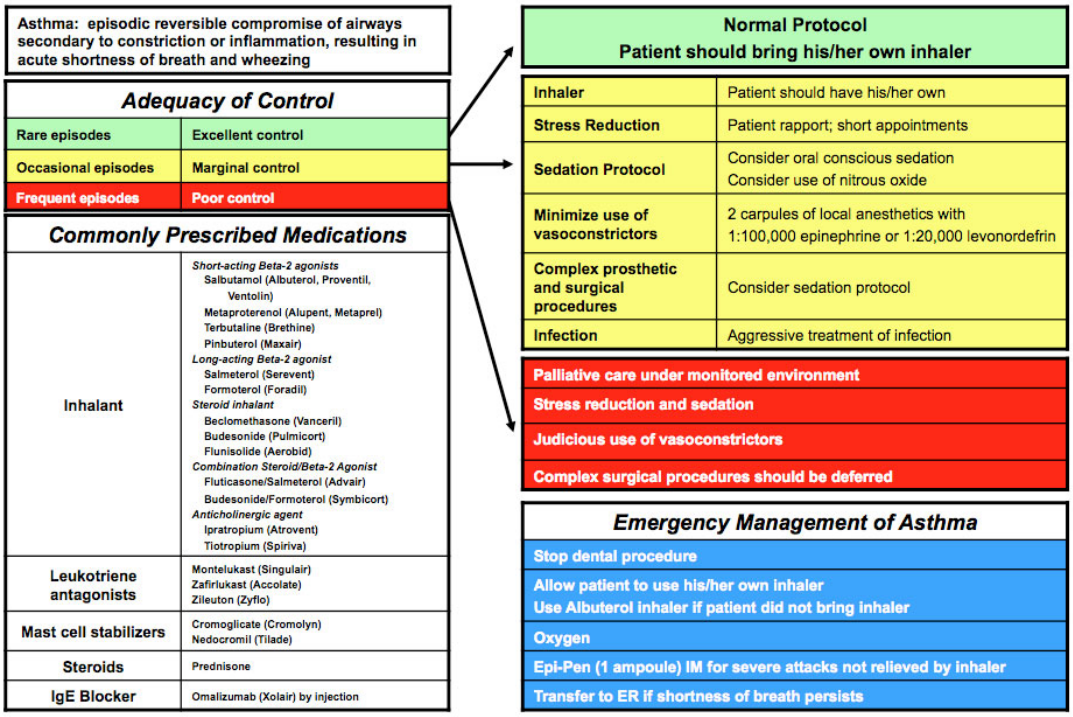
The frequency of the asthmatic attacks should be noted. Factors that would precipitate asthma should be noted. The medications taken should be recorded.

Severity of asthma is usually assessed by spirometry and pulmonary function tests. Measurement of **peak expiratory flow rate (PEF)** is a simple way of determining severity of asthma. Patients with severe asthma would have PEF of <50%. These patients should raise a **RED FLAG** and should be managed cautiously.

PEF > 90%	Mild disease
PEF 50-90%	Moderate disease
PEF < 50%	Severe disease

Dental Management of Patients with Asthma



PROTOCOL FOR INTRAVENOUS SEDATIO FOR PATIENTS WITH ASTHMA

- First morning case
- NPO after midnight
- Case limited to three hours
- Morning asthma medications with minimal amount of water
- Instruct patient to bring his/her own bronchodilator
- No hydroxyzine

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- Oxygen supplementation
- Limit use of vasoconstrictors
- D_5 ½ NS at 75 cc per hour

VIRAL HEPATITIS

Viral hepatitis is an infection of the liver by a virus. It is a contagious disease that affects approximately 500,000 patients in the United States every year. The majority of these patients have symptoms of an acute infection from which they recover. However, a small number of patients can progress on to have chronic hepatitis.

While there are many viruses that can cause hepatitis, the most common hepatitis in the United States are Hepatitis A, B and C.

Important laboratory tests that we use to assess liver function include:

- PT/INR, normal 1
- Platelets, normal >150,000
- Normal SGOT < 150 mU/ml
- Normal bilirubin < 1.5 mg/dl
- Normal alk p'ase < 4.5 BU

Liver normally has important synthetic and biotransformation function. In considering a patient for intravenous sedation, abnormal liver function can result in inadequate clotting factors and inadequate drug metabolism and excretion.

Hepatitis A

Hepatitis A has very low virulence and is usually a mild disease. Patients recover from the acute illness and have very limited sequellae.

Patients with hepatitis A can be managed with normal protocol.

Hepatitis C

Hepatitis C is a viral hepatitis transmitted largely by blood exposure. Exposure to other body fluid can sometimes cause hepatitis C. Patients with hepatitis C often would have mild symptoms of fever, malaise, nausea, vomiting and jaundice. While the acute disease is usually mild, patients with hepatitis C would often have persistent disease, leading eventually to cirrhosis and liver failure.

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Hepatitis C is now one of the leading causes of cirrhosis, following alcoholic liver disease.

Hepatitis C can be detected by determination of hepatitis C antibody in the serum. Extent of infection can be monitored by the determination of the viral load by measuring hepatitis C viral RNA.

Patients with hepatitis C should exercise the same precautions as for patients with hepatitis B with limited exposure to blood and body fluids of the infected patients.

We have now identified 6 genotypes of hepatitis C.
Patients with chronic compensated hepatitis C (genotype 1, which accounts for 75% of patients, 4, 5 and 6) who have sustained aminotransferase elevations and detectable HCV RNA should be treated.
Management of these patients is now largely revolving around ledipasvir and sofobuvir (Harvoni) from Gilead Science.

For patients with genotype 2 and 3, combination of sofobuvir and velpatasvir (Eplusa).

Patients with end stage liver disease should be considered candidates for liver transplantation.

In the dental evaluation and management of patients with hepatitis C, it is important to know PT/INR, CBC and platelet count, liver function tests. If there is no indication of a bleeding diathesis, one can proceed with normal protocol.

Hepatitis B

Hepatitis B is predominantly transmitted by a parenteral route. Patients with acute hepatitis B would have evidence of the virus in all bodily secretions including blood, sweat, saliva, semen and vaginal secretions. Hepatitis B is highly infectious and can be passed on from mother to infant and from spouse to spouse.
After exposure to the virus, there is an incubation period of about 12 weeks (range 6 to 24 weeks), during which the infecting virus replicates. The patient then has a prodromal phase when non-specific symptoms occur. The patients would have anorexia, nausea, low-grade fevers and malaise. Patients would then enter into an icteric phase when they have fevers, chills, malaise, arthralgia, anorexia, nausea and vomiting, together with clinical icterus. About 10% of the patients become chronic carriers

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and remain infectious after the acute illness. These patients are more prone to development of cirrhosis and hepatoma.
About 2 weeks prior to the onset of symptoms, the surface antigen to hepatitis B (HBsAg) is detectable in the serum. Shortly thereafter, enzymes associated with hepatocellular damage (SGOT, SGPT) begin to increase, denoting inflammation of the liver. Subsequently, antibodies to hepatitis B develop. Patients with antibodies to hepatitis B (HBsAb) have protective antibodies and are conferred life-long immunity. Unfortunately, about 10% of the patients never develop antibodies. These patients would have persistent antigenemia (HbsAg positive) with no antibody formation (HbsAb negative). Infectivity of the virus is usually associated with presence of the e antigen (HbeAn positive). These patients are chronic carriers and remain infectious and continue to have hepatocellular damage.
Active or persistent disease can be detected by the persistence of HbeAn, a marker of ongoing viral infection. Viral load can be measured by HB DNA in the blood.

Hepatitis B can be a very serious disease. Some patients with fulminant hepatitis can develop severe hepatocellular damage with resultant hepatic insufficiency and all of the complications thereof.

All dental professions who are HBAg and HBAb negative should have hepatitis B vaccination (series of three) and check for hepatitis B antibody status. On rare occasions, a second series may be necessary. Booster may be necessary every 5-10 years.

For patients with chronic “replicative” hepatitis B, lamivudine (Epivir), tenofovir (Viread) or entecavir (Baraclude) therapy should be considered. This should be continued for one year. Patients who lose HBeAg can stop the therapy while patients who retain HBeAg should have lamivudine continued. For patients with refractory disease, therapy with emtricitabine (Truvada or Emtriva) is often employed.

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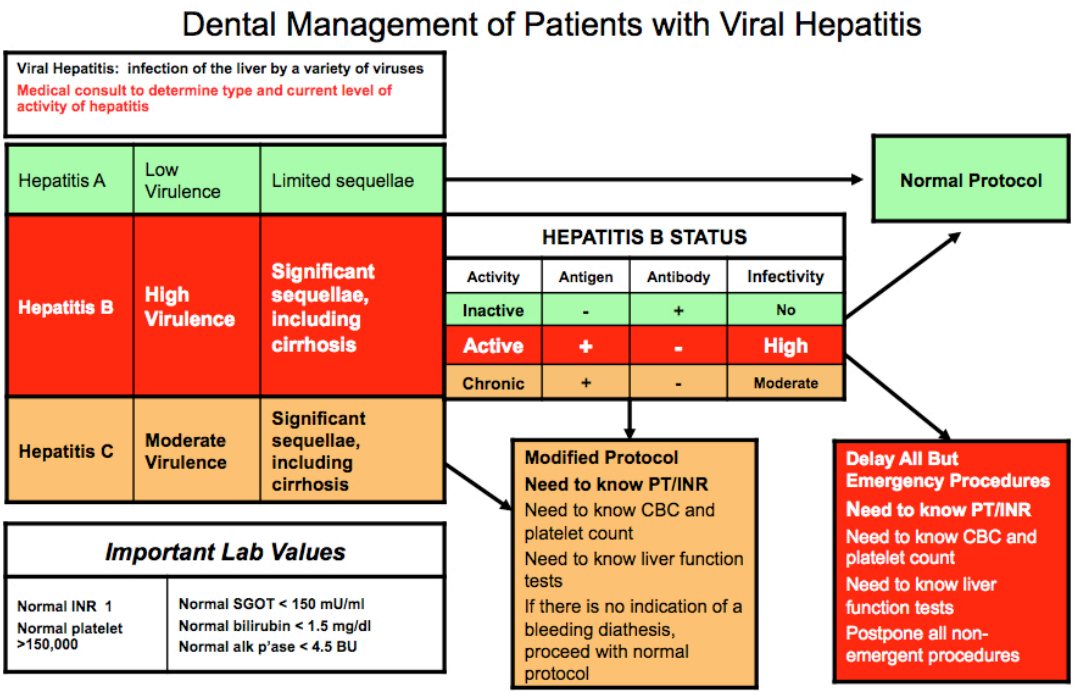

PROTOCOL FOR INTRAVENOUS SEDATION OF PATIENTS WITH HEPATITIS

- First morning case
- NPO after midnight
- **Defer procedures during active phase of hepatitis**
- Check INR, CBC and liver function tests
- Defer procedures if INR, CBC or liver function tests are abnormal
- D_5 ½ NS at 75 cc per hour

CIRRHOSIS

Cirrhosis is the result of severe or protracted injury to the liver causing loss of liver cells and progressive scarring. It is the fourth leading cause of death in young men between the ages of 35 and 54. The major cause of cirrhosis is alcoholic liver disease.

The patient with cirrhosis presents a special problem to the dentist. Inadequate synthesis of clotting factors may result in prolongation of prothrombin time and clinical bleeding. The inability to detoxify drugs may result in drug accumulation, and the use of even minor sedatives must be carefully weighed.

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Patients with cirrhosis pose a special problem to the dentist. Inadequate synthesis of clotting factors may result in prolongation of prothrombin time and clinical bleeding. Splenic sequestration and destruction of platelets would lead to thrombocytopenia with resultant bleeding. Inability of the liver to detoxify rugs may result in drug accumulation and the use of even minor sedatives must be carefully weighed.

Patient can also develop portal hypertension and the resultant complications of esophageal varices and ascties. Presence of portal hypertension is indicative of severe liver disease and these patients are NOT candidates for sedation.

Patients with cirrhosis may have abnormal liver function tests with elevated transaminases (SGOT, SGPT), alkaline phosphatase and bilirubin. These abnormalities can reflect the degree of ongoing inflammation and portal disruption.
Because of splenomegaly, there is increased sequestration and destruction of platelets in the spleen and patients can often have thrombocytopenia with significant clinical bleeding.
Compromises in hepatic synthesis can result in abnormalities in the levels of clotting factors. Patients often have elevated prothrombin time, accentuating the bleeding diathesis. The poor synthetic capability also results in decreased serum albumin. The degree of hypoalbuminemia and the degree of prolongation of PT will give the clinician an idea of the degree of compromise of synthetic activity.
In patients with alcoholic liver disease, dietary deficiency may result in decreased folate and B12 levels, resulting in macrocytic anemia. B12 and folate deficiency may also compromise platelet production.

Dental Evaluation of Patients with Cirrhosis

1. Potential bleeding problems: The patient should be asked about any history of abnormal bleeding. Clotting factor abnormalities and thrombocytopenia may be present in the patient with severe liver disease. Clotting factor deficiency is the result of inability of the cirrhotic liver to synthesize factors. Platelet deficiency is the result of platelet production difficulties and problems with sequestration in the patient with hypersplenism
2. The patient should be asked specifically about a history of encephalopathy or variceal bleeding since these patients are at much higher risk for any intervention.
3. Knowledge of their dietary restrictions and the medications they are on will also provide important clues about the severity of the liver disease. Patients with ascites are likely to have salt and fluid restriction and are on spironolactone and furosemide. Patients with a history of encephalopathy

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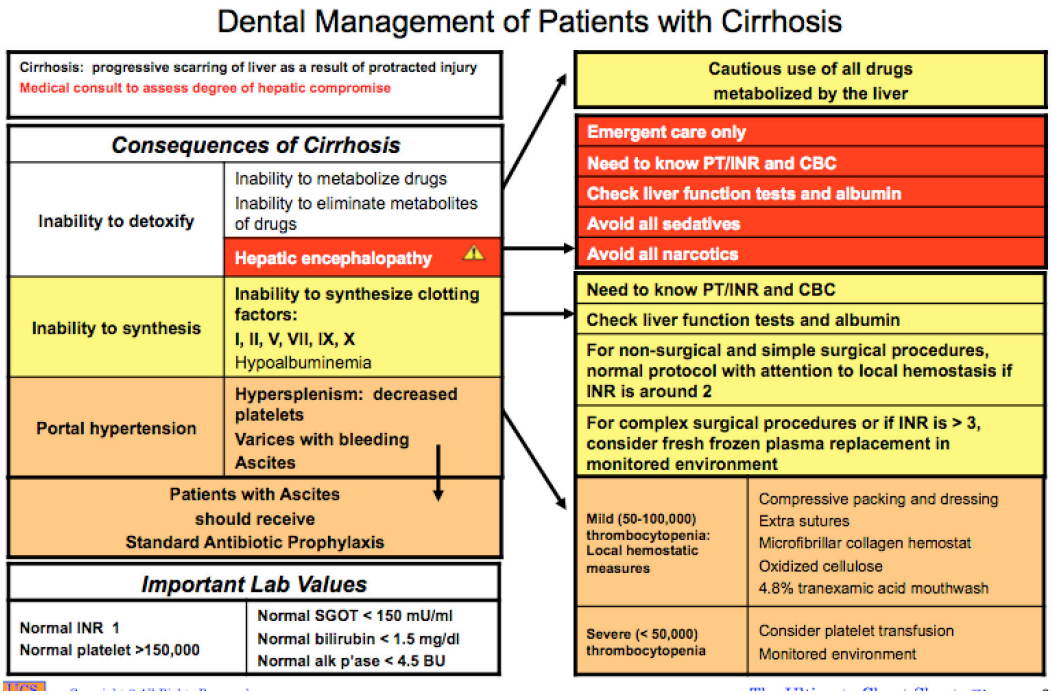
are likely to have protein restrictions and are on lactulose, neomycin or Metronidazole.

4. Labs that should be checked should include:
   a. Liver function tests
   b. CBC with platelet count
   c. PT/INR
   d. Albumen



PROTOCOL FOR INTRAVENOUS SEDATION FOR PATIENTS WITH CIRRHOSIS

- **Exclude all patients with history of hepatic encephalopathy, ascites and esophageal varices**
- Check INR, CBC and liver function tests
- Defer procedures if INR, CBC or liver function tests are abnormal
- D_5 ½ NS at 75 cc per hour

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KIDNEY DISEASE

The normal kidney has 5 major functions:

a. Excretion of nitrogenous waste
b. Excretion of drugs and drug metabolites
c. Maintenance of fluid and electrolyte balance
d. Acid base balance
e. Production of hormones
   -Renin
   -Vitamin D
   -Prostaglandin
   -Erythropoeitin

Because of the various functions the kidneys have, its failure will lead to:

i. Problems with excretion of drug and drug metabolites
ii. Uremic symptoms
iii. Fluid overload with resultant hypertension and congestive heart failure
iv. Metabolic acidosis
v. Excessive renin would lead to hypertension
   Absence of vitamin D would lead to hypocalcemia and hyperphosphatemia
   Absence of erythropoietin can lead to profound anemia

Nephrosclerosis and diabetes mellitus are the major causes of renal insufficiency.
Other causes include polycystic kidney disease, glomerulonephritis, interstitial nephritis, vascular disease, urinary tract obstruction and nephrotoxicity.

Patients with renal insufficiency have elevated BUN (>20 mg/dL) and creatinine (>1.5 mg/dL). They are usually anemic and would have hypocalcemia and hyperphosphatemia. PTH is often secondarily elevated and both erythropoietin and vitamin D levels are depressed.

Dental Evaluation of Patient with Renal Failure

Patients with BUN > 50 often have thrombocytopathy and care should be given to addressing issues of hemostasis

Many drugs used in dentistry are contraindicated in patients with renal insufficiency. Some of the contraindicated drugs include:

-Non-steroidal anti-inflammatory drugs
-Cox 2 inhibitors
-Tetracycline, except for Doxycycline
-Quinolone dose has to be adjusted

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Many of the drugs commonly used in dentistry are not affected. These include:

-Penicillin and derivatives
-Clindamycin
-Erythromycins
-Macrolides such as azithromycin and clarithromycin
-Cephalosporins
-Codeine, hydrocodone, oxycodone
-Acetaminophen

Hemodialysis

There are multiple issues to be addressed:

1. Access often determines need for antibiotic prophylaxis
   Patients with fistula do not generally need antibiotic prophylaxis
   Patients with AV graft should get antibiotic prophylaxis
   Patients with tunneled catheter should get antibiotic prophylaxis
2. Heparin during dialysis
   Heparin is generally used during hemodialysis
   Heparin is relatively short-lived and should not create problems with hemostasis beyond 4 hours after dialysis
3. Scheduling around the dialysis schedule
   Because of the use of heparin during dialysis, it is prudent to schedule dental procedures on a non-dialysis day.
4. Avoid drugs that are toxic to kidneys or excreted by the kidneys.

Peritoneal dialysis

There are two major forms of peritoneal dialysis, CAPD and CCPD
In both instances, the major risk for the patients is that of peritonitis.
For patients on peritoneal dialysis, standard antibiotic prophylaxis is appropriate.

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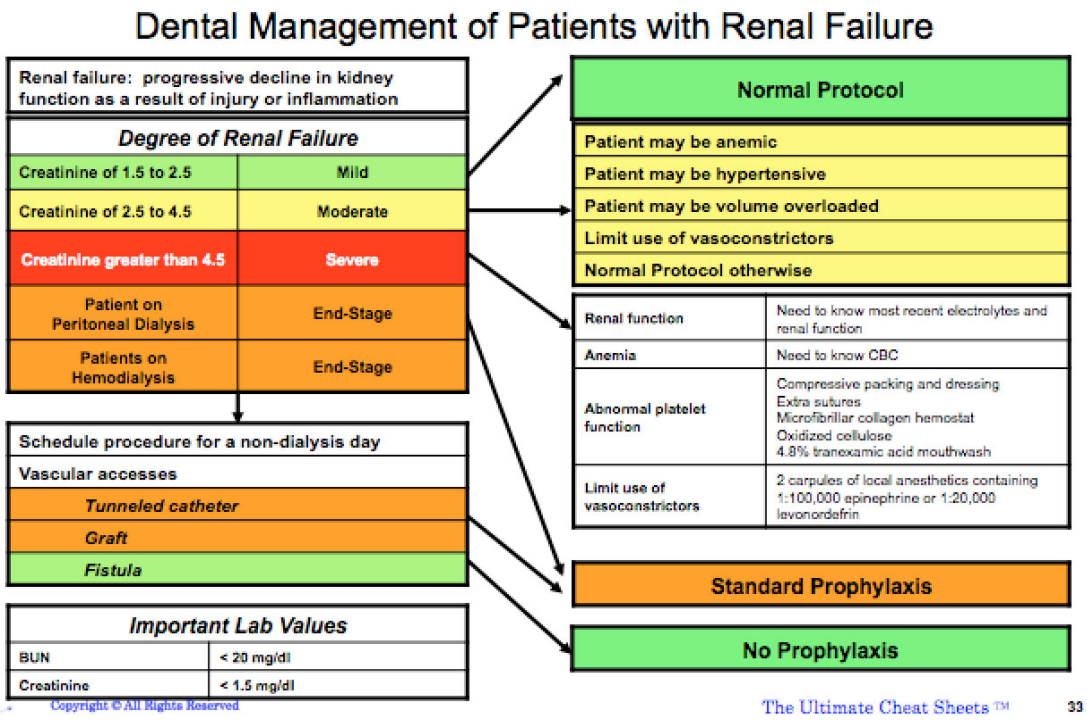

HIV INFECTION

It is estimated that the prevalence of HIV infection is about 1,185,000 in the United States (1 in 280 patients). 42,000 new cases are diagnosed annually. Unfortunately, one quarter of the patients with HIV is currently undiagnosed (252,000-312,000).

With the practice of universal precautions, caring for the HIV patient does not pose a special problem.

However, patients with HIV infection pose a special concern for sedation dentistry. Many patients are on:

Protease inhibitors
Anti-fungal agents

These should raise **Red Flags** since they will have significant interactions (category D drug-drug interactions) with many of the drugs that would be used for sedation dentistry.

Patients with HIV are not obligated to tell you of their disease but they are obligated to tell you all of the medications they are on.

There ares obviously significant advances in the field of HIV infection and attempts are now made to treat high-risk individuals with Truvada. The

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hope that we would be able to cure the infection is also increasing daily. The hope that we would be able to make a vaccine, however, continues to be elusive.

PREGNANCY

Since many of the drugs used in sedation dentistry are classified as Category D for the pregnant females: **There is positive evidence of human fetal risk**, but the benefits from use in pregnant women may be acceptable despite the risk (e.g., if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective). **Triazolam is classified at Category X, where the risk of the drug clearly outweighs the benefit.**

It is therefore extremely important to be sure that the patient is not pregnant before consideration of sedation dentistry. Any possibility that the patient might be pregnant should raise a **Red Flag**.

The following table explains the assignment of the various drug categories in the pregnant female:

Category A	Controlled studies in women fail to demonstrate a risk to the fetus in the first trimester (and there is no evidence of a risk in later trimesters), and the possibility of fetal harm appears remote.
Category B	Either animal-reproduction studies have not demonstrated a fetal risk but there are no controlled studies in pregnant women, or animal-reproduction studies have shown an adverse effect (other than a decrease in fertility) that was not confirmed in controlled studies in women in the first trimester (and there is no evidence of a risk in later trimesters).
Category C	Either studies in animals have revealed adverse effects on the fetus (teratogenic or embryocidal or other) and there are no controlled studies in women, or studies in women and animals are not available. **Drugs should be given only if the potential benefit justifies the potential risk to the fetus.**
Category D	**There is positive evidence of human fetal risk**, but the benefits from use in pregnant women may be acceptable despite the risk (e.g., if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective).
Category X	Studies in animals or human beings have demonstrated fetal abnormalities, or there is evidence of fetal risk based on human experience or both, and **the risk of the use of the drug in pregnant women clearly outweighs any possible benefit**. The drug is contraindicated in women who are or may become pregnant.

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UPDATE on PROTOCOL

PREMEDICATION FOR PROSTHETIC JOINTS POST-SEDATION ANALGESICS

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Harvard Medical School

2015 ADA Council on Scientific Affairs Recommendations on Prosthetic Joint Infections PUTTING IT INTO PRACTICE

INTRODUCTION

Approximately 200,000 primary total hip arthroplasties and 400,000 primary total knee arthroplasties were performed in the United States in 2003. It is projected that it will increase to 380,000 hip procedures and over 1,500,000 knee procedures by 2020.

Prosthetic joint infection rates range from 0.3% to 8.3% in the published literature. The infection can be caused by:

Entry of organisms into the wound during surgery
Hematogenous spread
Recurrence of sepsis in a previously infected joint
Contiguous spread from a local source

For decades, there has been debates about whether:

1. Dental interventions can cause bacteremia
2. Antibiotic prophylaxis can reduce incidence of bacteremia
3. Bacteremia from an oral source can lead to prosthetic joint infection.

HISTORY

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Initial guidelines were established in 1997 by AAOS and ADA and refined in 2003. The work group concluded that “The risk/benefit and cost/effectiveness ratios failed to justify the routine administration of routine antibiotic prophylaxis”. The recommendation is to use antibiotic prophylaxis only for the first 2 years after joint placement, for patients with high-risk joints and for patients with compromised immune status.

Unfortunately, the definition of what constitute high-risk joints and compromised immune status continues to change and it is not clear how obesity and diabetes crept onto the list.

By far the most unfortunate Information Statement from the Patient Safety Committee of the AAOS came in 2009 that opined “Given the potential adverse outcome and cost of treating an infected joint replacement, the AAOS recommended that all clinicians consider antibiotic prophylaxis for all total joint patients prior to any procedure that may cause bacteremia.”

This position was so contrary to clinical evidence that a furor soon ensued and in 2010, AAOMS believes that this Information Statement should not replace the 2003 consensus statement.

The 2012 recommendation is a very different kind of critical thinking, based entirely on Evidence Based Medicine. It replaces the 2009 AAOS Information Statement and states that it is important to note that an Information Statement is “an educational tool based on the opinion of the authors.”

NEW 2012 AAOS/ADA RECOMMENDATIONS: HOW WERE THEY DERIVED?

Goal is to examine the medical evidence (or lack thereof) with regard to the role of bacteremia from dental procedures and prosthetic join infection
Entirely evidence based
Multi-disciplinary: Equal representation from AAOS and ADA plus multiple other disciplines
Meets or exceeds all recommended Institute of Medicine standards
People with potential conflicts were excluded
People with strong biases were excluded

Evidence is based on review of available literature to judge the validity of the evidence. The evidence is **graded as:**

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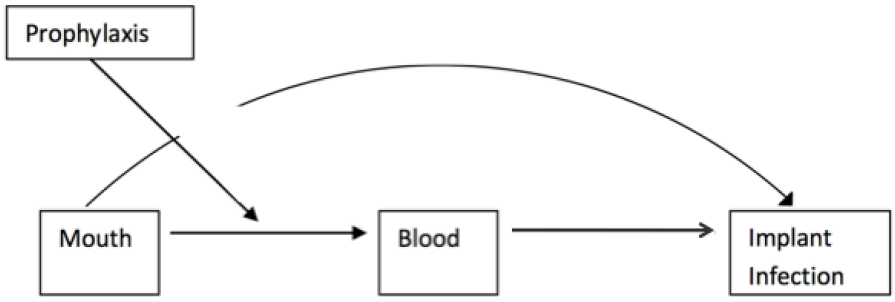
Strong
Moderate
Limited
Inconclusive
Consensus:
- No associated harm
- Relatively low cost
- Reflects current, routine clinical practice

The final guidelines reflect work from October 2010 to December 2012. The result is a 325-page dissertation that defined the issues of concern

QUESTIONS THE COMMITTEE ADDRESSED

1. Do dental interventions cause bacteremia?
2. Does antibiotic prophylaxis decrease the incidence of bacteremia related to dental procedures?
3. Does bacteremia from an oral source cause prosthetic joint infection?



Based upon evidence based medical evaluation, the Committee came to the following consensus:

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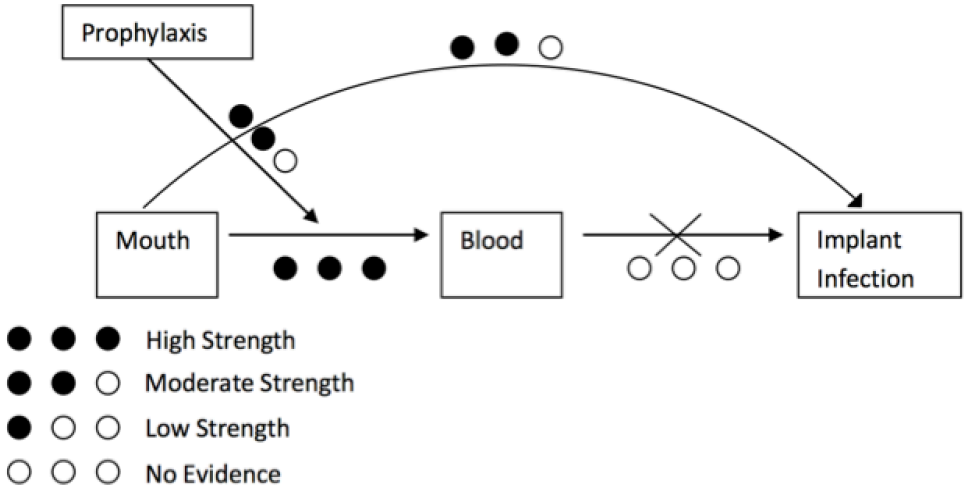

1. There is strong evidence that dental procedures cause bacteremia (High Strength)
2. There is moderately strong evidence that antibiotic prophylaxis decreases incidence of bacteremia
3. There is **NO EVIDENCE** that bacteremia from oral source is causally related to implant infection

The last statement is derived from extensive evaluation of evidence based literation but the most convincing data came from a study by Barbari, etal who connected a case controlled study examining 339 patients with prosthetic joint infection against 339 patients with similar demographics or comorbidities and compared the two groups for differences in dental visits (exposure), high and low-risk dental procedures, with and without antibiotic prophylaxis. They concluded that low and high-risk dental procedures performed within 6 months and 2 years of the hospital admission date were not significantly associated with increased risk of prosthetic hip or knee infection compared with no dental procedures. They also found that antibiotic prophylaxis did not decrease the risk of infection.

RECOMMENDATIONS with GRADES:

RECOMMENDATION 1: Practitioner might consider changing the longstanding practice of prescribing prophylactic antibiotics for patients who undergo dental procedures

This recommendation is founded in evidence that dental procedures are unrelated to prosthetic joint infection. Antibiotic prophylaxis does not reduce the risk of prosthetic joint infection.

GRADE: Limited

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- Practitioners should be cautious in deciding whether to follow a recommendation classified as Limited
- Should exercise judgment and be alert to emerging publications that report evidence
- Patient preference should have a substantial influencing role

Unfortunately, the Limited Grade of Recommendation leaves it as a free-for-all when it comes to the practical issues of pre-medication.

With the limited grade, patient, the orthopedic surgeon, the primary care physician and the dental professional all can decide that the patient can needs pre-medication. In practice, many orthopedic surgeons continue to insist on pre-medicating all joints forever, despite the total lack of evidence that there is any association between dental source of bacteremia and prosthetic joint infection.

The 2015 Recommendations is an attempt to clarify the issues.

2015 ADA Council on Scientific Affairs on The Use of Prophylactic Antibiotics Prior to Dental Procedures in Patients with Prosthetic Joints

Three additional case-control studies reviewed since the 2012 report. The best is still the original Barberi study. Three of 4 case control studies found no evidence that dental procedures is associated with prosthetic joint implant infection. One found a negative correlation (that dental procedures protected the patient from prosthetic joint implant infection).

CONCLUSIONS:

1. There is evidence that dental procedures are not associated with prosthetic joint implant infections
2. There is evidence that antibiotics provided before oral care do not prevent prosthetic join implant infections
3. There are potential harm of antibiotics including risk for anaphylaxis, antibiotic resistance and opportunistic infection like C difficile
4. The benefits of antibiotic prophylaxis may not exceed the harms for most patients

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5. Given this information in conjunction with the potential harm from antibiotic use, using antibiotics before dental procedures is not recommended to prevent prosthetic joint implant infection.
6. The individual patient's circumstances and preferences should be considered when deciding whether to prescribe antibiotics prior to dental procedures.

While these are very strong statements, the last point still leaves room for wiggle room.

NEW AAOS APPROPRIATE USE CRITERIA (AUC) 2016

The AAOS published in December of 2016 their Appropriate Use Criteria (AUC) that makes appropriate use of premedications a great deal clearer:

Immunocompromised status

- Stage 3 AIDS (CD4 count less than 200)
- Cancer patients on immunosuppression with fever and neutropenia
- Cancer patient on immunosuppression with severe neutropenic
- Rheumatoid arthritis on biologic disease modifying agents or prednisone > 10 mg/day
- Solid organ transplant on immunosupprssants
- Inherited diseases of immunodeficiency
- Bone marrow transplant patients (Pre-transplant and 100 days post-transplant)

Glycemic control

IN PRACTICE

The general assumption is that antibiotic prophylaxis is NOT necessary

Exceptions could be mandated by the patient's preference, the orthopedist's preference (communicated to you by fax or by letter on file), the patient's physician's preference or your own preference

In all instances, the rationale for giving antibiotic prophylaxis should be on file

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Possible Scenarios:

1. **Patient insists on pre-medication:** informed consent as documentation
2. **Communication with orthopedic surgeon:** send by fax, keep the stamped original:
   "In view of the 2015 ADA and the 2016 AAOS AUC recommendations, we would not be prescribing antibiotic premedication to our mutual patient unless you feel otherwise, in which case you should provide our office with your clinical rational and it would be most appropriate if you could write the prescription for pre-medication."
3. **Communication with primary care physician:** same as above, but generally not an issue
4. **The dental professional:** You are allowed to pre-medicate if you indicate in the file your rationale for so doing

If you are to prescribe pre-medication (which you probably should not):

No Penicillin Allergy	***Penicillin Allergy***
Cephalexin 2 gm **preferred because of the excellent anti-staphylococcal coverage** **Cephradrine 2 gm** **Amoxicillin 2 gm**	**Clindamycin 600 mg** **Cephalexin 2 grams (should not be used in patients with history of anaphylaxis, urticaria or angioedema to penicillins or ampicillin)**
All medications should be given 60 minutes before procedure	

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POST-SEDATION ANALGESIA

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PRE-EMPTIVE ANALGESIA

Studies examining pain control have repeatedly demonstrated that having analgesic on board prior to the onset of pain markedly enhance the efficacy of the analgesics. It is clearly understood that once pain has occurred, it significantly sensitizes the patient to future stimuli.

Optimal use of analgesics should follow the following guidelines:

1. Select analgesics based upon anticipated pain from the procedure
2. Start analgesics prior to the onset of pain
3. Use analgesics with long half-life to minimize the likelihood of breakthrough pain
4. Advise patient to take medication precisely as directed
5. Do not permit pain to break through

In the post-sedation patient, narcotics should be avoided and peripherally acting analgesics should be used. In the majority of the cases, analgesics should be adequate for management of mild to moderate post-operative pain.

ACETAMINOPHEN

Acetaminophen should form the backbone of analgesia in the post-sedation patient. Acetaminophen can be used singly or in combination with other analgesics. Acetaminophen is an analgesic but not an anti-inflammatory agent.

The new FDA guidelines now limit the maximal dose of acetaminophen to 3 grams per day. Most importantly, in patients with liver disease, the maximal dose of acetaminophen should not exceed 1.5 gram per day. This revision of guidelines has led to reformulation of a number of popular drugs such as the various formulation of Vicodin. It is important for the dental professionals to begin to prescribe agents singly (acetaminophen separate from oxycodone or hydrocodone) since it is probable that all combination narcotics would be off of the market in 2017.

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NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

It is now understood that there are two cyclooxygenases involved in the metabolic pathway of arachidonic acid in prostaglandin synthesis. Cyclooxygenase 1 is involved in the maintenance of gastric integrity, renal function and platelet adhesiveness. Cyclooxygenase 2 is involved in control of pain, inflammation, tissue repair and renal function. Traditional NSAIDs inhibit both Cox-1 and Cox-2 with resultant anti-inflammatory and analgesic effects.

NSAIDs is actually not a single class of drugs. There are many classes of drugs that are capable of inhibiting both Cox-1 and Cox-2:

Ibuprofen
Naproxen
Toradol
Ansaid
Dolobid
Meclomen
Voltaren
Orudis
Lodine

When used in maximal doses, there are little differences in the different NSAIDs. One should therefore be comfortable using one or two of these drugs.

Most dental professionals are comfortable with using ibuprofen, and the optimal dosing of the medication should be 400 mg q4h, 600 mg q6h and 800 mg q8h.
We would advocate using Naproxen Na 500-550 mg BID because of much better patient compliance.

Patient compliance has been examined and the dosing schedule appears to play an important role:

Dosage	Compliance
QD	73%
BID	70%
TID	52%
QID	42%

NSAIDs are contraindicated in patients with renal insufficiency.
Adverse effects of NSAIDs include GI toxicity including GI bleeding and anti-platelet effect due to reversible inhibition of thromboxane A.

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COX-2 SPECIFIC INHIBITORS

Cox-2 inhibitors have efficacy that is identical to that of traditional NSAIDs with fewer gastrointestinal side effects and no anti-platelet effects. It is therefore the preferred analgesics when hemostasis is a concern.

In studies of oral surgical pain, Cox-2 specific inhibitors have a long duration of action and have efficacy similar to narcotics such as oxycodone/acetaminophen.

For post-operative analgesia, celecoxib should be administered with 400 mg first dose and 200 mg BID thereafter for no more than 3 days.

Like all NSAIDs, celecoxib cannot be used in patients with renal insufficiency.

There has been cardiovascular concerns raised for Cox-2 specific inhibitors that have resulted in the withdrawal of rofexocib and valdecoxib from the market. However, it is now known that similar cardiovascular concerns apply to all prescription and OTC preparations of ALL NSAIDs and Cox-2 specific inhibitors.

A recent NEJM article examined the cardiovascular safety of NSAIDs:
24,081 patients with osteoarthritis and rheumatoid artritis randomly assigned to receiving ibuprofen, naproxen and celecoxib, treated over 20 months. The study compared cardiovascular deaths, MI or stroke in these patients. No difference in outcome were noted amongst the three groups.

The new labeling leads to increased awareness of these cardiovascular risks. All NSAIDs should be used very judiciously in patients with pre-existing cardiovascular disease. Most importantly, the new labeling calls for the use of the lowest dose of NSAIDs for the shortest duration possible. In

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dentistry, we cannot avoid using the highest dose for adequate analgesia but we certainly can limit the duration of NSAID use.

SUMMARY

In the management of post-operative pain in patients who have undergone sedation:

1. Narcotics should be avoided
2. Analgesic should be started before onset of pain (pre-emptive analgesia)
3. Acetaminophen has limited analgesic properties and no anti-inflammatory effect but is a good addition to any analgesic program
4. Ibuprofen and Naproxen are reasonable first-line drugs
5. Celecoxib is particularly important in procedures associated with significant bleeding
6. Clear instructions to avoid break-through pain

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Bleeding Disorders

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Formation of a definitive clot is a complicated process involving a number of physiologic events. When a blood vessel is damaged, marked vasoconstriction results. Platelets adhere to the damaged surface and aggregate to from a **temporary hemostatic plug**. Through two separate pathways, the intrinsic and the extrinsic pathways, involving a cascade of 12 circulating plasma proteins called clotting factors, the conversion of fibrinogen to fibrin is completed. Fibrin binds to the aggregated platelets to form a **definitive hemostatic clot**.

Finally, anti-clotting mechanisms in the fibrinolytic pathway are activated to prevent propagation of the clot and to allow for clot dissolution and repair of the damaged vessel. The fibrinolytic pathway is designed to limit the amount of clot that can form during the healing process. Successful hemostasis is therefore dependent upon adequate numbers of platelets, proper functioning of the platelets, adequate levels of clotting factors, and proper functioning of the fibrinolytic pathway.

In brief, platelets are responsible for the formation of a temporary plug and clotting factors are responsible for the formation of a permanent clot.

Bleeding can be the result of inability to form a temporary clot or the inability to form a definitive clot. Inability to form a temporary clot results from **inadequate platelet count (thrombocytopenia)** or **abnormal platelet function (thrombocytopathy)**. Inability to form a definitive clot results from **abnormalities in the clotting factors**.

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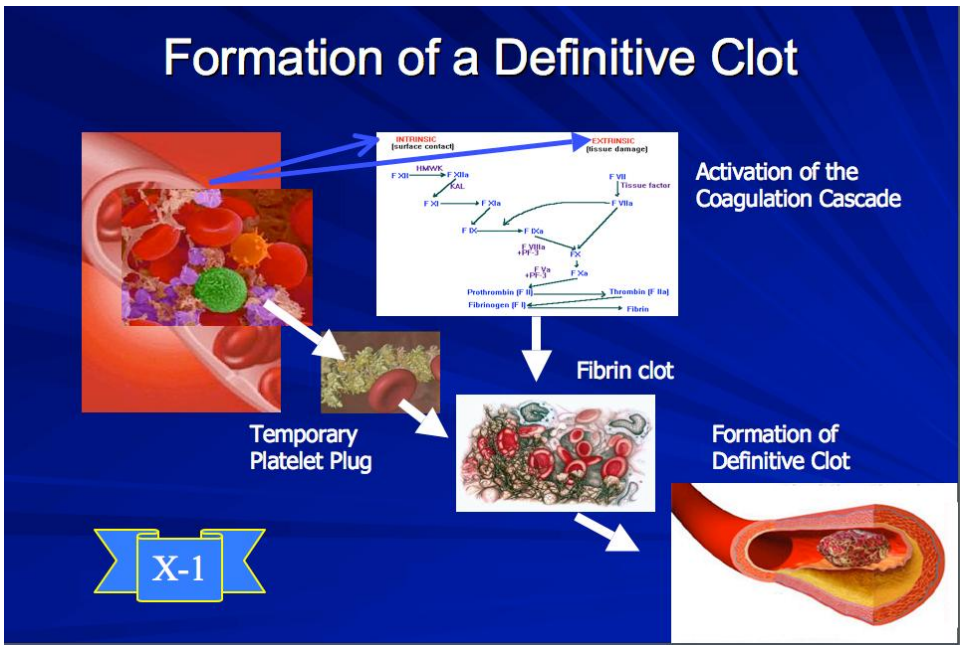

Platelet Disorder

The platelet count provides a quantitative evaluation of platelet function. The normal platelet count should be 100,000 to 400,000 cells/mm3. A platelet count of less than 100,000 cells/mm3 is called thrombocytopenia. Mild thrombocytopenia (platelet count of 50,000 to 100,000 cells/mm3) can result in abnormal bleeding post-operatively. Severe thrombocytopenia (less than 50,000 cells/mm3) can be associated with major post-operative bleeding and platelet transfusion is often necessary prior to any dental intervention.

Bleeding Disorders Secondary to Decreased Platelets

In general, conditions that result in decreased platelet orders are quite uncommon.

Bleeding Disorders Secondary to Abnormal Platelet Function

Abnormal platelet function (thrombocytopathy) indicates that despite normal number of platelets, the platelets do not function normally and form a temporary hemostatic plug ineffectively, with resultant clinical bleeding.

Abnormal platelet function can be assessed by the Ivy Bleeding Time. The bleeding time provides an assessment of the adequacy of platelet count and function. The test measures how long it takes

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a standardized skin incision to stop bleeding by the formation of a temporary plug. Using the standard Ivy method, the normal bleeding time is 5-9 minutes. The bleeding time is prolonged in patients with either thrombocytopenia or abnormal platelet function. This test is seldom invoked since the majority of patients with abnormal platelet function is already known to be on drugs that can cause platelet abnormalities: For example, a patient taking aspirin or Plavix may have bleeding time of 9-12 minutes. This can lead to mild intra-operative and post-operative bleeding.

Thrombocytopathy (abnormal platelet function) can be the result of either inherited or acquired disorders.

Inherited disorders account for less than 1% of patients who present with abnormal platelet function. The most common among these inherited disorder is von Willebrand's disease. In this disease, not only is there an abnormality in the function of platelets, there is also a deficiency in the production of Factor VII. Patients with this disease can therefore bleed both from a platelet abnormality and from Factor VII deficiency. The platelet function abnormalities in von Willebrand's disease can be corrected by administration of DDAVP.

Acquired disorders of platelet function are far more common than inherited disorders.

a. **Drug-induced defects:** by far the most common reason for thrombocytopathy is drug-induced thrombocytopathy. **Aspirin** and aspirin containing compounds are the most common drug to cause platelet function abnormalities. Aspirin inhibits cyclo-oxygenase 1 and 2 enzymes in the platelets in an irreversible fashion, resulting in decrease in formation of prostaglandin precursors. Once a platelet is exposed to aspirin, its function is irreversibly compromised. The effect of aspirin in susceptible patients therefore lasts the lifetime of the platelet population exposed. Patients exposed to even small doses of aspirin can have platelet function abnormalities for 7-10 days. This is not a dose-dependent phenomenon and patients on 81 mg of aspirin can have as much platelet function abnormality as a patient taking a much higher dose.

   Plavix is an agent that irreversibly blocks the $P2Y_{12}$ component of the ADP receptors on the platelet surface which prevents GPIIb/IIa receptor complex, thereby reducing platelet aggregation. Plavix would cause platelet function

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abnormalities for 7-10 days after last exposure. Plavix is used in a number of situations:

Patients with coronary disease
Patients with cerebrovascular disease
Patients with peripheral vascular disease
Patients with coronary stents

Plavix was originally envisioned by be more efficacious with less bleeding complications but this has not been substantiated in clinical trials.

One trial comparing Plavix vs ASA + PPI showed that the patients on Plavix had more bleeding complications.

Combination of **ASA and Plavix** is often employed, particularly during the first 6 months after stent placement. Combination therapy has also been invoked in a variety of other circumstances: in the CURE study, patients with acute coronary syndrome had better outcome on combination therapy but the combination was more likely to cause bleeding complications. In the CHARISMA study, addition of Plavix to ASA in patients with established vascular disease or multiple risk factors showed no benefit over ASA alone. Combination was associated with increased bleeding.

A recent study on single therapy vs combined therapy in patients with strokes showed that there was not additional benefit from combined therapy. There was an increased risk of bleeding complications in patients on combined therapy. This study was terminated early because of these findings.

A recent study in patients following CABG showed better saphenous vein graft patency, particularly in off pump bypass cases. However, patients on combined therapy had significantly more bleeding complications, making risk benefit evaluation difficult.

New Anti-platelet Agent: Prasugel (Effient)

Prasugel (Effient) is a new anti-platelet agent that is similar to Plavix and is usually used in patients after percutaneous coronary intervention or in patients with known Plavix allergy.

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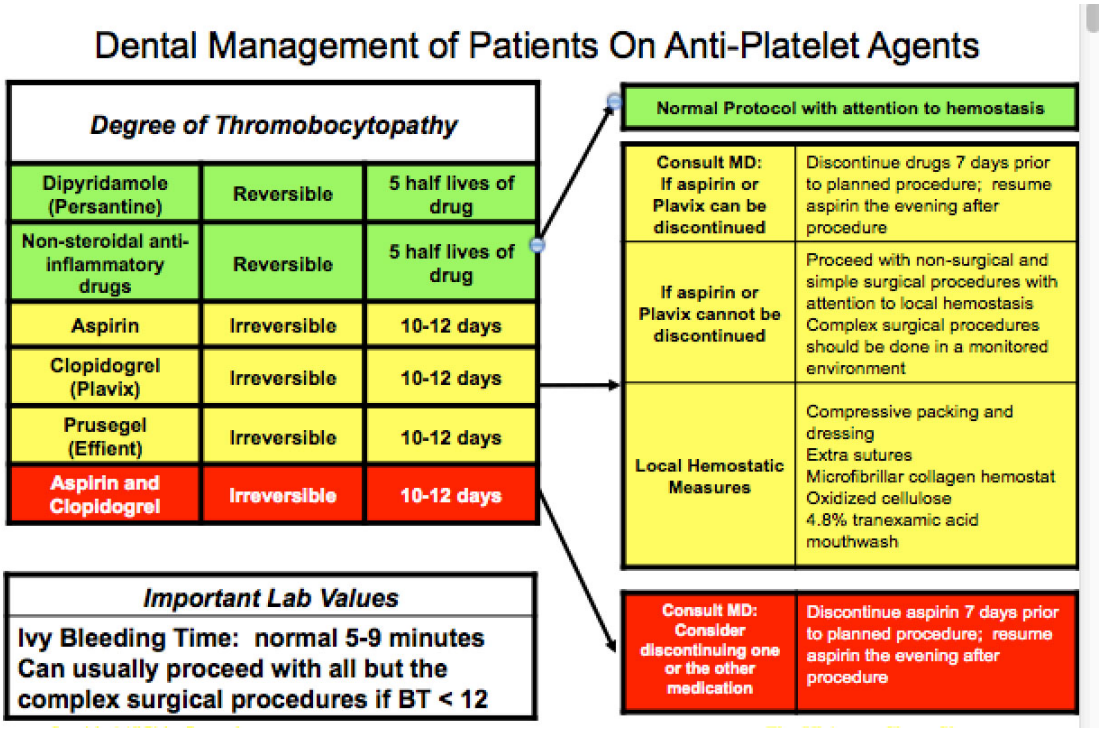
Non-steroidal anti-inflammatory drugs, on the other hand, causes reversible inhibition of cyclo-oxygenase 1 and 2. The effect of the non-steroidal anti-inflammatory drugs on platelet function therefore lasts only the lifetime of the drug and thrombocytopathy is usually reversed 24 to 48 hours after the last exposure to the drugs.

The COX-2 specific inhibitor (Celecoxib, Celebrex) does not affect platelet function.

Concurrent ingestion of alcohol after aspirin or non-steroidal anti-inflammatory drug exposure can markedly enhance thrombocytopathy.



Drugs to avoid in patients on anti-platelet agents include:

- Aspirin
- Non-steroidal anti-inflammatory drugs

Drugs that are safe include:

- All antibiotics
- Acetaminophen
- Celecoxib
- All narcotics
- All commonly used sedatives

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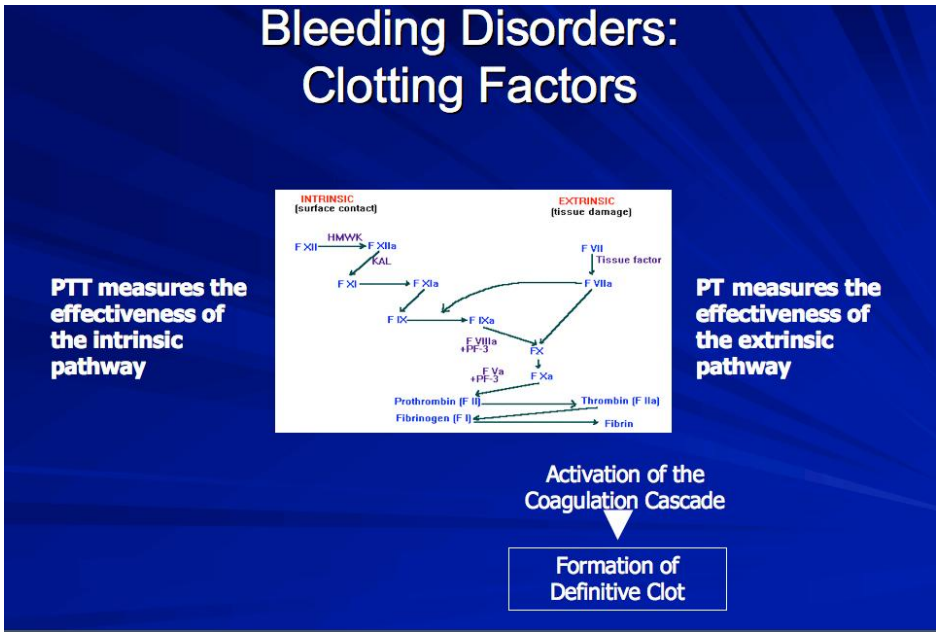
Bleeding Disorders secondary to Clotting Factor Issues

Two tests, the prothrombin time (PT) and the partial thromboplastin time (PTT), provide a reasonable measure of the integrity of the clotting factors.



The prothrombin time (PT) measures the effectiveness of the extrinsic pathway to mediate fibrin clot formation. A normal PT indicates normal levels of Factor VII and those factors common b both to the intrinsic and the extrinsic pathways (V, X, prothrombin, and fibrinogen). PT can be expressed in a number of ways: as PT in seconds or as INR, which is the most accurate way of following PT. INR takes into account of reagent variability and allows for the most accurate monitoring of PT. A normal INR is 1 and an INR above 2 would usually denote coagulopathy.

Patients taking coumadin have a prolonged PT/INR because of the interference of coumadin with the synthesis of vitamin K clotting factors. PT/INR is monitored frequently in these patients (weekly to monthly). A normal therapeutic range of INR would usually be in the 2-3 range. Patients requiring high-intensity anticoagulation may have INR in the 2.5 to 3.5 range.

Coagulation factor deficiency is due either to a congenital disorder or an acquired disorder.

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A number of **congenital clotting factor deficiencies** have been described, but three diseases, Hemophilia A (Factor VIII deficiency), B (Factor IX deficiency) and von Willebrand's disease, account for more than 90% of all inherited coagulation factor deficiencies.

Far more common than congenital factor deficiency is **acquired factor deficiency**.

Deficiency of vitamin K-dependent coagulation factors is the most common acquired disorder of coagulation. Factors II (prothrombin), VII, IX and X are made in the liver and require vitamin K for synthesis of their active forms. Among these factors, Factor VII has the shortest half-life (3 to 5 hours). Therefore, prolongation of PT (which measures Factor VII activity) is the first indicator of deficiency of vitamin K-dependent factors. Common causes of acquired disorders of coagulation include:

Oral anticoagulants: Coumarin anticoagulants competitively inhibit vitamin K action. Patients on anticoagulants will therefore have a prolonged PT.

Liver disease: with significant liver disease, synthesis of a number of coagulation factors is impaired. Vitamin K-dependent factors (II, VII, IX and X), as well as Factors I and V are all synthesized by the liver. Patients with severe liver disease can therefore have significant compromises of their ability to clot.

Dental Management of Patients on Coumadin Therapy

Indications for anticoagulation	
Atrial fibrillation	INR 2-3
Deep venous thrombosis	INR 2-3
Pulmonary Embolism	INR 2-3
Prosthetic valve	INR 2.5-3.5
Hypercoagulable states	INR 2.5-3.5

Important Lab Values
Normal INR 1

For most non-surgical and surgical procedures	**Check INR prior to procedure** **If INR is 2-2.5:** Proceed with attention to local hemostatic measures **If INR is > 2.5:** Consult MD and allow INR to drift down to safe range
Local Hemostatic Measures	Compressive packing and dressing Extra sutures Microfibrillar collagen hemostat Oxidized cellulose 4.8% tranexamic acid mouthwash **Topical thrombin**

Non-surgical and simple surgical procedures	INR 2.5 - 3.5	Proceed with attention to local hemostatic measures, including use of topical thrombin
Complex surgical procedures	INR 2.5 - 3.5	Consult MD Transient use of Fragmin for bridging Hold Fragmin on morning of procedure Resume Fragmin on evening of procedure Monitor PT/INR until patient is back in range

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Medical Management of Patients with Bleeding Disorders

For patients with platelet count much less than 50,000, platelet transfusions would be appropriate before major surgery

Aspirin and Plavix irreversibly inhibits platelet adhesiveness and would therefore inhibit the platelets for its lifetime. In order to prevent thrombocytopathy of these drugs, the drugs should be stopped for a week to 10 days before the procedure

Non-steroidal anti-inflammatory drugs inhibit cyclo-oxygenase reversibly and its thrombocytopathic effect would wear off as the drug effect wears off. Stopping non-steroidal anti-inflammatory drug for a day is usually adequate for the thrombocytopathy to wear off.

For patients with hemophilia, factor replacement should be undertaken prior to surgical procedures, particularly in instances of severe factor deficiency.

For patients with prolonged PT/INR secondary coumadin therapy or liver disease, this can be corrected slowly with vitamin K injections or rapidly with FFP transfusion.

Dental Evaluation of Patients with Bleeding Diathesis

Case #1

54 year old patient comes in seeking oral conscious sedation. He needs removal of the third molar. He gives a history of excessive bleeding with all of his previous dental procedures. He actually had to go to the hospital for transfusion on his last extraction.
Medical history is remarkable for hypertension for which he is on Cardura 4 mg bid.

What additional information do you need?

_____
_____
_____
_____
_____

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In general, CBC with platelet count, bleeding time, PT and PTT should address most of the concerns with hemostasis. In rare instances, specific tests such as platelet aggregation tests or Factor levels may be appropriate. The latter tests have to be coordinated with the patient's physician. These tests should be considered in patients with known risks:

1. A patient giving a strong personal or family history of bleeding should be screened.
2. A patient known to be on medications that can cause thrombocytopenia should be screened.
3. Patients with prior history of illnesses that can interfere with hemostasis should be screened (ITP, TTP, etc)
4. Patients on coumadin have to be screened.

Case #1

CBC normal
Platelet count 220,000
Ivy bleeding time 5 minutes
PT/INR 12/1.0
PTT <25 seconds

Detailed family and personal history are negative in detail.

What should you do?

_____
_____
_____
_____
_____

Case 2

54 year old gentleman with history of coronary artery disease comes in for consideration of oral conscious sedation. He has had two stents placed 2 years ago

PMH significant for hypertension and hyperlipidemia

Medications
ASA 325 qd
Plavix 75 qd

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Crestor 10 qd
Lisinopril 10 qd
Toprol XL 100 qd

He needs to have
Scaling and polishing of maxillary teeth
Caries removal
RTC 6,7
Maxillary teeth prepped and provisionalized

Anticipated to need 4 hours of surgery

What is his ASA status?

What should you do?

_____
_____
_____
_____
_____

1. Most of the problem with thrombocytopathy that the dentist would encounter comes from exposure to drugs that can impair platelet function. The most common drugs include aspirin, Plavix and non-steroidal anti-inflammatory drugs. Stopping these drugs at appropriate intervals prior to dental intervention should reverse the thrombocytopathy.
2. In instances where the patient has a known underlying disease causing thrombocytopathy, careful attention to hemostasis should be employed. Specific recommendations depend upon the underlying disease and the degree of the platelet function abnormalities.
3. Combined ASA and Plavix can cause excessive bleeding
4. It is important not to stop Plavix abruptly in a patient with a coronary stent since there are a number of reports of acute stent thrombosis
5. In patients with both ASA and Plavix, the physician should be consulted: usually one of the agents (usually ASA) cab be stopped
6. Local measures that will help hemostasis should be employed.

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There are a number of local measures that can help hemostasis:

1. Compressive packing and dressing
2. Extra sutures
3. Microfibrillar collagen hemostat
4. Oxidized cellulose
5. 4.8% tranexamic acid mouthwash
6. Platelet concentrates

Case 3

64 year old gentleman in for consideration of oral conscious sedation.

PMH significant for hypertension, diabetes mellitus and atrial fibrillation.

Medications:

5 mg of coumadin qd
Enalopril 20 qd
Metformin 500 qd
Glyburide 10 qd

Anticipated time of surgery 4 hours

What is his ASA status?

What should you do?

_____
_____
_____
_____
_____

PT/INR is 2.2
BP 120/68
HbA1c 6.6%

His physician does not care one way or the other with respect to his holding coumadin.

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What should you do?

_____
_____
_____

Case 4

54 year old patient with cirrhosis secondary to chronic alcohol excess comes in for consideration of oral conscious sedation

PMH significant for one bout of variceal bleeding necessitating transfusion of 4 units of blood. He has also had one admission for hepatic encephalopathy.

He needs 4 hours of surgery.

What is his ASA status?

What additional information do you need?

_____
_____

What sedation protocol would you consider for Case 4?

_____
_____
_____
_____

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ANTIBIOTIC THERAPY – THE TREATMENT OF ACUTE ODONTOGENIC INFECTIONS

In the seventies, the range of antibiotics available to treat acute odontogenic infections was limited. At that time most dentists utilized Pen VK, Erythromycin Stearate or E-Mycin and Tetracycline Hydrochloride. Penicillin was the first drug of choice because it was bactericidal. It had limited drug/drug interactions and was the cornerstone of therapy. In the presence of Penicillin allergy, the alternatives were Erythromycin Stearate or because of significant G.I. side effects, E-Mycin (enteric coated Erythromycin) which had less risk of side effects. A third choice was Tetracycline Hydrochloride; which suffers from the limiting factor that it could only be given an hour before or two hours after a meal. On a full stomach, it failed to absorb predictably. All of these drugs were given 250 mg. four times a day (qid). Today there are new dose schedules for some of the traditional antibiotics. There are new drugs in traditional categories and there are all new categories of antibiotics.

Also there has been a changing microbiota responsible for odontogenic infections. Classically, oral infections were “mixed” infections. That is, there were multiple species of bacteria found, most times including several streptococcus veridans subspecies unique to the oral cavity. While strep veridan organisms are still a significant part of odontogenic infections, today we are dealing with many more staphylococcus species. These species are many times resistant to conventional antibiotics. In addition, there is a broader range of resistant strains of anaerobes, both gram-negative rods and gram-positive cocci in the mix (Walker, C. Perio 2000, 1996).

One might think that in treating odontogenic infection, the key question is the potential for microbial resistance. Indeed, this is not the most important question. Although resistance must be considered, the most common cause for failure of therapy is failure to properly debride the wound. A dentist with an apparent failure at primary treatment for an odontogenic infection must first consider a failure to debride the wound thoroughly. This is true for both endodontic and periodontic infections. Prior to the forties, in fact, debridement was the only treatment the dentist could render. The frequency of advancing infections (spreading infections) was far greater than now. Today, we have the option of adding antibiotic therapy as an adjunct to our treatment. The treatment goals for the use of adjunctive antibiotics is to first and foremost improve debridement results and secondly, to prevent spreading infection.

If antibiotics are part of the treatment, we now introduce two issues as potential causes of treatment failure. Beyond failure to properly debride the wound, compliance problems and bacterial resistance problems must often be considered. We would like to develop an algorithm for antibiotic choice. Indeed, there are two separate algorithms that must be considered. The dentist

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in evaluating an infection must first decide on the level of infection. *Is it a localized or is it a spreading infection*. A localized infection is a classic endodontic fistula localized to the alveolus or localized swelling around the periodontium supporting a single tooth.

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Our first choice antibiotic in the treatment of the localized endodontic or periodontic infection should be a Penicillin. One might wonders about the persistence of Penicillin as a first choice given that it has had the longest time since its discovery in the early forties to develop increased resistance. Indeed, there has been increasing resistance to Penicillins. This is counterbalanced by the concept of *high dose safety*. The Penicillins are quite safe for the patient at higher doses. Dentists will recall that at one time a prophylactic recommended dose for Penicillin was 3000 mg. Because we can use higher dose Penicillins safely, the drug has remained effective in spite of the increased resistance developed over the recent decades.

Penicillins also have the persistent advantage of minimal drug/drug interactions. In a milieu of half a dozen or more prescribed drugs as part of some patient's daily life, the absence of drug/drug interactions is a significant advantage when considering an antibiotic choice. Furthermore, the Penicillins remain bactericidal with its preferred ability to directly eradicate the bacteria.

The three most commonly prescribed Penicillins in the United States are Pen VK 500 mg. qid, Amoxicillin 500 mg. tid and Amoxicillin 875 mg. bid. When considering the preferred choice, we have to consider the second most common cause for failure to successfully treat infection; *Patient Compliance Failures*. The risk of a patient not taking the antibiotic properly and, therefore, not maintaining a therapeutic level of the drug to successfully combat the infective organisms increases with the need for multiple daily doses. Greenberg in Clin. Ther. 1984 demonstrated a statistically significant difference in patient compliance based on dosage formulas. The article was a systematic review of multiple studies of drug compliance. Once a day *(qd)* and twice a day *(bid)* dosage forms of a drug yielded *70% plus* compliance. Greenberg's review of the literature showed, however, at a statistically significant level that once a drug required three time a day *(tid)* or four time a day *(qid)* dosage forms, compliance dropped under *50%.* This represents a dramatic potential failure for successful treatment. Our medical colleagues have exactly the same problem with regard to patient compliance and the treatment of medical problem related infection. (Schwartz, Perschy, et. al. "BID versus TID Clarithromycin…" Helicobacter, 1999 and Kardis, P. "Patient Compliance with Antibiotic Treatment for Respiratory Infections," Journal of Antimicrobial Chemotherapeutics, 2002 are among many other references. In general, therefore, the dentist should clearly prefer antibiotics that can be administered qd or bid. Because of the above considerations, our recommendation is *for a localized infection, the first drug of choice would be Amoxicillin 875 mg. given one bid for seven days.* It is notable that there is a newer formulation of Amoxicillin which is a once a day (qd) dosage. Unfortunately, this antibiotic is currently expensive and there is no statistically significant difference between QD or BID compliance. The statistical significance for compliance is at the BID / TID break.

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If we *consider a first choice for treating spreading infection*, we must consider the third risk for treatment failure; specifically, resistant bacteria. In a localized infection that does not improve to therapy, or a Spreading Infection the dentist can consider a broader spectrum antibiotic covering a maximum group of microbiota and specifically covering for staphylococcus and a broader scope of resistant anaerobes. In the setting of a spreading infection, there is the risk of severe complications if the spreading infection worsens. Risks of sepsis might require hospitalization. The recommendation, therefore, in the setting of a spreading infection, the doctor would want to choose an antibiotic that covers the maximum microbiota spectrum that could be the potential etiology for the odontogenic disease. For purposes of evaluation, we would define a patient as having a spreading infection if there was an odontogenic infection with any facial swelling, (this could be as little as an obliteration of the nasolabial fold at the corner of the nose), any cellulitis, any facial fistulization, involvement of the sinus, involvement of the retropharyngeal space or the findings of constitutional symptoms including fever and chills.

For this purpose we strongly recommend the drug Augmentin. Augmentin is an Amoxicillin preparation with the added drug Clavulanic acid. Clavulanic acid can be described to the patient as the supercharger of Amoxicillin. Augmentin has the Amoxicillin spectrum plus the extended spectrum including coverage for staphylococcus species and many more resistant anaerobes.

Two primary formulations of Augmentin exist; 500 mg. given tid and 875 mg. given bid. Because of the issue of patient compliance and the statistically better chance that the patient will take a drug given twice a day, we would recommend the 875 mg. form of Augmentin. There is also a second reason to recommend Augmentin at the 875 mg. dose to insure better compliance. Augmentin compared to Amoxicillin has an increased chance of a diarrhea side effect. Much of that is attributed to the presence of the second drug, Clavulanic acid, in the pill formulation. The manufacturer recommends the Augmentin 500 mg. to be prescribed tid. The Augmentin 875 mg formulation is given bid. Both pills carry an identical amount of Clavulanic acid (125 mg.). Therefore, by prescribing the bid dosage, you are only getting two-thirds of the Clavulanic acid dose found in the 500 mg. formulation dosage format.

In the treatment of a spreading infection, we not only want an antibiotic with an extended spectrum but we would generally choose a longer duration of therapy. *Therefore, our first drug of choice in the treatment of the spreading infection would be Augmentin 875 mg. given one bid for a ten day period.*

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PENICILLIN ALLERGY

What should the dentist consider for antibiotic choices in the setting of a Penicillin allergy. *Let's first consider localized infection*. The older macrolides, Erythromycin Stearate, enteric-coated Erythromycin or Erythromycin Ethylsuccinate are poor choices. There is significant G.I. toxicity and a mandatory four times a day dosage which makes for compliance problems. There is a huge resistance problem. First generation Erythromycins are most likely to have resistant species in an odontogenic infection. In today's milieu of prescription drug use, there are also many drug/drug interactions that contraindicate the use of Erythromycin. It is best for clinicians to not consider these medications as part of their armamentarium.

This is not the case for the newer second generation Erythromycins. The most commonly used second generation Erythromycins are Zithromax (Azithromycin) and Biaxin (Clarithromycin). Both of these drugs have better compliance considerations because of better G.I. tolerance, fewer G.I. complications and once a day, or twice a day, dosage formulations. In addition, the resistance issue is also better for both these drugs given that they both cover similarly to the Amoxicillin spectrum in addition to covering for staphylococcus and extended spectrum anaerobes. (not dissimilar to the Augmentin spectrum of coverage)

How should the dentist choose between these two drugs. Biaxin, unfortunately, shares with the older Erythromycins multiple drug/drug interactions. Both the older Erythromycins and Biaxin are processed by the cytochrome P450 system. This system, as we have seen earlier for many sedation drugs, metabolizes multiple drugs. However, it is a relatively low capacity system. If two such drugs are prescribed simultaneously; there is preferential metabolism of one while the other drug accumulates. Often the Erythromycin is processed normally while the secondary drug accumulates. This was such a significant problem with the drugs Seldane and Hismanal used for allergic rhinitis and Propulsid used for heartburn that life threatening arrhythmias occurred. These drugs because of this potential problem were removed from the market place. The first generation Erythromycins and Biaxin also interact today with the commonly used calcium channel blockers Cardizem, Calan and Isoptin. The Tennessee Medicaid Cohort published a study in the New England Journal of Medicine in 2008 that showed for a patient receiving one of these calcium channel blockers and taking first generation Erythromycin there was a 5.35 fold increased risk ratio for Sudden Death over the general population. Other drugs that interact with the Erythromycins include Digoxin and Norpace used to treat arrhythmias; unfortunately, *creating* arrhythmias in the presence of Erythromycin. Theophylline, the asthma drug caused arrhythmias and there is a significant risk for increases in the INR for Coumadin. Other potential interactions include myopathy with Statins and toxicity for multiple drugs including Tegretol, Busbar, Cyclosporine, Versed, Colchicine, Flomax and Diflucan.

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It is notable, however, that Zithromax (Azithromycin) is not metabolized by the Cytochrome P450 system and does not have multiple drug/drug interactions. Therefore, given its good compliance history, its good bacterial spectrum and its ease of dose, Zithromax is an excellent choice drug to treat odontogenic infection.

Our recommendation is that for the localized infection in the setting of Penicillin allergy that Zithromax be used. There are two common formulations for Azithromycin. Our first choice recommendation is Zithromax in a 500 mg. dose, to be given as a tripak (three pills with the sig. being one daily for three days) as directed by the package insert. An alternative and equally effective form of Zithromax is dispensing the 250 mg dosage form as a Z pack (six pills). The sig. for this drug is two pills the first day, then one daily for four days, as directed on the package insert. Both the 500 mg. dose tri-pak given over three days and the 250 mg. dose Z pack over five days have an active duration of action extending for ten full days. There are multiple references available for Azithromycin as an excellent choice in the treatment of odontogenic infection ranging from Cotter's article in Journal of Maxillofacial Surgery, 2003 entitled "Azithromycin for Odontogenic Infection" to Moore, JADA, 1990 "Dental Indications for Newer Macrolides."

Al-Belasy, Journal of Oral Maxillofacial Surgery, 2003 entitled "Azithromycin Efficacy…for Acute Infraorbital Space Infection." and Adriaenssen, J., Int. Med. Res. 1998 "Azithromycin Efficacy…Periapical Abscess Equivalent to Augmentin in Efficacy." are other excellent references.

Given Azithromycin's spectrum similar to Amoxicillin plus coverage for staphylococcus species and extended spectrum anaerobes, one would *consider Zithromax as a possibility for the treatment of spreading infection for the patient with Penicillin allergy. Clinically, however, we judge this drug to be only our second alternative to Penicillin.* Kuriyama in O.Microbio. Immunul. 2007 noted that the antibiotics best for treatment of oral anaerobic infection were Augmentin and Clindamycin as a broad-spectrum drugs. *Therefore, we opt to recommend Clindamycin 300 mg., dispense thirty or forty pills, one tid or one qid, as the prescription*.

Clindamycin has a similar advantage to Penicillin in that there are really no significant drug/drug interactions and in the milieu of patients with multiple prescription medications, it is easier to prescribe.

One downside to the Clindamycin story is that it is a tid or qid drug and, therefore, patient compliance is potentially more difficult. The most significant downside issue is *the association of clindamycin, when given as multiple doses with Pseudomembranous Colitis.*

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Clindamycin as a single dose prophylaxis drug, for example the current American Heart Association's SBE recommendations for prophylaxis is considered absolutely safe. There is only one case report in the English literature by Bombassaro and Wetmore et. al. in the Journal of the Canadian Dental Association, January of 2001. This case report involved the occurrence of pseudomembraneous colitis after single dose prophylaxis. The outcome, unfortunately, in the case was the death of the patient. The concern, however, for the dentist is generally a multiple dose safety issue.

The chance of pseudomembraneous colitis occurring as a result of the Clindamycin suppressing the G.I. microflora and allowing the overgrowth of clostridium difficile is approximately a one percent risk. The Physician's Desk Reference publishes a *black warning box about Clindamycin.* Clindamycin is the only antibiotic commonly used by dentists that has the black warning box. Prior to 2000 that black warning box carried in part the following citation; "Clindamycin use is associated with severe colitis which may end fatally…it should be reserved for severe infections where less toxic drugs are inappropriate." From 2000 to the present the PDR black box now reads "pseudomembraneous colitis reported may range in severity from mild to life threatening."

The dentist must closely advise the patient to discontinue the drug immediately if diarrhea is a problem. The dentist must be available through an easily accessed answering service and must stay in touch with the patient. If the diarrhea subsides in twenty four to thirty six hours, it is suggested that there was an intolerance reaction to Clindamycin and not developing pseudomembraneous colitis. If after twenty four to thirty six hours, the patient is still having substantial diarrhea, then pseudomembraneous colitis must be ruled out. This involves contacting the patient's physician and testing a stool sample for the presence and levels of clostridia difficile. The reason for this strict warning is that an article published entitled "The Changing Spectrum of Clostridia Difficile Associated Disease Implications for Dentistry" in JADA, Volume 131, No. 1, 2008, discussed the more aggressive and toxic form of clostridia difficile developing. The article also noted that the longer the treatment for the disease is delayed, the greater likelihood there will be megacolon and the risk for death.

Treatment for pseudomembraneous colitis involves the use of either Vancomycin (Vancocin) or Metronidazole (Flagyl) or the newer use drug Fidaxomycin (Dificid).

Because of this risk for pseudomembraneous colitis, the use of Clindamycin in dentistry should be limited to the treatment of a spreading infection in the presence of Penicillin allergy. Augmentin is clearly the first drug of choice for spreading infection given the absence of a black warning box. Physicians are generally much more diligent in avoiding recommending drugs that carry a black warning box. Clindamycin prescribed in multiple doses is primarily a dental drug. Physicians very rarely utilize this medication for extended treatment and simply limit Clindamycin use for premedication.

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There is also an area of relative contraindication for the use of Clindamycin.
In addition to being diligent in having a patient discontinue if diarrhea occurs and following up for the immediate cessation of that diarrhea, the thinking dentist should not utilize Clindamycin in any circumstance where there is a history of diarrhea to medications or where there is a history of diarrhea attached to a medical diagnosis. This would include patients with inflammatory bowel disease, irritable bowel syndrome and diverticulitis.

Metronidazole – The Piggy Back Drug

Metronidazole is a superb gram-negative drug but only gram negatives. It has no real gram-positive anti-microbial activity and, therefore, is simply an add on drug to another used to cover the gram-positive spectrum. It is an important addition to the dental armamentarium, however, because in Kuriyama's article in O. Microbio. Immunul. 2007, he noted that a best antibiotic against anaerobes in addition to Augmentin and Clindamycin is the third drug Metronidazole. Metronidazole can be prescribed as a 500 mg. tablet, one tid or one bid, along with a secondary drug. Combinations might include an Amoxicillin regimen or a Zithromax regimen and Metronidazole. Baumgartner writing in the endodontic literature suggested that the first drug of choice for a localized infection in endodontic abscess would be Amoxicillin.

In the presence of a partial or total failure to respond, repeat debridement is the first approach in addition to the piggyback of Metronidazole to Amoxicillin. In the setting of a local odontogenic infection with Penicillin allergy, it would not be unreasonable in today's milieu to start with Zithromax and in the absence of response add Metronidazole two days later.

The side effect concern with Metronidazole is the absolute level of alcohol intolerance. A patient cannot drink alcohol while on Metronidazole and for the one to three day period after the cessation of the drug. This can be a problem not just for the alcoholic but the patient who routinely enjoys alcohol and is socially appropriate. Inquiries must be made before prescription. Metallic taste is a common side effect and occasional G.I. distress. Contraindications to Metronidazole are seizure disorders, Coumadin therapy, Cyclosporine therapy and pregnancy. Dr. Fang and I summarize the above discussion in The Ultimate Cheat Sheets textbook on the page entitled "Antibiotic Therapy in the Treatment of Odontogenic Infection."

In summary, for a localized infection where antibiotic use is elected, our first drug of choice is Amoxicillin 875 mg. dispense fourteen with a Sig. of one bid. Our first choice in the setting of Penicillin allergy is Azithromycin 500 mg. dispense a tri-pak or Azithromycin 250 mg. dispense a Z-pack as directed. Clindamycin is only our third alternative in the treatment of localized infection because of the PDR's black warning box. For spreading infection, our first drug of choice is

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Augmentin 875 mg. dispense twenty. The Sig. is one bid. In the setting of a spreading infection with Penicillin allergy, Clindamycin by virtue of its superb anaerobic microbiota spectrum similar to Augmentin becomes our first choice alternative. It is Clindamycin 300 mg. dispense a sufficient number of pills for a ten day course (thirty to forty) with the Sig, being one bid or one qid. The above noted Azithromycin regimens would be our second alternative to Augmentin for spreading infection if Clindamycin was contraindicated. As a side bar, Metronidazole, as noted earlier, can be added 500 mg. twice a day or three times a day to an appropriate course of Amoxicillin and Azithromycin in the treatment of a spreading infection.

OTHER ANTIBIOTICS

There are other antibiotic alternatives that while not a first line drug have significant use in the dental setting. Newer Tetracyclines like Doxycyline or Minocycline given twice a day have an excellent literature supporting its use including Kuriyama, OOO 2000, noting that Minocycline among several other antibiotics are effective for “most major pathogens in oral facial odontogenic infections” and Jacinto in Int. Endo. J. 2006 referencing P. gingivalis (a gram negative anaerobic rod) is associated with purulent endodontic exudates and pain to palpation and is sensitive to multiple drugs including Tetracycline.

The second generation Ceftin (Cefuroxine) given as a 250 to 500 mg bid dose offers a fourth broader spectrum antibiotic for possible use in spreading infection beyond the preferred Augmentin, Clindamycin and Zithromax choices. Ceftin unfortunately is an expensive alternative with no generic form. The similarly expensive Fluoroquinilones, Ciprofloxacin (Cipro) 500 mg. bid or Levofloxacin (Levaquin) 500 mg. once a day with their excellent ability to penetrate into the sinus space as an alternative to Augmentin, are drugs beyond the scope of this lecture but ones that should be considered by the practicing dentist.

Recognizing the antibiotics that are recommended for utilization or drugs to avoid for Coumadin and Pradaxa therapy, the nursing mother or the pregnant patient are also considered in specific page summaries in *The Ultimate Cheat Sheets* and should act as a reference for follow up considerations of antibiotic therapy.

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CARDIAC RHYTHM REVIEW

Leslie S.T. Fang, MD PhD
Massachusetts General Hospital
Harvard Medical School

I. IMPORTANCE OF INTRA-OPERATIVE MONITORING

In all instances when IV sedation is employed medically (endoscopy, cataract surgery, arthroscopy or colonoscopy), intra-operative monitoring is mandatory. Just as a point of reference:

Cataract operation	30 minutes
Endoscopy	20 minutes
Arthroscopy	Less than an hour
Colonoscopy	30 minutes

How long is your sedation session?

Intra-operative cardiac monitoring provides important and rapid indication of any changes in the patient's clinical status, often long before changes in oxygenation (SaO_2). It is critical for the safety of the patient to be doing intra-operative cardiac monitoring. It also mandates that you have some comfort interpreting cardiac rhythm.

In this session, we will go through the basics of intra-operative monitoring and rhythm interpretation, touch base on some common rhythms and key in on rhythms we should not misinterpret.

II. INTRA-OPERATIVE MONITORING

Intra-operative monitoring is usually done with three precordial leads (and, in rare instances, a single lead). This would provide real time information. Usually, heart rate is automatically calculated and displayed and the dentist can set alarm for upper and lower limits of heart rates in every patient.

With intra-operative monitoring, important information that can be derived include:

- Rate
- Rhythm
- Narrow vs Wide Complexes
- Heart Blocks

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- Ischemia
- Infarction

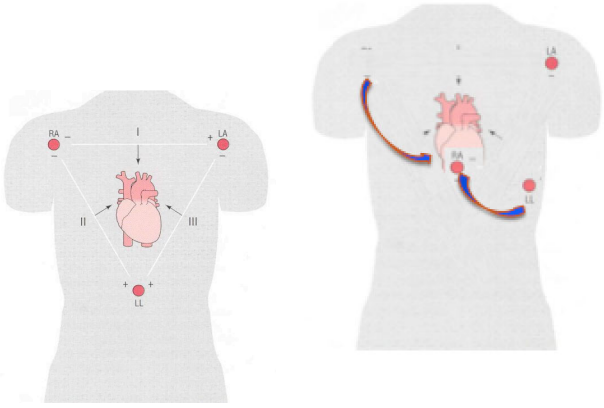
However, it is less likely to give accurate information about cardiac ischemia or ongoing infarction than the gold-standard 12-lead electrocardiogram.

If your cardiac monitoring is equipped with **3 leads**, placement of the 3 leads in a **modified V_5 lead system**:

- RA electrode on the middle of the manubrium
- LL electrode on the V_5 position
- LA electrode on the left arm
- Monitor lead II

The advantages of using the modified V_5 system for monitoring include:

- Some information about ischemia and infarction
- 89% of the ST segment changes can be detected



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In rare instances, only a single lead may be available for intra-operative monitoring. The good news is that we can still derive enough information to safely monitor most patients.

III. NORMAL EKG COMPLEX

A. COMPONENTS OF THE EKG COMPLEX

The normal EKG complex should have the following components:

- P wave
- PR interval
- QRS complex
- T wave
- ST segment
- QT interval

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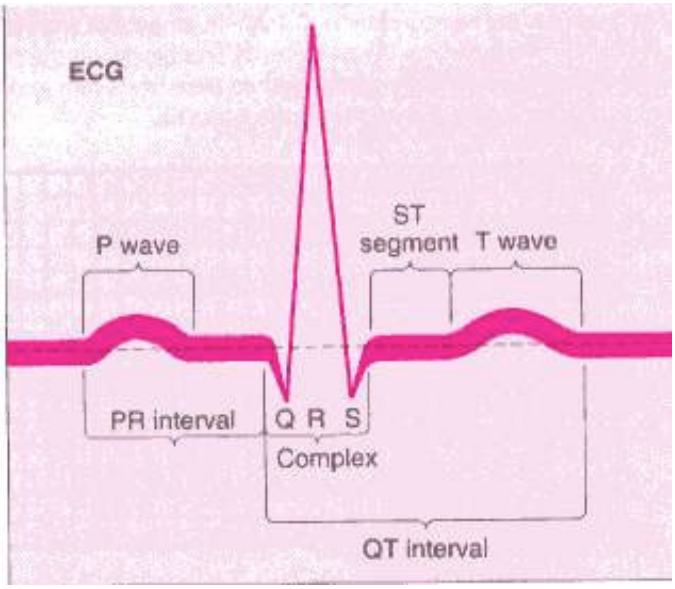

P wave: signifies depolarization of the sinoatrial node (SA node) in the atrium and the initiation of atrial contraction

PR interval: measures the rate of conduction from the SA node to the atrio-ventricular node (AV node); normal PR interval should be less than 0.12 seconds

QRS complex: measures the ventricular depolarization and the complex should be less than 0.08 seconds

T wave: measures repolarization

ST segment: measures the inteval between the end of ventricular depolarization and the beginning of repolarization; this segment can give important information about ischemia and infarction. ST segment depression is indicative of cardiac ischemia, whereas ST segment elevation is indicative of myocardial infarction

QT interval: measures the duration from the beginning of ventricular depolarization to end of repolarization and should be less than 0.40 seconds

B. MEASURING INTERVALS

The electrocardiographic display grid is designed to give rapid determination of time elapsed:

- Each small box on the EKG strip or the display grid represents 0.04 seconds

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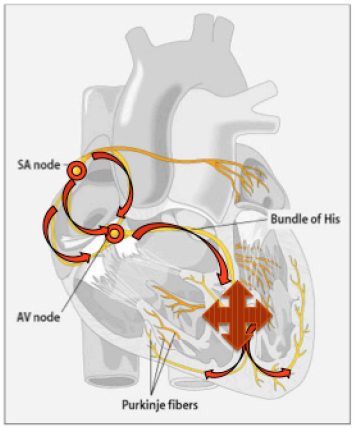
- PR interval should be less than 0.12 seconds: less than 3 small boxes on the grid
- QRS complex should be less than 0.08 seconds: less than 2 small boxes on the grid
- QT interval should be less than 0.4 seconds: less than 10 small boxes on the grid

Once you are familiar with looking at the monitor screen, some of these aberrancy would be obvious.

Electrical System: complete sequence



SA node depolarization causes atrial contraction. The signal from the SA node then travels to the AV node and down the Bundle of His and the Purkinje fibers, with resultant ventricular depolarization and contraction

The ventricle then repolarizes

IV. DETERMINANTS OF CARDIAC RHYTHM

Regardless of the mode of determination of cardiac rhythm (1 lead, 3 leads, 12 leads), the generated tracing should be able to give us information about 3 critical determinants of the cardiac rhythm:

- Rhythm
- Rate
- Narrow vs Wide Complex

Based on the accurate interpretation of these determinants, we should be able to generate important information on the patient being monitored

A. WHAT IS THE CARDIAC RHYTHM?

We will not be able to go into all of the intricacies of rhythm interpretation but would like to make sure that you are comfortable with the most common rhythms and the implications thereof. We will also highlight rhythms that are likely to deteriorate and cause clinical problems intra-operatively.

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B. DETERMINING HEART RATE

Heart rate can be rapidly determined based on the grid either on the display or on the electrocardiogram. Most monitors actually calculate and display the heart rate.

B. QRS COMPLEXES: NARROW VS WIDE COMPLEX

Narrow Complex: the QRS complex is less than 0.08 seconds; this denotes normal conduction

Wide Complex: the QRS complex is greater than 0.08 seconds; this denotes abnormal conduction; wide complexes often denotes electrical anomalies that is ventricular in origin

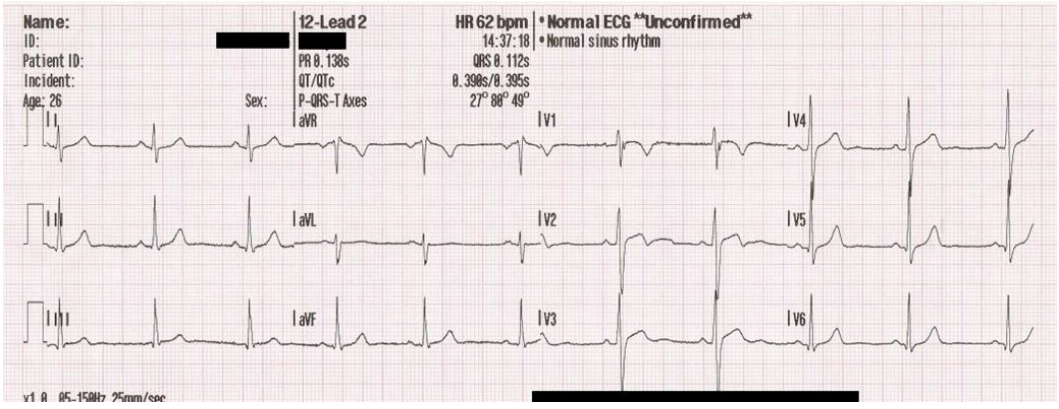
V. INTERPRETATION OF CARDIAC RHYTHMS

This is the fastest way to identify a rhythm not immediately obvious to you is to go through the algorithm and determine:

- Is the rhythm regular or irregular?
- Is the rate normal, slow or fast?
- Is the QRS complex narrow or wide? (Most important)

A. REGULAR VS IRREGULAR

1. In most instances, a regular rhythm at a normal rate with normal QRS complexes is **normal sinus rhythm** until proven otherwise:



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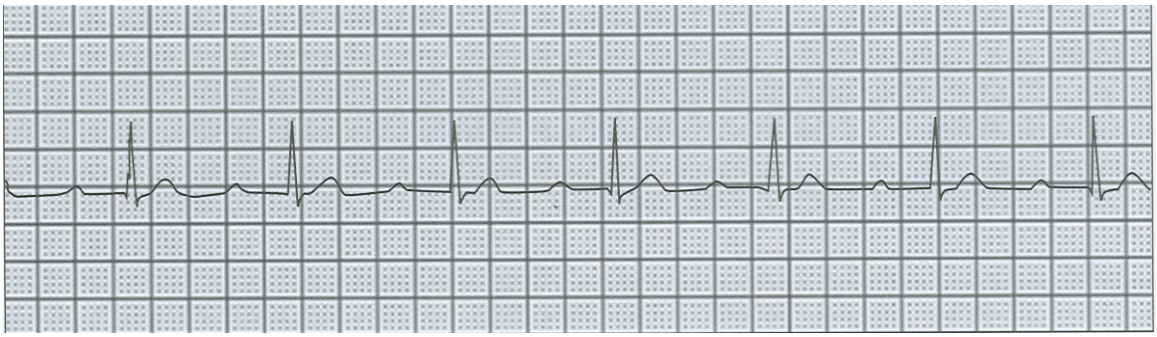
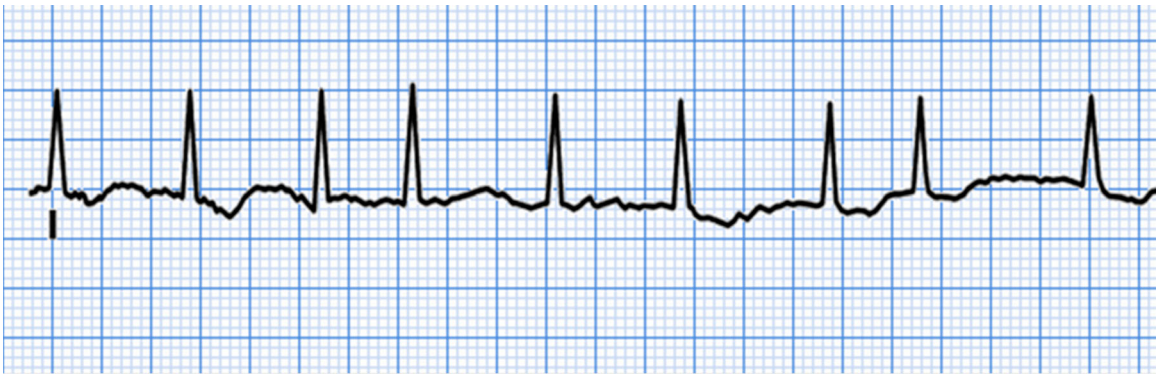
2. However, in rare instances, patients with **first degree heart block** can have similar appearance BUT:

PR interval is greater than 0.12 seconds



3. The most common irregular rhythm is **atrial fibrillation.** In atrial fibrillation, diseases affecting the sinoatrial node result in abnormal electrical pathways arising from different parts of the atria, resulting in an irregularly irregular rhythm. Because the electrical activity does go down the AV node and the normal conduction system, the QRS complexes are not affected.



Atrial fibrillation is an important rhythm to recognize. Patients who have new onset atrial fibrillation during a procedure are indicating cardiac ischemia and distress. Patients who are chronically in atrial fibrillation are usually on anticoagulation, which obviously has dental implications.

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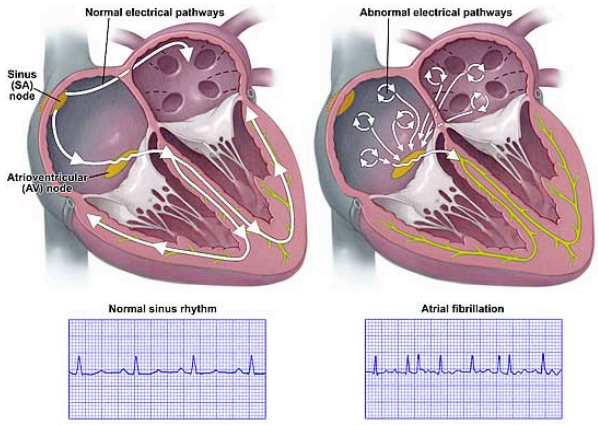

B. HEART RATE

Normal resting heart rate is between 60 to 80 beats per minute. Young athletes can get their resting heart rate down to the 40's and anxiety can drive the resting heart rate to rates in excess of 100 beats per minute. "Normal heart rate" therefore needs to be interpreted in context of the patient being monitored.

Heart rate greater than 100 bpm is termed **tachyarrhythmia or tachycardia**. In these patients, the QRS complexes would appear to be closer together than 3 large boxes on the grid.

Sinus tachycardia at 120 bpm:

Symptoms of tachyarrhythmia usually appear at heart rates greater than 150, particularly in older patients. Tachycardia can result in cardiac ischemia (increased demand) and lead to undesirable consequences.

Heart rate less than 60 bpm is termed **bradyarrhythmia or bradycardia.**

Sinus bradycardia at 50 bpm:

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Symptoms of bradycardia usually occurs at heart rate of less than 50 bpm. Patients with bradycardia usually complain of dizziness or light-headedness (cerebral hypoperfusion). In severe instances, it can result in cardiac ischemia as well (decreased supply).

C. NARROW VS WIDE COMPLEXES

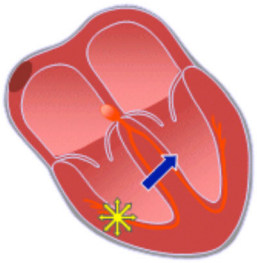
Narrow Complex: the QRS complex is less than 0.08 seconds; this denotes normal conduction

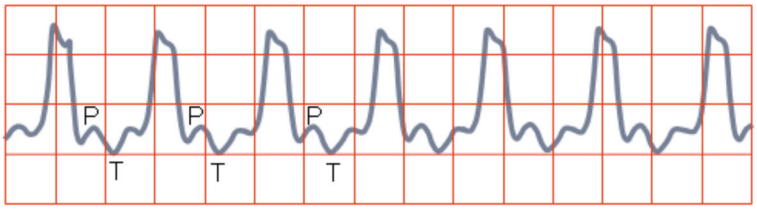
Wide Complex: the QRS complex is greater than 0.08 seconds; this denotes abnormal conduction; wide complexes often denotes electrical anomalies that is ventricular in origin

In general, wide complex rhythms should be carefully scrutinized since they often are precursors to trouble.

VENTRICULAR TACHYCARDIA

Impulses originate at ventricular pacemaker





Wide ventricular complexes. Rate > 120/min

Atrial flutter is now easily treated by use of a catheter to ablate the re-entry pathway.

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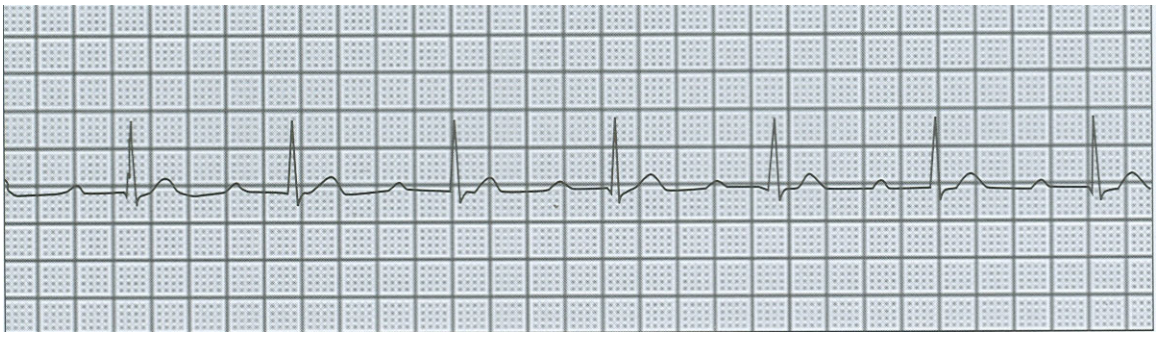
VI. SELECTED RHYTHMS

What is this rhythm?



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What is this rhythm?

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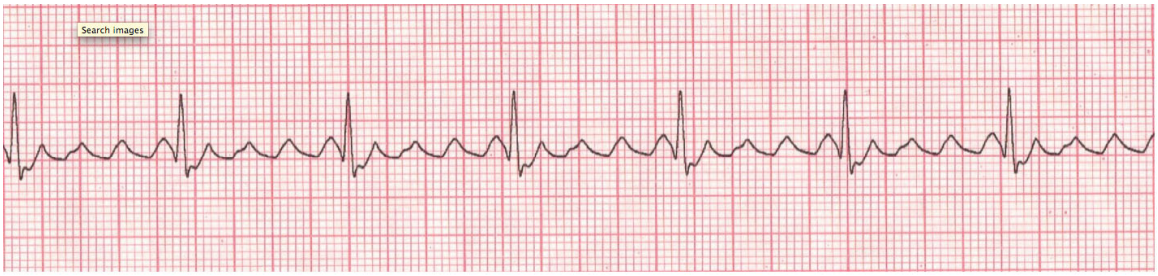
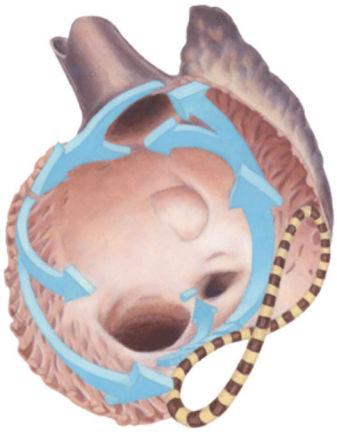
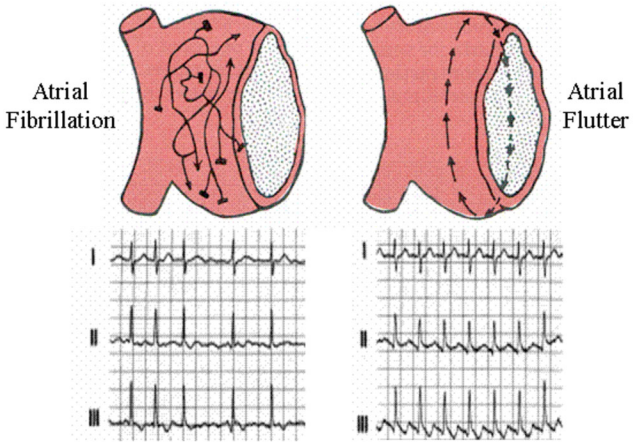
What is this rhythm?







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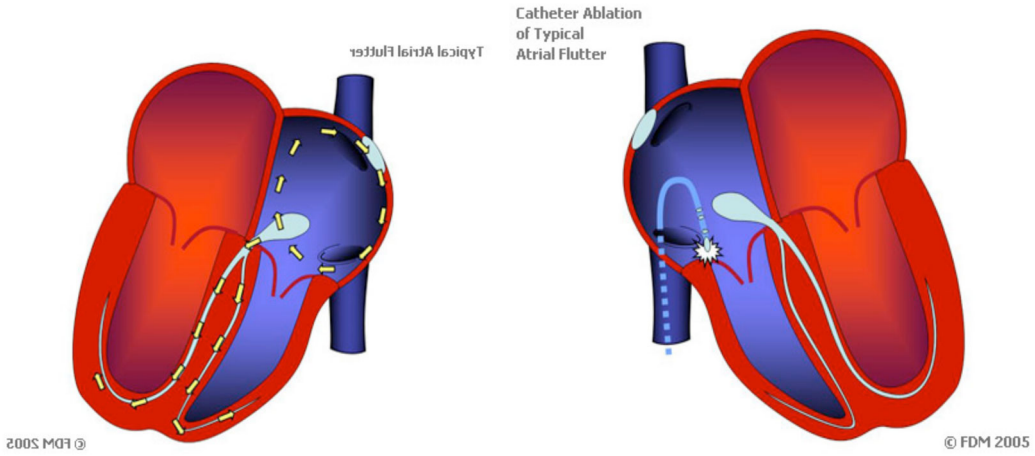
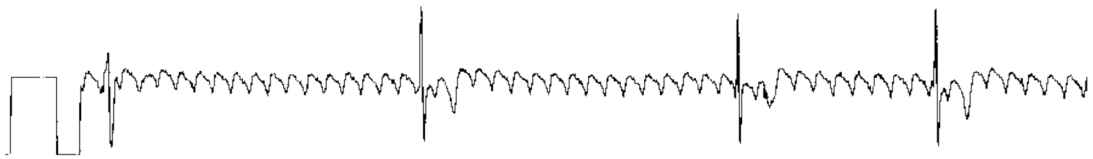
Treatment of atrial flutter:



Atrial flutter with high-grade block:



Because atrial flutter can deteriorate into symptomatic high-grade block, it is important to be able to recognize the flutter waves so as not to be caught unawares

What is this rhythm?

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_____
_____
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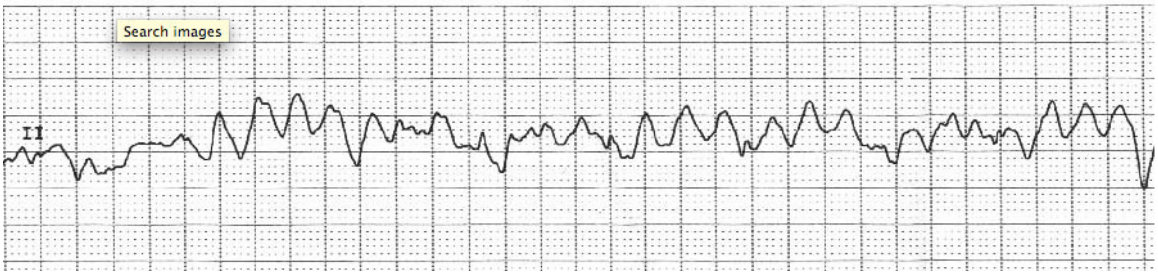
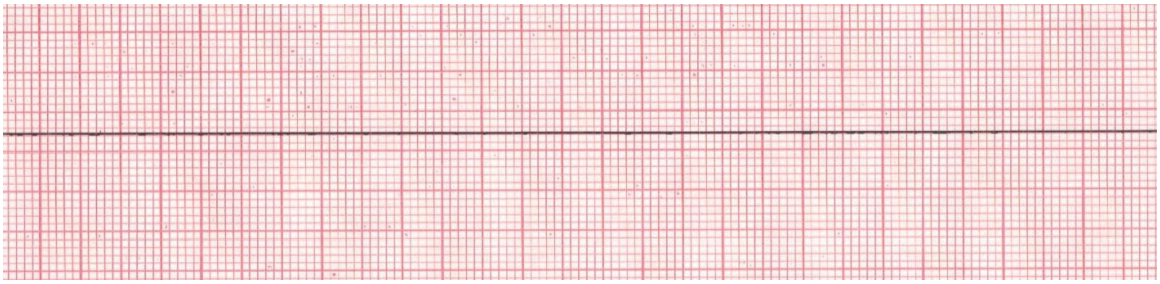
What is this rhythm?



The rhythm to end all rhythms?



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CASE STUDIES IN SEDATION DENTISTRY

SHOWCASING ASA III AND IV PATIENTS

HIGHLIGHTING NEW DRUGS, NEW PROTOCOLS, NEW GUIDELINES

Leslie S.T. Fang, MD PhD
Jerry Wellbrock, DDS

CASE 1

36 year old with a history of hypertension comes in seeking sedation dentistry.

He has had a 6-year history of hypertension that has been “impossible to control” and seeks sedation because his blood pressure is usually so high that dentistry was not possible.

On examination, his blood pressure was 178/110 with a pulse of 110. He admits that he is extremely anxious and is quite jittery and diaphoretic. His weight is 134 at a height of 5’ 5”.

Family history is significant for a brother with severe hypertension.

His medications include:

Omesartan 40 QD
Hydrochlorothiazide 25 QD
Amlodipine 10 QD
Minoxidil 10 QD
Hydralazine 100 TID
Metoprolol 100 BID
Diltiazem CD 300 QD

He will need 8 hours of dentistry:

What is his ASA categorization?

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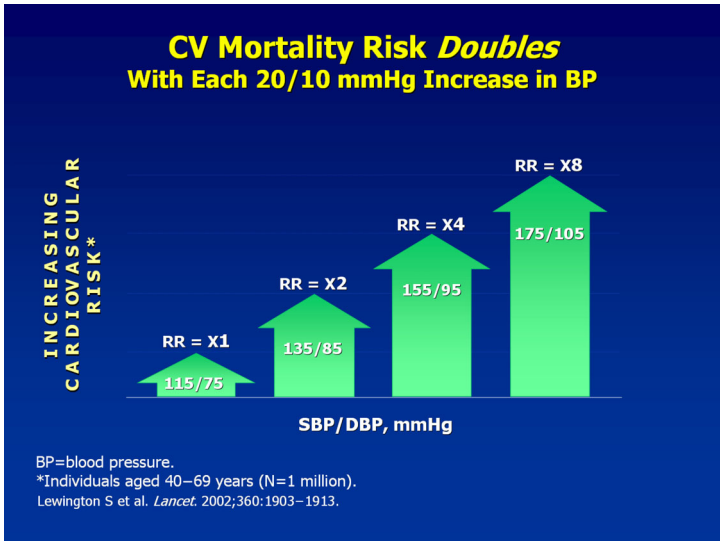
How would you sedate this patient? Is IV sedation safer?

Are there drug-drug interactions that we have to be aware of?

What kind of monitoring is necessary for this patient?

Management of Patients with Stage 2 Hypertension:

It is understood that anxious patients can present seeking sedation, hoping that sedation would decrease the blood pressure.
We have a young anxious patient with a strong family history of hypertension.
He is on 6 drugs and his blood pressure is still poorly controlled.
He hopes that sedation would be the solution since many patients would have blood pressure falling during sedation. However, it is still important not to sedate patients with Stage 2 hypertension in view of the significant cardiovascular risks that these patients pose:



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This is a patient on 6 drugs including Minoxidil. Minoxidil is a very potent vasodilator with significant side effects including fluid retention and glucose intolerance. The drug should be a red flag since it is generally reserved for patients with severe and intractable hypertension.

JNC 8 Guidelines and Sedation Implications

JNC 8 provides us with a glimpse into the magnitude of the problem with respect to the population that would be at risk and the adequacy of control of blood pressure.

One in three adults have hypertension in the US (73 million). Unfortunately, 39 million patients have blood pressure >140/90. Therefore, a lot of patients would arrive at the dental office having an elevated blood pressure (1 in 6 adults).

If the blood pressure is greater than 140/90 upon arrival, it would be reasonable to repeat the blood pressure in 5 minutes. If the blood pressure is below 160 systolic or 100 diastolic on repeat (Stage 1 hypertension), we can proceed with sedation. On the other hand, we should not sedate patients with persistent Stage 2 hypertension.

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CASE 1A 36-year-old patient except his BP is 160/96 on

He is on Verapamil 180 QD and HTZ 12.5 QD
Prilosec 20 qd
St. John's wart qd

Management of Patient with Stage 1 Hypertension:

What is his ASA status?

What potential drug drug interactions should we be concerned about?

What is the proper way to manage his hypertension medications through sedation?

How would you manage his BP through sedation?

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CASE 2

58-year-old gentleman with hyperlipidemia, a significant alcohol history and CHF secondary to dilated cardiomyopathy comes in for sedation dentistry. His history is also significant for having had a CRT-ICD placed a year ago to improve his cardiac output and “for his protection”.

He has been seeing another dentist but was told that he should seek dentistry under sedation because it would be safer.

His medications include:

- Coumadin titrated to INR of 2-3
- Amiodarone 200 BID
- Carvedilol 6.25 BID
- Furosemide 80 BID
- Metolazone 2.5 QD
- Prezcoxib 800/150 QD

He is allergic to penicillin.

On examination, his blood pressure was 98/60, his pulse rate was 60 and his rate of respiration was 18. He is 190 lbs. and 5’ 4”.

He will need 5 hours of dentistry:

What is his ASA categorization?

What additional information do you need?

How do you categorize his congestive heart failure?

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What is a CRT-ICD and how does it impact upon his sedation dentistry?

We have purposefully used a complicated case to push you to think about each of the component issues in this patient. You have elected not to sedate this patient but you are likely to see patients with various components of this case and will have to decide on what you would want to do depending upon your own comfort level.

Let's break out the component parts of this case and examine the issues arising from each of these components:

Categorization of Congestive Heart Failure:

Congestive heart failure is a growing concern: About 5.7 million patients in the United States have heart failure. One in 9 deaths in 2009 included heart failure as contributing cause. Heart failure costs the nation an estimated 30.7 billion dollars.

Degree of heart failure is usually gauged by the **Ejection Fraction (EF)** of the heart, i.e. what percentage of blood in the ventricle is ejected with every contraction:

Normal	> 45%
40-45%	Mild compromise
30-40%	Moderate compromise
<30%	Severe compromise

Functional status of the patient is also very important and the New York Heart Association has put forth this classification:

Class I	No limitation
Class II	Slight to mild limitation of activity
Class III	Marked limitation of any activity
Class IV	Symptoms at rest

Based on the EF and the NYHA classification, we can then risk stratify the patients. Our patients clearly has severe compromise.

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Management of Congestive Heart Failure:

Based upon the medications the patient is on, you can have a good idea of the degree of compromise from congestive heart failure.

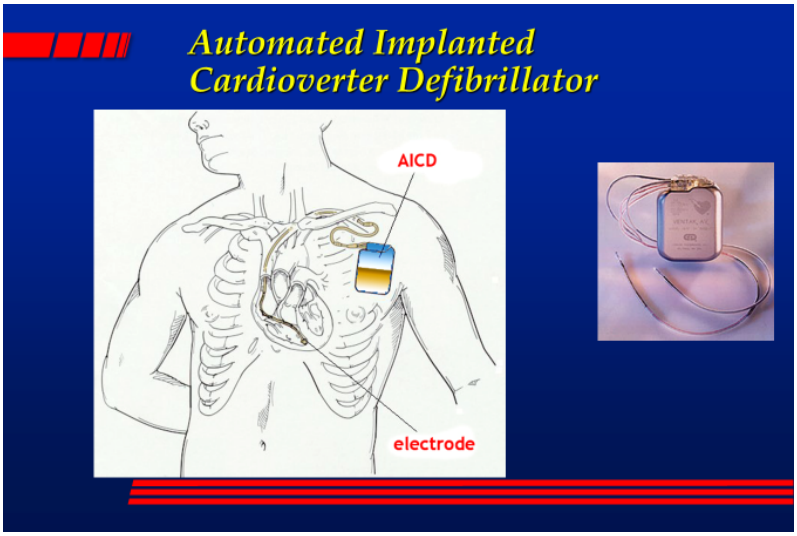
Most patients are on diuretics,

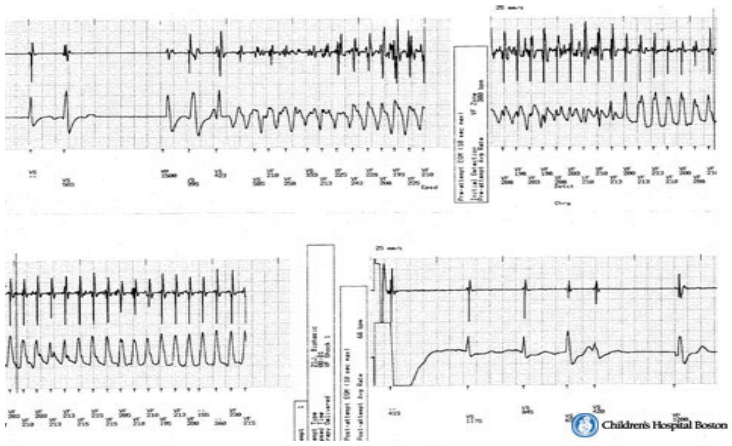
Patients with ejection fraction of <30% are at risk for clots forming in a baggy ventricle and are therefore **anticoagulated.**

Our patient is on Coumadin for low EF.

Management of Risk of life-threatening VT/VF:

Patients with ejection fraction of <30% are at risk for sudden death and are therefore candidates for automated implantable cardiac defibrillator (AICD or ICD):





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What would you do with his coumadin for his sedation dentistry?

How does amiodarone affect his candidacy for sedation dentistry?

What kind of monitoring is necessary for this patient?

How would you sedate this patient?

How does his being on Prezcovix affect your decision to sedate this patient?

What kind of monitoring is necessary for this patient?

If you were to sedate this patient, what would you use?

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Our patient clearly has anticoagulation and ICD implantation, indicating that he is at high risk of developing complications of congestive heart failure.

Also telling is the inclusion of **Amiodarone** in his medication regimen. This should be an automatic red flag since amiodarone is a drug with significant side effects (pulmonary fibrosis and hypothyroidism) and is reserved for intractable VT/VF or vary rapid AF. Our patient is on Amiodarone.

Management of anticoagulation

There is ample evidence that, as long as the patient is in therapeutic range, the risk of stopping anticoagulation in the patient is higher than the risk of bleeding complications.

Monitoring of Patients with Congestive Heart Failure:

Patients with mild congestive heart failure can be managed with pulse oximeter alone but patients with moderate or severe congestive heart failure should not be done without **cardiac monitoring** (and preferably, with capnography). While cardiac monitoring may be intimidating to some, the technology is now here to help with rhythm interpretation and help guide management. For management of such complicated patients, capnography is also rapidly becoming the standard of care. Capnography measures ventilation; pulse oximetry measures oxygen saturation which can be manipulated by adjustment of oxygen flow and may create a lag in recognition of respiratory compromise.

History of Excessive Alcohol Use:

This is presumably the etiology of his dilated cardiomyopathy. It is therefore reasonable to assume that his liver function is also compromised. If we were to sedate this patient, lorazepam would be a better choice since it is not metabolized by CYP3A4, an enzyme that is more sensitive to liver disease than glucoronidase that metabolizes lorazepam.

We have chosen a case that is very complicated and you have appropriately chosen not to sedate this patient. However, this case does help you understand all of the management issues pertaining to this disease that is rapidly increasing in prevalence.

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Case 3

38 year old with Type 1 diabetes mellitus and hypertension comes in for sedation dentistry.

He has been on an insulin pump for the last 8 years and has recently acquired a continuous glucose monitor. He has had excellent control of his diabetes with HbA1c of 7 over the past year. He has peripheral neuropathy, retinopathy and low grade proteinuria.

His medications include:

Continuous insulin infusion
Metformin 500 BID
Irbesartan 300 QD
Hydrochlorothiazide 12.5 QD
Doxycycline 50 BID

He has no known allergies.

On examination, his blood pressure is 120/84, P of 64 and R of 12. He is 5' 7" in height and 177 lbs. His oxygen saturation is 99% on room air.

He will need 9 hours of dentistry

Diabetes mellitus: 100 million diabetics and pre-diabetics

There are 1.5 million patients with Type 1 diabetes. These patients have an autoimmune disease that destroys selectively the beta cells of the pancreas, making them incapable of secreting insulin.

There are 24.5 million patients with Type 2 diabetes. These patients have endogenous insulin but has peripheral resistance, usually related to obesity.

Most worrisome is the fact that there are 74 million patients with pre-diabetes and these patients will undoubtedly incur significant burden on the medical and dental systems.

Treatment of Type 1 diabetes

Since patients cannot produce insulin, the major treatment modality is insulin. There are different forms of insulin: short acting, intermediate acting and long acting.

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However, the major emphasis in the Type 1 diabetic is the use of **insulin pump** whenever feasible. Barriers to using the insulin pump include patient compliance and financial barrier.
Insulin pump generally provides a very low dose continuous infusion of insulin, mimicking natural production of insulin. This can be adjusted according to the activity level of the patient. At meal times, the patient would inject a bolus to address the sugar and caloric intake for the meal.

Another important development in the treatment of Type 1 diabetics is the development of **continuous glucose monitoring.** This would allow for literally minute to minute adjustment of insulin infusion. However, the monitors track tissue glucose, which usually lags behind blood glucose. Nonetheless, we now have a means to tailor insulin infusion to tissue/blood glucose.

The glucose monitor can be coupled with the insulin pump, but the adjustment of the insulin infusion is still regulated by the patient.

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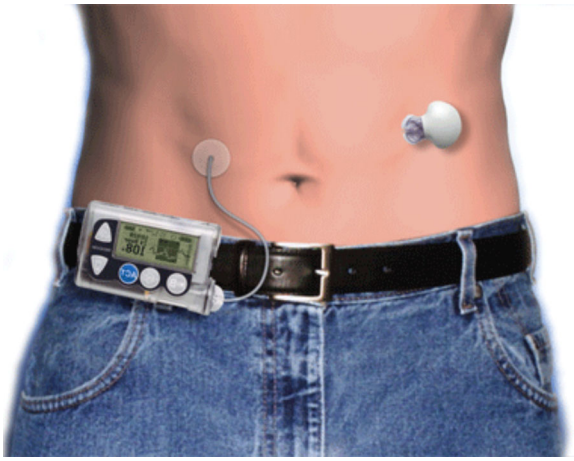

What is his ASA categorization?

What additional information do you need?

How would you sedate this patient?

What Protocol would you employ?

What would you do with his Doxycycline?

What would you do with the continuous insulin infusion through sedation?

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What is the proper way to manage his diet through sedation?

How does the diabetic triopathy (neuropathy, retinopathy and nephropathy) affect his sedation dentistry?

What kind of monitoring is necessary for this patient?

Management of Hypoglycemia

Mild hypoglycemia is characterized by sympathetic overdrive with weakness, jitteriness, nervousness, tremulousness, palpitations and excessive sweating. Unfortunately, these may lead the clinician to conclude that the patient is anxious and under-sedated.

Severe hypoglycemia results in lethargy, agitation and confusion and culminates eventually in seizures, coma and death.

It is therefore important to aggressively address the issue of hypoglycemia.

In the conscious patient, orange juice, cola or other glucose containing solution should be given to the patient, usually in three 4-ounce aliquots.

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Dental Management of Patients with Diabetes Mellitus

Diabetes Mellitus: an absolute or relative insulin insufficiency caused either by a low output of insulin from the pancreas or resistance of peripheral tissue to insulin

Type of Diabetes	
Type 1	Absolute insulin deficiency
Type 2	Peripheral insulin resistance

Adequacy of Control	
HgA1c < 6.5 %	Excellent control
HgA1c 6.5-8.5 %	Marginal control
HgA1c > 8.5 %	Poor control

Presence of Complications	
No	Low risk
Yes	High risk

Types of Complications
Retinopathy
Neuropathy
Nephropathy
Vasculopathy

Time of appointment	Morning appointment
Diet	Normal breakfast; protect oral intake
Stress Reduction	Patient rapport; short appointments
Sedation Protocol	Consider oral conscious sedation Consider use of nitrous oxide
All non-surgical and simple surgical procedures	Normal protocol Continue oral hypoglycemics or insulin
Complex prosthetic and surgical procedures	Consider sedation protocol Continue oral hypoglycemics or insulin
Infection	Aggressive treatment of infection

- Morning appointment with normal breakfast
- Continue oral hypoglycemics or insulin
- Simple restorative and surgical procedures under monitored environment
- Complex surgical procedures should be deferred
- Stress reduction and sedation

Emergency Management of Hypoglycemia
Stop dental procedure
Give glucose (orange juice, Gluco-gel, Insta-glucose)

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Case 4

67 year old with a history of Type 2 diabetes mellitus, hypertension and coronary artery disease comes in for consideration of sedation dentistry.

The patient has had diabetes for 11 years and has peripheral neuropathy. He has had hypertension since age 40 but this has been under good control. He developed dyspnea on exertion 5 months ago and had a positive stress test. He underwent coronary angiogram and was found to have a critical LAD lesion. He underwent atherectomy and placement of DES and is now on ASA and Brilinta. He has been completely symptom free since his intervention.

He is currently on:

Metformin 850 BID
Glipizide 10 QD
Januvia 100 QD
Irbesartan 300 QD
Metoprolol XL 100 QD
ASA 81 QD
Ticagrelor 60 QD
Fluconazole 100 BID

On exam, his blood pressure is 110/70, pulse of 60 and rate of respiration is 14. His weight is 243 lbs. and his height is 5' 8". His oxygen saturation is 98%

He will need 6 hours of dentistry:

Increasing number of percutaneous coronary interventions (PCI) are being done in place of CABG.

In this patient, he has had an atherectomy and placement of a stent and is therefore on DAPT (double anti-platelet therapy).

Atherectomy is a roto-rooter intervention that can open up even closed arteries. A distal basket is usually there to help with catching the debris from the procedure.

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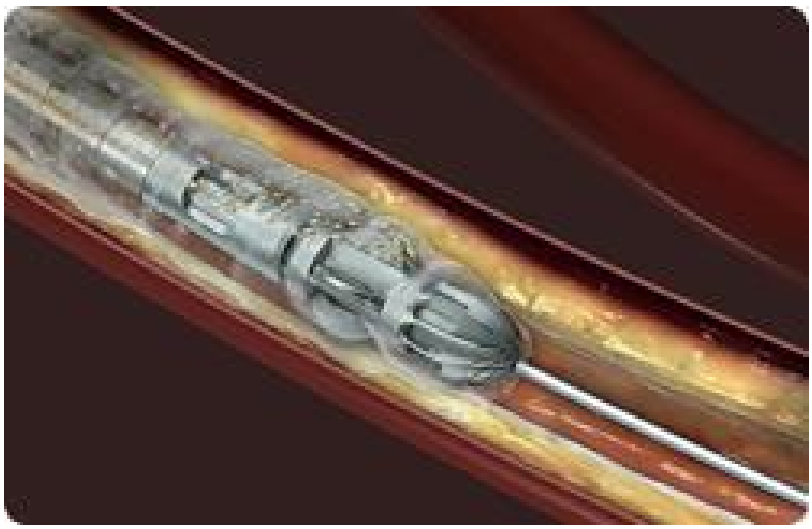

What is his ASA categorization?

What additional information do you need

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MANAGING A TYPE 2 DIABETIC THROUGH SEDATION

Diabetes mellitus is the sixth leading cause of death by disease in the United States. It is one of the most costly illnesses in the United States and diabetic complications such as blindness, renal failure and amputation contributes significantly to the cost.

Diabetes now affects one in 8 adults in the United States (8-9%). The prevalence is higher in the elderly and in certain ethnic groups such as blacks, Native Americans, Asian Indians and Pima Indians.

Relatives of diabetics have 2.5 times the risk of developing the disease.

There has been a 49% increase in the US in new cases diagnosed between 1990 and 2000 among adults. The rate of increase is expected to rise exponentially.

Currently, approximately 190 million people around the world have diabetes. Projections by the International Diabetes Institute suggest that this figure will rise to more than 330 million by the year 2025.

Diabetes currently affects 17 million patients in the US and accounts for $132 billion of the health care costs (1 out of 10 health care dollars spent).

An average dental practice of 1000 patients should expect to have 80-90 people with diabetes mellitus.

Medical Management of Type 2 Diabetes Mellitus

Patients with type 2 diabetes mellitus are advised to normalize weight by exercise and diet. They should be treated with oral hypoglycemic agents. With time, oral hypoglycemic agents may not be sufficient and insulin may have to be added.

Six different classes of oral antidiabetic agents are available.

A **Biguanides** such as Metformin act on intestinal absorption of glucose, decrease insulin resistance and also to enhance anaerobic glycolysis

B. **Sulfonylureas** such as Glyburide and Glipizide act to enhance insulin secretion. Chronic administration of Sulfonylureas also decrease insulin resistance

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A. **Glitazones** (Avandia, Actos) act primarily to decrease insulin resistance. Unfortunately, concerns have been raised about the cardiovascular safety of this class of drugs
B. **Alpha-glucosidase inhibitors** (Precose) act to decreases intestinal absorption of glucose
C. **Repaglinide** (Prandin) acts to stimulate pancreatic secretion of insulin
D. **Sitagliptin** (Januvia) acts to decrease insulin resistance

The United Kingdom Prospective Diabetes Study indicated that many patients with diabetes mellitus eventually require a combination of oral antidiabetic agents. A single oral antidiabetic agent should be used and gradually increased to the maximal dose. The first agent should be Metformin. If the patient's target is not reached within 2 to 3 months, the next level of antidiabetic agent should be introduced. While the majority of patients with type 2 diabetes mellitus can be controlled with combination of oral antidiabetic agents, insulin may be necessary in the later phases of the disease.

A patient on 4 or more drugs for control of blood sugar is usually a complicated patient

Diabetics should be taught to do home monitoring of blood glucose. This would involve the use of fingersticks and frequent determination of blood glucose over the course of the day. Therapy should be adjusted based on home monitoring.

Adequacy of Control

Although fasting blood sugar, urinary testing of glycosuria and glucose tolerance tests can all be used to evaluate adequacy of control, Hemoglobin A1c is now the gold-standard for determination of adequacy of control.

Hemoglobin A1c (HgA1c) is an important gauge of adequacy of control. The goal is to get a hemoglobin A1c level less than 6.5% (usually achieved with pre-prandial blood glucose levels of 70-126 mg/dl).

Current ADA guidelines regard HbA1c level of 6.5% as ideal control.

For every 1% decrease in HgA1c, there is:
21% decrease in mortality
21% decrease in diabetic complications
14% decrease in myocardial infarction
37% decrease in microvascular complications

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Unfortunately only 31-37% of the diabetic population in this nation has an HbA1c of less than 7%.

Complications of Diabetes

Patients with diabetes would develop a number of complications. These complications develop over years and progress from asymptomatic states to multiple complications.
In addition to the metabolic complications of diabetes, patients with diabetes mellitus may develop vascular, cardiovascular, renal and neurologic complications.

A. Large vessel complications of diabetes mellitus include peripheral vascular disease, coronary artery disease, and cerebrovascular disease.
B. Small vessel complications lead to diabetic retinopathy and diabetic kidney disease
C. Neurological complications include peripheral neuropathy with prominent sensory losses.
D. Autonomic insufficiency is seen in patients with severe disease and can result in postural hypotension, sexual impotence and abnormality in gastrointestinal motility
E. Diabetics are also more prone to infections because of compromised host responses.

These complications results in:

A. Diabetes being the sixth major cause of death in the US
B. Leading cause of renal failure
C. Leading cause for blindness
D. Leading cause for non-traumatic amputation
E. Diabetes as the major predictor of vascular events

Dental Evaluation of the Diabetic Patient

About 8-9% of the dental patients will have diabetes. These patients require careful dental evaluation:

A. The dentist should know the type of diabetes the patient has (Type 1 vs. Type 2)
B. The dentist should know the HgA1c in order to determine the adequacy of control
C. The dentist should know about presence or absence of diabetic complications

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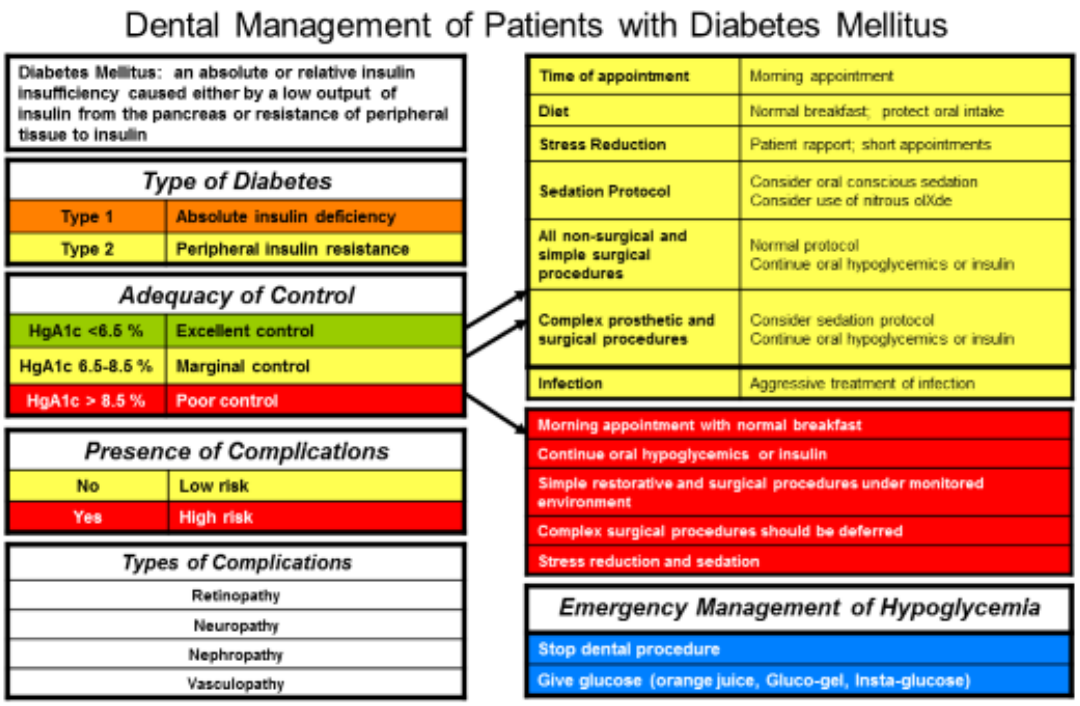
D. The dentist should know the treatment the patient is receiving for his diabetes.

Sedation of the Type 2 diabetic patient

A. Light non-fatty breakfast
B. Continue on oral hypoglycemic agents
C. Limit the length of the procedure to less than 3 hours
D. Make sure that the patient can resume oral intake
E. More liberal use of antibiotics



What form of sedation would you offer this patient?

What protocol would you employ?

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What would you do with the oral hypoglycemic agent?

What would you do with his Doxycycline

What would you do with his diet on the day of sedation?

How would you manage the ASA and Brilinta?

How does the atherectomy affect your sedation decisions?

What is the appropriate monitoring for this patient?

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CASE STUDIES IN SEDATION DENTISTRY II

Leslie S.T. Fang, MD PhD
Tony Feck, DMD

Case 5

60 year old lady in for consideration of sedation dentistry.

She has had hypertension “all of her adult life” but it has in general been under good control. She was diagnosed to have metastatic breast cancer 3 years ago, has had a right mastectomy and has recently been started on Avastin for metastatic disease to bone.

Her medications include:

Enalopril 40 QD

Atorvastatin 40 QD

Denosumab 60 SQ Q6M

Bevacizumab (Avastin) per protocol

She needs 3 extractions and eventual implants. She will need two sessions under sedation, the first 2 hours and the second lasting 3 hours.

What is her ASA categorization?

What other information do you need?

What form of sedation would you offer her?

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What concerns would you have?

MEDICATION-RELATED OSTEONECROSIS OF THE JAW: 2014 UPDATE SPECIAL COMMITTEE POSITION PAPER by AAOMS

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Definition

Change from BRONJ (bisphosphonate-related osteonecrosis of the jaw) to MRONJ (medication-related osteonecrosis of the jaw) because of growing number of cases of osteonecrosis of the jaw associated with other antiresorptives) denosumab: Prolia) and antiangiogenic agents (Avastin, Sutent, etc.)

Historical Perspective

Since 2003, there are more than 900 publications, more than 6,400 cases reported, and greater than 600 cases with oral bisphosphonates.

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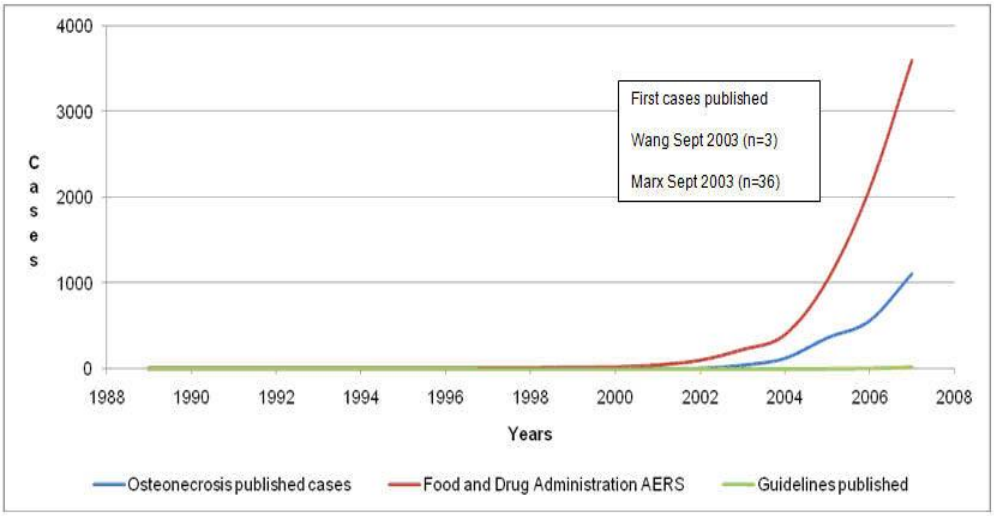
Timeline of Cumulative Cases of Osteonecrosis of the Jaw



FDA AERS = bisphosphonate associated ONJ logged into the FDA's Adverse Event Reporting System

Published cases= peer review publications

Clinical Implications for the Dentists

The dental professional will come across patients who are on medications that can cause osteonecrosis of the jaw:

1. 33 million prescriptions written world-wide for oral bisphosphonates
2. 30,000 cancer patients are receiving high-dose IV bisphosphonates for bone involvement by cancer
3. Increasing number of cancer patients on antiangiogenic agents

It should be stressed that 92% of the reported cases of MRONJ occurred in **cancer** patients who received high dose IV bisphosphonates. 8% of cases received oral or low-dose IV bisphosphonates for treatment of **osteoporosis**.

Among the bisphosphonates, IV zolendronic acid (Zometa) appears to be the leading offending agent.

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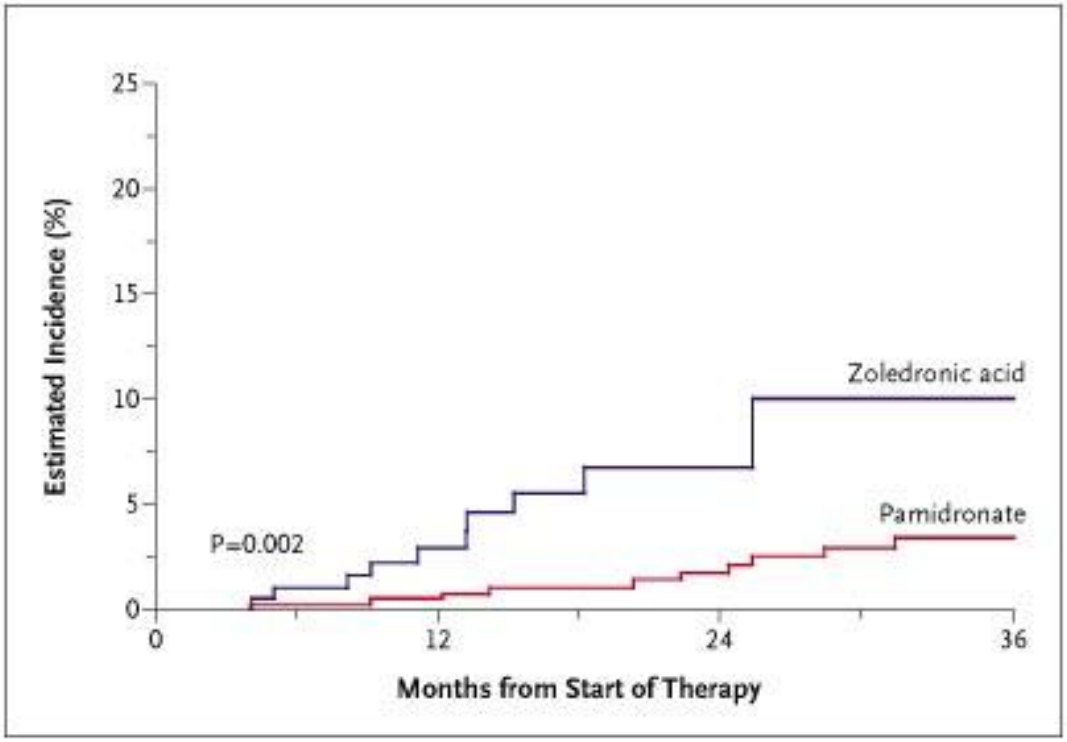

Diagnosis

1. Current or previous treatment with an anti-resorptive or antiangiogenic agent
2. Exposed bone or bone that can be probed through an intraoral or extraoral fistula(e) in the maxillofacial region that has persisted for more than 8 weeks.
3. No history of radiation therapy to jaws or obvious metastatic disease to the jaws.

Pathophysiology

Despite all that has been written, the pathophysiology of MRONJ is still not fully elucidated. Postulates:

1. Inhibition of osteoclastic bone resorption and remodeling
2. Inflammation/infection
3. Inhibition of angiogenesis

Risk Factors

1. Dose and duration of medication exposure
    a. High-dose IV bisphosphonate poses a higher risk than low dose IV or PO bisphosphonates
    b. In general, risk increases progressively with duration of therapy
2. Type of surgery

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   a. Dentoalveolar surgery
   b. Tooth extraction was the precipitating factor in 52-61% of cases
   c. In cancer patients exposed to IV bisphosphonates, tooth extraction was associated with a 33-fold increased risk for ONJ
3. Anatomical factors:
   a. ONJ is more likely to occur in the mandible (73%) than in the maxilla (22.5%) but can occur in both jaws (4.5%)
   b. Denture use was associated with a 2-fold increased risk for ONJ in cancer patients treated with IV bisphosphonates
4. Inflammation and infection are well recognized risk factor. Among cancer patients with MRONJ, pre-existing inflammatory dental disease was a risk factor among 50% of the cases.
5. Other factors:
   a. Corticosteroids
   b. Diabetes mellitus
   c. ?anemia
   d. ?tobacco use
   e. ?genetic predisposition (single nucleotide polymorphism)
6. C-terminal Telopeptide (CTX) is not validated and not recommended.

Incidence and Risks with Specific Agents

Cancer Patients

1. Risk of ONJ among cancer patients not given bisphosphonates ranged from 0% to 0.019%.
2. Risk increased by 50-100 fold among cancer patients exposed to zolendronate (Zometa, bisphosphonate) intravenously.
3. Risk is also increased by 50-100 fold among cancer patients exposed to denosumab (Xgeva, non-bisphosphonate anti-resorptive, a RANK-L antibody)
4. Risk of ONJ among cancer patients exposed to bevacizumab (Avastin, monoclonal antibody) is 0.2%
5. Case reports of ONJ for other antiangiogenic agents such as the tyrosine kinase inhibitors, sunitinib (Sutent) and sorafenib (Nexavar)
6. The risk is additive and combination of antiangiogenic agents with bisphosphonates markedly increase the risk.

Osteoporotic Patients

A larger population but a smaller problem:

1. Alendronate (Fosamax, Fosamax D), Risedronate (Actonel) and Ibandronate (Boniva) are associated with very low risk with incidence in the 0.04% range
2. Low dose IV bisphosphonates (Zoledronic acid, Reclast) is also associated with low risk
3. Denosumab (Prolia) is also associated with low risk

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Recommendations: Dental Evaluation:

PART OF YOUR NEW QUESTIONNAIRE

1. Are you currently on or have you been on
    a. Fosamax
    b. Fosamax plus D
    c. Actonel
    d. Boniva
    e. Prolia
    f. Zometa
    g. Aredia
2. If you answered yes:
    a. Duration of therapy
    b. Dose of medication
    c. If you are no longer on the medication, years since discontinuation
3. Any kind of cancer?
    a. What is the type of cancer you have had?
    b. Have you been treated with chemotherapy?
    c. What type of chemotherapy have you had?

Cancer Patients on IV Bisphosphonates

1. Inform patients of risks
2. Good oral hygiene
3. Regular dental care
4. Aggressive treatment of infection
5. Endodontic procedure preferred over extraction
6. Extract teeth only when the mobility is 3 or greater
7. Atraumatic tooth extraction
8. Chlorhexidine mouthwash
9. There is no data on the efficacy of drug holiday

Cancer Patients Prior to Initiation of IV Bisphosphonates

1. Thorough examination
2. Radiographic assessment
3. Identify acute infection and sites of potential infection and treat
4. Use of fluoride and chlorhexidine
5. Assess tooth mobility, periodontal disease, presence of root fragments, caries, periapical pathology, edentulism and denture stability
6. Address caries
7. Eliminate compromised teeth
8. Evaluate third molar
9. Eliminate ill-fitting dentures

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Patients with Osteoporosis

Risk for ONJ among osteoporotic patients exposed to oral bisphosphonates ranged from 0.00038% to 0.1%

Risk increases to 0.21% among patients with greater than 4 years of bisphosphonate exposure.

Risk stratification should include:

1. Duration of therapy >4 years
2. Age > 65
3. Oral infections
4. Pre-existing dental disease
5. Diabetes mellitus
6. Steroid use
7. Head and neck radiation

To a lesser extent:

1. Alcohol use
2. Tobacco use
3. Anatomical anomalies

Patients with less than 4 years of therapy with no other clinical risks

1. Inform patient that the risk of osteonecrosis is very small but cannot be completely eliminated
2. No alteration or delay in planned dental intervention
3. Good oral hygiene
4. Regular dental care

Patients with more than 4 years of therapy with or without other clinical risks

1. Inform patient of small risk of osteonecrosis
2. Good oral hygiene
3. Regular dental care
4. Aggressive treatment of infection
5. Consider non-surgical option
6. Discuss risks prior to implant placement or surgery
7. Treat one sextant or tooth at a time, where feasible, and allow for a 2 month interval before next intervention
8. Should consider “drug holiday” for 2 months prior to intervention, if feasible, and restart drug when osseous healing has occurred (No data to support or refute this recommendation)

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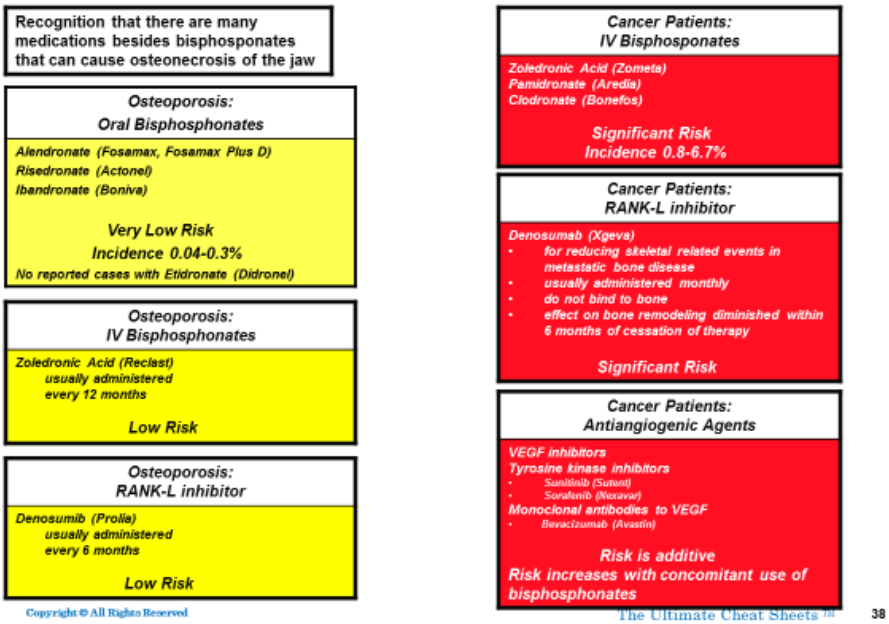
Medication Related Osteonecrosis of the Jaw (MROJN) 2014



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CASE 6

24 year-old with asthma, who has had frequent and severe episodes until she started a new medication 6 months ago. She has had no relapses since.

Her current medications include:

Xolair injection once a month,
ProAir inhaler on a prn basis
Slophylline 100 BID
And Symbicort BID.

She had had one hospitalization for severe asthma but did not require intubation.

On exam, her BP is 120/78 P 68 R 16 and her oxygen saturation was 97% on RA
She is 5' 2" and 180 lbs.

She now comes in and will require 4 hours of dentistry

What is her ASA?

DENTAL EVALUATION OF THE PATIENT WITH ASTHMA

Asthma is an episodic reversible compromise of the airways secondary to constriction or inflammation, resulting in acute shortness of breath and wheezing.

Asthma affects 7% of the population in the United States and the prevalence of the disease appears to be increasing. In urban areas, it is now estimated that one in four children has some degree of bronchospasm.

Asthma accounts for 5,000 deaths per year, and, unfortunately, young patients account for a large proportion of the deaths. Asthma also carries significant morbidity with loss of productivity.

Asthma occurs when there is edema and reactive changes in the small airways of the lungs.

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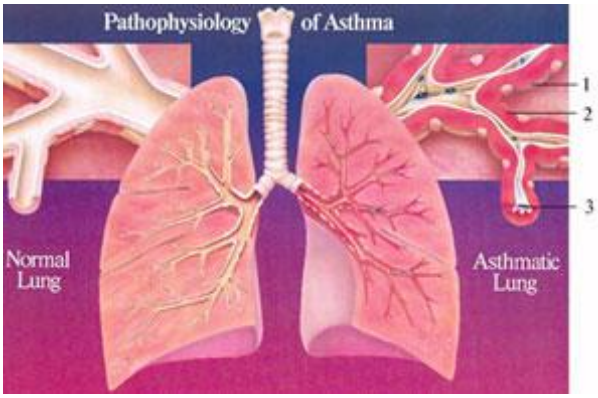

In the assessment of the patient with asthma, it is important to get a detailed history and a list of all of the medications the patient is on. Whenever possible, one should try and obtain some indication of the peak expiratory flow during an acute episode. The patient's physician should have serial pulmonary function on the patient and would be able to extract the PEF from these studies,

HISTORY:

In the evaluation of the patient with asthma, one should get a history of:

—Frequency of asthmatic attacks

—Severity of asthmatic attacks

—Precipitants of asthmatic attacks

—Stress can precipitate attacks

—Need for hospitalization

—Need for intubation

—? Attacks with prior dental procedures

MEDICATIONS:

There are numerous medications that the patient can be on but they belong to one of the following classes of medications:

—Bronchodilators

—Leukotriene antagonists

—Mast cell stabilizers

—Steroids

—IgE Blockers

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Most patients are on combinations of medications and any patient that is on 4 or more medications for the control of asthma is a complicated patient that can develop asthma much more frequently.

PEAK EXPIRATORY FLOW:

It is also important to know the peak expiratory flow during an acute attack since it will allow for immediate assessment of the severity of asthma in a given patient. This objective measurement will tell you how sick the patient might get should he/she develop an attack in the dental office. Peak expiratory flow is usually measured by a small hand-held flow meter or by spirometry in pulmonary function tests. This allows the patient to monitor his/her airway reactivity and permits early detection of an asthmatic attack.

Peak expiratory flow measures the percentage of air expired during expiration during an asthmatic attack and can give an accurate assessment of the severity of the attack;

—Normal PEF	100%
—Mild asthma	>90%
—Moderate asthma	50-90%
—Severe asthma	<50%

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DENTAL MANGEMENT OF THE ASTHMATIC PATIENT

In the management of the asthmatic patient, it is important to instruct the patient to bring his/her own inhaler to the visit. The patient should be instructed to take the normal morning medications. The appointments should be short and should be scheduled in the morning.

Dental procedures should be postponed if the patient presents with an acute respiratory illness, however mild. If the patient is audibly wheezing, has prolonged expiratory phase or is short of breath, dental procedures should be deferred.

Since a patient with asthma is often on adrenergic agents already, it is important to limit the use of vasoconstrictors. Erythromycin derivatives should be avoided since many patients are on theophylline. A small proportion of patients with asthma has aspirin sensitivity and aspirin should be avoided in these patients.

In the asthmatic, hydroxyzine should be used judiciously since drying up of the airway can provoke bronchospasm.

What additional information do you need from her MD?

What precautions do you need for sedating this patient?

How would you sedate the patient?

The patient will need post-operative antibiotics, what antibiotics are most appropriate and what should you avoid?

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Dental Management of Patients with Asthma

Asthma: episodic reversible compromise of airways secondary to constriction or inflammation, resulting in acute shortness of breath and wheezing

Adequacy of Control

Rare episodes	Excellent control
Occasional episodes	Marginal control
Frequent episodes	Poor control

Commonly Prescribed Medications

Inhalant	*Short-acting Beta-2 agonists* Salbutamol (Albuterol, Proventil, Ventolin) Metaproterenol (Alupent, Metaprel) Terbutaline (Brethine) Pirbuterol (Maxair) *Long-acting Beta-2 agonist* Salmeterol (Serevent) Formoterol (Foradil) *Steroid inhalant* Beclomethasone (Vanceril) Budesonide (Pulmicort) Flunisolide (Aerobid) *Combination Steroid/Beta-2 Agonist* Fluticasone/Salmeterol (Advair) Budesonide/Formoterol (Symbicort) *Anticholinergic agent* Ipratropium (Atrovent) Tiotropium (Spiriva)
Leukotriene antagonists	Montelukast (Singulair) Zafirlukast (Accolate) Zileuton (Zyflo)
Mast cell stabilizers	Cromoglicate (Cromolyn) Nedocromil (Tilade)
Steroids	Prednisone
IgE Blocker	Omalizumab (Xolair) by injection

Normal Protocol
Patient should bring his/her own inhaler

Inhaler	Patient should have his/her own
Stress Reduction	Patient rapport; short appointments
Sedation Protocol	Consider oral conscious sedation Consider use of nitrous oxide
Minimize use of vasoconstrictors	2 carpules of local anesthetics with 1:100,000 epinephrine or 1:20,000 levonordefrin
Complex prosthetic and surgical procedures	Consider sedation protocol
Infection	Aggressive treatment of infection

Palliative care under monitored environment

Stress reduction and sedation

Judicious use of vasoconstrictors

Complex surgical procedures should be deferred

Emergency Management of Asthma

Stop dental procedure

Allow patient to use his/her own inhaler
Use Albuterol inhaler if patient did not bring inhaler

Oxygen

Epinephrine IV or IM for severe attacks not relieved by inhaler

Transfer to ER if shortness of breath persists

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The Ultimate Cheat Sheets™

CASE 7

A 43 year old female with a dental implant comes in complaining of mild toothache at the implant site and a low grade fever. The patient has had an uneventful placement of the implant 6 months ago.

Her medical problems include systemic lupus erythematosus with hypertension, hyperlipidemia and chronic kidney disease (Stage 3).

She has severe dental phobia and requests that all intervention be done under sedation.

Her medications include:

Prednisone 12.5 mg QD
Plaquenil 100 QD
Amlodipine 10 QD
Diltiazem CD 300 QD
Hydralazine 100 TID
Metoprolol XL 100 QD
Atorvastatin 40 QD

She is allergic to penicillin (rash with amoxicillin).

On examination, her BP is 150/88 P of 90 R 14 T 100.6
Her height is 5' 4" and her weight is 102 lb.

There is tenderness on tapping the tooth but the tooth is not mobile. There is redness, edema around the implant site and bleeding with gentle probing as well as a slightly increased probing depth. There is no significant increase in probing depth or evidence of peri-implant attachment loss clinically.

What is her ASA status?

What diagnostic procedures do you need to address her presenting complaints?

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What are her risk factors for implant infection?

PERI-IMPLANTITIS

With more dentists placing more diverse implants into patients, there has been an increase prevalence of peri-implantitis. In most instances, this can be successfully treated but in rare instances, this can result in implant failure. The expectation is that >95% of implants will not fail.

Peri-implantitis is the destructive inflammatory process affecting the soft and hard tissues surrounding dental implants. The pathogens involved in this process are very similar to those found in association with periodontal disease.

In compound implants, between the actual implant and the superstructure placement are gaps and cavities into which bacteria can penetrate. Later, these can affect the adjacent tissue and cause peri-implantitis. Bone loss as the result of per-implantitis around an osseointegrated implant can lead to implant failure.

TERMINOLOGY:

1. **Peri-implant mucositis:** reversible inflammatory reaction in the soft tissues surrounding an implant
2. **Peri-implantitis:** inflammation with loss of supporting bone in the tissue surrounding an implant

ETIOLOGY:

Overall frequency is estimated to be 40-50% of peri-implant mucositis over 10 years and 5-8% peri-implantitis for a variety of implant systems over the same period.

The etiology of the implant infection is dependent upon the status of the tissue surrounding the implant, the implant design, the degree of roughness, external morphology and excessive mechanical load.

An increasing number of studies suggests that anaerobic plaque bacteria have an adverse effect on peri-implant tissue health, leading to peri-implantitis.

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MANAGEMENT GOALS:

1. **Control of infection**
   Oral hygiene
   Frequent recall
   2% chlorhexidine
   3% hydrogen peroxide

   Local application or insertion of tetracycline fibers for 10 days
   Local application of minocycline microsphere

   ? culture
   ? systemic antibiotics
      Ornidazole 500 mg BID for 10 days
      Metronidazole 250 mg TID or 10 days
      Amoxicillin 375 and metronidazole 500 mg x 10 days

2. **Detoxification of the implant surface**
   Ultrasonic scaler with Soft tip
   Air powder abrasive technique
   Gauze soaked in saline followed by citric acid application
   Air powder abrasive technique followed by citric acid
   Photodynamic therapy
   Er:YAG laser

3. **Regeneration of the alveolar bone**
   Autologous bone graft
   Autologous bone graft covered by membranes
   Full thickness flap

What are her risk factors for implant infection?

What would be the most reasonable approach to the management?

What would be the role of systemic antibiotics in this instance?

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On radiographic evaluation, it is clear that the left maxillary sinus appears to be opacified, how does that change your management?

She needs sedation for any dental intervention. How would you sedate her?

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CASE 8

36 year old reformed heroin addict comes in for dental evaluation. She has avoided dental care for at least 10 years and comes in now because of severe toothache. She reveals that she has severe dental phobia and cannot have dentistry without sedation.

She is on methadone maintenance but is otherwise on no medications.

She acknowledges that she has had a long hospitalization for infection of her heart valve and has been told that she needs premedication for all dental procedures. She also has had hepatitis "of some sort" but has not required treatment.

She has had no surgical procedures previously.

Her BP is 120/50, P 98 R 16 and T 100.4
Height 5' 8"
Weight 166 lb.
Oxygen saturation of 98%

On evaluation, he has significant oral pathology and will need at least 8 hours of dentistry.

What is her ASA?

What information do you need from her physician?

What is the implication of the heart valve infection?

What is the implication of the hepatitis?

What concerns would you have with her history of heroin addiction?

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What sedation protocol would you use?

What premedication would you use?

What would you do with her methadone therapy?

She will need dental intervention serially about 4 days apart, how does that affect the antibiotic premedication regimen?

Hepatitis C

Hepatitis C is a viral hepatitis transmitted largely by blood exposure. Exposure to other body fluid can sometimes cause hepatitis C. Patients with hepatitis C often would have mild symptoms of fever, malaise, nausea, vomiting and jaundice. While the acute disease is usually mild, patients with hepatitis C would often have persistent disease, leading eventually to cirrhosis and liver failure.

Hepatitis C affects 32 million patients in the US. And is now one of the leading cause of cirrhosis, following alcoholic liver disease.

15% of patients have active infection. 85% are chronic carriers and are infectious.

Hepatitis C can be detected by determination of hepatitis C antibody in the serum. Extent of infection can be monitored by the determination of the viral load by measuring hepatitis C viral RNA.

Patients with hepatitis C should exercise the same precautions as for patients with hepatitis B with limited exposure to blood and body fluids of the infected patients.

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In 2011, Gilead Sciences paid $10.4 billion to acquire Pharmasset, a company with a single drug. Sofosbuvir has been launched in December of 2013 and it is believed that Hepatitis C is now curable with a defined course of combined oral regimen. Unfortunately, Epclusa (sofosbuvir and velpatasvir) therapy is estimated to cost $80-100,000 per course of therapy.

Patients with hepatitis C and worsening liver function tests despite therapy should be considered candidates for liver transplantation.

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Patient Preparation, Dispensing, &Record Keeping

Patient Preparation

The initial appointment (even if the patient has emergency needs – remember they are not likely to tolerate any treatment without sedation) usually comes in two forms:

1. Sedation Consult
    - ✓ Limited time in schedule
    - ✓ Screening procedure
2. Comprehensive New Patient Appointment
    - ✓ Necessary for comprehensive care
    - ✓ Expedites treatment

The Sedation Consult (30-60 minutes) may consist of:

1. Emotional Exam/Dental History
2. Complete Medical History
3. Brief Intra-oral Exam
4. Discussion of Sedation Dentistry
5. Reappoint for Comprehensive Exam
   If, there is time available or the patient has emergency needs, and the patient is agreeable, then the comprehensive exam could be accomplished at this appointment, along with any of the following:
6. Radiographs
7. Pre-Sedation Work-Up
8. Financial Arrangements
9. Dispense or Prescribe Medication (after the sedation appointment is paid for)

The Comprehensive New Sedation Patient Appointment (1-2 hours) may consist of (some of these procedures may have already been performed during the consult):

1. Emotional Exam/Dental History
2. Medical History
3. Comprehensive Exam (including radiographs and study model impressions – remember to ask the patient for permission)
4. Discuss Sedation Dentistry
   If time is available, then any or all of the following can be accomplished (Remember that these patients don't want to have multiple appointments, so the more you can accomplish in these pre-treatment appointments the better):
5. Treatment Plan Presentation
6. Financial Arrangements
7. Pre-Sedation Work-Up
8. Dispense or Prescribe Medication (after the sedation appointment is paid for)

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Complete Medical & Social History

The sedation candidate requires an extensive evaluation of their medical and social history. This includes the following:

- Medical Conditions
- All Medications (including over-the-counter)
- Recreational Drug Use
- Date of Last Medical Evaluation & Physician(s) Contact Information
- The Possibility Of Pregnancy
- Tobacco Use
- Bathroom Routine (Frequency)
- Diet (Frequency, Amount of Sugar, Food Allergies)

A complete list of the patient's medications must be obtained and their potential for interactions with sedation medications obtained. This is best accomplished via a pharmacology software product such as Lexi-Comp Dental Drug Reference Library®.

The severity and control of the patient's medical conditions needs to be evaluated. This information can be gleaned from the patient interview, as well as the patient's medications. For many patients, including some ASAII and all patients where a severe systemic condition is suspected (ASAIII or greater), a medical consult with their treating physician is required.

The most efficient way to obtain such a consult is via facsimile. The practice contacts the physician's office alerting them that a patient under their care is in need of dental care under sedation and that verification of their medications, medical conditions is required for treatment. The physician's office is then informed that a fax is being sent that requires the physician's input into their medical viability for this care. They are told that their prompt attention to this matter is much appreciated and to expect the fax presently. Obtaining the name of the person you speak with is helpful. You should expect to have to follow-up with that individual repeatedly for return of the medical consult facsimile. There will be times when a phone call between the dentist and the physician will be necessary.

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Here is an example of the medical consult facsimile:

Dear Dr. _____,

Our office plans to provide dental care for a patient of yours, _____, under light oral conscious sedation using a sedative in the benzodiazepine class. He/She lists the following medical conditions and medications:

Please verify the patient's medications, medical conditions, your assessment as to the control of these medications, any concerns you may have, as well as your opinion of the patient's ability to undergo a dental procedure with sedation. Thank you for your prompt attention to this matter.

Comments

_____
_____
_____
_____
_____

Sincerely,

Your Name

TREATMENT PLANNING

Sedation patients have some peculiar traits that require the sedation team to adjust their normal treatment planning process. These include:

- **Have altered consciousness while sedated that may affect assessment and communication**
- **Often require longer appointments due to extensive dental needs**
- **Fear, gag response and sensitivity may require your examination and/or x-rays be done with sedation**
- **The Severely Debilitated Mouth can be a challenge to treatment plan** Patients who have not had professional dental care for many years oftentimes have had traumatic experiences in their past dental history that even pain, embarrassment, and social stigmas cannot overcome. These patients can be ideal candidates for minimal and moderate oral sedation. However, their oral condition can be extremely unhealthy, requiring complete reconstruction.

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With regard to treatment planning the sedation patient:

1. Be prepared to perform additional dentistry not treatment planned and do not charge for it.

   DO NOT add additional fees onto an existing treatment plan after pre-payment for services has been received from these patients. Therefore, be extremely careful in treatment planning these patients – include all potential treatment in your proposal. The severely debilitated mouth always holds surprises! For example, if a tooth may potentially need endodontic therapy, then treatment plan it for endodontic therapy. It is easier to refund then ask for additional fees.

2. Present the treatment plan to restore the entire mouth, not individual procedures, however, be prepared to adjust the time frame for financial considerations.

Sedation Work-Up

The Pre-Sedation Work-Up includes:

1. Baseline Vitals
2. Review & Sign Pre/Post Sedation Guidelines
3. Review & Sign Sedation Release Forms
4. Acquire Name & Phone Numbers of Responsible Party
5. Dispense Medication (after financial arrangements)

Patient Preparation Instructions (see next page)

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BEFORE AND AFTER INFORMATION FOR THE SEDATION PATIENT

Patient name: _____ Date:_____

BEFORE APPOINTMENT:

1. An adult (18 years or older) must bring you and escort you inside our office and not leave until you, the patient, are assisted by and assistant or doctor.
2. Take 0.25mg Triazolam (1 pill) one hour before your appointment, unless otherwise directed.
3. NO eating or drinking 8 hours prior to treatment, unless otherwise directed.
4. Wear loose, comfortable clothes to your appointment.
5. NO stimulants before your appointment (caffeine/nicotine), unless otherwise directed. (Nicotine: ___ hours prior to appointment.)
6. DO NOT wear contact lenses during your appointment.
7. **NO CHANCE OF PREGNANCY** _____ (Patient initials)
8. Leave all personal belongings at home (ex: purse, jewelry, watch, etc.) CORPORATE CENTER DENTAL CARE IS NOT RESPONSIBLE FOR BELONGINGS LOST BEFORE APPOINTMENT.
9. No grapefruit juice 1 week before your appointment.
10. No alcohol or recreational drugs 24 hours before or after your appointment.
11. **There has been no change in my medical history or medications since my sedation consult.** _____ (Patient initials)

AFTER APPOINTMENT:

1. An adult (18 years or older) must pick you, the patient, up promptly and stay with you for the duration of the day.
2. Following your appointment, keep hydrated as much as possible.
3. Take 2 extra strength Tylenol and 2 Advil every 8 hours after treatment for discomfort.
4. NO sedatives should be taken for 24 hours after appointment (alcohol included).
5. NO stimulants after appointment (caffeine/nicotine).
6. NO driving for 24 hours.
7. NO operating hazardous devices, heavy lifting or stair climbing until you have completely recovered.
8. Make no important decisions or plan on working for 24 hours.

PATIENT'S DESIGNATED DRIVER

Please designate below the name and telephone numbers for your designated driver (who must be 18 years of age or older):

Name of driver: _____

Phone Number: _____ Cell: _____

Upon releasing you to your designated driver, we will need to go over after care instructions specific to the procedures that were done during the appointment and you give your consent for us to do so. _____(Patient initials)

_____ _____

(Patient, Parent or Guardian) (Date)

_____ _____

(Witness) (Date)

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CONSENT FOR ANESTHESIA AND SEDATION

It is our moral and legal obligation to give you information necessary to make educated decisions in requesting treatment. The benefits of therapy are usually greater than the risk, but just as there are risks involved with driving a car, there are events that can occur with any type of treatment. These are being explained to inform and educate you…not to alarm you. Eliminating surprises will make your care go more smoothly. _____

Routine Aftermath…

1. **Minor oozing of blood from the surgery sites, if you are having teeth extracted, which will require you to use gauze pressure packs for the first 24 to 36 hours.**
2. **Postoperative discomfort and swelling which may require several days of home recuperation and appropriate medications.**
3. **Chapping of the lips caused by stretching the corners of the mouth during surgery.**
4. **Stiffness of the jaws and restricted mouth opening from several days to several weeks depending on the extent of treatment.**
5. **Bruising and soreness at the site of IV placement which may last for several days.** _____

Rare Occurrences…can include any event that might be remotely possible but unlikely to occur. These include but are not limited to: unexpected reactions to drugs (including allergies), which range from hives to heart failure. In rare instances, an infection (phlebitits) can develop in the arm at the site of the IV. This can be accompanied by redness, swelling and soreness of several weeks duration. The office staff has had training in managing these potential problems. _____

Medication, drugs, anesthetics and prescriptions may cause drowsiness and lack of awareness and coordination, which can be increased by the use of alcohol or other drugs. It would be wise not to operate any vehicle, automobile or hazardous device while taking such medications and/or drugs. Pain medication or the sedative agents can alter your judgment and work performance and you should plan accordingly. _____

Your consent and request for Anthony S. Feck, D.M.D. or Dr. Maxie J. Combs, D.M.D to perform the following treatment, procedure or surgery…

Full treatment as described in my treatment plan. _____

Your authorization for dental care with oral and/or intravenous (IV) administered sedation. _____

Your understanding that on rare occasions, conscious sedation may not allow you to undergo the dental treatment recommended, and other forms of anesthesia (including deep sedation or general anesthesia) may be required. Whether this is the case in your situation cannot be determined until the time of treatment. In some cases, treatment may have to be discontinued. Any charges for sedation and any treatment completed will remain your responsibility. _____

Your authorization for Dr. Feck, Dr. Combs, or any dentist working with him/her to use his/her judgment in managing unforeseen conditions which might unexpectedly arise during the course of the procedure. _____

Your understanding that lack of cooperation with our recommendation during your care may result in less than optimum result. _____

That you read and write English, understand the above information and have had the opportunity to review and discuss it as well as your health history including any serious problems or injuries. _____

That all statements requiring insertion or completion were filled in and inapplicable paragraphs, if any, were stricken before you signed. _____

That you are both mentally and physically competent to give this consent. _____

_____ _____

Witness **Date** **Patient, Parent or Guardian** **Date**

_____

Doctor **Date**

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(Your office name) AFTER SEDATION INSTRUCTIONS

1.) Patient cannot drive for 24 hours.

2.) Do not operate hazardous devices for 24 hours.

3.) A responsible person (18 years or older) must be with the patient until he/she has fully recovered from the effects of the sedation.

4.) Patient should not go up and down stairs unattended. Let the patient stay on the ground floor until recovered.

5.) Patient can eat whenever and whatever he/she wants.

6.) Patient needs to drink plenty of fluids as soon as possible (except alcohol).

7.) Patient may sleep for a long time or may be alert when he/she leaves. Attend to alert or sleepy patients the same. Don't trust him/her alone.

8.) Always hold the patient's arm when walking.

9.) Call us if you have any questions or difficulties. If you feel that the patient's symptoms warrant a physician and you are unable to reach us, go to the closest emergency room immediately.

10.) Patient should not carry, sleep next to or be left alone with the elderly or small children for a period of no less than 24 hours after the last dosage of medication.

11.) Drive directly home and call the office when you arrive at home and the patient is comfortable and safe.

Following most surgical procedures there may or may not be pain, depending on the patient's threshold for pain. The patient will be provided with medication for discomfort that is appropriate for him/her. In most cases, a non-narcotic pain regimen will be given consisting of acetaminophen (Tylenol) and Ibuprofen (Advil). These two medications **taken together** will be as effective as a narcotic without any of the side effects associated with narcotics. If a narcotic has been prescribed, follow the directions carefully. If you have any questions about these medications interacting with other medications the patient is presently taking, please call our office first, a physician and/or pharmacist.

(Your office name): 859-223-4644 (after hours 859-351-6539)

Companion's Signature_____ Date _____

Medications: Take only when checked
__ Tylenol (Acetaminophen) Take two every 8 hours
__ Advil (Ibuprofen) Take two every 8 hours
__Other:_____

_____

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Dispensing Medications

Medications that are dispensed from the office per the DOCS protocol such as diazepam, triazolam, or lorazepam should be carefully and accurately labeled in "child-proof" containers. Their containers are inexpensive and can be obtained from several medical/pharmacy supply houses. One such supplier is

Clark Containers 1-800-477-8425

The labels on these containers should include the following:

- Patient's Name
- Medication Name (Generic or Brand)
- Prescribing Doctor's Name & Contact Information
- Directions for Use
- Special Precautions
- Number of Authorized Refills
- Date of Prescription
- Date of Expiration (If Applicable)

* Your State may have additional requirements

Disposal Of Expired Drugs

The first step is to simply inform your regional DEA Field Division that you are in possession of expired Class IV drugs that you wish to dispose of and request a Form 41. You can find the number for your regional division in the blue pages of your phone book or by going to http://www.usdoj.gov/dea/agency/domestic.htm.

The DEA representative will advise you how to complete and submit the form. You may be required to do this by either standard mail or facsimile, depending on their preference. Complete the Form 41 (Registrants Inventory of Drugs Surrendered), and return it in the communiqué to DEA in triplicate.

Once the DEA has reviewed your completed Form 41, you will be contacted by an agent at your local DEA Field Division to inform you of the specific procedures you must follow while destroying the medication along with the permission to do so. You will be instructed to have at least two persons participate in the disposal of the medication. These must be persons who are licensed physicians, pharmacists, nurses, state or local law enforcement, or any combination thereof.

Once the drug has been destroyed, fill out the remainder of Form 41 and return it to the individual noted by your local DEA Field Division, also in triplicate. This

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will be used to certify that you have indeed taken the measures required by the DEA to correctly dispose of expired medications in your possession.

Proper disposal of these drugs should also be noted in your drug logbook in the inventory section. This is a good place to reference the location of the DEA documents in your office.

Oral Drug Inventory

TRIAZOLAM INVENTORY LOT:_____ EXP: _____
START AMOUNT:_____ X 0.25MG TABLETS

DATE	PATIENT'S NAME	#OF 0.25MG TABS	PRE LOAD	SEDATION VISIT	DISPENSED BY

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DIAZAPAM INVENTORY LOT:_____ EXP: _____
START AMOUNT:_____ X 5MG TABLETS

DATE	PATIENT'S NAME	#OF 5 MG TABS	DISPENSED BY

HYDROXYZINE INVENTORY LOT:_____ EXP: _____
START AMOUNT:_____ X 50 MG TABLETS

DATE	PATIENT'S NAME	#OF 50 MG TABS	DISPENSED BY

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Labels

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs.Feck & Combs 859-223-4644
Pt. Name:_____
5mg Valium
Take before bed the night before appointment.
NO REFILL

Drs. Feck & Combs (859)223-4644
Pt. Name:_____
0.25mg Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILLS

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Drs. Feck & Combs 859-223-4644
Pt. Name:_____
0.25 Triazolam
Do NOT eat/drink 6hrs. prior to appt.
Take pill 1 hr. before appt. NO REFILL

Record Keeping

Key Points:

1. The Drug Enforcement Administration (DEA) has a manual that specifically lays out the requirements of record keeping, storage, inventory for the prescribing and dispensing of a controlled substance.
2. To administer a controlled substance you must be registered with the DEA.
3. The oral and IV sedation medications in the recommended protocols of this course are schedule IV substances.
4. An inventory record must be maintained for two years.
   Inventory Record Requirements:
5. Keep all sedation medications and all other controlled substances in a locked cabinet.
6. When drugs are removed from the locked drug cabinet, care must be taken that they not be left out where others have access to them. Keys

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to the locked cabinet should be in a location known only to trusted personnel.

7. You must keep good records of the dispensing of all controlled substances. Chart Entries should include comprehensive chart notes including:
    - All procedures performed,
    - Sedative medications dispensed,
    - Dosages and times and routes of administration,
    - Local anesthetics administered,
    - Disposition of the patient upon arrival, as well as during the appointment, and at dismissal.
8. The pressure of the emergency oxygen supply should be verified on a regular basis. A record of this weekly inspection should be kept by a designated individual in the office near the oxygen source.

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Ride Name: _____
Phone #: _____

SEDATION VISIT DOCUMENT

0.25mg / 2mg
Enter TOP Dose=_____lbs.÷(100/25)= _____mg or _____ tabs of (Triazolam/Lorazepam) BMI:____

PRIOR TO SEDATION VISIT

Patient:_____ Date: _____
Medical Status: ASA I II III (circle one) Age: _____ Weight: _____
Medications& Dosage: _____
_____
Date of last MEDICAL exam:_____
Allergies to Drug and/or Food: _____
Diet Habits (e.g. Meals/Day) _____
Usual bathroom routine: _____ Amount of sugar in diet: _____
Smoker or Non-Smoker (circle one) Amount: _____
How many alcoholic beverages are consumed in a week? _____
Are recreational drugs used and if so, how often? _____
Baseline Vitals: Pulse _____ SpO2 _____ BP _____
___ Checked meds. in Lexi-Comp, printed copy and scanned in computer
Pre-Medication Prescription: _____ (Diazepam) *Dispensed*: ____
_____ (Triazolam, Lorazepam) *Called In*: ____
Specific Instructions:_____

DURING TREATMENT

Date: _____

Medication: Dosage	Time	Medication: Dosage	Time
_____		_____	
_____		_____	
_____		_____	
_____		_____	
_____		_____	
_____		_____	
_____		_____	

2% Lidocaine with 1:1000,000 epi: _____ Bathroom breaks _____ Food/Drink _____
3% Polocaine: _____ _____ _____
4% Septocaine _____ _____ _____
0.5% Marcaine with 1:2000,000 epi: _____
4% Citanest Plain: _____
General Nature of Dental Procedure: _____
_____

COMPLETION OF SEDATION VISIT

___ Pulse Ox print out copied and stapled to sedation visit document
___ All of patient's property returned to caregiver (glasses, watch, etc.)
___ Patient tolerated treatment well, was ambulatory, and oriented X3 when they left.

_____ _____
(Doctor's Signature) (Assistant/Hygienist's Signature)

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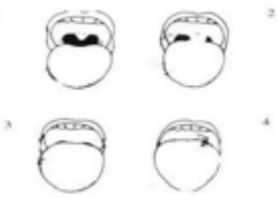
PATIENT_____ **DATE**_____

Drug Allergies:	**Previous Anesthesia History:** (IV or GA experiences)	**Pre-op vital signs:**
	_____ _____ _____	Height:_____ Wt: _____ BP:_____Pulse:_____ Resp:_____

Rating Scales (circle patients diagram and ASA risk assessment)

Mobility Status: (check one in each category) ____Ambulatory ____Wheelchair
Condition: _____ Stable _____Unstable
Activity: (Able to move voluntarily/on command) ___4 extremities ___2 extremities ____0 extrem.
Respiration: ___Able to deep breath and cough _____Dyspnea, shallow or limited breathing
Conscious: ____ Fully awake ____Arousable _____Not responding
Color: ___Normal ___pale, other ____cyanotic



Mallampati Assessment

	ASA classification
I	No organic disease
II	Mild or moderate systemic disease without functional impairment
III	Organic disease with definite functional impairment
IV	Incapacitating systemic disease that is a constant threat to life

Comments regarding pre-assessment:
Evaluating Dentist Signature: **Date:**

Fluid type & total ml used:

_____

Veinpuncture attempts:____

IV Start:	**SEDATION START:**	**Monitors used:** **Person:** _____ **B/P:** _____/_____ **Pulse:** _____bpm **Oximetry:** _____SpO2 **ECG:** _____ **Other:** _____
IV Cath Size:	**PROCEDURE START:**	
IV Site:	**PROCEDURE END:**	
IV Out:	**Pt. release:**	

Rating of Sedation (Check one)

No difficulty_____, slight difficulty_____, unable to accomplish treatment_____

Explaination of Rating: _____

Anesthesia Complications: (Circle which if any)

From sedation: N/V, paradoxical effect, excessive drowsness, NONE

From venipuncture: phlebitis, infiltration, hematoma, local infxn.NONE

Disposition Upon Dismissal

[ ] Tolerated procedure and anesthetic well, alert and oriented to time and place.
[ ] Escorted to companion's vehicle by wheelchair and seat belted into departing vehicle.
[ ] Return _____ days for post-op. Appointment Date._____
[ ] Return PRN
[ ] Verbal instructions given to Patient & caregiver [ ] _____
[ ] Written post-op instructions given Name & Relation of Person Accompanying Patient

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VERSED

5 MG PER 1ML CONCENTRATION:

1. 1 MG= .2 ML
2. 2 MG=0.4 ML
3. 3 MG= 0.6 ML
4. 4 MG=0.8 ML
5. 5 MG= 1.0 ML

FENTANYL

50 MCG 1ML CONCENTRATION

1. 25 MCG=0.5 ML
2. 50 MCG=1 ML
3. 75 MCG=1.5 ML

IV Drug Logs

Versed

Lot:_____

Exp:_____

Vial #:_____

Date	Patient Name	Amount Dispensed From Inventory	Amount Given to Patient During appointment	Amount Discarded	Witnesses

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Fentanyl

Lot:_____

Exp:_____

Vial #:_____

Date	Patient Name	Amount Dispensed From Inventory	Amount Given to Patient During appointment	Amount Discarded	Witnesses

Lorazepam

Lot:_____

Exp:_____

Vial #:_____

Date	Patient Name	Amount Dispensed From Inventory	Amount Given to Patient During appointment	Amount Discarded	Witnesses

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IV Sedation Case Examples

It has been said "experience is a great teacher". Indeed, there is much to be learned from our successes as well as our failures. The problem with learning from our own experiences is that it's akin to taking the test before we receive the lesson, and when we fail the test, the cost of that lesson can be significant.

Speaking of failures, it has been said that the greatest benefit from studying history is to not repeat the mistakes of the past. It can certainly be argued that the experiences of others therefore is a better way to learn than from our own experiences, as others have had to deal with the consequences of their lessons. This is especially true when the lesson is anesthesia, and a failed test can be life threatening for the patient.

This module is designed to present real-life scenarios, many taken straight from actual sedation cases in dental offices, to examine sedation misadventures in an effort to avoid repeating them. Keep in mind that misadventures can range from a sedation experience that didn't go as smoothly as expected, to morbidity, and even mortality.

Therefore**,** morbidity and mortality (M&M) reports can be insightful. However, these reports often have limitations:

- Incomplete, inaccurate
    - Reports to national regulatory agencies, governmental bodies, insurance companies, and professional societies are often limited in scope
    - The public often has limited access to closed claims
- Retrospective surveys tend to be inherently biased

Mortality Rate of Dental Anesthesia[1]

[1]Mortazavi, Baharyand, Yaser. *"Death Rate of Dental Anaesthesia",* J. Clin Diagn. Res., 2017 Jun; 11(6): ZE07-ZE09

This article reviewed more than 20 studies from 1955- 2012 with adequate data focusing on death associated with dental procedures

Findings:

- Found 218 deaths out of 71,435,282 patients (3 deaths per 1,000,000 persons) with the mortality rate of 1:327,684.
- Mortality rate per million dropped in half (6.2 per 1,000,000 vs. 3 per 1,000,000) since 1955 till the last report in 2012.
- In children, most cases died in the age of two to five years.
- Hypoxia was the most common cause of death

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- Cardiovascular, respiratory, and endocrine disorders, hepatic cirrhosis, septicaemia, and bacterial endocarditis were the most frequent underlying systemic disease in deceased patients.
- Although rare, death following general anaesthesia in dentistry, is a critical side effect mostly seen in patients with compromised health condition. Therefore, appropriate case selection in regard with patients' general health status as well as standard technical and equipment conditions are mandatory to diminish the risk of death during dental anaesthesia.

Author	Date	Type of Anesthesia	Death: People	Approx. mortality rate	Mortality rate per 1,000,000
Seldin	1955	General	15:2,429,148	1:161,943	6.2
Seldin	1958	General, Local	59:7,956,627	1:134,858	7.4
Driscol	1966	General	5:1,575,000	1:315,000	3.2
Driscol	1974	General, Sedation, Local	11:5,285,570	1:480,506	2
Tomlin	1974	General, Sedation, Local	29:7,956,000	1:274,344	3.6
Lytle	1974	General	3:1,295,000	1:431,666	2.3
Lytle	1980	General	0:1,285,000	0:1,285,000	-
Coplans	1981	General	56:14,473,000	1:258,446	3.9
Lytle	1989	General	7:470,000	1:672,000	1.4
D'Eramo	1992	General, Sedation, Local	2:2,082,805	1:1,000,000	0.9
Flick	1998	General, Sedation	1:151,355	1:151,355	6.6
Nkansah	1997	General, Sedation	4:2,830,000	1:707,500	1.4
Hunter	1997	General	0:1,126	0:11,226	-
D'Eramo	1999	General, Sedation	0:1,588,365	0:1,588,365	-
Deegan	2001	General, Sedation	19:14,206,923	1:747,000	1
D'Eramo	2003	General, Sedation, Local	2:1,706,100	1:853,000	1.1
Lee	2003	General	0:22,615	0:22,615	-
Rodgers	2005	Sedation	0:2,889	0:2,889	-
Flick	2007	General, Sedation	2:115,940	1:57,970	17
D'Eramo	2008	General, Sedation, Local	1:1,733,055	1:1,733,000	0.5
Braidy	2011	Sedation	0:1,167	0:1,167	-
Rodgers	2011	Sedation	0:3,320	0:3,320	-
Qiam	2012	General	2:34,277	1:1,639	610
Total	1955-2012	General, Sedation, Local	218:71,435,282	1:327,684	3

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Case Examples

The first five case examples are actual cases, and are typical examples from the literature in that limited information is available. This is in part because insurance carriers, who are invested mainly in the outcome of a suit or settlement, more than a retrospective analysis of the clinical details, report them. But mostly actual cases are light on details because the details are lacking in the documentation by the clinician involved, which in itself is a valuable teaching point for the healthcare team in providing any service to the public.

In each of these actual cases the case history will be presented, followed by an analysis in the form of pertinent questions regarding the preoperative evaluation, as well as the the procedure itself.

Case 1

33 yr. old male presented 8 a.m. with two painful third molars.

Because of a heavy travel schedule and being NPO since midnight, the patient and dentist agreed to remove the teeth and perform a lingual frenectomy with IV sedation.

PMH: Hx. of use of OTC appetite suppressants, none taken within 5 days of surgery. Hx. of hypercholesterolemia. Patient is 5' 6" and weighs 175 lbs.

Patient was confirmed NPO, monitors placed included a pulse oximeter, and nitrous oxide and oxygen were started. An IV was started in the right ACF, 2 assistants in the room.

At the start of the procedure, a total of 5mg midazolam, 100mcg fentanyl, were administered.

As the frenectomy began, the patient became more talkative and restless. The dentist gave an additional 2mg midazolam. Within 30 sec, the patient became acutely tachycardic with a heart rate >130/minute. The dentist called 911.

The dentist administered lidocaine 100mg within 2 min. of the onset of tachycardia, then bradycardia developed with a HR of 45/minute.

Epinephrine 10 ml of 1:10,000 was given, heart rate increased to 120/minute. Vital signs stabilized, with saturation at 98% and palpable pulses. Dentist administered 0.4mg naloxone and 0.2mg flumazenil.

EMT arrived and gave another dose of naloxone and flumazenil. The patient became more alert but saturation dropped and became hypotensive.

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In the emergency department, the BP was still low so fluid boluses were given. Admission CXR showed acute pulmonary edema and ARDS, so Lasix was begun.

Cardiac catheterization showed no significant findings. The cardiologist opined that the "patient received too much anesthetic."

No cause was ever identified, patient was hospitalized for 4 days then discharged without any injury or sequela. No suit or settlement.

From a pre-sedation patient evaluation perspective, what is (are) your main concern(s) with this patient?

1. That he did not have a pre-sedation workup appointment
2. His BMI
3. History of hypercholesterolemia
4. That the patient did not take premedication prior to the appointment
5. 1 and 2

What are possible reasons for the patient becoming more talkative and restless as the procedure progressed?

1. Hypoglycemia
2. Under-sedated
3. Psychological condition
4. Reaction to vasoconstrictor
5. All the above

Was it appropriate to reverse the patient?

1. We don't have enough information
2. No
3. Yes
4. All of the above

Case 2

70 yr. old male presented to have multiple extractions as part of a treatment plan for implants and fixed prostheses.

PMH: Hx of hypercholesterolemia, CABG 2 years earlier, taking Cardizem.

Patient is 5' 5" and weighs 205 lbs. Baseline BP was 146/84.

The plan, according to the consent, was IV conscious sedation. Patient was confirmed NPO, 2 assistants were in the room.

Monitors placed included automatic BP cuff and pulse oximeter, and supplemental oxygen was started. An IV was started in the right ACF.

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The night before, 5mg diazepam was taken at bedtime. The day of the procedure, 0.5mg of triazolam PO, 10mg of IV midazolam, and 100mcg of IV fentanyl was administered.

It is not clear at what intervals these drugs were given, or what the bolus dosages were.

The surgery was a difficult one, with multiple surgical extractions. The pulse oximeter ranged from 94-98% O_2 sat throughout the surgery.

There was wide variation in BP reported, with spikes as high as 220 systolic, with the lowest BP reading being 100/50.

Following surgery, the IV was removed, and the pulse was 92 and respirations were 12/minute. The patient appeared slow in regaining alertness.

The dentist stated that the patient was "confused and stuporous". His companion, his wife who is an RN, claimed the patient was "unresponsive."

Pupils were equal, round and reactive, the patient appeared diaphoretic and clammy, lips and fingernails were a "bluish-purple."

No further intervention during recovery. At time of discharge and for next several days patient was very lethargic. Wife also claimed he had nausea and vomiting with dizziness.

He was evaluated by a vestibular specialist, who diagnosed a peripheral vestibular problem. The patient had a dental implant placed about one year later.

An additional year later, during consultation with a neurologist, the patient learned he might have had a cerebellar or brainstem stroke.

MRI showed "encephalomalacia of left posterior fossa and right parietal lobe." MD stated it was an old finding, but difficult to confirm dental anesthetic as causative.

Dentist contacted carrier because he had learned the dental board had discussed the case. No suit ever filed - no sanction for dentist.

What, if any "red flags" should have alerted the clinician to increased risk to anesthesia?

1. Age
2. Baseline BP
3. BMI
4. 1 & 3
5. All the above

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What additional information is missing regarding the patient's risk of anesthesia?

1. Last medical evaluation
2. Medical consult
3. None of the above
4. Both of the above

Which of the following is true regarding the prescribed pre-sedation medication?

1. The dose of diazepam prescribed the night before was appropriate
2. The dose of the night before diazepam should have been cut in half
3. No diazepam should have been prescribed the night before

Which of the following is true regarding the potential reasons for the elevated blood pressure during the appointment?

1. The patient had too much local anesthetic
2. The patient was under-sedated
3. The patient was over-sedated
4. None of the above

Case 3

21 year-old female, ASAI presented to have 3rd molar extractions by OMS with IV conscious sedation.

5mg diazepam was prescribed for the night before.

Prior to the procedure, 5mg of midazolam was titrated over 5-7 minutes. Local anesthetic was then given followed by fentanyl (total of 100mcg).

Surgery was completed and the patient aroused, responding to verbal commands.

15 min later prior to being discharged, the patient became nauseated, and within 5 minutes felt better.

2 min later, respiratory arrest occurred.

Oxygen was administered, and the IV restarted.

0.8mg naloxone was administered, and spontaneous respiration resumed.

The patient awakened and said "I feel fine."

The OMS admitted her for observation. She was later dismissed from the hospital without further sequela.

Minor settlement = $2,999

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Which of the following is the least likely reason for the respiratory arrest?

1. Airway obstruction
2. Oversedation resulting in respiratory depression
3. Self-administered opioid
4. Aspiration of vomitus

Case 4

35 yr. old male had removal of a lower second molar four days prior.

Two days post-op, the patient developed firm swelling in the submandibular and submental spaces, with a temp of 102–103° F.

The dentist prescribed hydroxyzine, amoxicillin and metronidazole.

The patient called the night of the third day post-op stating the swelling was worsening.

Even though the office was due to be closed the next day, the clinician asked the patient to come to the office.

After examining the patient, the surgical plan was incision and drainage with intravenous sedation. Verbal consent was obtained, and NPO status was not stated.

An intravenous line and pulse oximeter were placed.

During the procedure, a total of 6mg of midazolam and 50mcg of fentanyl were administered, and penicillin G was given IV.

As the I & D began, there was a spontaneous rupture of pus.

The I & D was completed, the dentist gathered the used instruments and was bringing them into the next room when the pulse oximeter alarm sounded.

The dentist returned to find the patient with significant swelling of the tongue and trismus.

The dentist attempted to ventilate by mouth-to-mouth, then ran to get a bag/valve/mask and called 911.

Oral and nasal airways were placed. Oxygen saturation never exceeded 70%.

EMT arrived, performed nasal intubation. By that time patient exhibited decorticate posturing.

Pt. was admitted, ultimately discharged to rehab facility in a persistent vegetative state.

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Diagnosis was hypoxic encephalopathy.

Settlement out of court in the seven-figure range.

Which of the following is true in this case?

1. Verbal consent is sufficient as long as it is documented
2. When the doctor and patient are both the same gender, a third party in the operatory during sedation is not necessary
3. There are no conditions under which the sedated patient may be left unattended and unmonitored during the procedure
4. None of the above are true

Case 5

60 year-old female, ASAII presented in the afternoon for extraction of 1 tooth with IV sedation.

She was asked 3 times to confirm her NPO status.

She was administered 0.25mg triazolam one-hour prior to the appointment, 1 mg midazolam and 100mcg fentanyl just prior to the procedure.

After the tooth was extracted, an emesis containing meatballs/pasta/sauce occurred.

When she aroused, she apologized, saying her husband had asked her to have lunch with him to repair their marriage.

No suit, no settlement.

True or False - The sedation team has no potential liability in this case regarding the nausea/vomiting

1. True
2. False

Which of the following is a proper response to nausea/vomiting in this case?

1. Position the patient supine, turn the patient's head to the side and allow emesis
2. Do not assist the patient in clearing the mouth of vomitus as needed
3. Administer 0.4 mg of ondansetron
4. Use nitrous oxide to help calm the patient

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Case 6

52 year-old female presents for dental care, and chooses sedation to address her high fear of dentistry.

She had a medical evaluation eight months ago. She claims type-2 diabetes, hypertension, and high cholesterol.

She is taking Januvia, Metformin, Lisinopril, HCTZ, and Rosuvastatin.

Her BMI is 32.

She remarks of a frequent bathroom routine.

She thinks her HgA1c was 7 the last time it was checked. She checks her blood sugar twice daily and it usually runs between 120-140 mg/dL.

Her baseline vitals are BP – 132/84, BPM – 56, SpO2 – 95%.

What is your primary concern at this point?

1. The patient's age
2. The control of the patient's diabetes
3. The patient's baseline vitals
4. Her frequent bathroom routine
5. The patient's airway risk

Your check for drug interactions does not reveal any significant interactions.

Your consultation with the patient's PCP reveals the patient's last HgA1c to be 7.2. The physician feels as though the patient's medical conditions are under good control, and has no concerns regarding the patient's ability to tolerate dental treatment with conscious sedation.

Your instructions to the patient regarding her medications on the day of the appointment are:

1. Take all medications as usual
2. Withhold the Januvia and Metformin, take all other meds as usual
3. Take a half dose each of Januvia and Metformin, take all other meds as usual
4. Withhold all meds on day of appointment until after she returns home

What should the patient's oral pre-med dose be before the sedation appointment?

1. 2.5mg DZP night before, 0.125mg TZM loading dose day of
2. 2.5mg DZP night before, 0.25mg TZM loading dose day of
3. 5mg DZP night before, 0.125mg TZM loading dose day of
4. 10mg DZP night before, 0.25mg TZM loading dose day of

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During one of the hourly blood sugar checks, the patient's blood sugar is 95 mg/dL. The patient is properly sedated and is demonstrating no abnormal sign or symptoms. What should you do?

1. Administer flumazenil
2. Administer dextrose through the IV line
3. Feed the patient applesauce or yogurt
4. No intervention is necessary

Case 7

An 82 y.o. male presents for full mouth extractions and wants to be sedated. His last medical evaluation was nine months ago. He reports hypertension, high cholesterol, gout, sleep apnea, frequent bruising, frequent urination, and depression. His BMI is 24. He lists the following medications: bisoprolol, amlodipine, atorvastatin, allopurinol, sertraline, and 81mg aspirin. His baseline vitals are: BPM – 61, BP – 115/75, SpO2 93%.

What are your primary concerns at this point?

1. Advanced age
2. Airway
3. Sensitivity to medications
4. Medical condition status
5. All of the above

You require an updated medical evaluation. Your medical consult with his PCP reveals the patient's medical conditions to be under control. What should the patient's oral pre-med dose be before the sedation appointment?

1. 0mg DZP night before, 0.125mg TZM loading dose day of
2. 2.5mg DZP night before, 0.25mg TZM loading dose day of
3. 5mg DZP night before, 0.125mg TZM loading dose day of
4. 10mg DZP night before, 0.25mg TZM loading dose day of

During the appointment, the patient's BP rises to and remains elevated at ~145/88. The BPM is 88. SpO2 is 95%. The patient is conscious.

What are the possible reasons for the patient's increase BP and BPM?

1. He has to go to the bathroom
2. He is in pain
3. He is anxious
4. All of the above

The patient responds that he is comfortable, but needs to go to the restroom. After the patient uses the restroom, all vital signs return to normal. Later in the appointment, the patient's SpO2 drops to 86%. He is conscious and the airway is open.

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What should be the next step you take in your efforts to resaturate the patient?

1. Have the patient take a few deep breaths
2. Reverse the patient
3. Call 911
4. Check the finger clip

During the appointment you notice that the drip in the tubing chamber has stopped. What is the most likely reason?

1. The IV fluid bag is empty
2. The IV has clotted
3. The IV catheter is up against the wall of a vessel
4. The IV catheter is no longer in a vessel
5. Any of the above

Case 8

A 32 y.o. male presents to be sedated for placement of six dental implants. He claims asthma, and a high daily intake of sugar. His BMI is 38. He smokes 2 packs of cigarettes/day. He is on no medication, but does use an inhaler a couple times a year during the height of allergy season. He has had a medical evaluation within the last month. His BP is 132/85, BPM – 68, and SpO2 94%.

At this point, what are your primary concerns with regard to sedating this patient?

1. Asthma
2. Sugar intake
3. Smoking habit
4. Obesity
5. 2, 3, and 4
6. All of the above

Which of the following pre-sedation instructions are appropriate for this patient?

1. NPO six hours prior to the appointment
2. Bring his inhaler to the appointment
3. Smoke just prior to the appointment
4. All of the above

During the sedation appointment, this properly sedated patient starts to move around a lot, and act out of character.

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You suspect the reason for this is:

1. Allergic reaction to the benzodiazepine
2. Patient is experiencing hypoglycemia
3. Patient has undiagnosed bipolar disorder
4. None of the above

Case 9

22 y.o. female presents to have endodontic treatment and remarks of having a very high level of fear of dentists. She doesn't list any medications, but does list allergies to codeine and tramadol. She says ibuprofen irritates her stomach, and Tylenol doesn't work for her. Her last medical evaluation was two months ago. When asked specifically about any recreational drug use, she admits to being a recovering addict, and is currently taking suboxone. Her baseline vital signs are HR – 75, BP – 118/20, SpO2 98%.

What are your concerns with this patient?

1. Trust whether she is in recovery
2. High level of fear
3. Hyporesponse
4. All of the above

Your medical consult does indeed reveal her to be in recovery, with regular visits to confirm she is not using unprescribed drugs.

You do choose to sedate this patient.

Which of the following should be part of your pre-sedation appointment instructions for her?

1. Take 0.25mg of triazolam loading dose
2. Take suboxone as usual
3. 10mg of diazepam the night before
4. Discuss the potential for relapse from recovery

True or False. This patient <u>should not</u> be administered fentanyl as part of her sedation medication regimen:

1. True
2. False

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Case 10

A highly anxious 42 y.o. patient presents for extraction of remaining teeth with sedation. She mentions chronic bronchitis for the last eight years. She uses oxygen at night, and takes Norvasc, ipratropium, albuterol, and Lasix.
She smokes 1.5 packs of cigarettes/day X 24 years. Baseline HR - 86/min., BP - 118/70, O_2Sat - 91%.

Based on this information you should:

1. Treat the patient using an oral/IV combination protocol, utilizing 100% supplemental O2
2. Consult with the patient's physician regarding control of the respiratory disease
3. Treat the patient only if her FEV1% is 60% or higher
4. Refer the patient

You decide to treat the patient. Shortly after the IV is inserted and a total of 3mg of midazolam, and 50mcg of fentanyl is titrated to effect, the patient's O2 Sat numbers drop into the 80s. The patient is arousable, but fails to resaturate after opening the airway, checking the equipment, taking several deep breaths, and using supplemental oxygen.

What do you do now?

1. Panic
2. Reverse the patient
3. Call for help
4. Use positive pressure oxygen

Reversal resaturates the patient. The patient is not comfortable enough to continue without more sedation medication.

Which of the following is the appropriate course of action at this point?

1. Administer more medication
2. Dismiss the patient as soon as she satisfies dismissal criteria
3. Do not administer more medication and observe her for one hour
4. None of the above

Case 11

A 37-year-old male presents for cosmetic dentistry and requests sedation. He reports recently diagnosed HIV, with a normal T-Cell count. He is currently taking no meds. His baseline vitals are: HR – 88, O2 Sat – 98%, and BP – 120/78. Your medical consult reveals no contraindications to dental care with sedation.

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The night before the sedation appointment, the patient takes 5mg diazepam. On the day of the appointment he is administered 0.5mg of triazolam sublingual upon initial assessment.

Soon after the dose of triazolam, your patient informs you that he forgot to tell you that he has recently begun a regimen of Atripla. Your Lexi-Comp drug interaction analysis reveals an [X] interaction with triazolam.

What should you do?

1. Discontinue the appointment and send the patient home
2. Reverse the patient
3. Closely monitor the patient and be prepared to reverse if signs of oversedation
4. Administer O2 and call 911

Case 12

A 42-year-old female patient presents for sedation dentistry. After a comprehensive evaluation, you determine she is an acceptable candidate for oral conscious sedation in your practice. A pre-sedation work-up is performed and the patient is scheduled for a 3-hour sedation appointment.

30 minutes prior to the patient's visit, as you are reviewing the patient's records, you notice two lines on the patient's consent form that have not been initialed by the patient. The patient has been instructed to take 5mg of diazepam the night before, and 0.25mg of triazolam an hour before the appointment.

At this point, what should you do?

1. Proceed with the appointment as planned
2. Have the patients initial once they arrive
3. Have the responsible companion initial for the patient
4. Reschedule the appointment

Case 13

A 39-year-old female presents with moderate dental anxiety desiring to be sedated for dental implants. She claims no medical conditions, with her last medical evaluation six months ago. She says she does take something for her "nerves". Her BP is 118/80, HR – 90, SpO2 – 98%. Her medications include Abilify.

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Your research shows that Abilify is an atypical antipsychotic agent used for acute and maintenance treatment of schizophrenia, treatment of bipolar disorder, adjunctive treatment of major depressive disorder, and treatment of irritability associated with autistic disorder.

How should you proceed with this patient?

1. Have her not take the Abilify on the day of the appointment
2. Determine the reason she is taking the Abilify before sedating her
3. Since the Abilify is a [C] interaction, it is a non-issue

Case 14

A 45 y.o. man presents to be sedated for extensive dental treatment. He is taking Lexapro and Wellbutrin for depression. He has a past history of hepatitis B. He claims to be fully recovered but says he has to be careful taking some medications. He has been told he should never take Tylenol. He sees his physician every year, and a gastroenterologist every six months. His baseline vitals are: BP – 155/94, HR - 67, SpO2 – 96%.

What are your concerns with regard to sedating this patient?

1. Whether this patient is a carrier of hepatitis B
2. The patient's depression
3. The patients' compromised liver function
4. 1 & 3
5. All of the above

Your medical consult reveals that the patient is not a carrier but his ALT and AST are elevated, although not in the severe range. The physician "clears" the patient for sedation.

What should you do?

1. Treat the patient, eliminating the night before diazepam, using oral lorazepam on the day of the appointment, and carefully titrating the patient with IV lorazepam
2. Refer the patient
3. Since the physician cleared the patient, treat them as usual without any modifications

After the patient is successfully sedated, he calls you the next day complaining of pain and swelling in the area of the IV. You recommend warm compresses, and recommend an NSAID. The patient calls you the next day complaining of worsening redness and pain.

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What is the likely diagnosis, and recommended treatment?

1. Venospasm – reassure patient that discomfort and swelling will subside in 2-3 days
2. Extravascular Drug Administration – NSAIDS, antibiotics, and if no improvement refer to a vascular surgeon
3. Thrombophlebitis – elevate and limit activity of limb, moist heat, and NSAIDS

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The IV Sedation Facility Inspection

Every dentist desiring to perform IV sedation must be accredited by his/her state or provincial regulatory agency. This accreditation involves satisfying education requirements, both didactic and clinical, an application process, and in most cases, a facility inspection (that may include an on-site competency exam). Dentists that have completed post-doctoral specialty training may be exempt from some of these requirements.

This module is designed to assist the sedation team in preparing for and successfully completing the IV sedation facility inspection. However, the responsibility lies with the dentist applying for IV sedation accreditation to discover and satisfy the requirements for accreditation. There may be considerable variance in what each regulatory agency requires. This information can be determined by contacted the appropriate regulatory agency either directly or online at that agency's official website.

Whatever the regulatory agency requires, before scheduling the appropriate visit with representatives of that agency, the IV sedation team should be prepared with all of the required supplies, equipment, any facility modifications, knowledgeable in addressing questions from inspectors, and if necessary, ready to demonstrate practical competency in the IV sedation clinical procedure.

While this module is designed to prepare the dentist for the IV sedation facility inspection, every possible contingency involving the inspection process cannot be anticipated. Often the investigator(s) can be contacted directly for insights into how best to prepare for their visit. It is also highly recommended that the IV sedation team seek out peers who have been through the process to assist in preparation.

The facility inspection will be described in two parts – The facility checklist, and the on-site provider examination.

Facility Checklist

The supply and equipment checklist can be obtained in advance, making satisfaction of this requirement straightforward. Many of the items are obvious, and are covered as well as used as part of the IV sedation course. Other items may not be a part of the routine IV sedation procedure, and are necessary only for infrequent, if ever, use. An example, along with explanations of a facility checklist follows:

Operatory and Recovery Room

- **Minimum size of operatory room** – *This is for emergency personnel and equipment access. One facility checklist lists the minimum dimensions of 10 feet x 8 feet, or 80 square feet*
- **Minimum size door or opening to and from operatory room** – *Egress for gurney to enter and leave operatory. One facility checklist lists the minimum*

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dimensions of 35 inches net, or graphic documentation that an EMS gurney can be brought into the room

- **Minimum size of recovery room if present** – *Not all regulatory agencies require a separate recovery room, especially for conscious sedation. One facility checklist lists the minimum dimensions as 8 feet x 4 feet or 32 square feet, with a minimum door or egress from recovery room of 36 inches net, or evidence that EMS gurney can be brought into the room*
- **Minimum hallway from operatory room to exit** – *This is for emergency personnel and equipment egress. One checklist lists this as 42 inches in width*

Equipment

- **Oxygen systems: Primary with positive pressure** – *This source can be the oxygen that is supplied through a central or portable oxygen tank that may or may not be part of a combination oxygen/nitrous oxide delivery system. Attached to the oxygen delivery apparatus is a positive pressure device such as bag valve mask or a demand valve*
- **Oxygen systems: Secondary portable oxygen** – *This is a second source separate from the primary one, and is intended for use when the primary system is either depleted or unavailable to the site of the emergency need. It is a dedicated portable oxygen tank with regulator intended for use in emergency scenarios. Just like with the primary system, the secondary one can be attached to a positive pressure delivery device*
- **Suction system: Primary** – *This is the suction apparatus connected to the office high speed central evacuation unit*
- **Suction system: Secondary portable (non-electric, unless back-up generator available)** – *This backup suction unit is intended for an electrical outage, so it may not be necessary if the clinic has a back-up generator that can supply electricity to the suction unit. A manual suction unit, akin to a "turkey-baster" with a squeezable bulb can also suffice and has the added advantage of being usable away from the vicinity of the operatory suction device*
- **Operating light: Primary** – *This is the operatory light source normally used for illumination of the area being treated*
- **Operating light: Secondary surgical lighting or portable non-electric** – *Since the primary source is often powered by the facility's electrical system, a secondary source not dependent on this system, such as a batter-operated light is necessary during an electrical outage or when light is needed away from the vicinity of the operatory light source*
- **Operating chair/table with flat position** – *Best access to the airway, as well as the chest for emergency assistance is provided with the patient in a supine position. If the operatory treatment chair can be adjusted to this position, then it will satisfy this requirement. If it cannot, then another device (chair or table) that the patient can be moved to is needed*
- **Defibrillator or AED** – *A defibrillator is used to deliver a shock to restart the heart's electrical conductive system when interpretation by the clinician of the*

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patient's EKG deems it appropriate. The AED serves as both a device to interpret the heart rhythm and when appropriate deliver a shock.

Monitoring & Emergency Equipment

- **Stethoscope** – *for auscultation of heart and breathing sounds. The inspection may require a manual blood pressure measuring mechanism, or may allow for a automated device*
- **Sphygmomanometer** – *together with an inflatable cuff and gauge that measures pressure in mmHg, collapses the blood flow under the cuff, then determines pressure at which blood flow begins again (systolic), and eventually flows unimpeded (diastolic). The inspection may require a manual blood pressure measuring mechanism, or may allow for an automated device*
- **Pulse oximeter** – *for measuring heart rate and oxygen saturation. Devices can also measure other physiologic parameters such as blood pressure, respiratory rate, body temperature, end-tidal CO_2, and heart rhythm. The office being inspected should inquire as to the monitoring requirements for IV sedation by the governing agency.*
- **Oral airway –small, medium, large** – *An oropharyngeal airway (OPA) is designed to assist the emergency delivery of room air or 100% oxygen to an unconscious patient who is not breathing or not breathing adequately. This is accomplish by the OPA preventing the tongue and other soft tissue of the oropharynx from obstructing the upper portion of the airway. The proper size OPA is one in which the length extends extraorally from the corner of the mouth to the ear lobe of the ear*
- **Face mask –small, medium, large** – *Designed to fit over the patient's mouth and nose so that the patient can be ventilated with positive-pressure air or 100% oxygen. The correct size mask extends from the bridge of the nose to between the lower lip and inferior border of the chin, and provides a seal of the soft tissues so that gas doesn't leak when the mask is pressed against the face. The apparatus can also contain an attachment for measurement of expired CO_2*
- **Semi-advanced airway (LMA, Combi-Tube, King LT)** – *These devices can make ventilation more effective than a simpler airway device such as an OPA or nasopharyngeal airway (NPA), and may make regurgitation and aspiration of vomitus less likely. In addition, they require less training and expertise to place than an advanced airway device such as an endotracheal tube*
- A**dvanced airway** – *The advanced airway extends through the vocal cords and into the trachea, providing the most effective means by which to breathe for the unconscious patient who is not breathing. Placement requires training, and competency is attained through experience in placement. Therefore, its practical use by dental personnel may be limited. However, if required for the facility inspection, the sedation team should have the necessary equipment (laryngoscope, blades, endotracheal tubing), and a working knowledge of their application*

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IV access equipment

- **IV needle** – *For venipuncture access. The facility being inspected should verify any requirements such as any specific requirements (eg. needle/catheter combination, retractable needle, etc.,.)*
- **IV tubing and fluids** – *For continuous drip, administration of medications, and fluid replacement. The inspectors may have specific requirements as to the fluid constitution*

Emergency Drugs

- **Aerosol bronchodilator** – *For smooth muscle dilation of bronchial passages via beta2-adrenergic agonism. A* ***couple*** *of inhaled "****puffs****" of* ***Albuterol*** *self-administered is a common example*
- **Anticonvulsant** – *A long-acting benzodiazepine such as* ***Diazepam*** *injected IV at a dose of* ***10mg*** *is commonly recommended for this purpose*
- **Antihistamine** – *For treatment of mild allergic reaction, or as an adjunct to epinephrine in the treatment of anaphylaxis. An example is* ***25-50mg of diphenhydramine*** *injected IM or IV*
- **Antihypertensive** – ***Nitroglycerine*** *can be used for this purpose, because it results in systemic vasodilation, lowering blood pressure almost instantaneously. It can be administered by* ***tablet (0.6mg) sublingually every 5 minutes, up to 3 tablets in 15 minutes,*** *or by 1 to 2 translingual sprays (0.4mg/spray), every 5 minutes for up to 15 minutes. Nitroglycerine should not be used in patients taking drugs for erectile dysfunction*
- **Platelet inhibitor** – ***Chewing 325mg of aspirin*** *when a patient exhibits signs and symptoms of a myocardial infarction may lessen the impact of the event. Through aspirin's ability to inhibit platelet aggregation, it can prevent a clot from occluding a coronary artery. Chewing the aspirin results in better absorption through the oral cavity rather than absorption through the GI tract*
- **Vagal inhibitor** –*IV administration of* ***0.5mg of atropine****, treats symptomatic bradycardia by blocking the action of the vagus nerve on the heart resulting in increased heart rate*
- **Blood glucose elevating agent** –***3cc of IV 50% dextrose solution per minute up to 25 grams*** *is appropriate for the treatment of symptomatic hypoglycemia*
- **Sympathomimetic agent** – ***Epinephrine*** *through its alpha and beta agonism is indicated for* ***cardiac arrest at doses of 1mg every 3-5 minutes****, and* ***anaphylactic shock and bronchospasm not responsive to aerosol bronchodilators at a dose of 0.3-0.5mg***
  For different concentrations, use the following volumes for cardiac arrest (the correct amount may come in a pre-loaded syringe):

  1:1000 concentration (1mg/cc - this is 1 ampule) - 1 cc
  1:10,000 concentration (0.1 mg/cc) – 10 cc

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For different concentrations, use the following volumes for anaphylactic shock and bronchodilation (the correct amount may come in a pre-loaded syringe):

1:1000 concentration (1mg/cc - this is 1 ampule) - 0.3-0.5 cc
1:10,000 concentration (0.1 mg/cc) – 3-5 cc
1:20,000 concentration (0.05mg/cc) – 6-12 cc

- **Benzodiazepine antagonist** – ***Flumazenil (10 ml)*** *in a concentration of 0.1mg/cc given in* ***a dose of 0.2mg*** *to treat benzodiazepine overdose*
- **Narcotic antagonist** - ***Naloxone (1cc)*** *in a concentration of 0.4mg/cc given in a* ***dose of 0.4mg*** *to treat narcotic overdose*
- **Vasodilator** – For relief of chest pain, ***Nitroglycerine****, an effective vasodilator can increase oxygen to heart tissue. It can be administered by* ***tablet (0.6mg) sublingually every 5 minutes, up to 3 tablets in 15 minutes,*** *or by 1 to 2 translingual sprays (0.4mg/spray), every 5 minutes for up to 15 minutes. Nitroglycerine should not be used in patients taking drugs for erectile dysfunction*
- **Vasopressor** – *For the patient with symptomatic hypotension,* ***0.5-2 cc*** *(5-20 mcg, or 0.005 – 0.02 mg) of* ***epinephrine 1:10,000 every 1-5 minutes*** *will cause vasoconstriction of peripheral blood vessels, raising blood pressure. For different concentrations, use the following volumes (the correct amount may come in a pre-loaded syringe):*

  1:1000 concentration (1mg/cc - this is 1 ampule) - 0.05-0.2 cc
  1:10,000 concentration (0.1 mg/cc) – 0.5-2 cc
  1:20,000 concentration (0.05mg/cc) – 1-4 cc

Records

- **Patient medical history form** – An Example:

Health History

Patient Name (First, Middle, Last) _____

Date of Birth _____ Date of last **medical** exam:_____

What was this exam for? _____

Date of last **dental** exam: _____Have you been hospitalized in the last 5 years?

Yes _____ No _____ If yes, reason for hospitalization: _____

Are you currently receiving care? Yes _____ No _____ If yes, nature of care: _____

_____

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Please list the names/phone numbers of the physicians who are currently providing you care:

1.) _____ Phone #: _____

2.) _____ Phone #: _____

3.) _____ Phone #: _____

Do you have or have you ever had any of the following medical conditions? (Please check yes or no)

Migraines	Yes____ No____	Liver Disease (including Jaundice)	Yes____ No____
Epilepsy	Yes____ No____	Hepatitis, any form	Yes____ No____
Glaucoma	Yes____ No____	Thyroid Condition	Yes____ No____
Asthma	Yes____ No____	Sexually Transmitted Disease	Yes____ No____
Kidney Disease	Yes____ No____	HIV Positive/AIDS Related Complex	Yes____ No____
Snoring or Sleep Apnea	Yes____ No____	Do you use a CPAP?	Yes____ No____
Previous Biopsies	Yes____ No____	Cancer	Yes____ No____
Recurrent Illnesses	Yes____ No____	COPD/Respiratory Illnesses	Yes____ No____
Heart Murmur	Yes____ No____	Abnormal Heart Condition	Yes____ No____
Anemia	Yes____ No____	Abnormal bleeding from a cut	Yes____ No____
Arthritis	Yes____ No____	Sore/Enlarged Lymph Nodes	Yes____ No____
Dry Mouth	Yes____ No____	Slow healing mouth sores	Yes____ No____
Abnormal Blood Pressure	Yes____ No____	High/Low, What is it usually?	S_____/D_____
Diabetes	Yes____ No____	HgA1C: _____	Date last checked_____
Joint Replacement	Yes____ No____	Area and date of replacement:_____	

Other Infections _____

Do you have a medical condition that requires you to pre-medicate with antibiotics before dental treatment?

Yes____ No____ Reason: _____

Are you allergic to:

Tylenol/Acetaminophen Yes____ No____ Advil/Ibuprofen Yes____ No____ Sulfa Yes____ No____

Latex Yes____ No____ Penicillin Yes____ No____ Codeine Yes____ No____

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Please list other allergies (include drugs/medications, foods, seasonal, etc):

_____

Are you allergic to or have you ever had any unusual reactions to local anesthetic?

Yes_____ No_____ Explain: _____

Pharmacy Name: _____ Phone: _____

Please list any medications you are currently taking:

1.) _____ 6.) _____
2.) _____ 7.) _____
3.) _____ 8.) _____
4.) _____ 9.) _____
5.) _____ 10.) _____

Are you taking any Antacids? Yes ____ No ____

Are you taking Tagamet/Cimetidine? Yes ____ No ____If yes, how often? _____

Are you taking any herbal supplements/medications? Yes _____ No _____

If yes, which ones? _____

Are you a smoker? Yes ____ No ____ If yes, how much per day? _____ How long? _____

Women: Are you pregnant? Yes ____ No ____

Are you planning a pregnancy in the next 6 months? Yes ____ No ____

Are you nursing? Yes ____ No ____

_____ _____ _____

Patient Name (Please print) Patient/Guardian Signature Date

- **Patient anesthesia record** – *See Section 22 "IV Preparation, Dispensing, and Record Keeping" of this manual for this record-keeping form*
- **Office narcotic and scheduled drug recorded** - *See Section 22 "IV Preparation, Dispensing, and Record Keeping" of this manual for this record-keeping form*

Personnel

The office must keep the names of all current personnel, including the doctor(s) who may treat or assist with the IV sedation procedure, and a record of their CPR/BLS/ACLS training status (certification and expiration dates).

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On-Site Provider Examination

When the inspection procedure involves a competency demonstration, it can involve an oral exam, a venipuncture demonstration, a complete IV sedation procedure, or a combination of the above. This is where a consultation with other clinicians who have been through this experience within the same state/province in the recent past comes in handy. In addition, a request to one or more of the inspectors prior to their visit as to what information they may want the applying office to be prepared for is prudent.

The oral exam usually focuses on pharmacology, monitoring, or preventing and addressing emergency situations. The applying dentist should know the indications and dosages for all of the emergency drugs on the inspection checklist, as well as the dosages of all sedation medications along with their contraindications. Finally, the clinician should be able to interpret the meaning and interpretation of, as well as the response to aberrations in vital signs during IV sedation.

If part of the on-site inspection involves patient treatment, then proper patient selection can make for a less stressful, more successful outcome. Someone who has proven to be reliable, healthy, not too anxious, and has ample veins would be the best choice. If the clinician can practice the venipuncture procedure on the patient in advance, in order for both the patient and the sedation team to feel more confident.

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