INTRAVENOUS SEDATION IN DENTISTRY
6th Edition
Editor: Paul Templer

Continuing Education Course Manual

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INTRAVENOUS SEDATION

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Preface to the sixth edition:

The purpose of this manual is to provide the trainee in sedation with an overview of sedation in dentistry. It is complementary to the series of lectures and workshops. All aspects of sedation will not be covered and instead the focus will be on the use of intravenous midazolam as this is the initial safe technique we are aiming to teach.

More than ever patient safety is our paramount concern and we make no apologies for stressing this fact. Remember to always be safe – for your own and patients' sake – when practicing this valuable skill. Careful reading of the text will reveal repetition of material in several sections – this is usually because it is important.

I finish by quoting my predecessor and founding editor John Sinclair:

Skills in intravenous sedation techniques for dentistry are both an art and a science. Only experience can equip a dental sedationist with the knowledge to assess when this technique can be effectively used. Obtaining patient acceptance for this is an alternative to a general anaesthetic requires time and a great deal of tact, but the results are usually outstanding.

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Dunedin Hospital March 2019.

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NEW ZEALAND DENTAL ASSOCIATION

And

NEW ZEALAND DENTAL COUNCIL

Code of Practice
Conscious Sedation for Dental Procedures

1. INTRODUCTION

Sedation for dental procedures (with or without local anaesthesia) includes the administration by any route or technique of all drugs, which result in depression of the central nervous system. The objective of these techniques is to produce a degree of sedation of the patient, **without loss of consciousness**, so that uncomfortable procedures may be facilitated. The drugs and techniques used should provide a margin of safety, which is wide enough to render loss of consciousness unlikely. Loss of consciousness due to sedation constitutes general anaesthesia and carries specific risks. These guidelines are not intended for very light techniques such as nitrous oxide/oxygen mediated sedation.

These techniques are not without risk because of the:

Potential for unintentional loss of consciousness.

Depression of protective reflexes.

Depression of respiration.

Depression of the cardiovascular system.

Wide variety and combinations of drugs which may be used, with the potential for drug interactions.

Possibility of excessive amounts of these drugs being used to compensate for inadequate analgesia.

Individual variations in response to the drugs used, particularly in children, the elderly and those with pre-existing medical disease.

Wide variety of procedures performed.

Differing standards of equipment and staffing at the locations where these procedures may be performed.

It is important to recognize the variability of effect that may occur with sedative drugs, however administered, and that over-sedation, airway obstruction or cardiovascular complications may occur at any time.

1. GENERAL PRINCIPLES

The patient should be assessed before the procedure and this assessment should include:

1. A concise medical history, examination (including blood pressure measurement), performance of appropriate investigations and identification of risk factors. The American Society of Anesthesiologists' classification system is convenient for this purpose. (See Appendix. 1)

2. Informed consent for sedation as well as the planned procedure.

3. Written instructions for preparation for the procedure (**including the importance of fasting**), the recovery period, and discharge of the patient (including avoidance of driving, other dangerous activities, undertaking responsible business).

If the patient has any serious medical condition then the appropriate treating general medical practitioner and/or their specialist should be consulted prior to any planned treatment under sedation. If the patient is deemed to be seriously medically compromised then an anaesthetist should be present to administer sedation and to monitor the patient during the procedure.

The practitioner administering sedation requires sufficient knowledge to be able to:

4. Understand the actions of the drug or drugs being administered.

5. Detect and manage appropriately any complications arising from these actions. **In particular medical and dental practitioners administering sedation must be skilled in airway management and cardiovascular resuscitation.**

6. Anticipate and manage appropriately the modification of sedative drug actions by any concurrent therapeutic regimen or disease process, which may be present.

Techniques intended to produce loss of consciousness must not be used unless an anaesthetist is present.

A written record of the dosages of drugs and the timing of their administration must be kept as a part of the patient's records. Such entries should be made as near the time of administration of the drugs as possible. This record should also note the regular readings from the monitored variables.

Techniques, which compensate for inadequate local analgesia by means of heavy sedation, must not be used unless an anaesthetist is present.

3. STAFFING

If an appropriately trained medical or dental practitioner is not present to solely administer sedation and monitor the patient then there must be an assistant present during the procedure, appropriately trained in observation and monitoring of sedated patients, and in resuscitation whose duty shall be to assist the operating practitioner and monitor the level of consciousness and cardio-respiratory function of the patient.

The operator may provide the sedation and be responsible for care of the patient provided that the patient response to verbal commands or stimulation is continuously possible during the procedure.

If at any time spontaneous respiration and/or protective reflexes are lost, or the patient does not respond to verbal commands or stimulation, both the proceduralist and assistant must devote their entire attention to monitoring and treating the patient until recovery, or until such time as another medical or dental practitioner becomes available to take responsibility for the patient's care.

If general anaesthesia or loss of consciousness is sought for the procedure, then an anaesthetist must be present to care exclusively for the patient.

2. FACILITIES

The procedure must be performed in a location which is adequate in size and staffed and equipped to deal with a cardiopulmonary emergency. This must include:

An operating table, trolley or chair which can be readily tilted.

Adequate uncluttered floor space to perform resuscitation.

Adequate suction and room lighting including alternative means of providing suction and light in the event of a power failure.

A supply of oxygen and suitable devised for the administration of oxygen to a spontaneously breathing patient.

A self-inflating bag suitable for artificial ventilation together with a range of equipment for advanced airway management.

Appropriate drugs for cardiopulmonary resuscitation and a range of intravenous equipment. (See Appendix II.)

A pulse oximeter.

3. MONITORING

All patients undergoing intravenous sedation must be monitored continuously with pulse oximetry and this equipment must alarm when certain set limits are exceeded. There must be regular recording of pulse rate, oxygen saturation, and blood pressure.
Capnography must also be used According to the clinical status of the patient, other monitors such as ECG may be required.

4. OXYGENATION

Degrees of hypoxaemia occur frequently during intravenous sedation without oxygen supplementation. Oxygen administration diminishes hypoxaemia during procedures carried out under sedation and should be routinely available.

Pulse oximetry enables the degree of tissue oxygenation to be monitored and must be used on all patients during sedation.

5. TRAINING IN SEDATION FOR DENTAL PROCEDURES

An appropriately trained medical or dental practitioner should be present and be responsible for administration of sedation. The clinician is to be one of the following:

A dentist who has successfully completed relevant postgraduate training leading to an accredited qualification accepted by the relevant Health Authority.

A specialist anaesthetist.

6. DISCHARGE

The patient should be discharged only after an appropriate period of recovery and observation in the procedure room, or in an adjacent area, which is adequately equipped and staffed. Oxygen must be available in any area used for patient recovery.

The practitioner who administered the drugs, or another appropriately qualified practitioner should authorize discharge of the patient. The patient should be discharged into the care of a responsible adult to whom written instructions should be given. Transport should normally be by car.

Adequate staffing and facilities must be available in the Recovery Area for managing patients who have become unconscious or who have suffered some medical mishap. Should the need arise; the patient must be transferred to appropriate medical care.

APPENDIX I

The American Society of Anesthesiologists' classification of physical status:

Class I A normal healthy patient

Class II A patient with mild systemic disease

Class III A patient with a severe systemic disease that limits activity but is not incapacitating

Class IV A patient with an incapacitating systemic disease that is a constant threat to life

Class V A moribund patient not expected to survive 24 hours

APPENDIX II

Emergency drugs should include at least the following:

Adrenaline
Atropine
Dextrose 50%
Lignocaine
Naloxone (if Narcotic analgesics used)
Flumazenil
Portable emergency oxygen supply

GUIDELINES *– defined as 'a document offering advice'. These may be clinical (in which case they will eventually be evidence-based), or non-clinical.*

This document has been prepared having regard to general circumstances, and it is the responsibility of the practitioner to have express regard to the particular circumstances of each case, and the application of this document in each case.

Professional documents are reviewed from time to time, and it is the responsibility of the practitioner to ensure that the practitioner has obtained the current version. Professional documents have been prepared having regard to the information available at the time of their preparation, and the practitioner should therefore have regard to any information, research or material which may have been published or become available subsequently.

BEHAVIOURAL CONSIDERATIONS

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Dr P.A. Foreman's views:

Although great technical advances have been achieved in dentistry during recent years, the problems of anxiety and pain must still be overcome if the benefit of these advances is to be brought to all. Whether we as dentists accept it or not, the plain fact is that countless patients the world over still cannot bring themselves to seek regular dental treatment due to fear. In no other branch of the healing profession is pain, anxiety, and apprehension tolerated as it is in dentistry. As a consequence, many patients wait until their teeth are completely beyond repair, finally obtaining relief by the removal of all their teeth under general anaesthesia as the only "solution" to their plight. Others submit to regular dental treatment, but admit that visits to the dentist are an ordeal. Many dentists tend to discount these views. They are inclined to adopt the attitude that the patient is abnormal who complains of a persistent dislike of dental treatment. In short, they often will not accept the fact that there really is a problem.

My own experience confirms the opinion that it is fear which causes patients to avoid dental treatment, as a majority of patients attending my own practice for extensive restorative treatment under sedation have previously received only emergency treatment for the relief of pain. In fact, the average time since these patients last received dental treatment of any sort is almost four years. Most state that if an alternative to "conventional" methods of dentistry had not been available, they would have eventually had their remaining teeth extracted under general anaesthesia and replaced by full dentures – not through choice – but as the only way out of their dilemma. In the United States, the American Dental Association Council on Dental Therapeutics estimated in 1972 that 50% of the population was not receiving regular or complete dental care, mainly as a result of fear.

Some members of the dental profession still believe that if the public were able to receive Government financial assistance for dental treatment, their dental health would improve substantially. Unfortunately, in countries such as the United Kingdom, where the economic barrier to dental treatment has been largely removed by the State, this has not proved to be the case. No doubt this has been contributed to by intolerable third party interference and the enforcement of a rigid, non-preventively orientated government scale of fees which penalizes the conscientious dentist and rewards the one who is prepared to compromise quality for quantity. But the fact also remains that in the U.K. only 38% of the population avail themselves of treatment under the National Health Service, strongly suggesting that fear, not lack of funds, keeps may people away.

Adapted from Foreman, P.A., Behavioural Considerations in Patient Management, Anesthesia Progress 26:161 (Nov. – Dec.) 1979.

Introduction:

Since the advent of effective local anaesthetic drugs, it has been possible to virtually eliminate pain in dentistry, but the problem of anxiety still remains. Although the frightened patient often realises they are unlikely to suffer pain, their anxiety may be such that the response to even trivial stimuli is greatly exaggerated, leading to a stressful situation for all concerned, and difficulty in maintaining high standards of treatment. Ideally, complete psychological investigation of every apprehensive dental patient should be undertaken, and the reasons underlying the anxieties concerning the dental situation discovered and dealt with. However, due to an increasing population and shortage of qualified manpower, this is difficult within the limitations of a busy practice. Thus, a pharmacological means of circumventing the patient's anxiety may justifiably be considered, at least as a means of achieving initial dental fitness and cooperation. Once these have been achieved, patients are often more responsive to other approaches.

Until the 1960's, the problem of the fearful, apprehensive dental patient was partially "solved" by treatment under general anaesthesia. However, this had obvious disadvantages, including the risk of anaesthetic complications such as delayed recovery, nausea and vomiting, and succinylcholine pains; none of which endeared the method to the busy practitioner as a means of treating ambulatory patients. As well as these, the dangers of subjecting anxious high-risk patients to the emotional and physiological stresses of prolonged general anaesthesia need no elaboration.

The inevitable result was that over the years, many patients sought only emergency treatment for pain, eventually becoming edentulous through neglect of their teeth due to fear of dentistry. Obviously, what was needed was a means of bridging the gap between local and general anesthesia, so that dental treatment could be brought to nervous patients and others with the advantages, but without the risks and disadvantages, of general anaesthesia.

Since the 1960's, there has been growing acceptance of the use of inhalation and intravenous drugs for sedation in dentistry, particularly restorative dentistry, for very nervous patients who, because of fear, might otherwise receive less than adequate care; and also for those patients whose time is valuable and who prefer to complete their treatment in as few visits as possible. This interest was largely stimulated by the revival of nitrous oxide in dentistry as a sedative instead of as an anaesthetic, the introduction of inhalation agents such as methoxyflurane (Penthrane), and increasing use of the ultra short-acting intravenous anaesthetic methohexital sodium (Brietal Sodium, Brevital Sodium) and the non-barbiturate anaesthetics propanidid (Epontol), Althesin, Propofol, and others. Other techniques include the use of tranquillosedative agents, of which the most useful to date has been diazepam (Valium) and midazolam (Hypnovel, Versed) and the use of various drug combinations to produce sedation, as in the Loma Linda (Jorgensen) technique; or basal sedation on which small doses of methohexital or nitrous oxide are superimposed to deepen the level of sedation temporarily as required. (Berns, Shane, Foreman/Neels techniques).

The use of sedation can make dentistry very much more pleasant for both the patient and the dentist. It can also save the patient both time and money, and at the same time permit the dentist to work more efficiently and often to a higher standard; particularly when treating anxious patients. Also, more work can often be completed in one or two long appointments than in a series of short appointments. Our traditional “piecemeal” approach to dentistry is illogical and annoying to many patients, most of who would greatly prefer to complete their treatment in as few visits as possible.

Pharmacological techniques should always be considered as aids to treatment, not as a crutch or substitute for a good doctor/patient relationship. There is no doubt that the effectiveness of sedation can be greatly improved by the interpersonal relationship and degree of trust engendered between the doctor and his patient. Also important is an understanding of the factors that affect pain tolerance, and how we can supplement the use of drugs by various anxiety reduction techniques which will enable us to obtain the best of both worlds.

It is difficult to overemphasize the importance of managing the psychological as well as the pharmacological aspects of treatment. In order to manage patients effectively, we must consider many inter-related factors, and not look to any one particular modality – whether it is psychological or pharmacological – to solve all of our problems. Human beings, particularly anxious dental patients, are complex and their management is not always simple. Although increasing numbers of dentists employ various pharmacological techniques in the treatment of patients, it is essential to recognize that an understanding of behavioural factors is directly related to the success or failure of these techniques.

Major Dental Fears

What are dental patients afraid of? Next to the fear of pain, the most common fear is that of the local anaesthetic injection – which, paradoxically, is dentistry's greatest boon because it permits painless treatment. The sight and sensation of the local anaesthetic injection and its possible side and after-effects are closely followed by the sight, sound, and sensation of the drill as the most fear producing stimuli. Other fears include fear of the unknown; the surrender of control, as well as becoming immobilized and in a state of helplessness. (Unless absolute trust is present, certain personality types may respond to this situation with hostility and aggression). There is also a the fear of surgery and its attendant effects such as mutilation, side effects such as the loss of blood, the fear of general anaesthesia, and the fear of the after-effects of surgery. Other fears include tooth loss; representing aging, loss of attractiveness and sexuality, and degeneration of the body; as well as the fear of full dentures and their problems.

Finally, there is the fear of the dental operatory and its contents. Indeed, countless patients are simply afraid of the entire dental environment – its appearance, its smells, and its people.

What can be done to improve this situation? Firstly, it is important to recognize that although the threshold for recognition of pain (or pain perception) is approximately the same from one person to another; the degree to which each one reacts to pain (pain tolerance) varies tremendously

Factors Affecting Pain Tolerance

It is important to understand some of the factors which affect pain tolerance. Obviously, personal history is extremely important. Adult behaviour is often influenced by early experiences, prior traumatic dental experiences as a child (or even as an adult) having an important bearing on subsequent behaviour in the dental treatment situation. Vicarious sources such as family and friends' experiences and attitudes will also have a marked effect on the patient's attitude toward dentistry. So fear of dentistry is really a learned form of behaviour, as a result of direct or indirect experiences.

Cultural background can also affect pain tolerance. For example some cultures such as the Japanese, Chinese, and American Indians, tend to value stoicism and will be much less likely to express or complain about their discomfort. On the other hand some cultures are more demonstrative in expressing their pain.
Personality type is significant, with extroverts tending to be more tolerant of pain than introverts. The neurotic, introverted type with unstable ego defenses is often intolerant of even slight pain. Personality type is important when selecting a pharmacological technique. For example, nitrous oxide sedation is very much a "participation technique" and is more suited to the patient who likes to feel in control of situations. On the other hand, intravenous sedation is probably better suited to the more passive type of individual, as well as for those willing to entrust control to another. In both situations, trust in the operator is essential.

Age is a factor; younger people have less experience and so generally have a lower threshold for pain they regard as intolerable. Older people are generally more tolerant of pain however if they have a history of poor dental experiences they may become more apprehensive with time.

Gender differences are an interesting and controversial area. Studies produce conflicting results. Men show greater tolerance to pain when in a group – particularly if women are present. Women show a variation in sensitivity to pain related to the menstrual cycle with greatest sensitivity pre-menstrual. Several studies have shown women who have had children are more tolerant to pain generally but other studies have disputed this.

Perhaps the single most important factor of all in pain tolerance is the degree of anxiety present, research having clearly shown that the higher the degree of anxiety, the lower is the tolerance of pain.

Anxiety not only affects the degree of pain tolerance, but it has a marked effect physiologically. The autonomic nervous system is activated to trigger the "fight or flight" response, and the physiological side effects produced result in a vicious circle of increasing anxiety and physiological responses. Major changes that occur are an increase in heart rate and blood pressure, sweating, salivation, shift of blood from the viscera to the muscles, and release of glycogen from the liver in large quantities. Digestion slows or ceases altogether, and contraction of the stomach wall may occur. The latter is a major reason for the unpredictability of oral sedation.

The degree to which these responses occur is in direct proportion to the degree of anxiety present. The dental patient experiencing these changes usually becomes aware of his or her rapid heart beat or palpitations, as well as a feeling of internal pressure. If they were fighting or fleeing, these symptoms would not be noticed, but as this is not possible, the patient becomes aware of them and attempts to control them. However, not realizing that these sensations are normal and not due to some bodily malfunction, they become alarmed and even more anxious, thus intensifying the stimulus and setting up a vicious cycle.

Assessment of Anxiety

The degree of anxiety can be determined in a number of ways. Its physiological effects, as noted above, can be measured using a variety of physiological monitors. These may include measurements of blood pressure and heart rate, which are often elevated in fearful patients. Other, more effective means of measuring anxiety include galvanic skin conductance, electromyography, and plethysmography. This type of instrumentation has been used very successfully in biofeedback, a form of relaxation training which has gained wide acceptance and is also used in chronic pain management.

Whilst we are all familiar with the obviously anxious patient, it is important to be aware of covert behaviours which may also indicate underlying fears. For example, the patient who regularly fails or cancels appointments, attends irregularly, arrives late, requests minimal or emergency treatment; or who acts or talks aggressively. Others may be non-compliant concerning home care, oral hygiene, and post-operative instructions. Most anxiety, both overt and covert, is a result of prior conditioning both classical and operant, as well as through indirect, vicarious sources (modeling).

It is important that we understand the factors which may be contributing to our patients' anxieties, and learn how to deal with them on a one-to-one basis as well as trying to control them with the use of drugs. Often, pharmacology alone can lead to problems of loss of control, over-dosage, and complication – all of which might have been avoided with better doctor/patient relations and trust-building.

Many practices employing this philosophy also find it helpful to use additional aids such as a practice "welcome letter", and an anxiety questionnaire. One of the most useful of these has been that developed by Dr. Norman Corah, Professor of Behavioural Sciences at the New York State University School of Dental Medicine.

Anxiety Questionnaire

We realize that many people are nervous or frightened about going to the dentist. If you have such feelings, we would like to help you. The following information will help us make your dental experience more comfortable. Thank you for your co-operation.

Please indicate the answer that best describes your feelings:

You are going to the dentist today. How do you feel about it?

[ ] It does not bother me
[ ] I am a little nervous
[ ] I am afraid
[ ] I am very frightened
[ ] I am so frightened I sometimes break out in a sweat, or feel sick.

You are waiting in the waiting room, how do you feel about it?

[ ] It does not bother me
[ ] I am a little nervous
[ ] I am afraid
[ ] I am very frightened
[ ] I am so frightened I sometimes break out in a sweat, or feel sick.

You are in the dentist's chair, waiting for him to give you an injection. How do you feel about it?

[ ] It does not bother me
[ ] I am a little nervous
[ ] I am afraid
[ ] I am very frightened
[ ] I am so frightened I sometimes break out in a sweat, or feel sick.

You are in the chair waiting for the dentist to use the drill on your teeth. How do you feel about it?

[ ] It does not bother me
[ ] I am a little nervous
[ ] I am afraid
[ ] I am very frightened
[ ] I am so frightened I sometimes break out in a sweat, or feel sick.

Interview:

Name:
Specific fear:
Origin of fear:

Anxiety Reduction Techniques

What can be done to reduce patient anxiety, thereby increasing pain tolerance and hence the effectiveness of our sedative and anaesthetic techniques?

Dentist/patient relationship:

Probably the most important method of controlling anxiety, and upon which most other techniques are dependent, is for the dentist to establish a good therapeutic relationship with his or her patient. The quality of sedation is also dependent on the degree of rapport and trust the dentist has established with the patient. In order to do this, he or she must have a sound knowledge of the patient and their background, must be prepared to spend time discussing the patient's fears and anxieties, and must show a genuine concern for the patient's welfare and comfort. It is important to empathise with patients, and let them know that you understand how they feel. Equally important is the provision of sufficient time for discussion and patient education during the initial consultation appointment. This is time well spent, as it will result in a patient arriving for his or her treatment in a far more relaxed state of mind than would otherwise be the case. This in turn leads to more effective sedation involving the use of less drugs, more productive dentistry, and increased freedom from side-effects and after-effects.

Once this type of relationship has been established, the patient becomes more cooperative, more trusting, prepared to accept professional advice, and more open to suggestion. Under the circumstances, a maximum response to drugs is also more likely to occur, and the dosages used can be kept to a minimum.

Anticipation of reward:

It has been shown that tolerance of pain is increased when coupled with the prospect of reward. For example, only one-quarter of the men who were severely injured on the Anzio beachhead during World War II requested medication for pain. Many of these may have interpreted their injuries as blessings in disguise. For them the war was now over, and they would soon be sent home. By contrast, in civilian life, some three-quarters of patients recovering from the often less serious wounds of surgery complain of pain and ask for relief. Although these observations should perhaps now be viewed in the light of recent research into the release of endogenous opiates in response to stress, it is still true to say that tolerance for a painful stimulus is increased when stimulus is coupled with the prospect of reward, whatever the physiological mechanisms involved.

In dentistry, for example, verbal encouragement from the dentist before and during treatment as to how the procedure will improve the patient's appearance and enjoyment of food, etc. is important, along with commending the patient for calm behaviour and tolerance during a possibly uncomfortable operation. On the other hand, a patient who continually complains of minor discomfort should not be rewarded with attention and concern.

The act of non-reward will tend to decrease the probability of future complaints, just as the act of reward will tend to increase the tolerance of the patient who is well behaved during an unpleasant procedure. This is known as operant conditioning, where good behaviour is rewarded and poor behaviour is not.

Hypnosis and suggestion:

Hypnosis and, in particular, suggestion can play an important part in dentistry. Although hypnosis is only used by a minority of dentists, the value of positive suggestion should always be remembered in the management of patients. Positive suggestion can greatly enhance the quality of sedation particularly where good doctor-patient rapport is present.

Euphemistic language:

It is also important to try to avoid the use of emotive words such as pain, extract, and injection. Discomfort, remove and scratch are better terms and have a much less threatening connotation. It is sometimes useful to put ourselves in the patient's position.

Patient education:

Fear of the unknown is a very powerful stimulus to anxiety, particularly in children. The preparatory (pre-operative) interview is of the utmost importance. Time should be allotted at the initial consultation appointment, and to answer any questions they may have concerning this. In addition to discussing the dental procedure, time should be allowed for discussion of the sedation technique and its effects.

Patients may also be given printed material to take home for further study. In a landmark study by Beecher et al entitled, "The Value of the Pre-operative Visit by an Anaesthetist", the psychological effect of a pre-operative visit by the anaesthetist was compared with the effect of pentobarbital sodium (Nembutal) for pre-anaesthetic medication.

Patients who received 2mg/kg of body weight of Nembutal intramuscularly one hour pre-operatively became drowsy, but could not be considered calm. On the other hand, patients who received a 5-10 minute visit from their anaesthetist the night before their operations, informing them about the events which were to occur on the day of the operation and the anaesthetic to be administered, were not drowsy, but were significantly calmer on the day of the operation.

There is no doubt that patients facing a frightening situation become anxious and look for emotional support. Many patients obtain this support by seeking a realistic and well-informed view of the situation facing them, while others attempt to deny the existence of the impending danger. For both types, an authority supposedly able to modify the danger becomes invested with strong emotional significance, and statement made by this “danger control” authority can therefore assume enormous importance.

The placebo effect:

This is greatly influenced by the previous factors we have been discussing, in particular, the dentist-patient relationship. Various studies have shown this response to occur in anything from 17% to 92% of patients. For example, in one study of 2,628 oral surgery patients 67% suffered post-operative pain, and 75% of those patients given placebo tablets reported pain relief.

Placebos have been shown to be increasingly effective under conditions of increased stress. They have also been shown to have cumulative effects; addictive effects (leading to withdrawal symptoms when the placebo is withdrawn); and varying degrees of side effects, sometimes of a serious nature. Patients who respond to placebos also tend to get significantly more relief from post-operative pain with narcotic analgesics.

On the other hand, the placebo effect may be used to enhance the benefits obtained by specific remedies or procedures. The environment, along with the suggestions given patients, seems to be critical in determining the degree of placebo effectiveness. In order to obtain a maximum placebo response, the patient must trust and believe in their doctor, they must believe that they are receiving a real drug, and they must believe that the drug will reduce their discomfort. Confidence in the medication reduces anxiety and thus reduces subjective reaction to pain.

An intriguing development was the discovery that endorphins (natural opioid analogues produced by the body) possibly mediate placebo analgesia. One group of researchers found that Naloxone (an opioid antagonist) effectively blocked placebo analgesia in a group of post-operative oral surgery patients. This seems to suggest a possible biochemical basis for placebo activity, which could be utilized to our advantage in patient management.

Environment:

Staff should be friendly and sensitive, as well as efficient. The importance of the chair-side assistant and office staff to patient relaxation cannot be overemphasized. It is also worth noting that male patients will usually tolerate more pain in the presence of an attractive female (however this may not be an ideal guide to employment practices) conversely, a grim, humourless dental assistant is not the type to inspire confidence in an anxious patient.

Office and surgery décor should be warm and inviting, in order to help promote confidence and relaxation. Colour also plays a vital part in office and surgery décor.

Various colours are considered by some to have profound effects upon human emotions and physiology. For example, red supposedly has an excitatory effect on the central nervous system, resulting in increased blood pressure, respiration, and heart rate. On the other hand, Blue (particularly dark blue) has the reverse effect, exerting a calming action. Green is considered to be a colour that expresses firmness, consistency, resistance to change, and a wish to impress other; whereas yellow represents expansiveness, relaxation, and a release from problems and restrictions. Black and grey convey pessimism and gloom, but brown is a sensual colour, suggestive of physical comfort. It would thus appear that blues, browns, and yellows are perhaps the most useful colours for the purpose of increasing the patient's relaxation response.

Clothing worn by the doctor and staff is important, particularly with regard to colour and design. White, hospital-type uniforms are not favoured in dental practices, and most prefer the use of colours such as blue or brown.

Modeling:

Is a psychological technique which has been used with success in dentistry. A person who observes a model undergoing a procedure involving a feared stimulus but showing no negative responses tends to imitate the same fearless response. Modeling is particularly effective with children – for example, a young child can be permitted to see its mother undergoing a routine examination or prophylaxis, thus learning that dentistry is not a threatening experience (provided, of course that the mother is not herself a very anxious patient, in which case a negative learning process may occur).

Systematic desensitisation:

Another technique for reducing anxiety. This involves having a patient imagine themselves in a fear-producing situation while in a state of deep relaxation. After several pairings of the relaxed state with visualization of the feared stimulus, the fear-producing power of the stimulus is lessened. However, a considerable amount of time is required to produce lasting results, which can be a major drawback for the busy practitioner.

Biofeedback:

Is a practical means of teaching relaxation, which is now widely used, particularly in multidisciplinary pain clinics. Biofeedback involves the attachment of various types of physiological monitors to the patient, which is capable of feeding back audible and/or visual information as to the patient's physiological state from moment to moment.

The most useful biofeedback monitors appear to be those which measure pulse rate, blood pressure, respiratory rate, muscular contraction (particularly of the frontalis muscle) using a electromyograph (EMG); vasoconstriction (using a plethysmograph); and galvanic skin response and conductance.

When trained to use such equipment, it is possible for patients to learn how to achieve a state of deep relaxation quite rapidly. As it is almost impossible to remain highly anxious when deeply relaxed, it is apparent that biofeedback training may be useful in the management of anxious dental patients.

Music is of very real value. Background music of the “Muzak” type is found in many dental waiting rooms these days, and has a positive relaxing effect upon patients. Music can also be piped into the surgery and delivered via speakers, or directly to the patient via headphones. Alternatively, a patient may be offered the chance to bring in their own choice of music which they can listen to via headphones. This also permits the operator or assistant to converse with the patient if necessary. On the other hand, it is sometimes useful to increase the volume to a level which helps counteract threatening noises such as the sound of the air turbine. As far as the type of music is concerned, instrumental music of a soothing nature is usually best for relaxation purposes. By the same token, if music includes vocals, one must be careful that the lyrics do not trigger off an emotional response in the patient. It can be disconcerting if the patient burst into loud song (or tears) in the midst of a delicate crown preparation, so it is probably best to ask the patient which music they prefer after the doctor has pre-selected a list of suitable relaxing choices, or they can bring their own selection.

The dental chair should be comfortable and permit freedom of movement, which increases pain tolerance. On the other hand, discomfort and restricted movement decrease pain tolerance.

Audio-analgesia:

Also known as white noise - is a technique which has been used with only limited success in dentistry. It is based on the principle that it is easier to hear a single sound than one sound in the presence of another. This is known as “masking”. In dentistry, high volume “white sound” which is comparable to the sound of heavy rain or surf, has been used to mask the sound of dental noises. Unfortunately, it has not been shown to be particularly effective in reducing dental pain, except in a very high and potentially damaging decibel range. Audio-analgesia is also more effective in controlling slowly increasing painful stimuli, not sudden pain stimuli.

Nutritional factors:

These have also received increasing attention in the modification of behaviour and pain perception, several dietary substances having been shown to be precursors of neurotransmitters, such as acetylcholine (choline) and serotonin (tryptophan). Tryptophan-enriched diets have been shown in some trials, for example, to significantly increase pain tolerance levels by supposedly increasing brain serotonin .However other studies have not reproduced this effect so it may in fact be a variant of the placebo effect

Conclusion

The effectiveness of drugs used to control anxiety and pain in dental patients is directly related to the effectiveness of the behavioural approach. Moreover, the dividing line is becoming progressively harder to define, as research indicates that certain behavioural techniques may in themselves lead to the production within the patient of powerful endogenous analgesic agents. The complete answer to the problem of the anxious dental patient is multidisciplinary, integrating both behavioural and pharmacological approaches, along with a thorough understanding of the factors which influence pain tolerance and how these can be modified.

BASIC SCIENCE & PATIENT EVALUATION

Cardiovascular Physiology

Introduction to the CVS:

The cardiovascular system is comprised of:

- The heart
- The arteries
- The microcirculation (including the capillaries)
- The veins

Blood is pumped from the heart into the major arteries, where it is distributed through smaller and smaller branching arteries, then through arterioles into the capillaries. Here the major exchange of nutrients, gases and waste products occurs between the blood and tissues. The blood from the capillaries is collected into venules which join together to form small veins, and these drain into bigger and bigger veins which eventually empty into the heart through the superior and inferior venae cavae. By the time the blood reaches the veins, much of the energy imparted by the heart has dissipated, so the veins have valves to ensure that the blood keeps flowing in the right direction.

The heart has four chambers, arranged as a pair on the right and a pair on the left of a muscular dividing septum.

The two chambers on the right, the atrium and ventricle, are concerned with collecting the used systemic blood returning in the great veins and pumping it out into the pulmonary artery and through the lungs.

The atrium and ventricle on the left side collect the replenished blood returning from the lungs through the pulmonary veins and pump it out through the aorta (the major artery) to supply the whole of the body.

There are valves between each atrium and its ventricle, and between each ventricle and its corresponding major artery:

- Right atriventricular valve = tricuspid valve (has 3 leaflets)
- Left atrioventricular valve = mitral valve (bishops hat)
- Right ventricular outlet valve = pulmonary valve
- Left ventricular outlet valve = aortic valve

Essentially, there are two circuits of blood flow:

1. **Pulmonary Circuit:**

   Deoxygenated blood from the Right side of the heart pumped to the lungs and freshly oxygenated blood from the lungs back to the Left side of the heart.

2. **Systemic Circulation:**

   Oxygenated blood from the Left side of heart is pumped to the body tissues, and deoxygenated blood from the tissues is pumped back to the Right side of the heart.

Initiation of Heartbeat:

The heart is composed of specialised \muscle which contracts and relaxes in a rhythmic fashion.

Contraction = SYSTOLE

- blood pumped out through the pulmonary and aortic valves

Relaxation = DIASTOLE

- heart fills up with blood returning from the veins

Electric impulses spread throughout the Cardiac muscle to cause the relaxation and contraction. These events occur independently of any intervention from the nervous system, i.e. they are intrinsic properties of the heart itself.

Different parts of the heart vary in the rate that has the highest rate of spontaneous diastolic depolarization is the **sinoatrial** (SA) node, situated in the wall of the right atrium near the site of entry of the superior and inferior venae cavae. This acts as the heart's "pace-maker".

These cells reach threshold for depolarization earlier than their neighbours and the action potentials that result trigger action potentials in the other cells. Impulses originated in the SA node are conducted from cell to cell through the atrial and ventricular muscle via the intercalated discs. The arrival of an impulse at any given heart muscle cell causes that cell to contract.

Points to note:

- Cardiac muscle contractions are always brief twitches. There is a long lasting action potential and an associated long refractory period which means impulses cannot summate or "fuse" to provide smooth sustained contractions.

- Relaxation between each beat is essential for the heart to fill with blood to be pumped at the next beat.

- Heartbeat is **all or none**

- Cardiac muscle excites itself (cf. skeletal muscle which require a nerve impulse). Nervous supply to the heart influences rate and strength of contraction, but doesn't initiate the primitive heartbeat.

Neural Control of the Heart

Parasympathetic:

The vagus nerve sends fibres to the heart. Impulses in the vagus nerve liberates acetylcholine from nerve endings. This decreases the rate of diastolic depolarization in the SA node, lengthening the interval between cardiac impulse and **slowing** heart-rate.

There is no effect on ventricular contraction as the ventricles are virtually free of any parasympathetic innervation.

Sympathetic:

Sympathetic nerves reaching the heart supply all parts of the myocardium, but particularly the nodal areas. Impulses in these nerves release noradrenaline which accelerates the rate of diastolic depolarization, shortens the interval between cardiac impulses and **speeds up** heart-rate.

At times of stress, adrenaline is released from the adrenal gland – similar effect to noradrenaline. Adrenaline also causes cardiac muscle to contract more forcefully.

The ECG:

Passage of electrical impulse through the heart produces disturbance in the body's electrical field which can be detected with electrodes on the body's surface. Signals from these electrodes can be summed to give rise to the electrocardiogram.

Factors affecting Cardiac Output:

Cardiac output = volume of blood flowing out of either Right or Left side of the heart per minute.
Cardiac output is calculated by stroke volume (volume ejected during each heartbeat) x heart rate and measured in litres per minute.
SV is determined by venous return. The heart can only pump out the blood that comes into it. If venous return increases, SV increases as the myocardial muscle (in a healthy heart) will contract more forcefully if stretched ("Starlings Law of the Heart").
Cardiac output is also increased when the heart rate increases. Cardiac output falls when the heart rate slows (bradycardia) for example during a faint.

Blood Pressure:

Once it leaves the heart, blood flows through the rest of the body through a series of tubes. Fluid flow in tubes has a number of properties.

It firstly requires a pressure difference at each end of the tube – otherwise fluid cannot flow. The second is resistance to fluid flow. Some resistance is due to the tube and some is due to the fluid. A short tube has less resistance than a long one, a narrow tube more resistance than a wide one.

The resistance of a tube is directly proportional to the length, but is inversely proportional to the fourth power of the radius, i.e.

$$\text{Resistance} \sim \frac{\text{Length}}{\text{Radius}^4}$$

The circulation is very sensitive to small changes in the caliber of blood vessels.

Resistance to flow also depends on whether or not the flow is turbulent – smooth flow has less resistance than turbulent flow.

Blood pressure = Cardiac output x peripheral resistance

Peripheral resistance is determined largely by the arterioles. Placed at the centre of the microcirculatory bed, they control blood flow into the capillaries. Since their total cross sectional area is much smaller than that of the arteries or capillaries, they contribute a great deal of the total resistance to blood flow in the circulation, and changes in their diameter can cause profound changes in the total vascular resistance. Caliber of the arterioles is determined by smooth muscle in their walls which is subject to a number of influences from nerves, hormones and local metabolites.

- Noradrenaline – constriction
- Acetylcholine – dilation
- Bradykinin – vasodilatation
- Antidiuretic hormone (vasopressin) – vasoconstrictor
- Angiotensin – vasoconstriction
- Local needs – as a muscle contracts and does work it increases its requirement for blood supply. There is a local depletion of oxygen and an accumulation of carbon dioxide in the tissues. This increases blood flow by producing a direct dilator effect on the vessels. Accumulation of lactic acid and the drop in pH also causes dilatation in the arterioles.

Veins and venules represent the low pressure side of the circulation and changes in vascular tone here have little effect on cardiac resistance.

Baroreceptors:

One of the most important autonomic reflexes is concerned with the regulation of blood pressure. The carotid artery, which carries blood to the brain, contains an area near its bifurcation that contains a number of stretch receptors that are sensitive to distension in the artery. This is the Carotid sinus, and the receptors are baroreceptors.

If the pressure in the arteries rises, the carotid sinus becomes distended and impulses are discharged at a greater rate in the sensory carotid sinus nerve. This enters the medulla oblongata and fibres go to the cardiac and vasomotor centres of the medulla. As the impulse rate increases, the cardiac (vagal) centre is stimulated and impulses travel down the vagus nerve to slow the heart. At the same time the vasomotor centre is inhibited so that fewer impulses travel out along the sympathetic nerves throughout the body, and the peripheral arterioles relax. The result of these two actions is that cardiac output falls and peripheral resistance is lowered … BP comes down towards normal. The baroreceptor reflex is important in the moment by moment maintenance of arterial blood pressure. It helps the body deal with changes associated with alterations in posture, or with acute haemorrhage.

Respiratory Physiology

Introduction:

The goals of respiration are to provide Oxygen (O_2) to tissues and to remove Carbon dioxide (CO_2). To achieve these goals, respiration can be divided into 4 major functional events:

a) Pulmonary ventilation, i.e. the inflow and outflow of air between the atmosphere and the lung alveoli.
b) Diffusion of O_2 and CO_2 between the alveoli and the blood.
c) Transport of O_2 and CO_2 in the blood and body fluids to and from the cells.
d) Regulation of respiration.

The Respiratory Tract:

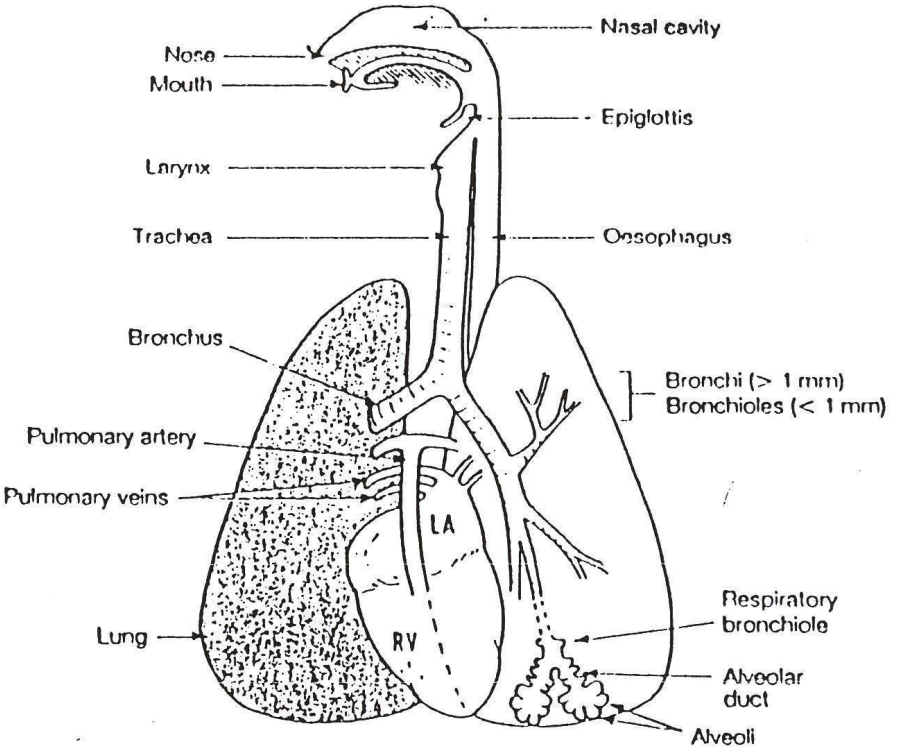


Fig. 16.1 Schematic representation of the upper and lower respiratory tract. LA, left atrium; RV, right ventricle of the heart.

On inspiration, air passes through the **upper respiratory tract** (nose, pharynx, and larynx) into the trachea. The conchae of the paired **nasal cavities** create a vast surface area which is line by ciliated columnar epithelium containing mucus-secreting cells; there are also coarse hairs or vibrissae at the nostrils and a dense vascular network in the submucosa. In the nasal cavities the air is **filtered** to remove foreign particles, **warmed** to 37ºC and **humidified**.

Sensory nerve endings of the trigeminal nerve detect irritants in the nasal mucosa and trigger sneezing. In the **pharynx**, sensory endings of the glossopharyngeal nerve detect irritants that cause aspiration reflex. The pharynx serves as a common passageway for food and liquid entering from the mouth and for gas entering from the nasal cavities. During swallowing, food and fluid are deflected from the entrance to the larynx by a cartilaginous flap called the **epiglottis**.

The **larynx** is a cartilaginous 'box' that contains the **vocal cords** separated by an aperture called the **glottis**, which closes during swallowing. Vibration of these cords as the air passes through the glottis produces sounds, the amplitude and pitch of which can be altered by the speed of air movement and the size of the glottis respectively. Changes in glottal aperture also occur in normal breathing. The glottis dilates during inspiration and constricts during expiration, the constriction increases airway resistance and therefore prolongs expiration; the constriction increases airway resistance and therefore prolongs expiration. Laryngeal muscles controlling the glottis are skeletal and are innervated by the recurrent laryngeal nerve. Another vagal branch, the superior laryngeal nerve, contains afferent fibres from mucosal irritant receptors which initiate the cough reflex.

The **lower respiratory tract** commences with the **trachea** which divides into the two main **bronchi**, one to each lung. These repeatedly subdivide within the lung unit until, after some 23 'generations' or divisions, the alveoli are reached Mucoserous glands in the submucosa and mucus-secreting goblet cells in the epithelium produce a fluid which contributes to humidification and helps trap particles and soluble pollutants. These are then moved towards the pharynx by the action of the cilia.

There are 300-600 million alveoli in the lungs and, since each **alveolus** at the end of a normal expiration is ~0.01 mm in diameter, they provided 50-90 m² of surface area for gas exchange. The alveoli abut with one another so that the wall (interalveolar septum) of one is shared with another. The alveolar wall is, in effect, a huge dense capillary network and has a number of very thin layers through which gas molecules must diffuse. These layers comprise the fluid lining layer, the alveolar epithelium, the interstitium and the capillary endothelium, and are referred to collectively as the alveolar-capillary membrane or air-blood barrier. The interstitium contains reticular and elastic fibres, which confer on the lungs some elasticity.

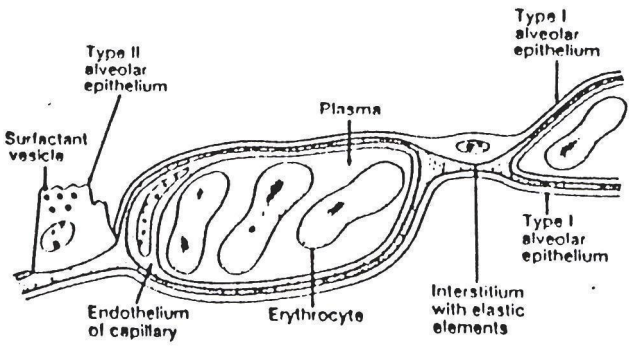


Fig. 16.3 Structure of the shared wall of two alveoli. The very thin fluid lining layer covering the alveolar epithelium is omitted.

Alveolar capillaries receive blood low in O_2 and high in CO_2 through the pulmonary circulation, while capillaries supplying the walls of the larger airways receive oxygenated blood from the aorta through the bronchial part of the systemic circulation.

A) Pulmonary Ventilation

Mechanics of Pulmonary Ventilation

The lungs expand and contract in two ways:

1) By downward and upward movement of the diaphragm to lengthen or shorten the chest cavity.
2) By elevation and depression of the ribs to increase and decrease the anteroposterior diameter of the chest cavity.

Normal quiet breathing is accomplished almost entirely by movement of the diaphragm. During inspiration, contraction of the diaphragm pulls the lower surfaces of the lungs downward. During expiration, the diaphragm relaxes and the **elastic recoil** of the lungs, chest wall and the abdominal structures compresses the lungs. During heavy breathing, extra required force to obtain rapid expiration is achieved by the contraction of the abdominal muscles which pushes the diaphragm upwards.

The second method for expanding the lungs is to raise the rib cage. The muscles which raise the rib cage are the **external intercostals**, the **sternocleidomastoid**, the **anterior serrate** and the **scalene**. The muscles which pull the rib cage downward during expiration are the **abdominal recti** and the **internal intercostals**.

Respiratory Rate

The normal respiratory rate is about 12 breaths per minute for an adult. It may be increased by exertion, anxiety, pregnancy, and in disease states. Children have higher respiratory rates which slow down to adult levels as they age. Respiratory rate may decreased in physically fit individuals or by sedation.

B) Diffusion of O_2 and CO_2 Between the Alveoli and the Blood

After the alveoli are ventilated with fresh air, the next step in the respiratory process is **diffusion** of oxygen from the alveoli into the pulmonary blood and **diffusion** of carbon dioxide in the opposite direction. The process of diffusion is simply random molecular motion of molecules intertwining their ways in both directions through the respiratory membrane.

Because the air in the lungs has a relatively high concentration of oxygen compared with the de-oxygenated blood in the pulmonary circulation, oxygen moves down its concentration gradient and the pulmonary blood becomes oxygenated.

Conversely carbon dioxide leaves the pulmonary blood and moves down its concentration gradient into the alveoli where it is breathed out.

C) Transport of O_2 and CO_2 in the Blood and Body Fluids

Once oxygen has diffused from the alveoli into the pulmonary blood, it is transported principally in combination with haemoglobin to the tissue capillaries, where it is released for use by the cells. In the tissue cells, oxygen reacts with various foodstuffs to form large quantities of carbon dioxide. This in turn enters the capillaries and is transported to the lungs.

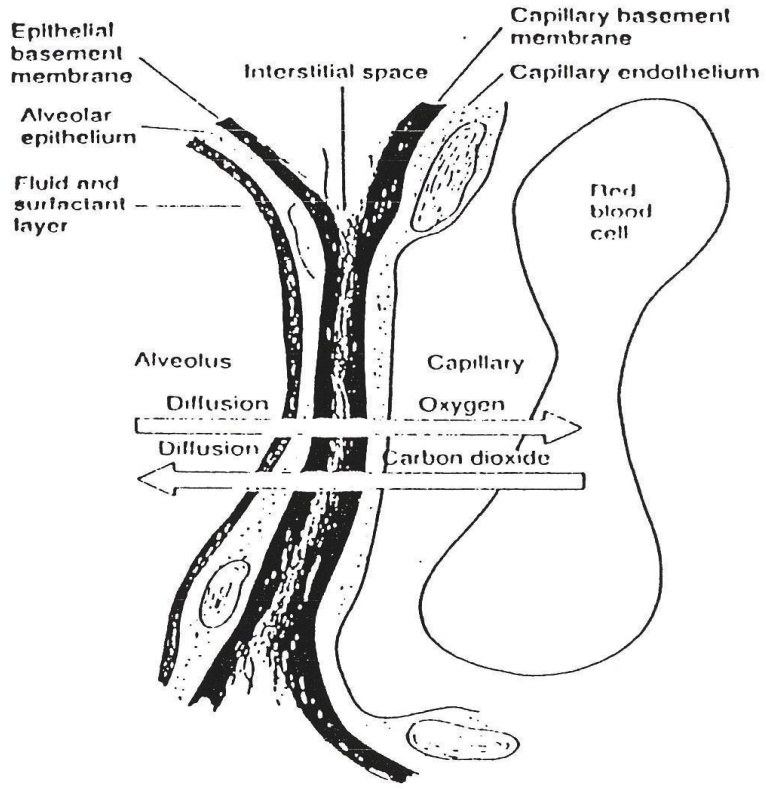


Figure 39-9. Ultrastructure of the respiratory membrane as shown in cross section.

D) Regulation of Respiration

Breathing occurs rhythmically. The rhythmicity of generated within the respiratory centres which are found in specific areas of the medulla oblongata (dorsal respiratory group, DRG; ventral respiratory group, VRG; and Botzinger complex) and pons (pontine respiratory group, PRG). The depth and frequency of the resting rhythm are determined by feedback to the respiratory centres from mechanoreceptors in the lungs and chest wall. Some stimuli, e.g. temperature and pain, affect breathing via higher brain centres (hypothalamus and limbic system) which then influence the respiratory centres. Breathing is also under appreciable voluntary control from the cerebral cortex (e.g. in breath-holding, taking large breaths or speaking) and this pathway bypasses the respiratory centres. Superimposed on the normal pattern of breathing are protective reflexes of short duration which are initiated, for example, by irritants as in coughing or sneezing. The principal control of ventilation is from chemoreceptors located peripherally (carotid and aortic bodies) and centrally (intracranial chemoreceptors). Breathing is more sensitive to stimulation by high CO_2 (hypercapnia) than low O_2 (hypoxia) so ventilation is matched through the CO_2 produced rather than the O_2 consumed.

PATIENT ASSESSMENT

Patient Selection

This section covers the many factors which must be taken into account before offering I.V. sedation to the patient.

Particular Indications:

1. Control of fear and anxiety.

2. Unpleasant procedures such as oral surgery, periodontal surgery and extensive crown and bridge procedures.

3. Relatively long appointments (up to 2 hours) e.g. multiple cavity preparations, crown and bridge work. Also suited to patients with limited time who wish to complete treatment in as few visits as possible, e.g. business people, mothers with small children.

4. Patients who suffer from severe gag reflexes may benefit from I.V. sedation.

5. Treatment of some physically and mentally handicapped patients e.g. cerebral palsy – provided one is an experienced sedationist... (These may be best hospitalized.)

An introductory “welcome letter” may also be helpful in many situations.

Example:

Please read the underlying statement before completing the medical, dental and anxiety questionnaires.

The goal of the modern dentist is to provide comprehensive, high quality oral health care for his/her patients. This includes the diagnosis, treatment and prevention of diseases of the teeth, supporting structure (gums and jaw bone) and the soft tissues of the mouth (tongue, cheek, etc.).

We would like to help our patients understand and appreciate fully the relationship of the mouth and teeth to one’s total health. It would not be possible to treat diseases of your teeth and gums if your mouth were separate and not a part of the rest of your body. The tissues and structures of the oral cavity are intimately connected by blood vessels and nerves to the other areas of your body. The same blood that passes through your brain, heart, stomach, etc., also passes through your teeth and gums. Disease or injury in other locations of the body may affect the oral cavity. The following few examples illustrate this relationship:

1. *A patient with a history of rheumatic fever requires antibiotic treatment before, during and after extraction of teeth, gum surgery, treatment of mouth infections etc. The antibiotics destroy germs that might enter the blood stream during surgery and travel to the heart causing problems on heart valves previously damaged by rheumatic fever.*

2. *The presence of diabetes usually requires antibiotic coverage because the diabetic patient is more prone to infection as a result of changes in blood sugar levels and the tissues therefore do not heal as well.*

3. *It is important that the dentist be aware of any drug allergies you might have. For example, the administration of penicillin to a patient allergic to this drug could result in a serious medical emergency.*

4. *All medications that you are presently taking should be made known to the dentist, since they must be considered in relation to your dental treatment. For example, if you are taking sleeping pills, tranquillisers or antidepressants at home, it is important to relate this dosage to any sedation administered to prior to dental treatment. If the dentist were to give or prescribe antibiotics or pain medication without the knowledge of the same or similar drugs prescribed by your physician, a serious medical reaction may occur.*

5. *Your psychological attitudes in respect to dentistry are important when planning necessary dental treatment. For example, a patient with high blood pressure may experience further increases in blood pressure if he or she is nervous. In such cases medication may be administered prior to dental treatment to lessen anxiety and thus avoid increases in blood pressure.*

It is important that your health status is determined prior to dental treatment. This will not only enable us to provide a high quality oral health care service for you, but it will also help to reduce the risk of a medical emergency occurring before, during or after dental treatment.

It is therefore very important that you complete the following health questionnaire accurately and completely. Please ask if there is anything you are unclear about.

Thank you for your co-operation.

Medical History and Evaluation

PHYSICAL ASSESSMENT

History:

The pre-operative medical and dental history must be:

(a) Concise and relevant to the dental situation.
(b) Examined in detail for discrepancies and omissions, and anything in doubt clarified before proceeding with treatment.
(c) Be signed by the patient and the doctor.
(d) Be revised regularly and prior to each new course of treatment.

A printed, dated and signed medical history form is the best method (see example). This requires the patient to think specifically about each question, and there is less chance of a patient forgetting or thinking something is not important. Reports of deliberately misleading medical histories (some of which have led to fatalities) and the withholding of important medical information by patients, indicate the importance and possible medico legal implications of a medical history which has not been signed by the patient.

From the medical history it should be possible to assess most past and present medical conditions. Anything in doubt must be fully discussed with the patient, and where further information appears desirable before treatment, the patient's doctor should be consulted. A telephone call is usually sufficient, and the response is normally one of appreciative co-operation. Tell the G.P. what you propose doing, the need for effective sedation of the patient you are treating, and the drugs you intend to use to provide this. Discuss hospitalization vs. treatment in the office if the patient is a borderline case.

Signed, informed consent must be obtained prior to treatment of any description.

Always keep prevention in mind. High-risk patients should be hospitalized and not accepted for treatment in the dental office. All medical histories must be regularly revised at future appointments.

Examples of Health Questionnaires and Consent Forms

Example 1.

CONFIDENTIAL HEALTH QUESTIONNAIRE

In order to provide the best and safest dental treatment, we need to know of any medical problems which may affect your treatment.

Please tick the box only if the answer is yes.

NAME: _____ Mr/Mrs/Miss/Ms
(Surname) (First Names)

ADDRESS: _____

_____

TELEPHONE NO:

Home: _____

Bus: _____

DATE OF BIRTH: _____

OCCUPATION: _____ **HEIGHT:** _____ **WEIGHT:** _____

NAME OF MEDICAL PRACTITIONER: _____

1. Are you receiving any medical treatment at the present time? [ ]
2. Have you ever been admitted to hospital at any time in the past year? [ ]
3. Have you ever had any of the following?

Rheumatic Fever	[ ]	Epilepsy	[ ]
Heart Trouble	[ ]	Anaemia	[ ]
High Blood Pressure	[ ]	Bleeding Disorders	[ ]
Asthma	[ ]		
Arthritis	[ ]	Gastric Problems	[ ]
Hepatitis	[ ]	Cold Sores	[ ]
Chest Problems	[ ]	Depressive Illness	[ ]
Migraine Headaches	[ ]	Kidney Trouble	[ ]

Diabetes	[ ]	Recreational drug use	[ ]
Other	[ ]		

4. Are you taking any tablets, medicines or drugs?
   If so, please list: _____

   _____

5. Have you any allergies that you are aware of? [ ]
   If so, please list: _____

   _____

6. Do you have any artificial joints or a pacemaker? [ ]

7. Have you ever experienced excessive bleeding or bruising from cuts, scratches or dental treatment?

8. Have you ever had contact with the AIDS virus? [ ]

9. Have you ever had a reaction to an anaesthetic? [ ]

10. Women: Are you pregnant now? [ ]

Are there any other aspects concerning your health that you think we should know about?

_____

_____

Name of Parent or Guardian:
(For patients under 16 yrs)

Signed by: Patient / Parent / Guardian Date: _____

Has there been any change in your previous medical history or medication since your last course of treatment? [ ]

DETAILS: _____

CONSENT

1. The medical history I have given is true and correct to my knowledge.

2. I consent to the Surgical Procedure for _____ and have had explained and understand the complications of such procedures under:

   Local Anaesthetic / Local Anaesthetic and Sedation / General Anaesthetic

Signature: _____ Date: _____

Example 2.

PATIENT QUESTIONNAIRE

NAME: _____ MR/MRS/MISS/MS
(Surname) (First Names)

ADDRESS

_____

_____

TELEPHONE NO: _____ DATE OF BIRTH: _____
Home

_____ OCCUPATION: _____
Business

Name of last Dentist: _____ Date of last visit: _____

Person who referred you: _____

If under 20 – Name and Address of Parent/Guardian: _____

_____

If you have Dental Insurance please name the company: _____

Name of Medical Practitioner: _____

Other questions or information: _____

_____

_____

Form Updated – Dates: _____

_____

_____

_____

Vital Signs in Dentistry – A Summary

BLOOD PRESSURE:

Blood Pressure consistently above 140/90 mm HG in the resting, non-anxious patient represents arterial hypertension. Three consecutive measurements should be made when in doubt, and the lowest is assumed to be correct.

Mild hypertension
140/90 – 160/95
sedation if indicated

Moderate hypertension
160/95 – 200/115
stress reduction

Severe hypertension
Above 200/115
medical emergency

Mild hypertension rarely produces symptoms. Measurement at the initial physical evaluation will identify the disease at a readily treatable stage. This service is a public health contribution of incalculable value. Refer severely hypertensive patients to the G.P. for further management unless dental treatment is an emergency.

PULSE RATE:

Pulse rate varies from 60 to 90/min in the normal adult. In the threatening dental environment, 110/min is arbitrarily chosen as the upper limit of normality.

A rate of 40 to 60/min in the superbly conditioned athlete is considered normal (sinus bradycardia). A pulse rate less than 60/min in the non-athlete may indicate heart block; a pulse rate greater than 110/min may indicate acute or chronic heart disease. Generally a rate less than 60 or more than 110/min may in the adult dental patient is worthy of investigations.

The appearance of bradycardia (pulse rate less than 60/min) in a patient with previously normal rate may indicate an impending faint or less commonly cardiac arrest, particularly in the presence of distress (sweating, weakness, shortness of breath, or chest pain); immediate medical assistance should be called.

PULSE RHYTHM:

Atrial fibrillation is suspected with a pulse that appears totally irregular. This is a serious finding if the patient hasn't been previously diagnosed and/or treated. Again referral to their G.P. for accurate diagnosis and management is indicated before continued dental treatment.

Premature ventricular contractions (PVCs) are recognized as an occasional pause in an otherwise regular rhythm. Whereas PVCs may occur in healthy patients, when they are recognized at a rate of 5/min or more in a patient with heart disease, medical consultation is needed.

PVCs invariable accompany a heart attack and always precede cardiac arrest. When PVCs are recognized at a rate of 5 per minute, or more, in the distressed patient (sweating, palpitations, weakness, shortness of breath, or chest pain), immediate medical assistance should be called.

BODY TEMPERATURE:

Body temperature by mouth ranges from 36-37.5C in the healthy individual.

RESPIRATION:

Normal rate for the reasonable apprehensive adult is 16-20 breaths per minute. A rate greater than 20/min should be investigated – but may just be due to anxiety.

HEIGHT AND WEIGHT:

Investigation is in order for an unexplained significant weight change. Grossly overweight or underweight patients represents significant risk during elective dental therapy, and both types of patients are very sensitive to depressant drugs.

FREQUENCE OF MEASUREMENT:

Measurement of blood pressure in an adult patient at the first physical evaluation is considered by many as a moral obligation. The practitioner dedicated to comprehensive care also determines pulse rate and rhythm , and measures the body temperatures as indicated by symptoms and signs.

Excerpted from: McCarthy, F.M., Vital Signs – The six minute warnings, 100: 682-691, May 1980, Journal of the American Dental Associations.

History Notes

When investigating the medical history, watch in particular for:

1. **Cardiovascular disease:** Congestive heart failure – valvular disease
   Recent coronary – myocardial infarct
   Conduction problems
   Angina
   Hypertension
   Congenital heart disease
   Rheumatic heart disease
   Anaemias

   Patients with some of the above conditions should be treated in a hospital with the increased level of monitoring and medical back up that this implies. If you are unsure consult the patient's physician. Hypertensive patients will usually be on medication: **make sure they have taken their tablets**. Some antihypertensive drugs potentiate or prolong the action of analgesics, sedatives, and tranquillosedatives. They may also predispose to postural hypotension. Well patients with a history of cardiac disease may be considered for treatment in the dental surgery, if agreed by the patient's physician, once you have become an experienced sedationist. In such cases, careful monitoring and titration of sedation will be required, and supplemental oxygen may be beneficial.

   Tachycardia (increased heart rate) is potentially harmful in patients with angina or ischaemic heart disease, Drugs which may increase heart rate such as local anaesthetic solutions containing adrenaline should be used with care. Use aspirating dental syringes, in order to avoid the likelihood of intravascular injection, which is reported to be as high as 10% of dental local anaesthetic injections, particularly inferior alveolar (mandibular) blocks.

2. **Respiratory disease:**
   Asthma
   Bronchitis
   Bronchiectasis
   Emphysema
   Pulmonary Oedema

   Many patients have a history of mild asthma, and for light sedation present few problems. A history of severe asthma warrants medical consultation. All asthmatics should bring with them their usual inhaler. In an acute attack use the patients own (blue) inhaler to help them. If this is not available any ventolin inhaler will help. If this does not work rapidly summon help give oxygen and consider small doses of adrenaline. (See section on Emergencies).

   Bronchitis and other respiratory conditions do not normally present the same problems for sedation as for general anaesthesia Patients with moderate to severe emphysema should be treated in the hospital environment.

1. **Endocrine dysfunction**

(a) Diabetes

Do not treat uncontrolled diabetics in the dental surgery. Controlled diabetics may be treated four hours after normal insulin and a light meal. If severe apprehension is present it may be advisable to reduce insulin on the day of treatment, following consultation with the patient's physician, as such patients may not eat at all. Many mild diabetics are controlled by diet alone, and in these patients it is important to schedule appointments between normal meal times. Avoid long procedures, keep patient well oxygenated, and see they have a meal after treatment.

Note: It is advisable to consult with the diabetic's physician before administering sedation.

(b) Hyperthyroidism (thyrotoxicosis)

Do not treat anyone with uncontrolled thyrotoxicosis – they require urgent medical attention. Signs include high metabolic rate, tachycardia, nervousness, and weight loss. Co-existing heart disease may be present. Once treated ensure the patient takes their usual medication and proceed cautiously. If you are concerned consult with their endocrinologist before scheduling any treatment.

(c) Adrenal Insufficiency

Must be assumed a possibility if the patient has been taking corticosteroids within the preceding six months. E.g. in arthritis, ulcerative colitis, asthma, some allergic conditions. Continue steroid therapy before, during, and after treatment. If a patient has been on a significant dose of prednisone (greater than 5 mg) for a period of longer than 3 months or a lower dose for a longer duration (i.e. 2.5 mg for several years) then they may be vulnerable to adrenal insufficiency and hypotension. Discuss the patient with their G.P. or physician and consider "steroid cover" for the procedure.

4. **Epilepsy**

Midazolam decreases the likelihood of a seizure. The patient should be instructed to take his/her usual medication. Avoid stress, keep to short appointments.

5. **Liver Disease**

The liver has large reserves and has to have suffered significant damage before clinically important problems occur. A patient with immanent or pre-existing liver failure or active hepatitis should not be treated except as an emergency. Mild liver impairment can occur and has a variable effect on drug metabolism and distribution. Metabolism of midazolam is not usually effected but remember - as always - titrate small doses of midazolam to clinical effect.

6. **Pregnancy**

While not an absolute contraindication during the second and third trimesters, the use of sedation should generally be avoided during pregnancy, particularly the first trimester. A history of complications during early pregnancies or any suggestion of hypertension calls for medical consultation. In advanced pregnancy use the semi-reclining position to reduce pressure on the inferior vena cava. Remember that any sedative given to the mother will affect the unborn child via the placenta. Also, transmission can occur to an infant via breast-feeding.

7. **Drug Therapy**

A number of drugs may react with the local and general anesthetics, sedatives, and tranquillisers administered in the course of dental treatment. Some of these drug interactions may lead to serious complications. An up-to-date drug catalogue must be available for constant reference in order to check the uses, side effects and interactions of any drugs the patient may be taking. The possibility of illegal drug consumption must also be kept in mind.

Remember to always titrate the dose of midazolam to clinical effect. Once your patient is adequately sedated they do not need any more. The interactions between drugs can be complicated.

For example:
A patient scheduled for dental sedation is taking lorazepam for the treatment of panic disorders. Lorazepam is a long acting oral benzodiazepine and in the same class of drug as midazolam. The patient might:

1. Require less sedation than you expect due to the pre-existing effect of the lorazepam which they have taken before attending your surgery.

Or

2. Require more midazolam to achieve adequate sedation due to down regulation of their benzodiazepine receptors because of chronic use of lorazepam.

Watch in particular for:

(a) **Monoamine oxidase inhibitors (M.A.O.I.)**

e.g. tranylcypromine (Parnate)
selegiline (Eldepryl)
phenelzine (Nardil)
moclobemide (Aurorix)

These were some of the first types of medication to successfully treat depression. Due to side effects (a risk of cerebral haemorrhage through a sudden rise in blood pressure is always present when M.A.O.I.'s are combined with foods containing amines such as cheese, broad beans, yeast extracts, etc., as well as C.N.S. stimulants such as amphetamines) they have almost vanished from medical practice except in the very occasional elderly

patient. MAOI's interact with all types of sedatives, anaesthetics, and analgesics so patients on them are difficult to manage.

Moclobemide (Aurorix) is a modern MAOI which has been altered to cause much less severe and significant reactions it may be continued safely and is safe to use with midazolam sedation

(b) **C.N.S. depressants, e.g. barbiturates, narcotic analgesics**

The effect of anaesthetic and sedative drugs, including midazolam, may be potentiated in patients taking C.N.S. depressants. However clinically this may not be apparent – remember to titrate midazolam to effect.

(a) **Tricyclic antidepressants**

e.g. amitriptyline (Amitrip, Laroxyl, Tryptanol)
clomipramine (Anafranil, Clopress)
and others

Inadvertant intravascular injection of local anaesthetic solutions containing adrenaline may lead to an exaggerated hypertensive response in patients taking tricyclic antidepressants. Care must therefore be exercised.

(d) **Cardiovascular drug**s.
Antihypertensive Drugs: A large and frequently changing group. See Mimms New Zealand drug manual.

(1) Neurotropic

Centrally acting amine synthesis blockers
e.g. methyldopa (Aldoment, Prodopa)

Reserpine and rauwolfia alkaloids
Clonidine (Catapres, Dixavit)

Peripherally acting ACE inhibitors
e.g. captopril (Capoten)
enalapril (Renitec)
lisinopril (Prinivil, Zestril)

Adrenergic neurone blocking drugs
e.g. bethandine (Esbatal)

Alpha-adrenoceptor blocking agents
e.g. prazosin (Minipress, Hyprosin, Pratsiol)
Beta-adrenoceptor blocking agents
e.g. propanolol (Inderal, Angilol, Cardinol)
metoprolol (Betaloc, Lopresor, Mycol)
(there are ten chemical variants)

Combine alpha- and beta-adrenoceptor blocking agents
e.g. labetalol (Trandate, Albetol, Hybloc)

Calcium blocking agents
e.g. verapamil (Isoptin, Civicor, Verapamil, Univer)
nifedipine (Adalat, Nical, Nyefax)

Direct acting vasodilators
e.g. hydralazine (Appresoline, Supres)

Ganglion blocking agents
e.g. trimetaphan (Arfonad)

Diuretics e.g. Frusemide

Combinations:

All these drugs, by definition, lower blood pressure - as can midazolam. Generally this is beneficial however care must be taken when sitting these patients up as many antihypertensive medications can cause postural hypotension

ii. Cardiac Glycosides – digoxin (Lanoxin)

Avoid the use of atropine and scopolamine, which may produce tachycardia. Adrenaline (endogenous or in local anaesthetics) increases myocardial sensitivity and may precipitate arrhythmias.

(e) **Tranquillisers**

i. Major Tranquillisers

(1) antipsychotics

(a) butyrophenones
- droperidol (Droleptan)
- haloperidol (Haldol, Serenace)

(b) dibenzoxazepines
- loxapine (Loxapac)

(c) diphenylbutylpiperidines
- fluspirilene (Imap)

(a) lithium (Lithicarb, Lithomyl)

(e) thioxanthenes
- flupenthixol (Depixol)
- thiothixene (Navane)

(1) phenothiazines

e.g. chlorpromazine (Largactil)
fluphenazine (anatensol, Modecate)
thioridazine (Aldazine, Melleril)
prochlorperazine (Stemetil)
thiopropazate (Dartalan)
trifluoperazine (Stelazine)
pericyazine (Neulactil)
perphenazine (Trilafon)
methotrimeprazine (Veractil, Nozinan)
pipothiazine (Piportil)

Extrapryramidal tract symptoms (tardive dyskinaesia and dystonia) may occur with the pheothiazines. Their hypotensive effects may also be potentiated by sedative and anaesthetic drugs.

i. Minor Tranquillisers

e.g. meprobamate (Equanil)
hydroxyzine (Atrarax, Vistaril)
benzodiazepines (many listed – see list in Pharmacology) of Benzodiazepines section, and New Ethicals Catalogue)

Synergism may occur with anaesthetic and sedative drugs, e.g. diazepam, midazolam, methohexital, pethidine and other opioids, nitrous oxide, etc(but not always).

ii. Antihistamines

e.g. promethazine (Phenergan)
dextrochlorpheniramine (Polaramine)
diphenhydramine (Benadryl)
trimeprazine (Vallergan)
astemizole (Hismanal)
terfenadine (Teldane)
(many others listed – see New Ethicals)

Antihistamines (especially Phenergan, Vallergan, and Valoid) can be sedating on their own and may therefore increase the sedative effects of midazolam. If possible, antihistamines should not be taken for 24 hours before sedation. The newer "non-sedating" antihistamines such as Teldane cause less sedation and so probably do not require the 24 hour stand down period.

(f) **Stimulants/ Drugs of abuse:**

e.g. amphetamine (Benzedrine)
dextroamphetamine (Dexedrine)
methamphetamine
methylphenidate (Ritalin)
cocaine

With the exception of methylphenidate, most of these drugs have been withdrawn from the market as having limited therapeutic advantages. However, illicit manufacture and supply continues.

This group of drugs is subject to widespread abuse. Patients taking them illegally will usually omit mention of this when completing the medical history form. Such patients often leave the medical history unsigned, and it is only after this is pointed out that the information may be reluctantly provided. Tactful questioning of suspect patients will often uncover a history of drug abuse, and the dental practitioner must always be alert to this possibility. If there is any doubt, do not treat the patient.

Patients taking prescribed stimulant drugs (e.g. Ritalin) must be warned not to take them for several days prior to treatment, as satisfactory sedation is often impossible to obtain in the presence of C.N.S. stimulation

(g) **Anticonvulsants**

e.g. diphenylhydantoin (Dilantin)
valproate (Epilim)
clonazepam (Rivotril)
carbamazepine (Tegretol)
phenobarbitone (Gardenal)
ethosuximide (Zarontin)
acetazolamide (Diamox)
clobazam (Frisium)
diazepam (Valium)

clormethiazole (Hemineurin)
primidone (Mysoline)

Dilantin and other anticonvulsants are commonly prescribed to control epilepsy. Midazolam and other sedative and anaesthetic agents are not contraindicated, but apprehension prior to treatment may lead to a seizure.

(h) **Corticosteroids**

See adrenal insufficiency

(i) Illegal Drug Consumption

This is a possibility which must always be kept in mind. Suspect patients who are unwilling to sign the medical history, and those who express an unusual interest in drugs used for sedation. Never treat if you suspect they have recently taken something (as risk of drug interactions).

Examples: Alcohol + benzodiazepines or other C.N.S. depressants may lead to respiratory depression.

Cannabis + benzodiazepines or other C.N.S. depressants may produce restlessness, disorientation, difficult (or impossible) to treat, and/or delayed recovery.

Cocaine + psychoactive and sedative/anaesthetic agents may lead to agitation, confusion etc.

N.B: Patients who have a past history of drug abuse are also much more likely to be resistant to drugs used for sedation.

Routes of Drug Administration

In general, oral methods of drug administration for premedication and sedation leave much to be desired. Because of different rates of gastric absorption and factors such as individual tolerance, age, habits, medical history, and degree of pre-operative apprehension, oral premedication is very unpredictable, particularly in adult patients. In fact, in very anxious individuals who have the greatest need for effective premedication, the stress of an impending dental operation may well delay the absorption of the drug until the least desirable time – for example during recovery from IV sedation or a general anaesthetic. A further disadvantage or oral premedication is that patients need to be supervised from the moment the drugs are taken, and accompanied to and from the dental office.

Intradermal, subcutaneous and intramuscular premedication are more reliable than oral premedication, but are relatively slow to take effect and are seldom painless. Also, all of these methods share the great disadvantage of oral premedication in that dosage must be determined arbitrarily, thus the effects will range from under-dosage to over dosage. The intravenous route in dentistry is outstanding for its ability to create a predictable and effective level of an effective agent, particularly for the ambulatory patient.

Advantages of the I.V. Route:

2. **Accuracy** - Because the effect is seen quickly, dosage need never be arbitrary. Administration can be carefully adjusted to the reaction of the patient until the desired result is achieved, thus minimizing the risk of over dosage.

3. **Rapid Drug Action** - The full effect of an intravenously administered drug is seen almost immediately, at the most within a few minutes (latency).

4. **Relatively pleasant** - Provided a careful venipuncture technique is employed, the administration of intravenous drugs should be painless. Sharp, sterile, fine gauge disposable catheters should be used, as well as surface obtundent on venipuncture site, e.g. EMLA patches.

5. **Increments** - Further increments of the drug may be readily administered as required ("titration") to maintain optimum drug action during treatment. Facilitated by the use of an indwelling catheter.

6. **Supervision** - Direct supervision of the patient during the time of peak action of the drug, which is not always possible with other routes of administration e.g. oral.

7. **Relatively Fast Rate of Recovery** - The peak drug effect is seen at the commencement of treatment, followed by a falling off of drug action during the operation and is always much faster than with the use of other routes of administration. The duration of after-effects is thus considerably reduced.

8. **Emergencies** - Usually best handled by the intravenous route e.g. acute adrenal insufficiency, acute allergic reactions.

Disadvantages:

1. Limited usefulness in young, uncooperative children. (Nitrous oxide sedation and/or oral sedation may be better or even general anaesthesia for the totally intractable).

2. Local complications may possibly occur, such as intra-arterial injection, thrombophlebitis and venous thrombosis.

1. Systemic complications due to over dosage may occur if rules for safe I.V. administration are not observed.

Precautions:

4. Intravenous procedures are apparently simple, but in fact require a very high degree of responsibility and care by the operator and ancillary personnel. Over dosage can easily occur (consider both drug concentration and speed of injection). Always observe the patient very closely during induction and always use an incremental dose. Never use an arbitrary "rule of thumb" dosage technique e.g. weight/dosage estimation, factors such as age, habits (alcohol, smoking, drugs), medical history and degree of anxiety are of great importance, and should be taken into consideration

   For safe I.V. administration – use dilute solutions where possible and a slow, titrated rate of injection; combined with careful observation of the patient.

2. **Staff** - Intelligent, interested, skilled and responsible ancillary staff are essential to the safe and efficient operation of an intravenous practice.

3. **Equipment and Facilities** - Proper office equipment (including emergency equipment) and facilities, as well as experience in their use, are essential. (Refer to lectures on technique and emergency procedures).

4. **Post-operative Care** - Adequate recovery facilities must be provided. The patient must be kept in the office until collected by a responsible person and supervised during recovery.

Training in I.V. sedation procedures and the allied pharmacology, and experience under supervision are of course essential before such procedures are undertaken. Although patients readily accept these methods, their use can only be justified if the safety factor equals or surpasses that of others in more general use. Also, the standard of treatment attainable must at least equal that attainable through other methods. If these criteria cannot be met, there is no place for these techniques in dentistry. Experience to date, however, has shown that safety and a high standard of treatment can be achieved by skilled, conscientious dental practitioners who are trained in the proper use of I.V. sedation.

Apart from the advantage to both patient and dentist in the removal of anxiety and stress from the dental situation, other benefits also result from the safe use of I.V. sedation. Complete treatments are often possible in one long appointment, rather than spread out over a series of short appointments. Although still traditional in dentistry, this piecemeal approach is inefficient for the dentist and is often a nuisance for patients with limited time at their disposal. Another important benefit is the reduction of emotional and physiological stress in patients.

Stages of Sedation and Anaesthesia

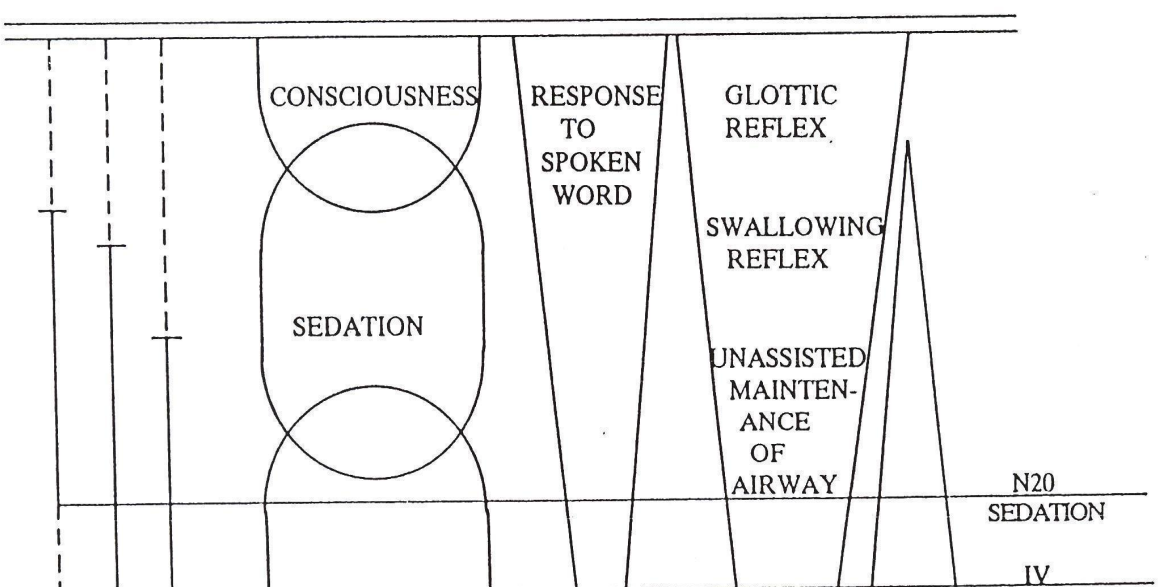
Since 1937, the Stages of Anaesthesia as devised by Guedel have been the yardstick by which depth of anaesthesia has been judged throughout the world. However, the Guedel signs and stages were described for ether anaesthesia and are unsuitable and ineffective to describe modern anaesthesia and sedation.

The second stage of anaesthesia, according to Guedel, was termed the stage of delirium. Professor Sir Robert Macintosh of Oxford University modified this in 1940 by suggesting that it was only potentially so; the patient reacting to pain stimuli without the restraint of self control. There is still no yardstick by which to define the level of a patient under administration of intravenous sedation drugs. Any one description would neither fit all patients, nor fit individual patients on different occasions, as it is impossible to relate a specific pain stimulus and individual to a level of consciousness. Many superimposed factors modify the reaction from person to person and from one occasion to another.

In modern dentistry we may wish to produce a level of amnesia or sedation, as a background to local anaesthetics. Next, a level of analgesia is recognized. Deeper still, general anaesthesia may be required to cover more intense stimuli.

Much dentistry can be carried out with little discomfort to the patient in the amnesia/analgesia zones which are very safe as there is minimal depression of normal protective reflexes. Below these levels the transition from consciousness to surgical anaesthesia is not sharply demarcated, particularly with the intravenous agents, where Guedel's Stage II is virtually non-existent. The type of dentistry planned will determine the level of pain control to be selected. Stage III general anaesthesia is now seldom required in dentistry, with exceptions such as major oral surgery and the management of some types of handicapped patients. Wherever possible it is advisable to keep sedation as light as possible and to supplement with local anaesthesia as necessary to provide analgesia. In this way the continued presence of the patient's protective mechanisms is assured, and because of lower dosages, a more rapid and complete recovery is likely to occur.

Stages of Anaesthesia and Sedation (simplified)



Requirements of the Ideal Sedative Agent for Dentistry:

1. **Safety** - Low Toxicity wide therapeutic safety margins, reliable, predictable actions, rapid, complete recovery following treatment.

2. **Selectivity** - Anxiolytic. Retention of patient co-operation (verbal contact maintained). Amnesia for unpleasant procedures. Muscle relaxant (especially muscles of mastication – long procedures). Analgesia (less important when supplemented with local anaesthesia/analgesia).

3. **Freedom from side effects** - Local, such as thrombophlebitis, Systemic such as respiratory and/or circulatory depression.

5. **Freedom from drug interactions**

6. **Simplicity of administration**

7. **High degree of patient acceptance**

8. **Higher quality of treatment possible than with other means**

Venipuncture:

Venipuncture Technique

After positioning the patient comfortably in the supine position:

1. Sit close to the patient.
2. Support the patient's arm (knee, table arm board) and immobilize the wrist.
3. Select the vein (inspection, palpation before occluding the vein).
4. Stop venous flow (tourniquet or hand pressure).
5. Clean the skin with a swab (alcohol or chlorhexidine) if you want.
6. Estimate the centre of the lumen and immobilize the vein.
7.
8. Enter at 30°, align the vein, block off vein with finger or thumb, withdraw needle-leaving catheter in place.
9. Place valve or cap on end of cannula.
10. Ensure tourniquet is released (assistant should double check).
11. Inject a few drops of solution, it should inject easily, this is to check you are in the vein. Alternatively inject a few mls of saline.
12. Execute slow rate of injection (with close observation of the patient throughout).
13. Extract cannula with one quick movement at conclusion of treatment.
14. Exert pressure on the vein at least 30 seconds (sterile swab) to prevent haematoma.

Complications of Venipuncture: Prevention and Management

POTENTIAL COMPLICATIONS WITH USE OF ANTECUBITAL FOSSA:

1. Entry into brachial artery or its superficial ulnar branch. Avoid median basilica vein, use cephalic or median cephalic vein. Always palpate first.

2. Damage to the median nerve.

DISADVANTAGES OF DORSUM OF HAND FOR VENIPUNCTURE:

The dorsum of the hand or wrist is commonly used in medical general anaesthesia, where the patient is lying flat on an operating table, and it is thus more convenient. However, in the dental chair, the antecubital fossa is more suitable and easier to immobilize. There are also a number of other disadvantages related to the dorsum of the hand in the dental situation.

1. More difficult entry as skin tends to be tougher than the antecubital fossa.

2. Veins are usually smaller (increased risk of thrombophlebitis).

3. Veins poorly supported in connective tissue – tend to slide away from needle.

4. Veins often thickened, with narrowed lumen in older patients.

5. Greatly increased risk of haematoma production due to above factors.

6. Haematoma in this region cannot be easily concealed by patient, unlike haematoma in antecubital fossa, with possible damaging effect on professional reputation.

7. Anchorage and stabilization of cannula more difficult during lengthy procedures in the dental chair (unlike operating table). Cannula may slip out of vein thus necessitating new venipuncture, and increasing likelihood of haematoma.

COMPLICATIONS OF VENIPUNCTURE:

The complications of venipuncture are due mainly to faulty injection technique. They can cause great inconvenience and possible severe and permanent disability to the patient, damage to the reputation of the administrator, and in some cases may lead to litigation.

1. **Haematoma** (the most common complication)

   Cause: Leakage of blood into surrounding tissues.

   Prevention: Immediate pressure on vein after removal of needle or after failed venipuncture. Use antecubital fossa in preference to back of hand.

   Treatment: Local heat packs, pressure, massage, time.

2. **Extravenous Injection**

   Cause: Catheter lumen partially or completely outside vein.

   Prevention: Never inject unless absolutely certain the lumen of the catheter is completely within the vein. Catheter location must be checked frequently.

   Treatment: A small extravenous deposit (one or two drops) of solution will usually disperse and be absorbed without harm if the site is immediately massaged. Larger amounts may require dispersion by side infiltration of the area with up to 10 ml of 1% procaine solution with hyaluronidase ('Hyalase'). Lignocaine is of no value as it is not a vasodilator.

   Injection of large amounts of solution extravenously can only be considered negligent. One or two small drops of solution may however, be unavoidable on occasion, e.g. very small veins, needle half-in, half-out (allowing positive aspiration, but only partial injection into the vein).

3. **Intra-arterial Injection**

Can be considered negligent, particularly if an amount greater than one or two small drops is injected.

   Cause: Catheter lumen within artery instead of vein.

   Prevention: Careful examination and palpation before applying tourniquet. Avoidance of areas where arteries are known to be present (watch for aberrant superficial ulnar branch of the brachial artery). Careful checking of aspirated blood for colour, pulsation. Always start injecting a very small initial test dose. Stop if patient complains of pain radiating down arm from forearm to the hand and fingers (differentiate from pain radiating up the arm due to irritant or cold solution). Never inject if in any doubt as to catheter location.

Symptoms: Severe burning pain radiating down arm. Blanching of the skin. Weakness or absence of radial pulse (late symptom indicating thrombosis). Thrombosis eventually leads to gangrene and possible amputation if not treated successfully.

Treatment: Leave needle in artery. Inject up to 10 ml 1% lignocaine to help relieve pain, dilute drug injected, and relieve arterial spasm, then remove cannula. If pain persists or doubt exists as to prognosis, patient must be hospitalized for anticoagulant therapy and possible brachial plexus or stellate ganglion block. Full notes must be sent including time of injection, site of injection, drug(s) used, dosages and solution percentages, and any treatment given.

NO sedation and dental treatment should be carried out following suspected intra-arterial injection, as symptoms could be masked resulting in irretrievable damage.

4. **Injection into Nerve**

Cause: Probing in area where important nerves are known to be present e.g. medial nerve in inner aspect of antecubital fossa. Sensory or motor paralysis may result. Risk increased with use of larger gauge, long bevel needles.

Prevention: Only visible, superficial veins in the outer aspect of the antecubital fossa should be chosen wherever possible, particularly by those inexperienced in venipuncture.

Treatment: Refer for medical consultation if necessary and if pain persists.

5. **Thromphlebitis**

Cause: May be caused by an irritant solution remaining in contact with the vein wall or being injected into the vein wall; or by damage to the vein wall during venipuncture or catheter insertion; or prolonged use of an IV Catheter.

Specific Causes:

a) Irritant solutions – Many sedative and anaesthetic drugs are irritant to varying degrees e.g. thiopental, early diazepam preparations, hydroxyzine, etc. Any restriction to venous flow which allows the solution to remain in contact with the vein can result in irritation which may produce thrombosis. Concentration of solution and speed of injection are important.

Preventing by dilution where possible, slow injection into large veins, and post-injection flushing with normal saline or 5% dextrose in water where irritant solutions used (particularly where pain noted during injection). Thrombophlebitis tends to recur in some patients (local allergic reaction).

b) Large needles – Greater risk of damage to small veins. Similarly with use of I.V. catheter.

c) Long bevels – greatly increase the risk of the 'half-in half-out' position, particularly with restless patients where the arm and the wrist are not properly immobilized.

   Prevent by using large veins wherever possible, by ensuring needle and arm cannot move during administration, and checking for damaged needle points by careful observation and wiping suspect needles across sterile gauze.

d) Smaller veins – More easily damaged during venipuncture and subsequent patient movement. Greater risk of haematoma, perivascular leakage.

   Avoid where possible, watch for 'half-in half-out' position, and apply immediate pressure following removal of needle to reduce risk of haematoma formation. Use short bevel needles if possible.

e) Restricted venous return – May be due to tight sleeve, incompletely loosened tourniquet, arm board or elbow support straps too tight. Any tendency to venous stasis may result in thrombosis formation.

   Prevent by instructing patient to wear loose clothing, and checking tourniquet and arm board or elbow support straps are not too tight.

f) Arm movement – May result in vein damage from needle, or tightening of arm strap, clothing or tourniquet.

g) Inflammation – May be due to perivascular leakage from failed venipuncture to certain drugs or solvents, e.g. diazepam. Pain may be noticed running up arm during injection.

Reduce risk of subsequent thrombophlebitis by flushing with several ml of water for injection, or saline after administration to remove remaining traces of solution from the vein.

h) Infection – due to neglect of proper sterilization precautions. Indefensible. Always use disposable items where possible, autoclave others.

Treatment: Rest arm (sling)
Local heat, warm soaks.
A non-steroidal anti-inflammatory analgesic may be prescribed provided treatment is started early or prescribed prophylactically. Prophylactic antibiotic therapy should also be considered.

Severe cases may require hospitalization for anticoagulant therapy (heparinisation) + antibiotic therapy.

6. **Posture Hazards**

Faulty posture may cause injury to nerves, muscles or joints.

a) Brachial plexus: Stretching of the cords of the brachial plexus may occur in a sedated or unconscious patient if the arm is allowed to be abducted and externally rotated. Varying degrees of paralysis may result, either temporary or permanent, depending on the amount of trauma. Prevent by keeping the patient's arms adducted (close to side) and internally rotated.

a) Radial nerve: May be injured by pressure on the lateral side of the arm at the insertion of the deltoid muscle.

b) Ulnar nerve: May be injured by pressure at the elbow behind the medial epicondyle of the humerus or on the medial side of the forearm.

c) Back injury: May result from lack of cushioned support.

d) Neck injury: May result if the head of the sedated or unconscious patient is allowed to fall backward without support. The older split headrest type chairs are particularly dangerous in this respect. Extreme care with patients who have a past history of whiplash injury.

PHARMACOLOGY

Benzodiazepine Pharmacology

Midazolam (Hypnovel or in the U.S.A. Versed) is a sedative belonging to the benzodiazepine (BZ) group of drugs, which also includes chlordiazepoxide (Librium), nitrazepam (Mogadon), triazolam (Halcion), lorazepam (Ativan), temazepam (Euhypnos), oxazepam (Serepax), diazepam (Valium)and many others.

Presentation:

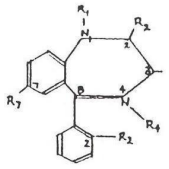
1. **Midazolam:** 7.5 mg tablets; and ampoules for injection (5 mg/5 mls and 15 mg/3 ml).

Benzodiazepine Pharmacology

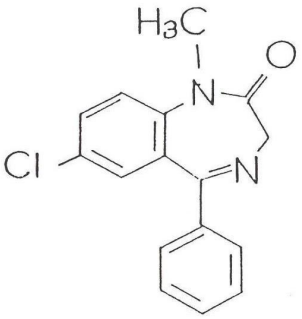
The 1,4-Benzodiazepine nucleus is the basis of a very large number of Benzodiazepine derivatives that either possess the typical agonist properties of anticonvulsant, muscle relaxant, hypnotic, sedative and tranquillising effects, or do not (so-called “antagonist” Benzodiazepine derivatives). These notes will consider two Benzodiazepines only Midazolam (Hypnovel), and flumazenil (Anexate).

The clinically useful Benzodiazepine agonists are 1,4-benzodiazepines, and most contain a carboxyamide group in the 7-membered heterocyclic ring structure. A substituent in the 7 position, such as a halogen or a nitro group, is required for sedative-hypnotic activity. Both Midazolam and Flumazenil exhibit an additional Imidazole ring structure. Midazolam exhibits a pH-dependent ring-opening phenomenon, whereby the Benzodiazepine ring closes at pH values >4.0 but opens reversibly at pH values <4.0. Physiological pH of 7.4 maintains the closed ring structure and may enhance lipid solubility. In an acidic aqueous solution, Midazolam is water-soluble and the parenteral formulation therefore does not contain organic solvents (such as propylene glycol in early Diazepam preparations) and is instead buffered to an acidic pH of 3.5. Once Midazolam enters the body, the pH rapidly increases to 7.4 and the solubility properties of the drug change. At physiological pH, Midazolam becomes highly lipid-soluble which increases the rate at which it enters the brain and increases the rate of onset following I.V. administration. Midazolam is the most lipid-soluble Benzodiazepine currently available, followed next by Diazepam. The water-solubility of Midazolam, results in a low incidence of injection pain and venous thrombosis.

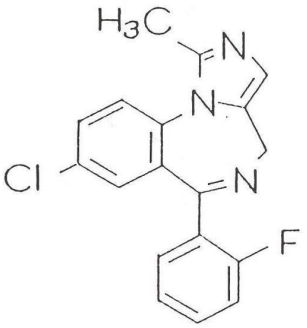
Chemical Structures



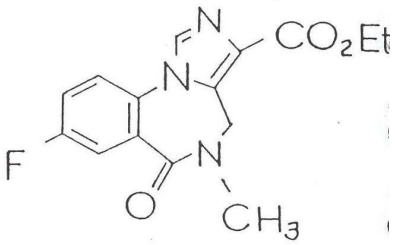
1,4 BENZODIAZEPINE NUCLEUS



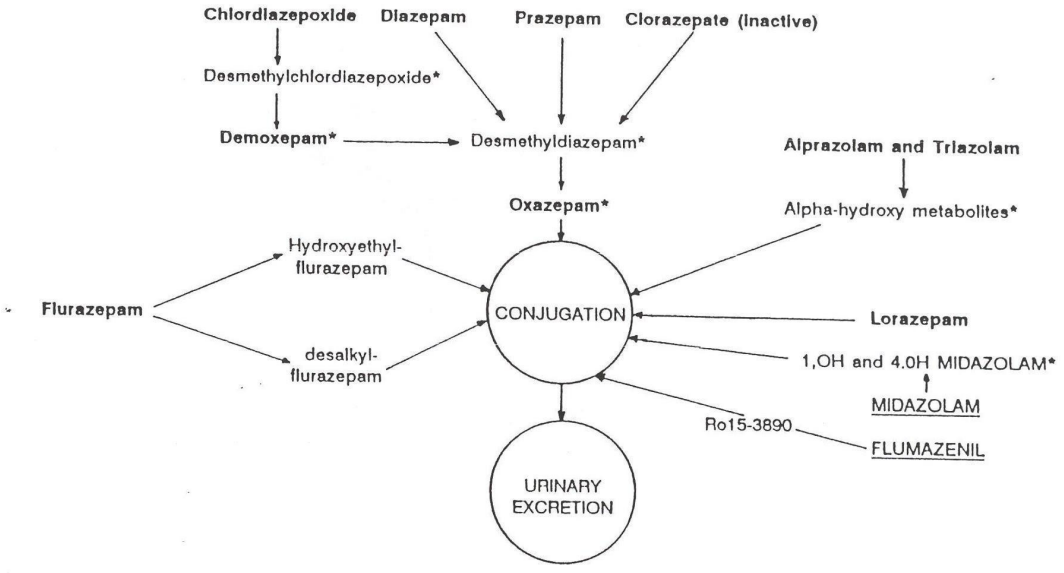
DIAZEPAM



MIDAZOLAM



FLUMAZENIL



Biotransformation of benzodiazepines (* = active metabolite)

Gamma-aminobutyric acid (GABA) is an important inhibitory neurotransmitter in the brain, and glycine is the major inhibitory neurotransmitter in the spinal cord and brain stem. Agonist Benzodiazepines augment or facilitate GABA-ergic neurotransmission, producing sedation and anticonvulsant activity, while other central nervous system effects, such as anxiolysis and muscle relaxation, appear to be due to glycine-mimetic effects in the spinal cord and brain stem. Specific receptors for the Benzodiazepine's were first identified in the central nervous system in 1977. The highest concentration of receptors is found in the cerebral cortex, followed by the hypothalamus, cerebellum, corpus striatum and finally, medulla. The identification of receptors (specific high-affinity binding sites) for the Benzodiazepines has led to the development of selective antagonists that bind to the Benzodiazepine receptor and prevent Benzodiazepine agonist drugs from occupying receptor sites. The Benzodiazepine antagonist flumazenil (Anexate) is described later. The regional distribution of the Benzodiazepine receptors parallels the distribution of GABA receptors, which suggest that, although Benzodiazepines do not appear to bind together receptor sites, they probably have some influence on GABA-containing neurons, since the overall effect of Benzodiazepines binding to their receptors is to enhance the inhibitory effects of GABA. The relative receptor-binding affinities appear to correlate to some extent with the relative potencies of Benzodiazepines. In rat models, the rate of association with the Benzodiazepine receptor for Midazolam is slightly greater than for Diazepam and more than thirty-fold greater than for Lorazepam (Ativan), indicating that Midazolam and Diazepam should have a more rapid onset of action. Conversely, Diazepam and Midazolam dissociate from the receptor faster than Lorazepam and therefore Lorazepam has a longer duration of action than the other two Benzodiazepines. Midazolam has about twice the affinity for Benzodiazepine binding compared with Diazepam and this contributes to the greater milligram potency of Midazolam (approximately 2.5 x that of Diazepam).

In addition to the agonist Benzodiazepine and antagonist Benzodiazepine molecules that bind to brain Benzodiazepine receptor sites, a third group of anxiogenic Benzodiazepine receptor compounds exist, not themselves Benzodiazepines, some of which are naturally occurring (D-carbolines). These molecules can block the effects of Benzodiazepine agonists, and when administered alone produce effects such as anxiety reaction, pro-convulsant activity and seizures. These compounds have been termed "inverse agonists" and are of theoretical interest in terms of their possible role as natural endogenous mediators of anxiety reactions.

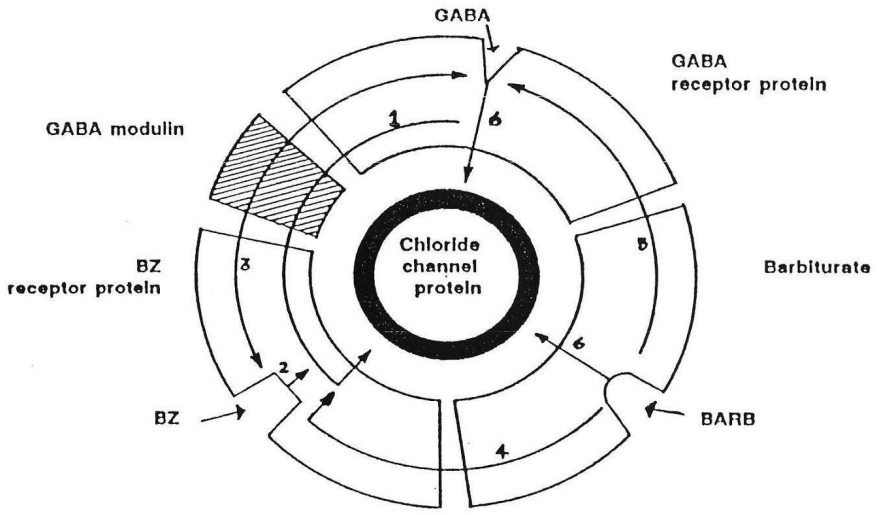


Figure 21-5. Proposed model of the GABA-benzodiazepine-chloride channel receptor complex. GABA binding to its receptor activates the chloride channel through a coupling mechanism involving GABA-modulin and the benzodiazepine (BZ) receptor protein (1). Binding of benzodiazepines (BZ) enhances this coupling function (2). and increases GABA binding in a reciprocal relationship (3). Binding of barbiturates (BARB) also enhances the coupling function (4). increases the affinity of GABA for its receptor (5). and may directly activate the chloride channel at high concentrations (6). GABA = gamma-aminobutyric acid.

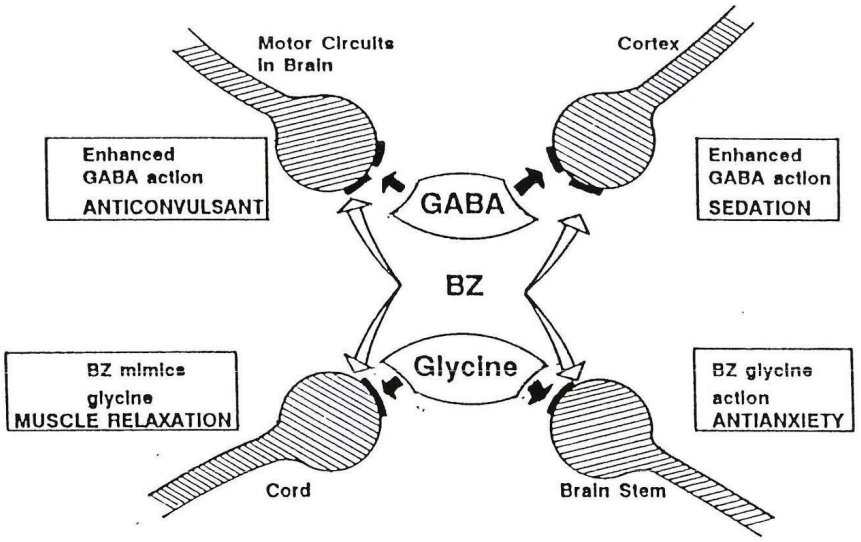
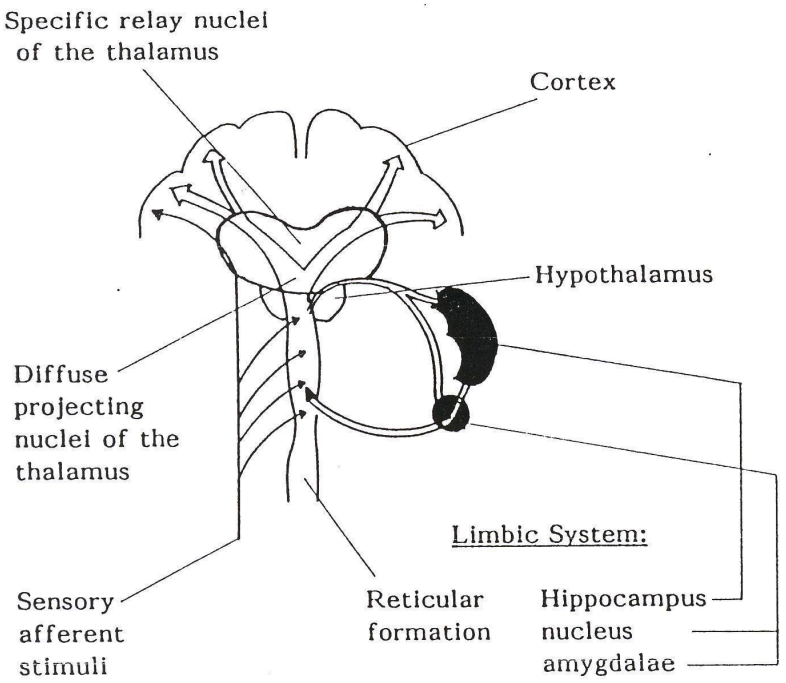


Figure 8.11. Pharmacologic mechanisms for the benzodiazepine drugs. The schematic diagram illustrates the actions of -aminobutyric acid (GABA) and glycine in presynaptic nerve terminals. The benzodiazepine drugs (BZ) facilitate the inhibitory actions of GABA and mimic the inhibitory actions of glycine. (With permission from Richter JJ: Current theories about the mechanisms of benzodiazepines and neuroleptic drugs. *Anesthesiology* 54:66-72, 1981.)

BENZODIAZEPINES



The principal sites of action (shown in black) of Benzodiazepine in doses producing a demonstrable effect on the C.N.S.

Benzodiazepine Pharmacodynamics

1. **Central Nervous System:**

The Benzodiazepines interfere with interneuronal transmission, producing skeletal muscle relaxation due to inhibition of polysynaptic spinal reflexes (more pronounced with Diazepam) and sedation due to inhibition of the limbic system – the area of the cortex which is believed to mediate emotional influences on cortical activity. The continuing activity of reverberating circuits among the neurons of this system is thought to contribute to anxiety states. Because of their pronounced anxiolytic activity, the Benzodiazepines have found widespread use in the treatment of anxiety states and insomnia, as well as in the field of premedication.

Obviously, a drug which will selectively inhibit the activity of the limbic system and thus emotional response to external stimuli (anxiety, fear, rage etc.) also has potential for the management of anxious and apprehensive dental patients.

The limbic system comprises the amygdala, hippocampus, and cingulated gyrus. Experimentally, stimulation of the amygdala is followed by electrical activity in the hippocampus, but after injection of Diazepam or Midazolam the hippocampal response to stimulation of the amygdala is reduce; though not to stimulation of the hippocampus of the opposite hemisphere. This suggests that the amygdale may be the site of action of these drugs, breaking the patterns of neuronal activity responsible for anxiety.

This highly selective action of the benzodiazepines is of great clinical importance. Instead of general C.N.S. depression, such as that produced by the barbiturates, Midazolam in clinical dosages acts mainly upon the area of the brain responsible for anxiety – the limbic system – without significant respiratory, autonomic, or extrapyramidal activity. However if the dose is increased beyond that required for anxiolysis generalized CNS depression (coma) can occur.

The Benzodiazepines do not possess analgesic properties, thus routine supplementation with local anaesthesia is required for painful procedures. An apparent “analgesic effect” noted with some apprehensive patients who, following administration of Midazolam do not react to painful stimuli when local anaesthesia is withheld, is probably due more to the marked anxiolytic and relaxant actions of the drug reducing the normal response of the patient to painful stimuli. (Decreased anxiety leads to increased pain tolerance – see first section).

Amnesia (anterograde) is an important and useful feature of Midazolam. Perception is unaffected, but memory consolidation is affected markedly. Even when amnesia is minimal, patients usually agree that their dental treatment has been much more tolerable instead of an ordeal. The degree and duration of amnesia shows wide variation between patients, and increases with dosage.

In general, approximately 10 minutes of good amnesia follows the administration of Midazolam when administered I.V., which covers the administration of local injections. This is followed by up to an hour or more of impaired memory function. It is therefore important to remember that although a patient may appear to be awake and alert after treatment, they may in fact still be amnesic for some time. Amnesia may also be reinforced during treatment by distracting the patient's attention from the dental procedure by various means e.g. conversation, questions, music, etc.

2. **Cardiovascular System:**

Large doses of Benzodiazepines given to patients with heart disease can cause haemodynamic instability. The small doses used for sedation are not usually associated with significant cardiovascular changes, apart from those associated with anxiety reduction (fall in heart rate and blood pressure). Likewise, any anti-arrhythmic effect at low dose is secondary to anxiety reduction.

3. **Respiratory:**

Anaesthetic doses of Benzodiazepines cause dose-dependent respiratory depression, which can lead to apnoea. Small titrated intravenous doses do not affect the ventilatory response to carbon dioxide much, so that doses of midazolam used for premedication and sedation are not often associated with clinically important respiratory depression in healthy individuals. However there is a wide patient variability in the sensitivity to the effects of benzodiazepines including respiratory depression so always monitor your patient ! The drugs do not cause bronchoconstriction, but in patients with chronic obstructive pulmonary disease, the respiratory depressant effects of Midazolam may be greater and more prolonged than those associated with normal, healthy patients, and the same applies to the elderly.

N.B. The above applies to the use of Midazolam as a sole agent. When used in combination with respiratory depressants such as opiates where potentiation may occur, apnoea and severe hypoxemia are likely. Numerous reports of morbidity and mortality have appeared in the literature in such instances, particularly with Midazolam / opiate combinations. These combinations are not advised for routine use in the dental surgery due to their inherent risks.

Similarly, the likelihood of respiratory (and circulatory) depression is increased by the use of premedication, particularly with opiates, and must be carefully monitored. (In general, the use of premedication is unnecessary and undesirable prior to most dental procedures, particularly where good communication skills and doctor/patient relations are present.)

It is also important to realize that Benzodiazepine muscle relaxation can easily cause partial or complete upper-airways obstruction with little direct depression of the respiratory centre. Clearly, the doctor and assistant must ensure that the chin is properly supported and the airway kept patent throughout treatment.

4. **Muscle Relaxation:**

There are several important implications for dentistry in this property of Benzodiazepines.

Gag-reflexes are obtunded, due to the Benzodiazepine action on polysynaptic reflexes to interfere with interneuronal transmission, although monosynaptic reflexes (knee-jerk) are unaffected as is stimulation of muscles through its motor nerve. Thus Benzodiazepines are useful for patients with dystonic and athetoid types of cerebral-palsy, and severe bruxism may also be temporarily controlled by prescribing oral Benzodiazepines – best taken before bedtime. Trismus following dental extraction may also respond to oral treatment with Benzodiazepines.

The half-way ptosis or “Verrill sign” is no longer considered of major importance as most patients are relaxed, amnesic and co-operative at dose levels well below those required to produce ptosis. EVE signs are now recommended as a useful clinical test that avoids over sedation.

Benzodiazepine Pharmacokinetics

Midazolam is metabolized within the liver by hydroxylation to the major metabolite 1-Hydroxymidazolam and a secondary metabolite 4-Hydroxymidazolam. Both metabolites are conjugated and then excreted in the urine as glycuronides. Although the metabolites have pharmacological activity, they are probably of little clinical importance. Very little unchanged Midazolam (less than 1%) is excreted in the urine. Since Midazolam is highly lipid-soluble it rapidly crosses the blood-brain and placental barriers to gain access to Benzodiazepine receptors in the C.N.S. Following intravenous administration of 5 mg of Midazolam, concentrations decline bi-exponentially with a distribution of half-life of 30 minutes and an elimination half-life that may range from 1-4 hours (compare with Diazepam in table). The total clearance approximates 50% hepatic blood flow. Oral Midazolam undergoes substantial first-pass hepatic extraction so that about 50% of the drug does not reach the systemic circulation.

The table shows that Midazolam has a relatively large volume of distribution, short elimination half-life, and a high clearance, giving the drug a short duration of action and rapid recovery after single dose intravenous administration. This makes midazolam particularly suitable for short procedures (30-60 minutes), such as cavity preparation and short surgical cases.

Accumulation is less likely to occur following repeated doses of Midazolam due to its high clearance and rapid re-distribution.

Midazolam elimination half-life is also increased and clearance reduced in elderly males. Midazolam volume of distribution is increased in obesity leading to a prolonged elimination half-life with no change in clearance. This calls for dose reduction – on a per kilo basis a 120kg person does not need twice as much as a 60kg person but they may well need more. There is minimal alteration of Midazolam clearance in chronic renal disease. Although plasma protein binding of Midazolam (94-96%) is reduced in renal disease, the pharmacokinetics of unbound Midazolam is unchanged.

Great care should be exercised if midazolam is used in people with neuromuscular conditions such as myasthenia gravis. These patients already have or can have weak muscles and midazolam can aggravate this with dangerous (respiratory) consequences.

Comparison of Pharmacokinetics

	Diazepam	Midazolam	Flumazenil
Protein binding %	96	40	
Volume of distribution (L/Kg)	1.1-1.7	0.63	
Plasma clearance (ml/min)	265	690	
Distribution half-life (minutes)	30	5	
Elimination half-life (hours)	2	0.97	

Clinical Significance of the Pharmacodynamics

There is a delay of at least 1-3 minutes before the onset of effect following I.V. administration ("latency") and a wide individual variation in response to the drug. The normal dose range of Midazolam required to produce adequate sedation, relaxation, and co-operation in most adult patients lies between 1-8 mg. However, factors such as age and current drug therapy will require dosage to be modified. Elderly patients in particular are often much more sensitive to most drugs, and dosage must be greatly reduced in such cases, sometimes to as little 0.25-1 mg midazolam in very elderly people. For these reasons, a slow rate of injection is essential, along with close observation of the patient during administration. This is often referred to as titration.

Important Note: Always keep in mind Midazolam is a specific anxiolytic drug and should relieve anxiety and produce amnesia well before other signs, such as drowsiness, ptosis etc, become evident. The patient need not necessarily be made to look "sleepy".

Duration of clinical action varies, but in most cases up to 30 minutes of sedation occurs with Midazolam. Many patients will tolerate an extension beyond this period without distress although apparently fully recovered. However, as Midazolam has a half-life 2-4 hours there will still be an appreciable time following apparent recovery before the drug is completely detoxified. It is thus essential to see that all patients are taken home and supervised after treatment, even if they appear quite "normal", due to the possibility of prolonged sedative and amnesic after-effects in isolated cases
With Midazolam, an upper maximum dose limit of 10 mg is advised. Paediatric dosage should be titrated on the basis of 0.05-1 mg/kg, bearing in mind that the dose response is poorly related to weight.

As Midazolam affects psychomotor function for several hours following administration, driving motor vehicles, operating machinery, and undertaking important business matters must be prohibited the same day, as must the use of alcohol.

Chapter Summary: Midazolam

Presentation	Aqueous solution 5 mg/5 ml amps 15 mg/3 ml amps
Pain on injection	Uncommon
Venous Thrombosis	Uncommon
Anterograde Amnesia	Excellent (brief) amnesia.
Onset of action	May be delayed – slow titration important, signs and symptoms not obvious.
Signs and Symptoms	Anxiolysis Sedation (may be delayed) Slurred speech uncommon Nystagmus uncommon Ptosis uncommon
Respiratory and CV depression	Minimal at sedative dosages; more likely with rapid administration (especially respiratory depression) or combined with other C.N.S. drugs (hypoxaemia)

PHARMACOLOGY –FLUMAZENIL (ANEXATE)

This imidazodiazepine derivative is structurally similar to Midazolam but essentially lacks the agonist properties of the clinically used Benzodiazepines. Thus given alone even in enormous doses, it has no clinical actions. However, in the presence of an agonist Benzodiazepine, flumazenil blocks the central nervous system effects of sedation, amnesia, anxiolysis, muscle relaxation, ataxia, anticonvulsant, sedative and amnesic properties. It is specific for the Benzodiazepine receptor and therefore does not antagonize the effects of barbiturates, opiates or ethanol

Following intravenous administration the onset is rapid (within 5 minutes) but the duration of effect of the usual dose (0.1-0.5 mg) is relatively short (about 1-3 hours) in keeping with its short elimination half-life of about 60 minutes. The administration of flumazenil to patients sedated with Benzodiazepine produces no appreciable cardio-respiratory changes apart from reversal of any depression. The dose required to reverse BZ sedation is approximately 0.4 mg intravenously with an initial starting dose of 0.1 mg: further 0.1 mg increments maybe given up to a maximum of 1.0 mg. (Midazolam remaining in the I.V. line is totally compatible, so no flushing is required.) Although intellectually the patient may appear completely reversed, ataxia may persist and be apparent when the patient is asked to stand. The pharmacokinetics dictate that re-sedation is a distinct possibility with flumazenil reversal of Midazolam sedation so that patients must not be discharged prematurely. However, (except in the aged or infirm) return of significant sedation is not likely in Midazolam doses up to 10 mg.

Advantages of Flumazenil:

1. Use as an emergency drug for over sedated patients.

2. Reversal of untoward reactions to BZ agonists e.g. paradoxical excitation.

3. Accelerated recovery in certain situations, e.g. elderly patients with slow recovery.

4. Interruption of sedation in certain situations, e.g. surgical placement of prosthesis, endoscopy, certain neurosurgical procedures (trigeminal nerve ablation).

5. Reversal of sedation at end of procedure (controversial) See: “Midazolam – Is Antagonism Justified?” in reference section.

Presentation I.V. - 0.5mg/5 ml, 1 mg/10 ml

Disposition:

Liver-metabolised to inactive glycuronides; metabolite is independent of midazolam. Does not affect midazolam pharmacokinetics and vice versa.

Toxicity, Precautions, Contraindications:

Vein tolerance as good as midazolam. Contraindicated in BZ addiction as may precipitate sudden withdrawl crisis.. "it is inadvisable to risk administering I.V. sedation to patients who would not normally be sedated due to their age, psychological make-up or medical history, merely because an antagonist is available."

Cyclopyrrolone derivatives used as hypnosedatives (Zopiclone = "Imovane") though not Benzodiazepines, act on the Benzodiazepine receptor and are reversed by Flumazenil.

Chronic Benzodiazepine Habituation:

Patients consuming chronic high-dose Benzodiazepine medication will be resistant to the recommended I.V. sedation doses of agonist Benzodiazepines and it may in fact be quite difficult to sedate even with large doses. Sudden withdrawal of Benzodiazepines medication in these patients can cause an acute withdrawal syndrome that can include convulsions, and extreme anxiety. Because of this the use of Flumazenil in these patients is contraindicated. Presumably this applies also to Zopiclone abuse.

MIDAZOLAM – IS ANTAGONISM JUSTIFIED? I

Under the above title, an editorial in the Lancet of July 16th 1988 considers the use of midazolam itself for sedation, largely in endoscopic procedures, and also the reversal of its effects by flumazenil.

There is, apparently, an increasing tendency for minor surgical (including endoscopic) procedures to be carried out by the operator under sedation provided by midazolam. There is also, apparently, a lack of appreciation of the possible dangers on the part of the non-anaesthetically-trained administrators. This has reached such a point in the USA that their Food and Drug Administration (FDA) has been called upon to forbid the use of midazolam for conscious sedation in any patient, and its administration for any reason at all to any person over the age of 60 years.

To some extent, this stage of affairs can be considered as an over-reaction to the initial lack of appreciation of the drug's potency. It is also an indictment of the common medical habit of giving drugs on a rigid rule-of-thumb, dose-for-weight basis, instead of a careful titration against observed effect. There is no doubt that the early recommendations were influenced by the popular and widespread use of diazepam; an eminently safe drug with which a relative overdose normally presented few, if any, problems. The endoscopist inexperienced in sedation techniques could take his ampoule of diazepam, multiply weight by the standard dose factor, and inject the resultant quantity with every expectation of success. The time the patient took to recover was really immaterial, given an adequate nursing presence.

In dental use, the extra potency of midazolam was recognized very soon, as was its much enhanced effect in the elderly. This happy state of affairs was due, almost entirely, to the universal dental habit of titrating the dose incrementally against the patient's responses. Where this was not done, undesirable side effects were common, and fatalities all too frequent. Midazolam was released for use in the USA in May 1986: by January 1988, six deaths associated with an overdose of midazolam had been reported to the FDA, despite the fact that in November 1987, the recommended dosage in the USA was reduced to 0.07 mg/kg for adults and 0.05 mg/kg for elderly patients.

The UK has not been exempt. In addition to four deaths reported to the Committee on Safety of Medicines (CSM), there have been reports of respiratory depression, respiratory arrest, cardiac arrest, hypotension and heart block. The majority of those patients had been given midazolam for conscious sedation, and at least one of the fatalities had been given a dose greater than that recommended by the manufacturer.

“Sedation for endoscopy”, runs the editorial, “is undertaken usually by the endoscopist, little or no monitoring used, and the procedure is often carried out in a dark environment; consequently, cardiac or respiratory depression may go unnoticed for some time. In addition, the endoscopist may be unfamiliar with techniques of cardiorespiratory support if such measures become necessary.” Yet this situation is not only tolerated; it is implicitly accepted. Where is the campaign against the endoscopist-sedationist? After such a condemnation, it is unwise to condemn the trained, experienced and conscientious dentist sedationist and to demand ever more elaborate and expensive monitoring equipment with the intention (overt or covert) of making the whole process so complicated for him as to become unviable.

Happily, the editorial itself exhibits no such bias. It makes exactly those recommendations of technique which are standard in dental practice, and which have contributed so much to its safety.

“There is little doubt”, it says, “that midazolam can be used safely for conscious sedation if the correct dose is used, due allowance is made for the patients’ age, and appropriate precautions are taken. There is considerable inter-individual variation in response and the dose must be titrated carefully; the introduction, in both the UK and the USA, of a formulation containing 2 mg/ml facilitates titration.” There is more, and it is all good sense.

The editorial also considers the increasing use of flumazenil, the first direct benzodiazepine antagonist, to reverse the sedation of midazolam; either for correction of an overdose; for accelerating the speed of recovery; or for the convenience of the operating or nursing staff. It condemns all three, and these matters are considered further in this issue of the Digest.

Unfortunately, the Lancet does not follow the habit of it’s sister journal, the BMJ and attribute it’s editorials. One does not know whom to congratulate. We must hope that he or she is an anaesthetist, and a dental anaesthetist at that!

MIDAZOLAM – IS ANTAGONISM JUSTIFIED? II

Ever since the introduction of Naloxone (Narcan) as an antagonist to the opiate drugs the search has been on for an effective antagonist to the benzodiazepines.

With the introduction of flumazenil (Anexate), this situation has changed dramatically. For the first time, it is now possible specifically to reverse the action of the benzodiazepine, particularly midazolam, and produce what appears to be a remarkable recovery. The use of this drug for this purpose has been widely advocated both in dental and in medical circles.

Reading the literature, one could be forgiven for believing that a new day had dawned. In fact, the drug has given rise to unexpected problems; both pharmacological and ethical.

First, flumazenil has an elimination half-life of less than one hour: less than one-half that of midazolam in the normal healthy adult and very much less than that of midazolam in those who are elderly, or who have impaired metabolisms

Second, flumazenil works by displacing the midazolam from its receptor sites: it does not affect its elimination from the body, so that when the flumazenil itself is eliminated, that portion of the midazolam which has not been metabolized will still be available and active. Consequently, there is always the possibility that patients may become re-sedated after their discharge. This effect will be dose-related, but it will always present a potential danger.

Third, there may be a temptation, where the sedation is less than ideal, to increase the dose of midazolam to an undesirable degree and then to use flumazenil to reverse the sedation at the end of treatment. This is a highly dangerous attitude: the cardio-respiratory depression which is always present when midazolam is used is also dose-related. The mortality and morbidity which have been recorded as associated with the use of midazolam have most often stemmed from over-dosage, and it seems likely that this follows an exponential curve. Carefully titrated against the patients responses, and never exceeding the recommended maximum dose level, midazolam seems to be entirely safe, but its dangers increase dramatically as the boundaries are disregarded.

Fourth, the pressures of a busy operating list may lead to the use of flumazenil as a means of getting rid for the patient sooner than a natural recovery would allow. This has, indeed, been advocated as helping to reduce the pressure on both medical and nursing staff. Thankfully, this sort of condition does not normally arise in dental practice: nor should it be allowed to. Not only is it doubtful morality to subject a patient, for no good therapeutic reason, to a drug which has a drastic effect upon their metabolism; but since they will be discharged to an unknown situation, the very real possibility of re-sedation presents an unacceptable hazard.

If midazolam itself is not to become the subject of antagonism, it should not itself be carelessly antagonized.

INTRAVENOUS SEDATION TECHNIQUE

INTRAVENOUS SEDATION INFORMATION SHEETS

What is Intravenous Sedation?

Intravenous Sedation in dentistry is a modern technique of sedation which takes the fear out of dentistry and makes dental treatment a very pleasant experience.

It is suitable for most patients, however, if you are not in good health or are taking any medicines or tablets, you should tell your dentist so that the sedation can be modified to suit your needs. Before the examination of your teeth you will be asked to fill in a medical history form. Please do this thoroughly and if you do not understand any question, discuss it with the dentist or one of his staff.

The technique involves the injection of small amounts of a sedative into a vein in the arm. This injection is painless and the sedative soon takes effect. The patient becomes very relaxed and very drowsy and the dental treatment can be carried out quite comfortably. Local anaesthetic injections are sometimes necessary, however, these will not cause you any discomfort. Quite often the procedure can be carried out without a local anaesthetic in complete comfort. Because of the amnesia (forgetfulness) produced by these sedative agents it seems to the patient that he has been asleep for most of the treatment however, he has merely been sedated and has not been unconscious as is a patient under general anaesthetic.

On completion of the treatment the patient rests for a short while before being escorted home. While you will feel quite alert after treatment, the sedative agents are not completely eliminated from the body for some hours afterward – hence the need for an escort home. No food or drink should be taken for some hours before the sedation treatment session; however, you will be given a cup of tea or coffee while you are resting after treatment. Your dentist will provide you with detailed pre- and post-operative instructions.

Intravenous sedation is a very pleasant way to enabling dental treatment to be carried out. Your dentist will be pleased to explain it further to you.

Pre-Operative Instructions

- Please report details of drugs being taken, especially sleeping drugs, tranquillizers, or cortisone preparations.

- No food or drink should be taken within four hours of the appointment time.

- Please do not wear tight clothing – sleeves especially should be easily drawn up past the elbow.

- Contact lenses are best removed at the appointment time.

- Please visit the toilet prior to entering the surgery.

Instructions following sedation.

- The patient must be accompanied by another person.

- Any patient accepting a sedation appointment must specifically agree:

  i) Not to drive a vehicle or operate machinery that day, after the sedation.
  ii) Not to undertake responsible business matters.
  iii) Not to drink any alcohol, until the next day.

(Reverse side of above)

The following appointment times have been reserved for you. Before any sedation appointment, please note carefully the instructions overleaf. If unable to attend, please give sufficient advance notice for the time to be otherwise allotted.

N.B. Appointments failed without notice may be charged for.

2
3
4

NOTE: In your own interests, and that of other patients, you should not attend for appointments if you develop any cold, influenza, sore throat or other infection. In this case please telephone to make alternative arrangements.

Post-Operative Instructions (A)

(given to person accompanying patient home)

Instructions to Patient:

You must **NOT** drive, or operate any machinery today. You must **NOT** undertake any responsible business matters today. You must **NOT** drink alcohol today.

Instructions to Person accompanying Patient:

This person has had dental treatment carried out with the aid of intravenous sedation. Occasionally, some of the sedative drugs used may produce drowsiness for several hours, particularly after long appointments.

You are therefore requested to:

1. See the patient safely home, and arrange to stay for several hours, particularly after long appointments.

2. See that the patient obeys the above instructions.

Post-Operative Instructions (B)

(given to person accompanying patient home)

Instructions to Patient:

PLEASE RETURN HOME AND REST; We strongly recommend that you have a responsible adult to stay with you for the first six to eight hours.

ACTIVITIES: The medicine which was used to put you into a sedated state will still be acting in your body for ten hours so you may feel a little sleepy. This feeling will slowly wear off.

YOU SHOULD NOT UNDERTAKE THE FOLLOWING FOR AT LEAST 24 HOURS:

1. Drive a car.
2. Drink any alcohol, including beer.
3. Make important decisions, such as sign important papers.
4. Travel alone by public transport.
5. Use hazardous machinery.
6. Engage in sports, heavy work or heavy lifting.

Intravenous Sedation Technique:

1. **Preparation of solution for injection**

   Midazolam:

   Using a 5 ml disposable syringe, draw up the contents of either a single 5 mg (5 ml) ampoule of midazolam, i.e. 1 mg/ml or 15 mg/3 ml the more concentrated solution. Label the syringe.

   Note: For dental sedation purposes, it is suggested that the midazolam preparation containing 5 mg/5 ml be used in preference to the 15 mg/3 ml amp. The 5 mg/5 ml ampoule may be further diluted if desired, to 10 ml, with water for injection. This will result in a 10 ml syringe containing 1 mg midazolam/2 ml solution. The higher dilution has the advantage of providing greater control of dosage during administration.

2. The patient is seated in a chair, lowered to the supine position, made comfortable, and kept warm. (It is advisable that a blanket be kept handy to place over the patient's legs and body during long appointments to reduce heat loss.) Initial oximetry, BP and pulse recordings are now taken.

   Note: The routine use of the supine position (or semi reclining position in a contour dental chair) is necessary where sedation procedures are employed, to guard against possible hypotension and decreased cerebral oxygenation. Although midazolam is relatively free of adverse cardiovascular effects, the manufacturers recommend the patient be kept supine for up to one hour following I.V. administration to reduce the risk of postural hypotension. In fact, the use of I.V. sedation and the supine or semi reclining position is complementary to modern seated, four handed dentistry.

3. A tourniquet or similar restriction is applied to the patient's arm and a suitable large vein in the anticubital fossa is selected for injection. Preferably, the vein should be in the outer (radial) aspect to avoid any risk of injection into the brachial artery, or into an aberrant ulnar branch of this artery. This may be present in up to 18% of patients, and is detectable by observation and palpation. (Refer to section on Venipuncture).

4. After swabbing the skin with alcohol, a few drops of ethyl chloride or other coolant can be applied to the injection site, so that painless venipuncture can be carried out. Following venipuncture, the catheter position is checked by aspiration of blood, taped in position, and the tourniquet is released.

5. A second aspiration is carried out to ensure the tip of the catheter has not been moved or kinked during deflation of the vein, and a test done of a few drops of solution is injected followed by a wait of 30 seconds to check for allergy, hypersensitivity and needle location. Administration of the drug should be slow.

During administration, regular aspiration checks should be carried out to see that the catheter remains correctly placed. Background noise must be kept to a minimum during administration, and the doctor should employ reassurance and suggestion as the drug takes effect. This will also increase relaxation and lead to a maximum response.

6. **Signs and symptoms of drug effect:**

   The first symptom noted by the patient may be relaxation, loss of fear, drowsiness and lethargy and difficulty in focusing. Signs apparent to the operator include obvious loss of fear (which may be dramatic in some patients), onset of sedation, slurred speech, muscular relaxation, and lateral nystagmus. Another sign mentioned earlier is ptosis, which was once considered a useful indication of optimal dosage. However, ptosis is not invariable present, and may not occur in some patients until dosages approaching over dosage.

   Also, when the administrator is engaged in conversation with the patient during induction, and the patient is responding and possibly looking around, ptosis is seldom a reliable sign. Always remember that adequate relaxation can usually be achieved well short of ptosis, particularly with the use of good communication skills by the doctor.

   The amount of midazolam required is usually between 4-10 mg with a mean of around 7 mg. However, age, medical history, and current drug therapy must be considered when assessing correct dosage. Some patients may be unduly sensitive to benzodiazepines and show a marked reaction to as little as 0.5 – 1 mg midazolam (this applies particularly to the elderly, as noted earlier, in whom the titration rate must be markedly reduced).

   A maximum dose of 10 mg midazolam should be adhered to, except in rare circumstances, as higher dosages may produce undesirable side effects and more prolonged recovery.

1. Local anaesthetic injections are placed as required, using an aspirating syringe to minimize the risk of intravascular injection. After administering local anaesthetics, a further set of monitoring recordings should be taken.

   The use of large volumes of local should be avoided to reduce the risk of toxicity, as well as vasoconstrictor side-effects. This risk may be further reduced by using local anaesthetics such as prilocaine (Citanest) which is 60% less toxic than Lignocaine. Prilocaine is also available with a synthetic vasoconstrictor, felypressin (Octapressin), which produces fewer cardiovascular effects than adrenaline and nor-adrelaine.

   Research clearly shows that even dilute amounts of adrenaline cause measurable cardiovascular effects. The American Dental Association Guidelines recommends a maximum allowable healthy adult does of 0.2 mg adrenaline (10 cartridges of 2% Lignocaine with adrenaline 1:100,000 or 5 cartridges of 1:50,000); and that patients at cardiovascular risk should receive no more than 0.04 mg adrenaline (2 cartridges of 2% Lignocaine with adrenaline 1:100,000, or 1 cartridge of 1:50,000).

Although good sedation and amnesia are usually provided by midazolam it is still necessary to use a gentle approach when giving local injections, in order to avoid any sudden painful stimulus which may result in a calm patient becoming restless, applying surface anaesthetic to the injection site and using sharp disposable needles, it is common to find the patient later remembers nothing of this part of the treatment (particularly where suggestion, and diversion of the patient's attention are employed).

Distraction is a potent re-enforcer of amnesia with benzodiazepine and interferes with the memory consolidation process. It should be used wherever possible.

7. When good local anaesthesia has been obtained, dental treatment is carried out with the aid of mouth packs, props and nurse-assisted operating as necessary. Continue to monitor oximetry throughout the procedure, along with intermittent BP and Pulse checks as necessary, with a final set of recordings at the conclusion of treatment.

Contraindications to Intravenous Sedation

1. Inadequate training and experience of administrator and office staff.
2. Inadequate office equipment and facilities, including emergency equipment.
3. Inadequate office recovery facilities.
4. Absence of suitable veins.
5. No suitable escort arranged following completion of treatment.
6. Informed consent of patient, or parents of juvenile, not obtained.
7. Unco-operative patient (refuses to obey pre- and post-operative instructions regarding food, driving, alcohol etc.)
8. Medical contraindications: poor risk patients requiring hospitalization
9. Doubtful medical history. Always consult patient's physician if in doubt, never treat patient. Watch for illegal drug consumption, recent alcohol.
10. Pregnancy, particularly the first trimester.

Relative contraindications:

1. Young children with small veins and poor co-operation.
2. Poor co-operation during induction.
3. Local anaesthesia unlikely to be effective, e.g. acute abscess.
4. History of difficult behaviour under sedation (a small percentage of patients can only be managed satisfactorily under general anaesthesia, mainly children).

Monitoring

Heart rate and blood recording before and during treatment are essential, and continuous pulse oximetry is mandatory. Careful attention to the airway and respiration, and to clinical signs-such as eye signs, is essential. Mechanical monitoring assumes greater importance with increasing depth of sedation. Where midazolam is used, the patient should only be sedated and be able to hear and respond to the operator throughout; but this does not eliminate the need for continual monitoring of the patient's physiological parameters.

The principles of monitoring the sedated patient are outlined in the NZDC guidelines. The single most important item is the presence of a trained observer **CONTINUOUSLY** from the moment of first drug administration to the recovery of full consciousness. While operating in the mouth, the dentist must delegate some of the monitoring tasks to a trained assistant, who should also keep a simple monitoring record (section 8). A fundamental point is that the sedated patient's level of consciousness is reduced and therefore the are at risk for obstructing(or losing) their airway. A patient with an obstructed airway can suffer severe harm within minutes. Surgery design and work practice must ensure that the sedated patient is **never unattended**.

Blood pressure recordings before and during treatment are extremely important. Apart from detecting undiagnosed hypertension, BP trends may make it easier to diagnose inadvertent intravascular adrenaline injection, angina, etc. Regular and careful attention to airway and respiratory movement (especially the onset of paradoxical movements indicating upper airway obstruction, or of prolonged expiration seen in acute asthma) and level of consciousness are essential. With midazolam sedation, the patient should be able to hear and respond to the operator throughout.

Pulse oximetry and capnography are mandatory electronic aids to monitoring sedated patients, who invariably have at least slight respiratory depression. The rationale for oximetry is that it reliably detects arterial oxygen de-saturation, well before experienced clinicians are able to pick up the first signs of hypoxia. Capnography allows real time monitoring of breathing so any issues can be addressed before problems arise. Familiarity with the design, function, use and limitations of these instruments is essential.

Pulse Oximetry

All practitioners using intravenous sedation require a knowledge of pulse oximetry.

Pulse oximeters measure the arterial oxygen supplied to vital organs. They do not detect cell hypoxia.

Hypoxic brain damage can occur with cyanosis, or change in blood pressure, heart rate, or cardiac arrhythmias, therefore instruments that measure oxygenation are invaluable. Traditional monitors measuring pulse, blood pressure and ECG rapidly detect circulatory alterations, but dangerous changes in oxygenation are not evident until the patient is seriously affected. Risk factors which may contribute to hypoxaemia include poor function of the lungs or heart or sleep apnoea.

HISTORY:

- In 1860 the spectrophotometer was invented. A device which measures different substances absorbance at different wavelengths.

- It was noticed that saturated and unsaturated haemoglobin (Hb) transmitted light differently. Oxyhemoglobin (HBO2) is more transparent to red light than reduced Hb.

- First oximeters were used in aviation research during World War II.

- By 1950 pulse oximeters were being used but they had limitations. They were unable to distinguish between arterial and venous blood.

- In 1950 the Japanese developed the concept of two wavelength pulse oximeters, which solved this. Currently some oximeters are capable of measuring many wavelengths and are able to measure all four forms of haemoglobin. They can determine fractional saturation.

Haemoglobin is a protein molecule which is a vehicle for oxygen transport to tissues. The loose and reversible combination of Hb and O2, allows Hb to pick up and unload O2 according to metabolic need and demand.

The pulse oximeter measures the amount of O2 bound to Hb (HbO2) as a ratio, comparing it with the amount of Hb that is available for binding (functional haemoglobin).

Problems and limitations:

- The pulse oximeter assumes that only arterial blood pulsates.
  The three major sources of artifact which interfere with pulse oximetry are ambient light, low perfusion (low AC to DC signal ratio) and motion.

- **Ambient light**: Fluorescent lights emit 660nm radiation which may interfere substantially with oximeter function. This can be minimized by covering the sensors with an opaque shield. Radio frequency interference from diathermy also can introduces artifact.

- **Perfusion**: Reduced finger perfusion due to hypotension, hypothermia, or vasoconstrictor drugs will reduce the accuracy. Extremes in vasodilatation result in signal loss. Monitoring of gravely ill patients is not reliable.

- **Patient motion**: Results in a high AC to DC signal ration. Manufacturers have tried different approaches to try to overcome this problem. One way is by increasing signal – averaging time, i.e. measurements are taken over a longer time period and the effect of intermittent artifacts are less, **but** this results in a slower response time to an acute change in SaO2.

- **Coughing**: Can produce increase venous pressure waves that exceed arterial pulsation leading to transient artifacts.

- Altered haemoglobin: Carboxy Hb (HbCO = haemoglobin bound to carbon monoxide) and MetHb can affect oximeter accuracy. Both raise the SPO2 above actual SaO2. (MetHb is less than 1% in normal people but may be increased by some medications and HbCO in a non-smoker is less than 2%).

- Heavy **smokers** may have 10% HbCO. The oximeter reads SpO2 = 97% when actual SaO2 is only 90%.

- **Dyes**: Medical dyes (like methylene blue or indocyanine green) and some nail polishes (especially blue or dark false nails) tend to depress displayed SPO2 - to fix turn the pulse oximeter sideways to avoid the nail. Bilirubin does not affect or interfere with accuracy.

- **Anaemia**: Patient may still have SaO2 of up to 100% but has insufficient amount of Hb to meet metabolic needs. The pulse oximeter may still be able to detect adequate pursuable changes to process saturation values.

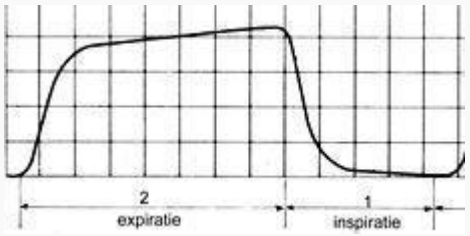
Dental IV sedation: for ease of access we place on finger, away from operative site.

Capnography

(This section uses the Wikipedia article as its basis but I have modified it. There are also lots of good websites and videos available on the internet if you are interested –Ed note.)

Capnography

Intervention



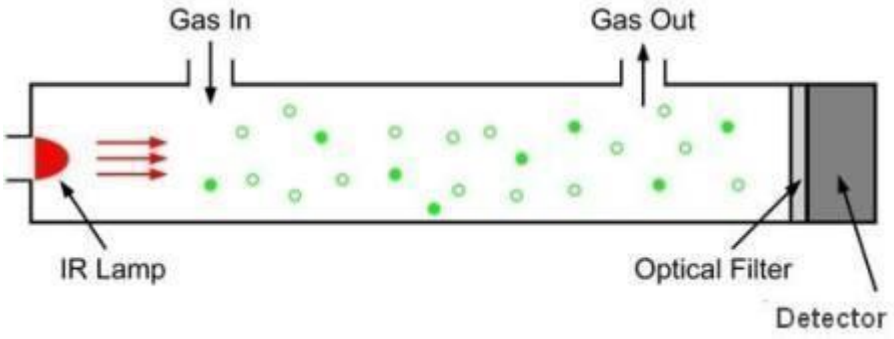
Typical capnogram. Expiration phase on the left, inspiration on the right.

Capnography is the monitoring of the concentration or partial pressure of carbon dioxide (CO_2) in the respiratory gases. Its main development has been as a monitoring tool for use during anaesthesia and sedation. It is usually presented as a graph of expiratory CO_2 (measured in millimeters of mercury, "mmHg") plotted against time.

The capnogram is a direct monitor of the inhaled and exhaled concentration or partial pressure of CO_2, and an indirect monitor of the CO_2 partial pressure in the arterial blood. In healthy individuals, the difference between arterial blood and expired gas CO_2 partial pressures is very small. In the presence of most forms of lung disease, and some forms of congenital heart disease (the cyanotic lesions) the difference between arterial blood and expired gas increases and can exceed 7mmHg.

How does a capnograph or capnometer work?

Capnographs usually work on the principle that CO_2 absorbs infrared radiation. A beam of infrared light is passed across the gas sample to fall on a sensor. The presence of CO_2 in the gas leads to a reduction in the amount of light falling on the sensor, which changes the voltage in a circuit. The analysis is rapid and accurate.

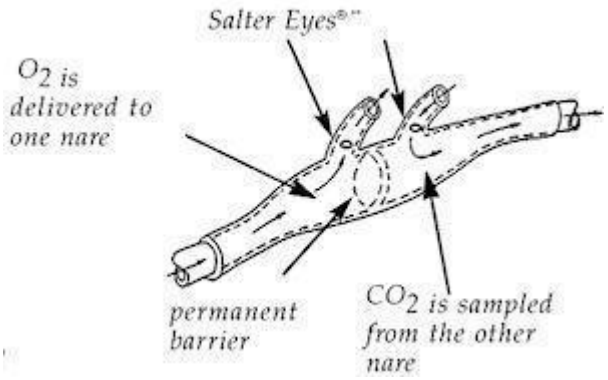


Diagnostic usage:

Capnography provides information about CO_2 production, pulmonary (lung) perfusion, alveolar ventilation, respiratory patterns, and elimination of CO_2 from the body. The shape of the curve is affected by some forms of lung disease; in general there are obstructive conditions such as bronchitis, emphysema and asthma, in which the mixing of gases within the lung is affected.

Conditions such as pulmonary embolism and congenital heart disease, which affect perfusion of the lung, do not, in themselves, affect the shape of the curve, but greatly affect the relationship between expired CO_2 and arterial blood CO_2. Capnography can also be used to measure carbon dioxide production, a measure of metabolism. Increased CO_2 production is seen during fever and shivering. Reduced production is seen during anesthesia and hypothermia.

Use in sedation



During sedation in dentistry the CO_2 is typically monitored by using nasal prongs which also supply oxygen. Capnography directly reflects the elimination of CO_2 by the lungs. Indirectly, it reflects the production of CO_2 by tissues and the circulatory transport of CO_2 to the lungs.

Capnography has been shown to be more effective than clinical judgement alone in the early detection of adverse respiratory events such as hypoventilation, and apnoea, which may be due to oversedation and/or airway obstruction thus allowing patient harm to be prevented. During procedures done under sedation, capnography provides more useful

information, e.g. on the frequency and regularity of ventilation, than pulse oximetry.

Capnography provides a rapid and reliable method to detect life-threatening conditions (malposition of tracheal tubes, unsuspected ventilatory failure, circulatory failure and defective breathing circuits) and to circumvent potentially irreversible patient injury.

Capnography gives the first indication a patient has slowed or stopped breathing - it has thus become the most valuable monitor for ensuring safety in sedated patients.

Remember 1.almost all the serious complications in sedation are due to respiratory depression
2. You have to be alerted to a problem before you can fix it.

Blood Pressure Monitoring

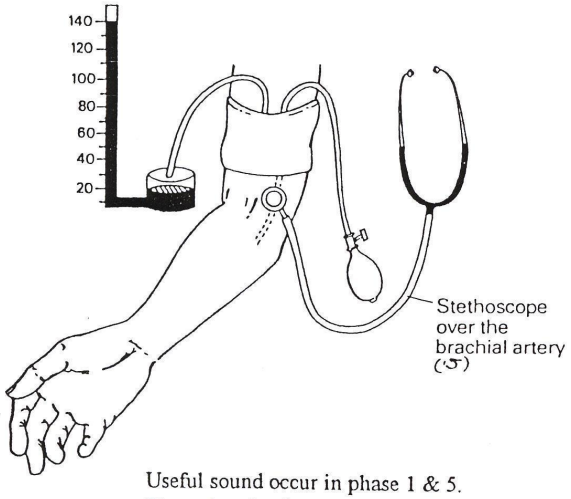
BP is defined as the amount of force exerted against the walls of an artery by blood. It is measured in mm Hg. The force of contraction of the heart, the amount of blood pumped per heart beat and how easily blood is able to flow through the vessels all influence BP.

BP has two components and both these are measured. The **systolic level** (highest pressure) occurs when the heart contracts and the **diastolic** (lowest level) occurs when the cardiac muscle is relaxed. They are written with systolic level over diastolic level.

Normal BP range 100/60 – 150/90mmHg. BP is influenced not only by age, sex and amount of blood in the system, but factors like emotion, pain, exercise, body size and medication can affect pressure.

History:

- 18th Century, Rev. Hales first measure BP by inserting a tube into the artery of a horse and seeing the blood level rise 9 feet.
- 19th Century, Sicipone Rocci (Italian Physiologist) invented the **sphygmomanometer (sfig-mo-ma-Nom-eter)** which is a device for measuring BP, however this device could only measure systolic pressure.
- Dr Korotkoft (Russian Surgeon) developed a technique of measuring both systolic and diastolic pressures. With the aid of a stethoscope he discovered that if a cuff was pumped up and then slowly released, then it was possible to hear different sounds as the pressure dropped. The different sounds indicated different phases of the heart beat.



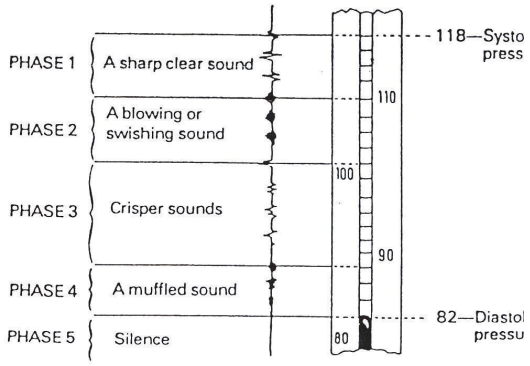


Fig. 5 The Korotkoff sounds.

Useful sound occur in phase 1 & 5.

Phase 1 the first sound is hears = systolic press

Phase 5 the sound disappears = diastolic pressure.

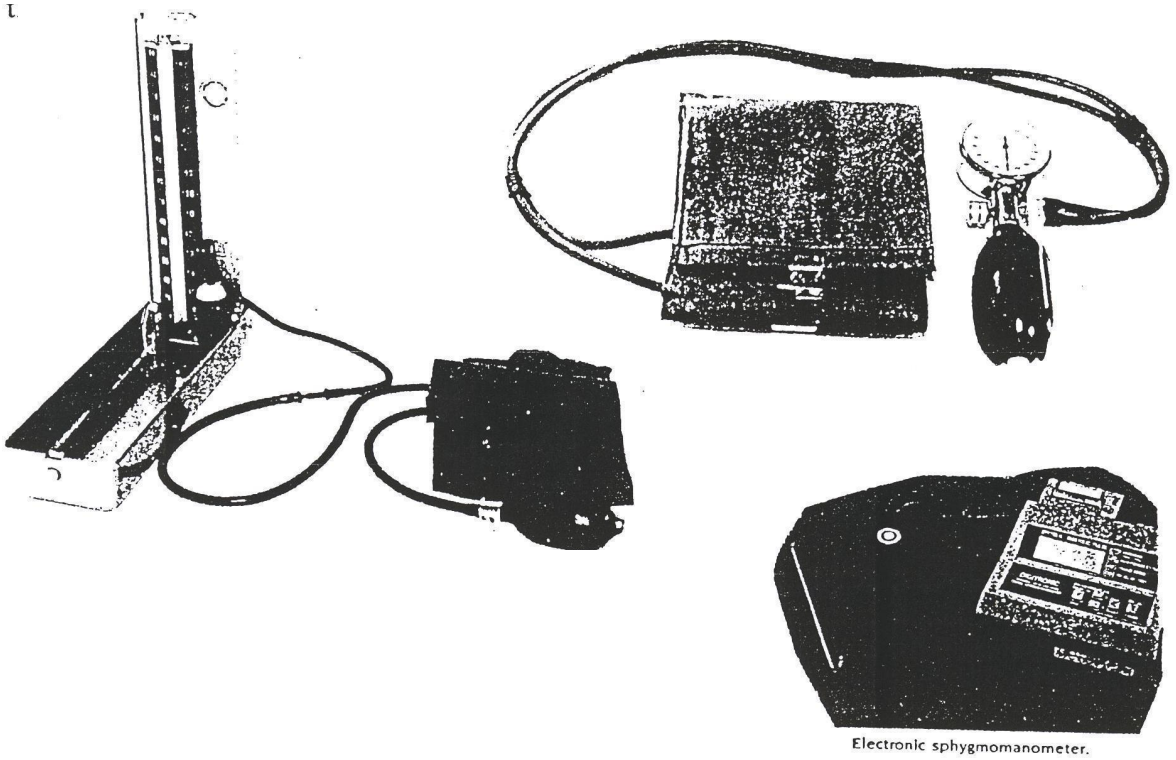
(Phase 4 & 5 are generally within 10mmHg some controversy as to whether it is 4 or 5 which reflects diastolic pressure. Consensus favour 5)

Equipment there are three types of sphygmomanometer:

- Mercury sphygmomanometer
- Aneroid sphygmomanometer
- Electronic sphygmomanometer

Generally, BP is taken on an arm, measuring the brachial artery. Both mercury and aneroid sphygmomanometers require clinical skills and the use of a stethoscope (auscultatory – technique of listening to sounds with a stethoscope / Palpation methods are possible).

They all have a pressure cuff and the mercury and aneroid have a measuring scale. The electronic sphygmomanometer is automatic and once the cuff is applied in the correct position, then the pulse and BP are automatically calculated and displayed by the unit



Electronic sphygmomanometer.

Most semi-automated machines work on one of the following principles:

- The detection of Korotkoff sounds
- The detection of arterial blood flow by ultrasound
- Phase-shift method which measure pressure changes between two segments of a double cuff.

- Oscillographic detection of arterial pulsation with a double arm cuff.
- Tonometry – depends on the principle that displacement of a force-sensitive transducer over a superficial artery can be made linearly proportional to the arterial BP (most common method today).

Technique of taking BP

- Well maintained equipment.

- Bare arm (no tightness of clothes) and relaxed patient.

- Cuff applied around arm. (Arm is supported and level with 4th intercostal space at the sternum.

- Palpation of brachial artery, placement of stethoscope which is held firmly and then the cuff inflated rapidly. When the pulse sound disappears, increase 30mmHg above this level. The point at which the pulse first disappears is around the level of the systolic pressure (a guide as to when to expect the first sound).

- Slowly let the pressure out of the cuff (2-3mmHg per heart beat/second). The appearance of sound = systolic pressure and the disappearance of sound = diastolic pressure. These are then recorded to the nearest 2mmHg).

(Initially, examination of blood pressure measure both arms and if there is a difference of more than 10mmHg then the arm with the highest pressure is used. When recording BP, it is helpful to record additional information e.g. which arm, position of patient, presence of unusual circumstances e.g. anxiety.)

Measuring BP in children: Use smaller bladder size (cuff) and paediatric diaphragm on stethoscope for measuring BP (other methods may have to be used – measure leg).

Elderly patients often have medical conditions which influence BP. Many may have raised systolic pressure (Hypertension). BP regulating mechanisms in the elderly are less effective and some may be susceptible to a fall in BP when standing (postural hypertension). Many are on antihypertensive drugs (can use leg and use popliteal fossa/artery – area behind knee).

Sources of Errors

Faulty equipment:

- Mercury meniscus not at zero-calibrations not correct.
- Scale dirty.
- Air vent blockage slows mercury flow.
- Cuff bladder not applied correctly or not adequately maintained (perished rubber).
- Cracked tubing or non airtight tubing.

Observer:

- Insufficient clinical ability.
- Observer biases – round off figures i.e. fit man round down and obese man round up.
- Digit preference – preference for terminal digits 0 & 5 though 5mmHg does not appear on many scales.
- Viewing distant angle incorrect. (View within 1 metre – vertical level to see real meniscus.
- Slow relaxed method – if rush and release pressure too fast then this leads to understating the systolic pressure and overestimating the diastolic pressure. (It takes 5 minutes to measure BP correctly.)

Patient:

- BP varies greatly within a day – Circadian rhythm.
- Other factors also influence BP. (Exercise, meals, smoking, alcohol, temperature, bladder distension and pain).
- Obesity (Actual and artifact – cuff not able to be applied and used correctly).
- Arrhythmia – change in stroke volume (blood flow).
- Postural position (Normal people have no difference in BP provided the arm is supported at heart level. Vertical displacement of arm = hydrostatic pressure increases as arm is dropped leads to an error as large as 10mmHg. If arm is unsupported then muscles are working and this increases diastolic pressure by up to 10%).

  Repeated measures of BP (inflation's) can cause venous congestion. Need to deflate cuff between readings and leave 15 seconds between successive measures.

BP is reasonably accurate provided potential areas of error are reduced and/or eliminated.

EQUIPMENT AND ORGANISATION

Equipment and Facilities

Minimum basic requirements for safe intravenous conscious sedation procedures:

1. Intelligent, highly trained, efficient and responsible nursing assistance.

2. High velocity suction which is essential for the rapid removal of water, saliva, blood and the debris of cavity preparation.

3. Mouth packing must be completely effective, so that the airway is protected at all times against inhalation of the slightest trace of foreign material. Such a barrier may either be a rubber dam or sponge pack.

4. Resuscitation apparatus must always be immediately available, and the dentist and his staff must be fully conversant with emergency procedures. Facilities for the administration of oxygen under pressure should be present in every dental surgery, and cylinders must be checked regularly. This should be a portable cylinder with regulator, connector tubing and mask, dedicated for use in emergency.

5. Emergency drugs should include pressor agents, adrenaline, hydrocortisone, antihistamines, and benzodiazepine and narcotic antagonists. Drugs, however, should generally be regarded as aids to basic emergency procedures, except in cases such as acute allergic reaction or adrenal insufficiency. (See section on emergencies for specific emergency drugs).

6. The dental chair should permit treatment of the patient in the supine position, and should allow for the legs to be raised above the level of the head if necessary.

7. Proper sterilization facilities such as autoclaving or dry heat are essential.

8. Full and accurate treatment records should be kept.

9. Adequate recovery facilities must be provided. The patient must remain on the premises until a responsible person can take him/her home. The surgery recovery area should be kept warm, particularly during winter months. As most anaesthetic and sedative drugs produce peripheral vasodilatation, a blanket placed over the patient's legs will help prevent heat loss during long procedures.

10. Drug storage facilities are important, and secure storage is a requirement of law.

Practice Organisation:

It is essential that I.V. sedation be a team effort with each member of the team having a defined role with defined duties and responsibilities. These should be written down as a protocol for all members of the team.

There should be a written protocol for routine care as well as emergency situations. Problems and difficulties are more likely to arise with departures from the standard routines. If you choose to deviate from your routines, know why you are doing so and acknowledge the risk benefit ratio.

You will need to develop a particular protocol for your own individual situation and practice. If your staff are not appropriately trained, it is your responsibility to train them or arrange for their training. You will also need to check that your premises, facilities and equipment comply to the current guidelines.

Facilities And Equipment:

The facilities have been well defined in the governing policy document on sedation for dental procedures and this includes the following:

1. A facility which is adequate in size, staffed and equipped to deal with a cardio pulmonary emergency.

2. A chair which can be tilted readily to the horizontal position.

3. Adequate uncluttered floor space to allow external cardiac massage on the patient should this prove necessary.

4. Equipment suitable for measurement of the patient's blood pressure.

5. Adequate suction and room lighting.

6. A supply of oxygen and suitable devices for administration of oxygen to a spontaneously breathing patient.

7. A means of inflating the lungs with oxygen (e.g. a range of laryngeal airways and self-inflating bag suitable for artificial ventilation).

8. Appropriate drugs for a cardio pulmonary resuscitation and a range of intravenous equipment.

9. A pulse oximeter and a capnography monitor.

Specialised Equipment:

- Tourniquet
- 10 ml syringes
- Drawing up needles (18 gauge)
- Steriwipe disinfectant patches
- Tegaderm patches
- Intravenous cannulas
- Mouth props
- Mouth packing devices
- Tray for intravenous equipment
- Sedation drugs and their reversal drugs
- Nasal prongs and a means of connecting these to a continuous oxygen supply

Routine Care:

Receptionist's Responsibilities:

- Gives patient medical history form
- Gives patient information form on sedation and pre-operative instruction forms
- Checks appropriate forms are signed, and that patient understands the requirement for fasting, transport and an escort
- On arrival of the patient for sedation the receptionist checks compliance with the pre-operative instructions

Dental Assistant Responsibilities:

- Checks emergency oxygen, suction and lighting prior to first patient each day
- Prepares all equipment so that it is ready for dentist
- Escort the patient to the surgery, rechecks and records pre-operative compliance
- Removal contact lenses and partial dentures if necessary
- Places protective glasses and checks for comfort. Protect elbows, arms and calves – cover with a blanket for long procedures
- Places nasal prongs and connects to supplemental oxygen
- Assists dentist with monitoring and sedation. Records blood pressure, pulse, saturation and monitors pulse oximeter throughout procedure, recording parameters at specified intervals
- Assists with clinical task but particularly responsible for protection of airway with suction
- Assists patient to recovery area. **(No sedated patient should be left unattended** by the office staff until the clinician responsible for the sedation has discharged the patient to the care of the escort.)
- Discharges the patient after authorization from the dentist with written post-operative instructions and verbal instructions to the escort
- Safe disposal of drugs and contaminated I.V. equipment

Routinely (monthly) checks emergency equipment and drugs noting expiry date of drugs

Dentist's Responsibilities:

- Is ultimately responsible for the team and their activities
- Must ensure that the correct drug at the correct dosage is appropriately given
- Staff must be trained and a written protocol of responsibilities and duties for all staff developed
- Provide written pre-operative instructions, post-operative instructions, medical history, consent forms and monitory/record of sedation forms

Leadership is by example – do what I do, not what I say

EMERGENCIES:

By their very nature, emergencies occur rarely and your team will have minimal or no experience in managing emergencies. It is therefore essential to have regular practices of emergency situations, so that each person knows:

- Who does it
- How to do it
- Why do it
- Who gets the emergency drugs and equipment
- Who calls the emergency team on the telephone
- Who records the time
- What is each person's responsibility during CPR

You should practice your emergency drill every six months and update your CPR at a recognized course every two years.

If possible, all emergency equipment and drugs should be sited together. This location should be known by all staff and clearly identified. Current emergency numbers for crash team, ambulance etc. should be in a prominent position beside the telephone.

Receptionist's Instructions:

- Phone 111
- Name of dentist
- Address of surgery
- Nature of problem
- **DO NOT HANG UP**

THE IMPORTANCE OF THE DENTAL ASSISTANT:

In addition to being competent at the usual dental assisting procedures, the assistant has an important additional role to play when intravenous sedation is being used. They should also obviously be trained in medical emergency management, although medical emergencies are perhaps even more likely to occur as a result of treating fearful patients without, rather than with sedation. The most common complication is that of syncope – the common fainting attack, which usually occurs when treating a frightened patient under local anaesthesia who has missed breakfast.

The following points are indicative of a well organized dental office using I.V. sedation:

- Everything should be ready before the patient arrives so that when they do arrive they are taken promptly to the treatment area. If a mildly anxious patient is kept waiting, an active imagination can result in having to deal with a terrified patient in the surgery, making treatment much more difficult. It can also result in a much less effective sedation, increased drug requirements and increased risk of complications.

- Welcome the patient in a friendly manner – try to put them at ease. Pre-operative reassurance is very important to anxious patients.

- Seat patient comfortably in the chair. Check they are adequately fasted and that they have someone to take them home after treatment (the receptionist will also do this, but you need to double check).

- Make sure that if the patient wears dentures or contact lenses, that they remove them before treatment starts. **Don't lose the contacts!**

- Take base line recordings.

- Place protective glasses and nasal oxygen prongs.

- Assist as required with venipuncture. During venipuncture, a reassuring hand can be placed on the patient's shoulder, or some very nervous patients may wish to have a hand held.

- During the induction phase, some quiet conversation can help with the relaxation, which is the aim of the drug-administration, but be careful not to allow some nervous patients to become too 'chatty'.

- Maintain a calm reassuring manner without interfering with the sedation process.

- Assist the dentist with continual monitoring, perform periodic monitoring recordings.

- During the work, especially if the lower jaw is being operated on, supporting the jaw may be required to protect the airway.

- Maintain thorough and careful suction to remove filling debris and do not allow moisture to get behind a mouth pack. Ask the dentist to stop working if necessary.

- At the end of the procedure, take care of the patient's appearance before assisting them through to the recovery area.

- Patients may benefit from a recovery drink of coffee or tea, prior to leaving the premises. They must never be allowed to leave the building unescorted.

COMPLICATIONS AND EMERGENCIES

COMPLICATIONS AND EMERGENCIES

Introduction

No text on sedation or anaesthesia can be considered complete without including a section on the prevention and management of complications and emergencies. Although many firmly believe that serious medical emergencies are, in fact, less likely to occur when anxious patients are properly sedated, it must also be recognized that the use of drugs may increase the risk in some situations. Fortunately, the great majority of emergencies can be prevented, provided certain basic rules are employed. It is the purpose of this section to emphasise the means of prevention, as well as management.

Sedation for dental procedures includes the administration by any route or technique of all forms of drugs which result in depression of the central nervous system. The objective of these techniques is to produce a degree of sedation whereby rational communication with the patient is continuously possible, so that the uncomfortable and/or stressful procedures may be facilitated. The drugs and techniques used should provide a margin of safety which is wide enough to render unintended loss of consciousness unlikely.

It is important to understand the variability of effects which may occur with sedative drugs, however administered, and that over-sedation or airway obstruction may occur at any time.

To ensure that standards of patient care are satisfactory, equipment and staffing of the area in which the patient is being managed should satisfy the requirements as laid down in the Sedation Practice standard of the New Zealand Dental Council. It is the responsibility of the practitioner to ensure the practitioner has the current version, since policy documents are reviewed from time to time.

Complications can occur before, during and after sedation procedures and it is important to understand how firstly to prevent these from occurring, and how to recognize and manage complications if they arise.

PREVENTION

The prevention of dental office complications and emergencies depends upon the following factors:

1. Patient selection and evaluation.

2. Pre-operative patient management.

3. Patient care during treatment.

4. Post-operative patient management.

The above factors are in turn directly dependent upon the training, skill, and experience of the dentist and the skilled use of correct, well-maintained office equipment and facilities.

1. **Patient selection and evaluation**

   The pre-operative medical and dental history is vitally important (see section on evaluation). A signed, dated medical history questionnaire is the best approach, provided it is used as a guide and not as a substitute for further investigation during the patient interview. A questionnaire requires the patient to think specifically about each question, and reduces the possibility of significant facts being forgotten or overlooked. It should be concise, but relevant to dental treatment under sedation and must be carefully checked following completion by the patient. Anything in doubt must be clarified, in consultation with the patient's physician if necessary, before proceeding with treatment.

   The old adage, "never treat a stranger", has never been truer than in these days of increasingly sophisticated medical treatment and continually multiplying potent pharmacotherapeutic agents.

   Further discussion of patient selection, physical diagnosis and evaluation is unnecessary here, as the subject has been fully covered in the section on Patient Evaluation.

1. **Pre-operative patient management**

   Having decided that pharmacosedation is indicated, it is important to present it in such a way that the patient will readily accept it. Those referred by other patients are usually less apprehensive, but some are afraid of anything outside their experience. It is essential that a strong doctor/patient relationship built on trust is established wherever possible before treatment is carried out. The patient's fears must be considered, and reassurance provided at the consultation appointment. The procedure should be explained, without going into unnecessary detail, and any questions the patient may have, answered. Avoidance of complications and the effectiveness of the sedation technique employed are directly related to the interpersonal relationship established before the treatment appointment.

Pre-operative instructions must be stressed, and strictly adhered to by the patient. These should include the restriction of food and drink for 3-4 hours pre-operatively, removal of dentures and contact lenses, attendance to toilet requirements and arranging for a responsible escort following treatment. Observance of these regulations must be checked before treatment is commenced.

Failure to follow instructions should result in cancellation of the appointment. Patients accepted for treatment who have not obeyed instructions will quickly tell others of such casual attitudes, and the risk of future complications and emergencies will increase accordingly. If rules are made, they must be kept, and those who break the rules must be refused treatment. It is surprising how quickly the word spreads among patients that “the doctor means what he says” (or doesn’t mean what he says) and such cancellations are extremely rare in a well-run practice.

A light carbohydrate meal is permissible 4-6 hours prior to light sedation, and clear fluids up to 2 hours before, depending on techniques and depth of sedation.

Do not keep anxious patients waiting. Have everything in readiness so that when the patient arrives, he or she is taken immediately to the treatment area. If a mildly anxious patient is kept waiting, an active imagination may result in having to deal with a highly anxious patient in the surgery, thereby decreasing the effectiveness of the sedation technique, increasing drug requirements as well as the risk of complications.

2. **Patient care during treatment**

The supine position (or semi reclining position in a contour dental chair with the head and feet at the same level) should be used routinely wherever sedation techniques are employed. This will greatly reduce the risk of syncope and its potentially serious sequelae. Use of this position is, of course, complementary with modern operating techniques.

There are, however, some disadvantages which must be considered:

(a) The risk of foreign bodies entering the pharynx is increased, e.g. amalgam and tooth debris, calculus, reamers, burs, etc. Proper mouth packing or the use of rubber dam is essential for protection, along with effective use of suction by an alert dental assistant. Where oral sponge packs are used, they should be replaced frequently before they become saturated, and discarded when fraying occurs. Spare suction tips should be readily available in case of blockage.

(b) The risk of gastric regurgitation is more likely in the supine position, although uncommon. Hiatus (oesphageal) hernia predisposes towards this. It is therefore doubly important to stress pre-operative fasting instructions in such patients. On the other hand, should regurgitation or vomiting occur, it is more easily managed than with a patient who is seated upright.

(c) Women in advanced pregnancy are usually more comfortable in a semi-reclining than a supine position, where fainting may occur due to pressure from the foetus on the inferior vena cava, resulting in obstruction venous return.

Obese patients, acute asthmatics, patients with hiatus hernia, and those with cardiac insufficiency and orthopnoea may also be more comfortable in the semi-reclining position. However these cases should only be attempted by very experienced practitioners as they are all high risk.

(d) The risk of foreign bodies entering the patient's eyes is increased, and the use of a protective shield should be considered.

The dentist and his/her staff must be properly trained in the care of the unconscious patient, know how to position the patient correctly, and be capable of maintaining a patent airway during treatment. Although the techniques described in this manual are conscious patient sedation techniques when correctly employed, incorrect use, over-dosage, hypersensitivity, etc., may lead to deeper levels of sedation approaching general anaesthesia. Even patients treated with light sedation can doze off, and their respiratory efficiency may be impaired if the airway is not properly cared for. During treatment, the assistant should support the patient's chin and watch respiratory movements. A common early warning sign of partial respiratory obstruction is snoring, a sign which should not be ignored.

The dental assistant should be trained to monitor the carotid pulse, eye signs, and the patient's colour; as well as respiration. An assistant can also be trained to record blood pressures. The assistant should be observant, and report anything unusual such as respiratory disturbances, swallowing attempts, needle blockage, pulse oximetry changes, etc. Particle of amalgam and tooth debris should be removed immediately with suction, and she/he should request "time out" from operating in order to remove debris from difficult to reach areas.

3. **Post-operative patient management**

Following treatment, patients should be slowly raised to the seated position for a short time before being asked to stand, to prevent postural hypotension. They should then be taken to the recovery area, supported if necessary; to await a responsible escort to take them home. During this immediate post-operative period they must be supervised by the auxiliary staff.

Recovering patients must never be left unattended. Hypoxemia / apnoea can easily occur, leading to potentially disastrous complication. Even if the patient appears “awake” at the end of the treatment, it is easy for them to relapse into sleep, shallow respirations, and hypoxia.

N.B. A pulse oximeter should always remain attached to the patient during the recovery period.

Post-operative instructions must be clearly established and agreed to before treatment. The patient should not be permitted to leave the office unaccompanied. A copy of the post-operative instructions should be given to the person escorting the patient home, and the important points stressed. Driving, operating machinery, drinking alcohol and undertaking important business matters must be prohibited for the remainder of the day. It is impossible to predict the degree and duration of sedative and amnesic after-effects in individual patients. Medico-legally, strict observance of these instructions is very important. It is this author’s experience that it is far better to be over-cautious than to take chances, and that patients appreciate this concern for their welfare.

Other post-operative precautions should include the avoidance of hot stoves, hot baths, and responsibility for the care of small children for several hours after treatment.

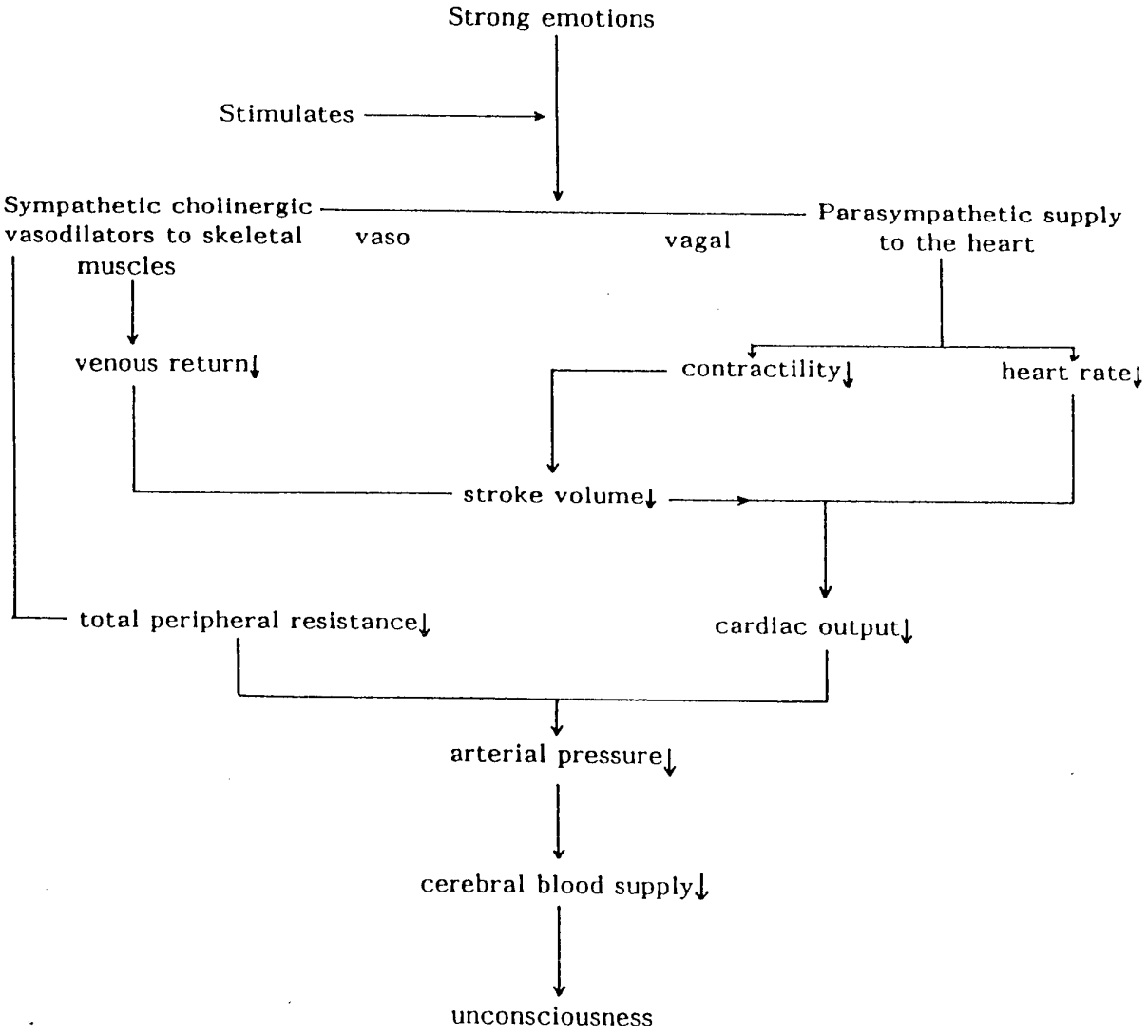
Complications and Emergencies

Fortunately major complications are rare and can be successfully managed by the practitioner. They can occur before, during or after sedation and may or may not be related to sedation itself. Figure 1 shows the schematic diagram of the major problems seen during I.V. sedation.

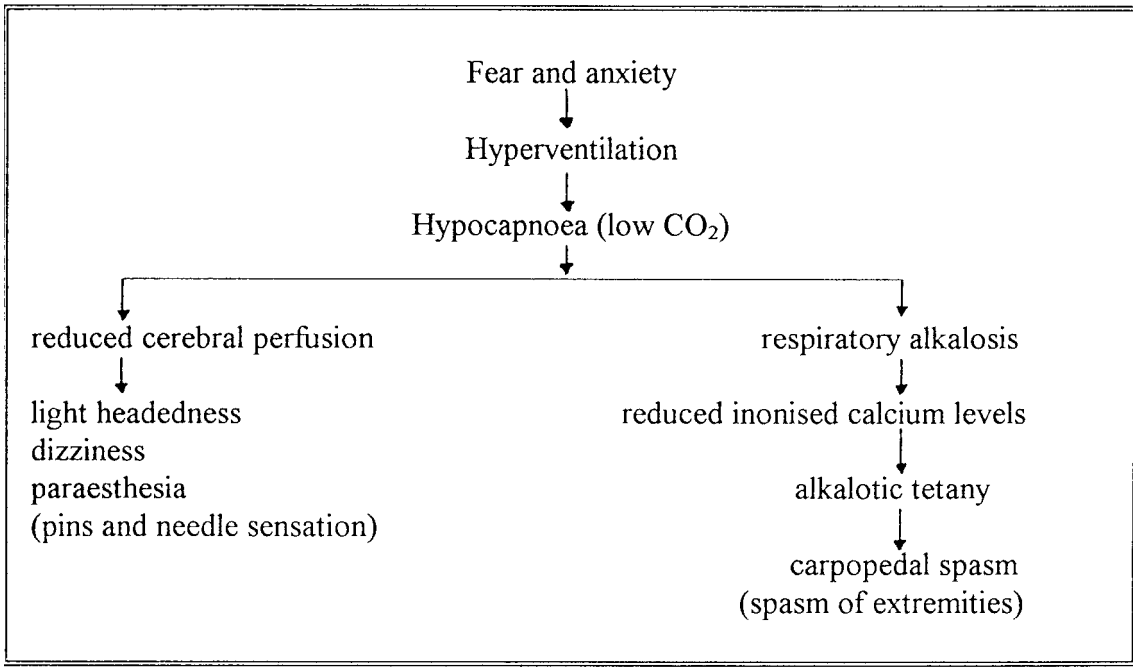
Figure 1.

Unrelated to sedation	**Local complications**	**General complications**
Fainting Hyperventilation Asthmatic attack	Fear of needle Difficult veins Accidental removal of needle	Respiratory problems - hypoventilation - apnoea
Angina Myocardial infarction	Extravasation Intra-arterial injection	Cardiovascular problems - Hypotension - Arrhythmias
		Sensitivity Anaphylaxis Amnesia Dreams/fantasies Vomiting/regurgitation

Fainting is one of the commonest of problems seen in the dental surgery. Fainting may be defined as loss of consciousness due to cerebral ischaemia. Figure 2. gives the schematic diagram for the onset of fainting.



Hyperventilation is seen not uncommonly in poorly selected and prepared patients who come for I.V. sedation. The following facts may be important in its symptomatology (Figure 3).



How will you deal with such a situation?

There is the possibility that a variety of medical conditions like acute asthmatic attack, angina, myocardial infarction and stoke may occur any time in the surgery, though these are fortunately very rare. Epilepsy, either a grant mal or other types, may be precipitated due to the stressful situation in the dental surgery for the patient. A hypoglycaemic attack is far more common and dangerous in a diabetic patient, particularly one who is on regular insulin regimen, than problems associated with hyperglycaemia; this is particularly so in a fasting patient and in whom adequate precautions are not taken regarding the insulin regimen.

How will you diagnose angina or myocardial infarction?
What will you do if the patient develops an acute asthmatic attack?
Can you differentiate between a stroke, attack of epilepsy, hypoglycaemic attack and overdose due to the intravenous sedative drug?
Have you got a plan of action to deal with a concurrent medical emergency in your surgery?

Local complications associated with intravenous sedation include **difficult venipuncture**, either due to poor veins or a patient who is petrified at the thought of having to have a painful injection. EMLA local anaesthetic cream application 45-60 minutes before venipuncture can guarantee against major discomfort during intravenous cannulation. It can easily be applied to the back of the hand,antecubital fossa, or where veins can easily and conveniently be found. Simple venous tourniquet, along with

opening and closing the fingers as well as tapping over the veins, should bring the veins into prominence. If not, application of a warm towel or dipping the hand or arm in warm water, for 10-15 minutes should help. Use of **cannula** in preference for a needle, and firm taping of the same, should almost ensure against **accidental removal** from within the vein and assures continued venous access, which is of vital importance in an emergency A careful technique of insertion and removal of the cannula should prevent any **extravasation** of blood. Injection of a small amount of normal saline or 5% dextrose solution into the cannula prior to injection of sedative drug should confirm any possibility of extravascular injection and then could be corrected in time.

What do you do to avoid venipuncture problems in your patients?
Does your current technique of venipuncture guarantee you continued venous access at all times during the treatment as well as in the immediate post-operative period, for dealing with an emergency?

Systemic complications can occur. These include major cardio-respiratory problems. Arguably the commonest and perhaps the most easily treatable complications are ***respiratory problems***. The commonest mechanism of occurrence is due to relative overdose, either due to larger than intended dose, **too rapid** an injection of a **large bolus** or by use of **combination** of drugs (e.g. benzodiazepines and opiates), all leading to **hypoventilation** or **apnoea**. Inaccurate **dilution** of the drug as well as incorrect labeling can cause problems. Opiates shift the normal carbon dioxide response curve to the right, increasing the chances of hypoventilation. Similarly drugs like benzodiazepines reduce the hypoxic response and hence tend to prevent the normal response to hypoxia of increasing the ventilation. Hence a combination of the two drugs can be a problem indeed, unless particular attention is given to monitoring. Respiratory problems can also be precipitated by higher than expected degree of sedation and partial respiratory obstruction and if left undetected, leads to hypoxia. **Snoring** is an early warning of possible problems. Careful observation of respiration as well as continuous use of pulse oximetry can prevent problems from developing.

Have you a set routine for diluting the drug and identifying the same during the procedure?
Do you consider a combination of drugs for I.V. sedation a distinct hazard?
What are the ways **you** monitor the respiratory function during I.V. sedation?

Cardiovascular problems include **hypotension** as well as various forms of **arrhythmias**. Frequent monitoring of **blood pressure** can often demonstrate changes in blood pressure brought on by the sedation technique. Always remember that fainting can also occur during the procedure, leading to a fall in blood pressure.

Do you monitor the blood pressure during I.V. sedation?
What methods do you use to assess the cardiovascular function during I.V. sedation?
Do you consider it important to monitor the cardiovascular system using methods other than pulse oximetry?

Sensitivity reactions to drugs can occur, but this can easily be prevented by using the titration method for achieving the level of sedation; always give small increments of the drug. Anaphylaxis to benzodiazepines is very rare indeed. **Amnesia** is a well known effect of benzodiazepines, often used to our advantage. But it can persist and may not be reversed by flumazenil. **Dreams and fantasies** can occur in any patient; this is often related to the dose of benzodiazepines used. Presence of a female assistant is always necessary during the treatment performed by a male practitioner on a female patient.

Nausea and regurgitation can occur during I.V. sedation as much as during general anaesthetic if the depth of sedation is deep and adequate attention is not given to these issues. It is currently advocated that a light carbohydrate meal 4-6 hours before scheduled sedation is acceptable and perhaps ideal; patients should be encouraged to have clear fluids (coffee, tea or fruit juice) until about 2 hours before the procedure. This actually has shown to reduce the gastric acidity and volume, both potentially dangerous situations in an unconscious patient, as well as prevent necessary dehydration, caused by prolonged fasting.

What are your present instructions to patients regarding fasting before I.V. sedation?

There are many issues you should consider during I.V. sedation in order to avoid major complications. Do you give particular consideration to the following?

Age of the patient
- Change of pharmacokinetics with age
- Change of pharmacodynamics with age
- Precautions in the elderly

Pre-existing **medical conditions**
- ASA grading

Medication intake by the patient
- Keep regular medication as they are
- Special requirements for diabetic patients

Criteria for **selection** of patients

Position on the table

Use of **oxygen** during sedation

Post-operative period

Use of reversal agents

Do you monitor patients during recovery period?

Resuscitation when needed

Equipment **trolley**

Drugs for CPR training

How do you **maintain the skills** of CPR?

What is the ideal set up?

Management

1. **<u>Unrelated to Sedation</u>**

   a) **Fainting:**

   - Loss of consciousness due to cerebral ischaemia.
   - The presentation is an anxious patient, sweating, complaints of nausea, pale appearance, slow heart rate, low blood pressure, and unconsciousness.
   - May occur in a patient who is lying horizontally and can occur at any time during treatment.
   - Effects may last 30-40 minutes.
   - Continue to observe patient after regaining consciousness as fainting may recur.
   - May need to abandon treatment.

   Treatment:

   - Place patient in head down position and raise legs.
   - Administer oxygen and monitor breathing and pulse.

   b) Hyperventilation:

   - Rapid breathing/increase in respiratory rate.
   - The presentation is rapid breathing, pins and needles sensation in extremities, spasm of hands and feet, feet, feeling faint, clouding of consciousness.
   - The symptoms are mainly due to low CO_2 in the blood due to hyperventilation.

   Treatment:

   - Reassurance
   - Possibly re-breathing into a bag (this helps to build up CO_2 in the lungs)
   - Use of some form of sedation/patient management for next visit.

   c) Asthma Attack:

   - Presents as coughing and wheezing, sweating, tachycardia, cyanosis in severe cases.
   - Caused by bronchospasm which results from hyperirritability of the tracheobranchial tree.
   - May be rapidly progressive.

Treatment:

- Use of 'Ventolin' type inhaler.
- In severe cases Call an ambulance and ready patient for transfer to hospital. Administer supplemental oxygen and additional ventolin.
-
-

d) Angina:

- Presents as pain or tightness over chest, arm or throat region due to decreased blood flow to a portion of the heart muscle.

Treatment:

Rest, oxygen administration, Nitrolingual spray or glyceryl trinitrate tablet ('anginine') 0.6 mg sublingually.
Consider immediate transfer to hospital via ambulance.

e) Myocardial Infarction:

- Sudden onset of pain or discomfort over chest, arm or neck, collapse, hypotension and shocked appearance.
- Pain may not be a feature in a third of cases.

Treatment:

- Oxygen/C.P.R.
- Immediate transfer to hospital by ambulance.

f) Stroke:

- Presents as a sudden feeling of being unwell, collapse, altered levels of consciousness and slurred speech, dizziness, loss of vision.

Treatment:

- Care of airway and oxygen.
- Immediate transfer to hospital.

g) Epileptic Attack:

- 'Grand Mal' type of seizure is initiated by a sudden cry caused by the spasm of the diaphragmatic muscles, followed by loss of consciousness. Unco-ordinatied beating of limbs may follow and patient may then become comatose. Then, generally within a few minutes, the patient regains consciousness with headache and confusion.

Treatment:

- Care of airway
- Transfer to hospital

h) Hypoglycaemic Attack:

- Presents in patients giving a history of insulin use and inadequate control of diabetes. Fine tremor, sweating, dilated pupils, agitation and convulsions or coma.

Treatment:

- 50% dextrose intravenously (5-10 ml FAST) – expect a prompt recovery.
- If unavailable – sugar or sweet drinks to conscious patient; dissolve sugar under tongue in unconscious patient.
- Far more common and dangerous than hyperglycaemia. This is particularly so in fasting patient and in whom adequate precautions are not taken regarding the insulin regime.

i) Hyperglycaemic Attack:

- High blood sugar levels causing dehydration, which is more life threatening than the hyperglycaemia itself.
- Uncommon, since patients attending for sedation would presumably have fasted for several hours.

Treatment:

- Dehydration treated first (I.V. saline) then move to hospital for insulin and ongoing support.

2. <u>Local Complications</u>

a) **Fear of Needles:**

Sight of needle:
- look other way
- careful technique of needle to keep out of patients vision
- distraction

Fear of pain:
- good technique
- use of EMLA cream, Ethyl Chloride spray, small amount of local anaesthetic prior to venipuncture.

b) **Difficult Veins:**

- Use of tourniquet
- Opening/closing of fingers
- Tapping over vein
- Warm towel/dipping hand into warm water

c) **Accidental Removal of Needle:**

Use of a cannula in preference for a needle and firm taping of the same should almost ensure against this and also ensures continuous venous access, which is of vital importance in an emergency.

Side Effects and Complications of Intravenous Sedation:

LOCAL It is advisable that large veins are selected for injection wherever possible, combined with slow injection and use of fine gauge needles. Great care must be taken to ensure the needle is correctly placed within the lumen of the vein before commencing administration, as perivascular injection or leakage may cause pain.

SYSTEMIC: Midazolam in clinical dosage produce sedation, muscular relaxation and amnesia. The latter will often last up to 30 minutes or more following I.V. administration, which can be very useful in dentistry. However, post-operative amnesia may persist in some patients for several hours after treatment. It is thus mandatory that all patients are accompanied home following treatment, and are supervised during the next few hours – no matter what the patient's own assessment of his or her condition may be. Driving motor vehicles, operating machinery, undertaking business matters, and drinking alcohol must be prohibited for 24 hours after treatment.

Higher dosages of benzodiazepines may lead to prolonged drowsiness, dizziness, ataxia and disorientation (particularly in elderly patients). Over-dosage may produce tachycardia, hypotension, respiratory depression, nausea and vomiting, and coma.

Note: The speed of injection plays an important part in the production of side effects during I.V. drug administration. For this reason (apart from the latency of midazolam) it is advised that a slow rate of injection be used routinely.

The benzodiazepines have a marked synergistic effect with central depressants such as barbiturates and alcohol, which could lead to respiratory and circulatory depression
Midazolam, in particular, markedly potentiates undesirable side effects of opiates, such as hypoxaemia, respiratory depression and hypotension.

Emergency

YOU KNOW WHAT TO DO: DO THEY?

No longer does the dentist work in isolation. No longer can one man work entirely single-handed in the surgery, performing all the tasks himself, particularly where anaesthesia or sedation are used. Dentistry is nowadays a team operation. Most teams, with practice, become exceedingly skilled in the routines of quiet, uneventful dentistry. But what happens when that quiet is broken? What happens when it ceases to be uneventful? Will the team swing into action just as smoothly then?

The emergency may not be your fault, but what happens afterwards most assuredly is. It may never happen. To most, it never will. But skills become rusty with disuse: would you care to go for a cruise in a ship whose lifeboats were rusted to the davits?

A pause for thought, perhaps?

Of course, you have:

1. carefully selected the patient and, if the intravenous route is to be used, checked for suitable veins;

2. taken a detailed medical history and consulted with the patient's doctor, if in doubt;

3. checked that pre-sedation instructions have been obeyed;

4. used an 'approach dose' induction technique;

5. obtained the lightest practicable level of sedation.

Fine. But does your team know:

1. How to use the emergency aspirator? (Do you even have one?)

2. How to use the resuscitator? (When did you last blow the dust off it?)

3. How to use the emergency oxygen supply? (If you want it now, how many seconds before the patient actually gets it?)

4. How to inflate the lungs? (Reservoir bag, squeeze bag, Guedelairway, mouth-to-mouth – or what?)

5. How to perform external cardiac massage? (In those first few horrible seconds, who does what, and with which, and to whom, and in what order?)

6. Who rings for the ambulance, and when? Who contacts the hospital? (Do they know the 'phone numbers without looking?)

4. If you were not there, could they still do it? (You have sprung to your feet and knocked yourself out on the operating light. You will recover – will the patient?)

All satisfactory? Good. So the lifeboat drill is sound, and the crew know their stations. But are there equipment and supplies in the boat and are they any leaks?

1. Are your emergency drugs to hand, all together, sterile syringes and needles in the same box, succinct instructions in the lid? Are any of the drugs out of date? Do you know that for certain? Who checks them, and how often?

2. Where are the airways and the KY Jelly? Are they all there? Have they all got tapes on? Who checked them last, and when?

3. Are the batteries in your laryngoscope still sound? Have you spares and a spare bulb? Are they regularly checked? By whom?

4. Have you still got your Magill forceps? Has anybody oiled the joint recently?

All in order? Good! But is your team really as good as all that? Honestly? If it is not, it should be, should it not? It is up to you: a team cannot be led from behind.

Nothing succeeds like success, and there is no greater boost to morale than feeling oneself part of a team which is thoroughly on top of its job; one which it is a privilege to join. This is as true in general dental practice as in the cup winning sports team.

In Summary:

Remember that intravenous sedation is a simple, safe, technique that will help many of your patients to have a more pleasant dental experience. If you listen to the voices of experience, follow the guidelines, and use common sense you will find it a safe and stress free addition to your practice.

Good luck and do try it !