CONTINUING EDUCATION
COURSE MANUAL

INTRAVENOUS SEDATION IN DENTISTRY

7TH EDITION

The New Zealand Society for Anaesthesia and Sedation in Dentistry Inc. (NZSSD)

https://www.dentalsedation.co.nz/

Continuing Education Course Manual

Intravenous Sedation in Dentistry

6th Edition

Edited by Paul Templer

Revised March 2023 by Graham Shaw and Don Macalister

INTRODUCTION

ACKNOWLEDGEMENTS

The Editor wishes to thank the members of the Education Committee of the New Zealand Society for Sedation in Dentistry for their contributions.

Credits

Some images in this manual are used with the kind permission of Wim J.E.P. Lammers, MD, PhD, formerly Professor, Dept of Physiology, College of Medicine & Health Sciences, UAE University, Al Ain, United Arab Emirates, and formerly Honorary Professor, Bioengineering Institute, University of Auckland.

PREFACE TO THE 6TH EDITION

The aim of this manual is to provide trainees with an overview of sedation in dentistry. It serves as a complementary resource to a series of lectures and workshops. Although we cannot cover every aspect of sedation, we will focus on teaching the safe technique of intravenous midazolam as the initial skill.

We prioritise patient safety throughout this manual and emphasise the need for caution during practice to ensure the safety of both the practitioner and patients. You may notice some repeated information in certain sections, which is intentional as the information 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.*

Paul Templer
Department of Anaesthesia
Dunedin Hospital
March 2019

NZDA CODE OF PRACTICE, DCNZ PRACTICE STANDARD, & MISUSE OF DRUGS ACT

This course manual was developed in conjunction with the **NZDA Code of Practice for Sedation** and the **DCNZ Practice Standard for Sedation**. While some of the information may be repeated in both documents, our aim is to emphasise the significance of this information.

Familiarity with both documents is essential as they outline the standards we must follow to comply with our governing bodies. It is crucial that you read and understand them thoroughly.

Please note that both documents are subject to regular review, and NZDA and DCNZ will typically inform practitioners when a document has been updated. You must review and comply with any future changes.

NZDA Code of Practice for Sedation

To load the CoP (PDF) in your browser, click here »

Or copy & paste this link into your browser:
https://www.nzda.org.nz/assets/files/Standards__Guidelines/Codes_of_Practice/CoP_Sedation_for_dental_procedures.pdf

DCNZ Practice Standard for Sedation

To load the Standard (PDF) in your browser, click here »

Or copy & paste this link into your browser:
https://www.dcnz.org.nz/assets/Uploads/Practice-standards/Sedation-practice-standard-April-2017.pdf

Misuse of Drugs Act 1975

To view the Misuse of Drugs Act 1975 (web page) in your browser, click here »

To download a **PDF** version of the Misuse of Drugs Act 1975, go to the web page and click the "Print/Download PDF [1.0MB]" link at the top-right of the page.

Or copy & paste this link into your browser:
https://www.legislation.govt.nz/act/public/1975/0116/latest/DLM436101.html

CHAPTER 1: BEHAVIOURAL CONSIDERATIONS

For many people, even the thought of a routine dental check-up can be daunting and cause feelings of shame.

Sedation in dentistry aims to provide patients with a comfortable, respectful, and non-threatening dental experience. It allows people to overcome their fears and reservations, enabling them to take care of their dental health.

By removing the anxiety associated with dental procedures, sedation dentistry helps patients move beyond avoidance and take control of their oral health.

Despite significant advancements in dental technology in recent years, the issues of anxiety and pain must still be addressed if everyone is to benefit from these developments. The reality is that a vast number of patients worldwide continue to avoid regular dental treatment due to fear, regardless of whether dentists acknowledge this fact.

In dentistry, discomfort, anxiety, and apprehension are tolerated far more than in any other healing profession.

Although modern dentistry has made significant technical advances, dental anxiety remains a major obstacle to providing care to all. Despite the availability of effective local anaesthetics that can eliminate pain, many patients still experience anxiety that can cause exaggerated responses even to minor stimuli, creating stress for everyone involved and making it difficult to maintain high treatment standards.

While a complete psychological assessment of every apprehensive dental patient would be ideal to discover and address the underlying causes of their anxiety, this is often impractical in a busy practice due to a shortage of qualified staff. Therefore, it may be justifiable to consider pharmacological interventions to alleviate patient anxiety as a means of achieving initial dental fitness and cooperation. Once these are established, patients may become more receptive to other approaches.

Sedation can greatly improve the dental experience for both patients and dentists. It can save time and money for patients while allowing dentists to work more efficiently, especially with anxious patients. Additionally, more work can often be completed in one or two long appointments compared to multiple shorter appointments. The traditional approach of treating dental issues bit by bit is often frustrating for patients who would 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. It is essential to understand that the effectiveness of sedation can be greatly improved by building a strong interpersonal relationship and trust between you and your patient.

Additionally, it is important to have a good understanding of the factors that affect pain tolerance, and how we can supplement the use of drugs with various anxiety-reduction techniques. By doing so, we can achieve the best of both worlds and provide optimal care for our patients.

The importance of managing both psychological and pharmacological aspects of treatment cannot be overstated. Effective patient management requires consideration of many interrelated factors, and no single modality - whether psychological or pharmacological - can solve all problems.

Managing human beings, especially anxious dental patients, is complex, and not always straightforward. While more dentists are using various pharmacological techniques, we must recognise that success or failure of these techniques is directly linked to an understanding of behavioural factors.

Major Dental Fears

What are dental patients afraid of? After pain, the most common fear among dental patients is the local anaesthetic injection, despite its role in providing painless treatment. The sight and sensation of the injection, as well as its possible side effects, are closely followed

by the sound and sensation of the dental drill as the most anxiety-provoking stimuli.

Patients may also fear the unknown, the loss of control, and becoming immobilised and helpless during treatment, which can trigger hostility and aggression in some individuals lacking trust.

Other fears include surgery and its potential effects such as mutilation and blood loss, as well as the fear of general anaesthesia and its after-effects. Furthermore, patients may fear tooth loss, which can represent ageing, loss of attractiveness and sexuality, and degeneration of the body, as well as the challenges associated with full dentures.

Fear of the dental surgery and its contents can also be a significant concern. Many patients experience anxiety simply due to the dental environment - the appearance, smells, and the people involved in the treatment process.

Factors Affecting Pain Tolerance

What can be done to improve this situation? It is important to recognise that although the threshold for recognising pain (or pain perception) is approximately the same from one person to another, the degree to which each person reacts to pain (pain tolerance) varies tremendously.

Research has clearly shown that the degree of anxiety present is perhaps the single most important factor in pain tolerance. It is

well-established that the higher the degree of anxiety, the lower the tolerance of pain.

Anxiety not only affects pain tolerance, but it also has a marked physiological effect. The autonomic nervous system is activated, triggering the "fight or flight" response, and the physiological side effects produced create a vicious cycle of increasing anxiety and physiological responses.

The major changes that occur are an increase in heart rate and blood pressure, sweating, salivation, a shift of blood from the viscera to the muscles, and the release of large amounts of glycogen from the liver. Digestion slows or ceases altogether, and the stomach wall may contract. The latter is a significant reason for the unpredictability of oral sedation.

Reducing patient anxiety will increase pain tolerance and therefore the effectiveness of your sedative and anaesthetic techniques. But how can we achieve this?

ANXIETY REDUCTION TECHNIQUES

Dentist/Patient Relationship

The most important method of controlling anxiety is for you to establish a good therapeutic relationship with your patient. The quality of sedation is dependent on the degree of rapport and trust established with your patient.

To achieve this, you must have a sound knowledge of your patient's background, be prepared to spend time discussing their fears and anxieties, and show genuine concern for their welfare and comfort.

Empathising with your patient and allowing sufficient time for discussion and patient education during the initial consultation is essential.

When you are skilled in establishing a good relationship, you will find your patient becomes more cooperative, trusting, prepared to accept professional advice, and open to suggestion.

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, research indicates that certain behavioural techniques may lead to the production of powerful endogenous analgesic agents.

The complete solution to the problem of anxious dental patients is multidisciplinary, integrating both behavioural and pharmacological approaches, along with a thorough understanding of the factors that influence pain tolerance and how these can be modified.

These are the views of P.A. Foreman, a pioneer of New Zealand dental sedation and a former teacher and mentor.

CHAPTER 2: BASIC SCIENCE & PATIENT EVALUATION

CARDIOVASCULAR PHYSIOLOGY

The cardiovascular system includes...

- the heart,
- arteries,
- veins,
- and microcirculation (including capillaries).

Blood is pumped from the heart into the arteries, which distribute it through smaller and smaller branching arteries and arterioles until it reaches the capillaries (the smallest blood vessels in your vascular system). This is where the major exchange of nutrients, gases, and waste products occurs between the blood and tissues.

Blood from the capillaries then flows into venules (the smallest veins), which join together to form small veins that flow into bigger and bigger veins before eventually flowing into the heart through the superior and inferior venae cavae.

To ensure that the blood keeps flowing in the right direction, veins have one-way valves.

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

The atrium and ventricle on the right side collect the used systemic blood returning in the great veins and pump 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 that supplies the entire body.

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

Right atrioventricular 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

There are two circuits of blood flow: pulmonary circuit and systemic circulation.

Pulmonary Circuit

The pulmonary circuit pumps deoxygenated blood from the right side of the heart to the lungs, and freshly oxygenated blood from the lungs back to the left side of the heart.

Systemic Circuit

The systemic circuit pumps oxygenated blood from the left side of the heart to the body tissues, and deoxygenated blood from the tissues back to the right side of the heart.

Initiation of Heartbeat

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

The contraction, known as systole, pumps blood out through the pulmonary and aortic valves.

The relaxation, known as diastole, allows the heart to fill up with blood returning from the veins.

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

Different parts of the heart vary in rate, but the sinoatrial (SA) node, situated in the wall of the right atrium near the site of entry of the superior and inferior venae cavae, has the highest rate of

spontaneous diastolic depolarisation. This acts as the heart's pacemaker.

These cells reach threshold for depolarisation earlier than their neighbours, and the action potentials that result trigger action potentials in the other cells. Impulses originating from the sinoatrial 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.
- The heartbeat is all or none.
- Cardiac muscle excites itself, unlike skeletal muscle, which requires 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

Fibres from the vagus nerve extend to the heart. Impulses in the vagus nerve cause acetylcholine to be released from nerve endings, which decreases the rate of diastolic depolarisation in the SA node. This lengthens the interval between cardiac impulses and slows heart rate. There is no effect on ventricular contraction, as the ventricles are virtually free of any parasympathetic stimulation.

Sympathetic

Sympathetic nerves that reach 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 depolarisation, shortens the interval between cardiac impulses, and speeds up heart rate.

During times of stress, adrenaline is released from the adrenal gland. This has a similar effect to noradrenaline, as well as causing the cardiac muscle to contract more forcefully.

The ECG

The passage of an electrical impulse through the heart produces a 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 is the volume of blood flowing out of either the right or left side of the heart per minute. It is calculated by stroke volume (the volume ejected during each heartbeat) multiplied by heart rate and measured in litres per minute.

Stroke volume (SV) is determined by venous return. The heart can only pump out the blood that comes into it. If venous return increases, SV increases because the myocardial muscle (in a healthy heart) will contract more forcefully if stretched ("Starling's Law of the Heart").

Cardiac output also increases 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 - arteries, veins and capillaries.

Fluid flow in tubes has a number of properties. It requires a pressure difference between one end of the tube and the other; otherwise, fluid will not flow.

Resistance to fluid flow is due to the tube's length and diameter, and the fluid. A short tube has less resistance than a long one, and a wide tube has less resistance than a narrow one.

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

Resistance ~ Length / Radius ^ 4

Circulation is very sensitive to small changes in the caliber (diameter) 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 is calculated by multiplying cardiac output by peripheral resistance.

Peripheral resistance is largely determined by the arterioles, which 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 to the total resistance to blood flow, and changes in their diameter can cause profound changes in the total vascular resistance.

The 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 causes constriction
- Acetylcholine causes dilation
- Bradykinin causes vasodilation
- Antidiuretic hormone (vasopressin) is a vasoconstrictor

- Angiotensin causes vasoconstriction
- Local needs, such as when a muscle contracts while doing work, increase its requirement for blood supply. There results a local depletion of oxygen and an accumulation of carbon dioxide in the tissues, which increases blood flow by directly dilating the vessels. The accumulation of lactic acid and the the drop in pH also cause dilatation in arterioles.

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

Baroreceptors

The carotid sinus, located at the bifurcation of the carotid artery that carries blood to the brain, contains stretch receptors that are sensitive to distension in the artery.

These receptors, called baroreceptors, are important in the regulation of blood pressure.

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. These impulses enter the medulla oblongata, where fibres go to the cardiac and vasomotor centres.

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 fewer impulses travel out along the sympathetic nerves throughout the body, and the peripheral arterioles relax.

These actions result in a decrease in cardiac output and peripheral resistance, lowering blood pressure towards normal.

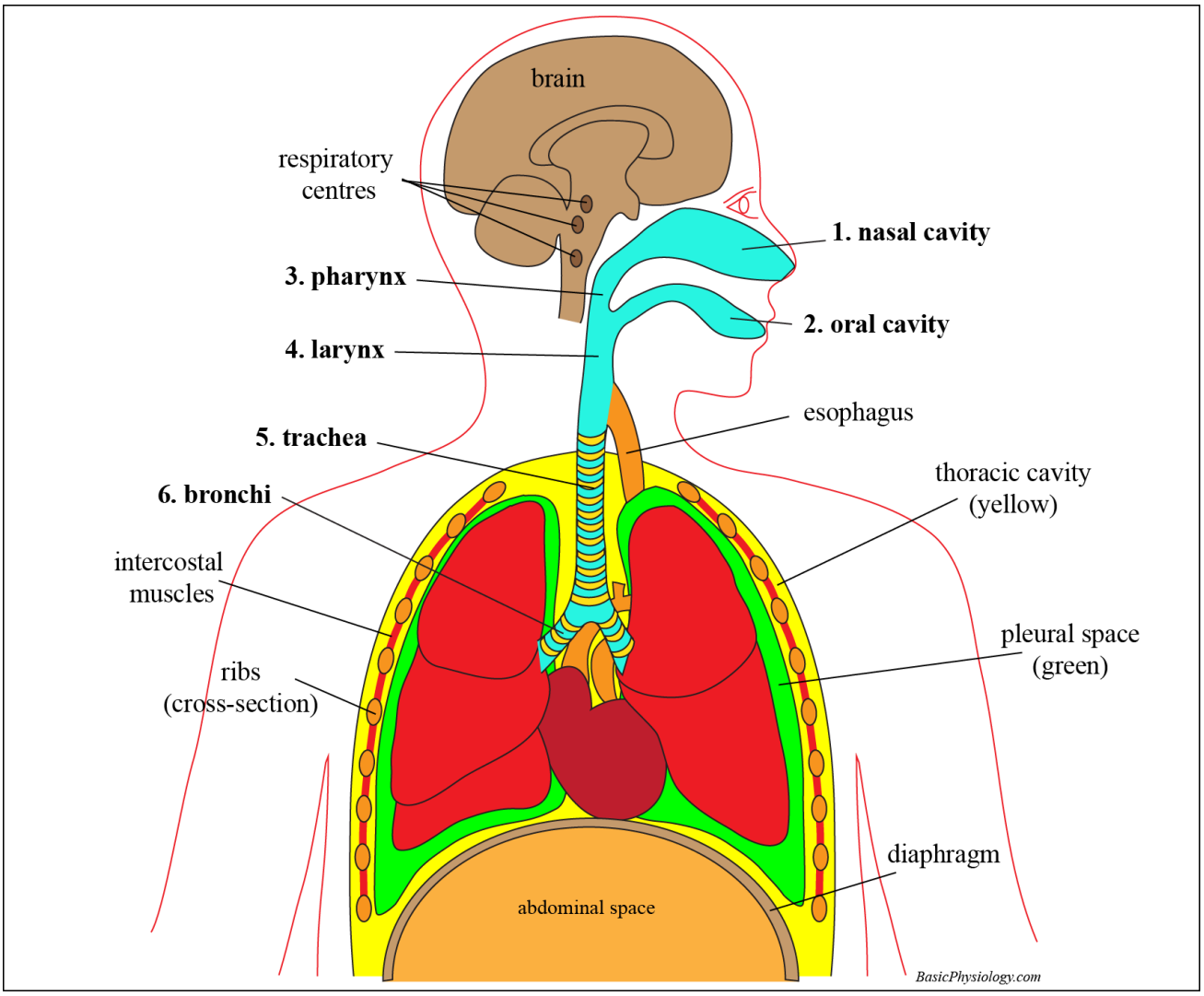
The baroreceptor reflex is essential in the moment-by-moment maintenance of arterial blood pressure, helping the body deal with changes associated with alterations in posture or acute haemorrhage.

RESPIRATORY PHYSIOLOGY

The purpose of respiration (breathing) is to supply oxygen (O_2) to tissues and remove carbon dioxide (CO_2). To accomplish these goals, respiration can be divided into four major functional events:

1. **Pulmonary ventilation**: the inflow and outflow of air between the atmosphere and the lung alveoli (tiny, balloon-shaped air sacs located at the end of the bronchioles, the branch-like tubes in the lungs).
2. **Diffusion of O_2 and CO_2**: the exchange of gases between the alveoli and the blood.
3. **Transport of O_2 and CO_2**: the movement of gases in the blood and body fluids to and from the cells.
4. **Regulation of respiration**.

The Respiratory Tract



When we breathe in, air travels through the **upper respiratory tract** (nose, pharynx, and larynx) into the trachea. The paired nasal cavities have a large surface area lined with ciliated columnar epithelium that contains 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, air is **filtered** to remove foreign particles, **warmed** to 37°C, 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 gas entering through the nose. 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 structure that contains the vocal cords separated by an aperture called the **glottis**, which closes during swallowing.

When air passes through the glottis (the space between the vocal cords in the larynx), it causes the vocal cords to vibrate and produce sounds. The amplitude and pitch of the sounds can be altered by the speed of air movement and the size of the glottis.

Changes in glottal aperture also occur in normal breathing. The glottis dilates during inspiration and constricts during expiration, which increases airway resistance and prolongs expiration.

The laryngeal muscles, which control the opening and closing of the glottis, are made up of skeletal muscle tissue and are innervated (supplied with nerve fibres) by the recurrent laryngeal nerve (RLN).

Another nerve branch that comes off of the vagus nerve is the superior laryngeal nerve. This nerve contains afferent fibres, which

means that it carries signals from the mucosal irritant receptors to the brain. These signals then initiate the cough reflex, which is a protective mechanism of the respiratory system to clear irritants or foreign particles from the airways.

The **lower respiratory tract** starts with the **trachea**, which divides into the two main **bronchi**, one to each lung. These bronchi repeatedly subdivide within the lung unit until, after about 23 divisions, the alveoli are reached.

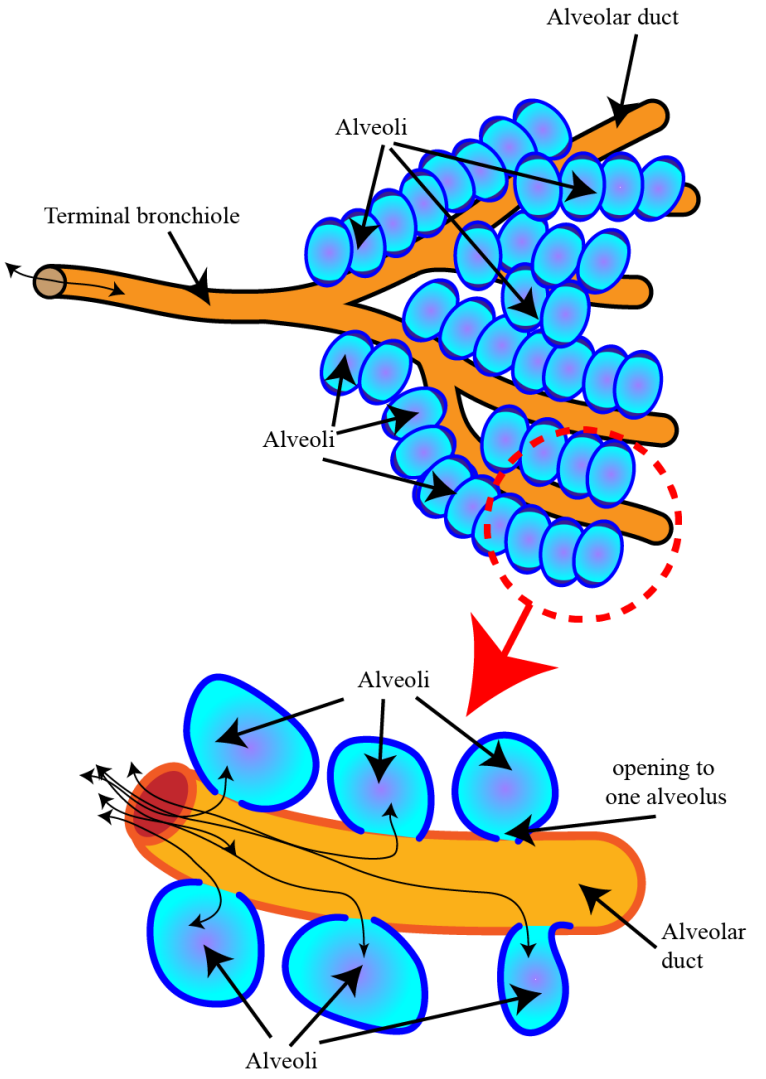
Mucoserous glands in the submucosa and mucus-secreting goblet cells in the epithelium produce a fluid that helps humidification, and helps trap particles and pollutants. The cilia then move these particles towards the pharynx.

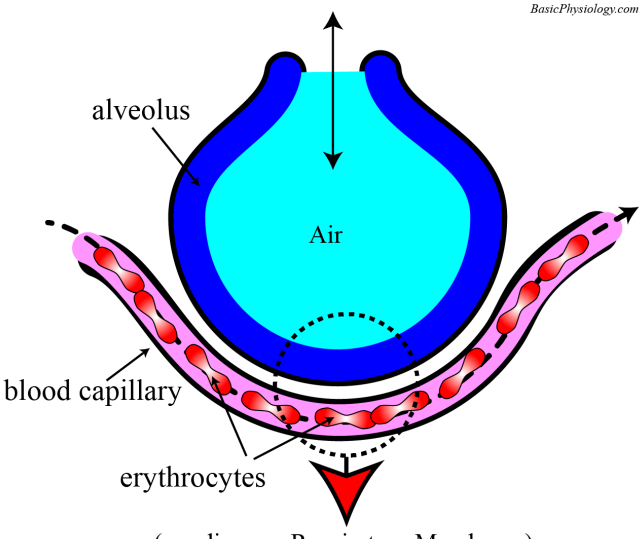
There are 300-600 million alveoli in the lungs, Each **alveolus** has a diameter of about 0.01 mm at the end of a normal expiration. Together, they provide 50-90 m^2 of surface area for gas exchange.

The alveoli abut with one another, so the wall (interalveolar septum) of one alveolus is shared with another. The alveolar wall is, in effect, a dense capillary network with several 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, collectively referred to as the alveolar-capillary membrane or air-blood barrier.

The interstitium contains reticular and elastic fibres, which provide the lungs with some elasticity.





(see diagram Respiratory Membrane)

Alveolar capillaries receive blood with low O_2 and high CO_2 levels through the pulmonary circulation.

Capillaries that supply the walls of larger airways, on the other hand, receive oxygenated blood from the aorta through the bronchial part of the systemic circulation.

A. Pulmonary Ventilation

Mechanics of Pulmonary Ventilation (How Breathing Works)

The lungs expand and contract in two ways:

1. The diaphragm moves up and down to change the length of the chest cavity.
2. The ribs move in and out to change the diameter of the chest cavity.

Normal breathing is mainly driven by the diaphragm. When we breathe in the diaphragm contracts and moves down, pulling the lower surfaces of the lungs down with it. This creates space for the lungs to expand and fill with air.

When we breathe out, the diaphragm relaxes and moves up, aided by the **elastic recoil** of the lungs and chest wall, which helps push air out of the lungs.

During heavy breathing, such as during exercise, the abdominal muscles can apply extra force to help push air out of the lungs by pushing the diaphragm upward.

The second way the lungs can expand is by expanding the rib cage. The muscles that raise the rib cage when breathing in are the **external intercostals**, the **sternocleidomastoid**, the **anterior serrate**, and the **scalene**.

The muscles that contract the rib cage when breathing out are the **abdominal recti** and the **internal intercostals**.

Respiratory Rate

Adults normally breathe about 12 times per minute. However, this can change depending on factors such as activity level, anxiety, pregnancy, and certain medical conditions.

Children tend to breathe faster than adults, but their respiratory rate slows down as they get older.

It is common for physically fit individuals or those who have been sedated to have a slower respiratory rate.

B. Diffusion of Oxygen and Carbon Dioxide in the Respiratory Process

The transfer of oxygen and carbon dioxide between the air sacs (alveoli) in our lungs and our blood is a crucial part of the respiratory process.

This process, known as diffusion, occurs naturally as molecules move randomly across the respiratory membrane.

As fresh air fills the lungs, oxygen molecules diffuse through the alveoli into the bloodstream. Meanwhile, carbon dioxide molecules move in the opposite direction, diffusing from the bloodstream through the alveoli, where they are exhaled.

The air we breathe in contains a higher concentration of oxygen compared to the deoxygenated pulmonary blood in our lungs. As a result, oxygen moves naturally from the air into our bloodstream following a concentration gradient.

The concentration gradient in this case refers to the difference in concentration of oxygen between the air in the lungs and the bloodstream, which are separated by the lung membrane, or alveoli. Oxygen flows from the region of higher concentration (the air in the lungs) to the region of lower concentration (the bloodstream) until the concentration is balanced on both sides of the barrier, thereby oxygenating the pulmonary blood.

Conversely, carbon dioxide moves down its own concentration gradient, from the bloodstream (higher concentration) through the alveoli into the lungs (lower concentration), where it is exhaled back into the air.

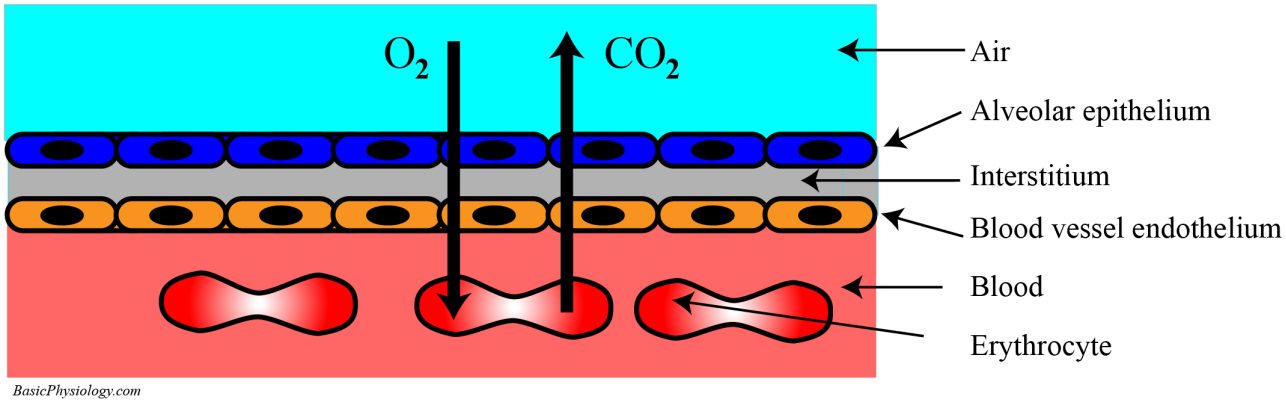
C. Transport of Oxygen and Carbon Dioxide in the Blood and Body Fluids

Once oxygen is in the bloodstream, it travels through the body and is transported to the cells, where it is used for energy.

The oxygen is usually carried in combination with haemoglobin, a protein in red blood cells.

As the cells use oxygen, they produce carbon dioxide, which is then transported back to the lungs and exhaled.

The respiratory membrane:



D. Regulation of Respiration

Breathing occurs in a rhythmic pattern, with the rhythm generated by respiratory centres located in specific areas of the medulla oblongata and pons. These centres include the dorsal respiratory group (DRG), ventral respiratory group (VRG), Botzinger complex, and pontine respiratory group (PRG).

The depth and frequency of the resting rhythm are determined by feedback from mechanoreceptors in the lungs and chest wall.

Certain stimuli, such as temperature and pain, can affect breathing through higher brain centres like the hypothalamus and limbic system, which then influence the respiratory centres.

Breathing can also be voluntarily controlled by the cerebral cortex, as seen in activities like breath-holding, taking large breaths, or speaking. This voluntary control bypasses the respiratory centres.

In addition to the normal breathing pattern, there are protective reflexes of short duration, such as coughing or sneezing, which are often triggered by irritants.

The primary control of respiration comes from chemoreceptors located both peripherally (in the carotid and aortic bodies) and centrally (within the brain).

Breathing is stimulated more by high levels of carbon dioxide (hypercapnia) than low levels of oxygen (hypoxia). As a result, respiration is regulated based on the amount of carbon dioxide produced rather than the amount of oxygen consumed.

CHAPTER 3: PATIENT ASSESSMENT

Patient Selection

This section discusses the various factors that need to be taken into account before offering intravenous (IV) sedation to a patient.

Specific Indications

1. Management of fear and anxiety.
2. Unpleasant procedures, such as oral surgery, periodontal surgery, and extensive crown and bridge procedures.
3. Lengthy restorative, periodontal, or surgical appointments. IV sedation is also suitable for patients with limited time who want to complete their treatment in as few visits as possible.
4. Patients with severe gag reflexes may benefit from IV sedation.
5. With the appropriate experience, IV sedation can be used in special needs dentistry.

MEDICAL HISTORY AND EVALUATION

Physical Assessment

History: The pre-operative medical and dental history should be...

- Concise and relevant to the dental situation.
- Thoroughly examined for discrepancies and omissions, with any doubts clarified before proceeding with treatment.
- Signed by both the patient and the doctor.
- Regularly updated and reviewed prior to each new course of treatment.

A printed, dated, and signed medical history form is the most effective method (see appendix). This approach encourages patients to carefully consider each question, reducing the likelihood of them forgetting or overlooking important information.

Instances of deliberately misleading medical histories or withholding crucial information highlight the importance and potential legal implications of having a signed medical history.

The medical history should enable the assessment of most past and present medical conditions. Any uncertainties must be discussed with the patient, and if further information is needed before treatment, the patient's doctor should be consulted. A phone call is typically sufficient, and doctors are usually appreciative and cooperative. Inform the general practitioner (GP) about the

proposed treatment, the need for effective sedation, and the drugs intended for use. Discuss hospitalisation versus in-office treatment for borderline cases.

Signed, informed consent must be obtained before any treatment. If multiple treatment options are possible (e.g. deep caries requiring endodontics, or a cracked tooth needing extraction), include consent for any necessary treatment variations.

Always prioritise prevention. High-risk patients should be hospitalised and not treated in the dental surgery. Regularly revise all medical histories at future appointments.

SUMMARY: VITAL SIGNS IN DENTISTRY

Blood Pressure

Blood pressure consistently above 140/90 mm Hg in a resting, non-anxious patient indicates arterial hypertension. If in doubt, take three consecutive measurements and consider the lowest one correct.

- Mild hypertension: 140/90 to 160/95; sedation if needed.
- Moderate hypertension: 160/95 to 200/115; stress reduction.
- Severe hypertension: Above 200/115; medical emergency.

Mild hypertension rarely causes symptoms. Measuring blood pressure during the initial physical evaluation can identify treatable hypertension, providing significant public health benefits.

Refer severely hypertensive patients to their GP for further management, unless dental treatment is an emergency.

Pulse Rate

Normal adult pulse rates range from 60 to 90 beats/min. In a dental setting, 110 beats/min is considered the upper limit of normal.

A rate of 40 to 60 beats/min is normal for highly conditioned athletes (sinus bradycardia).

A rate below 60 beats/min in non-athletes may indicate heart block.

A rate above 110 beats/min may suggest acute or chronic heart disease.

Generally, rates below 60 or above 110 beats/min in adult dental patients warrant investigation.

Bradycardia (pulse rate below 60 beats/min) in a patient with a previously normal rate may signal an impending faint or, less commonly, cardiac arrest. If the patient shows signs of distress (sweating, weakness, shortness of breath, or chest pain), call for immediate medical assistance.

Pulse Rhythm

If a patient's pulse appears to be completely irregular, it may indicate the presence of atrial fibrillation - a heart condition characterised by an abnormal heart rhythm. If they haven't been previously diagnosed or treated, refer them to their GP for accurate diagnosis and management before continuing dental treatment.

Premature ventricular contractions (PVCs) appear as occasional pauses in an otherwise regular rhythm. If PVCs occur at a rate of 5/min or more in a patient with heart disease, medical consultation is necessary.

PVCs often accompany heart attacks and precede cardiac arrest. If PVCs occur at a rate of 5/min or more in a distressed patient (sweating, palpitations, weakness, shortness of breath, or chest pain), call for immediate medical assistance.

Body Temperature

Oral body temperature in healthy individuals ranges from 36°C to 37.5°C.

Respiration

The normal respiration rate for a reasonably apprehensive adult is 16-20 breaths/min. Rates above 20 breaths/min should be investigated, but may just be due to anxiety.

Height & Weight

Investigate unexplained significant weight changes. Extremely overweight or underweight patients pose significant risks during elective dental therapy and are highly sensitive to depressant drugs.

Frequency of Measurement

Measuring blood pressure in adult patients during the first physical evaluation is considered a moral obligation. Practitioners dedicated to comprehensive care also determine pulse rate and rhythm and measure body temperature as indicated by symptoms and signs.

HISTORY NOTES

When examining a patient's medical history, pay close attention to the following conditions...

1. Cardiovascular disease

- Congestive heart failure and valvular disease
- Recent coronary events and myocardial infarction
- Conduction issues
- Angina
- Hypertension
- Congenital heart disease
- Rheumatic heart disease
- Anaemias

Patients with any of these conditions require thorough assessment.

Newly diagnosed or unstable patients should only be sedated in a hospital or high-care setting.

Stable patients may be suitable for sedation once you gain more experience. If uncertain, consult the patient's physician.

Hypertensive patients are typically on medication, so **ensure they have taken their tablets**.

Some antihypertensive drugs can enhance or prolong the effects of analgesics, sedatives, and tranquillosedatives, and may also predispose patients to postural hypotension.

Patients with a history of cardiac disease may be considered for treatment in a dental surgery if approved by their physician and once you become an experienced sedationist. In such cases, careful monitoring and titration of sedation are necessary, and supplemental oxygen may be beneficial.

Tachycardia (increased heart rate) can be harmful for patients with angina or ischaemic heart disease. Exercise caution when using drugs that may increase heart rate, such as local anaesthetic solutions containing adrenaline.

Use aspirating dental syringes to minimise the risk of intravascular injection, which has been reported in up to 10% of dental local anaesthetic injections, particularly in inferior alveolar (mandibular) blocks.

2. Respiratory disease

- Asthma
- Bronchitis
- Bronchiectasis
- Emphysema
- Pulmonary Oedema

Many patients have a history of mild asthma, which typically poses few issues for light sedation. However, a history of severe asthma necessitates medical consultation.

All asthmatic patients should bring their usual inhaler. In the event of an acute attack, use the patient's own (blue) inhaler. If unavailable, any Ventolin inhaler will suffice. If the inhaler is ineffective, promptly seek help, administer oxygen, and consider small doses of adrenaline (see *Chapter 8: Emergencies in the Dental Surgery* for more information).

Bronchitis and other respiratory conditions generally do not present the same challenges for sedation as they do for general anaesthesia.

Patients with moderate to severe emphysema should be treated in a hospital setting or cautiously by experienced sedationists.

3. Endocrine dysfunction

(a) Diabetes

Do not treat uncontrolled diabetic patients in the dental surgery. Controlled diabetics may be treated 4 hours after their normal insulin dose and a light meal.

If severe apprehension is present, consider reducing insulin on the day of treatment after consulting the patient's physician, as such patients may not eat at all.

Many mild diabetics are managed by diet alone; for these patients, schedule appointments between normal meal

times. Avoid lengthy procedures, maintain proper oxygenation, and ensure they have a meal after treatment.

Note: Consult the diabetic patient's doctor before administering sedation.

(b) Hyperthyroidism (thyrotoxicosis)

Do not treat patients with uncontrolled thyrotoxicosis - they require urgent medical attention.

Signs include a 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 concerned, consult with their endocrinologist before scheduling any treatment.

(c) Adrenal insufficiency

Adrenal insufficiency should be considered a possibility if a patient has taken corticosteroids within the preceding 6 months, e.g. for arthritis, ulcerative colitis, asthma, or 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 longer than 3 months or a lower dose for an extended duration (e.g. 2.5 mg for several years), they may be vulnerable to adrenal insufficiency and hypotension.

Discuss the patient with their GP and consider "steroid cover" for the procedure.

4. **Epilepsy**

Midazolam reduces the risk of seizures. Instruct the patient to take their usual medication. Minimise stress and schedule shorter appointments.

5. **Liver Disease**

The liver possesses substantial reserves, and significant damage must occur before clinically relevant issues arise.

Patients with imminent or pre-existing liver failure or active hepatitis should not be treated, except in emergency situations.

Mild liver impairment can occur and may variably affect drug metabolism and distribution. Midazolam metabolism is typically not impacted, but as always, titrate small doses of midazolam to achieve the desired clinical effect.

6. **Pregnancy**

Although not an absolute contraindication during the second and third trimesters, sedation should generally be avoided during pregnancy, especially in the first trimester.

A history of complications in early pregnancies or any indication of hypertension warrants medical consultation.

In advanced pregnancy, use the semi-reclining position to reduce pressure on the inferior vena cava.

Keep in mind that any sedative administered to the mother will affect the unborn child through the placenta. Transmission can also occur to an infant via breastfeeding.

7. Drug Therapy

During dental treatment, various local and general anaesthetics, sedatives, and tranquillisers may be used. However,

Some drugs can interact with the local and general anaesthetics, sedatives, and tranquillisers used during dental treatment. This can cause serious complications.

Therefore, it is important to have an up-to-date drug catalogue to check the uses, side effects, and interactions of any drugs the patient may be taking. It is also important to consider the possibility of illegal drug use.

When administering midazolam, the dose should be adjusted gradually (titrated) until the desired clinical effect is achieved. The goal is to give your patient the right amount of midazolam to achieve the desired level of sedation without giving too much, which could cause adverse effects.

Drug interactions can be complicated, as seen in the example of a patient taking lorazepam for panic disorders. Lorazepam is a long-acting oral benzodiazepine, similar to midazolam. The

patient may require less sedation than expected due to the pre-existing effect of lorazepam. On the other hand, they may require more midazolam to achieve adequate sedation due to down-regulation of their benzodiazepine receptors from chronic use of lorazepam.

It is important to watch for certain drug interactions, such as...

(a) Monoamine Oxidase Inhibitors (MAOI)

Although MAOIs are rare in clinical practice, patients on them are difficult to manage as MAOIs interact with all types of sedatives, anaesthetics and analgesics.

If you ever encounter a patient on MAOIs, it will likely be an elderly patient who has been on them for a long time. Try to avoid sedation as the results may be unpredictable.

However, Moclobemide (Aurorix) is a modern MAOI that has been altered to cause much less severe and significant reactions, making it safe to use with midazolam sedation.

(b) Central Nervous System (CNS) Depressants, e.g. barbiturates, narcotic analgesics

Anaesthetic and sedative drugs, including midazolam, may have a stronger effect on patients who are taking CNS depressants. This is because CNS depressants can enhance the sedative effects of other drugs, leading to a stronger sedative effect than expected.

However, this may not be clinically apparent, so it is important to monitor such patients closely when administering midazolam, and adjust the dose as needed to avoid over-sedation or other adverse effects.

(c) Tricyclic Antidepressants

Tricyclic antidepressants, such as amitriptyline (Amitrip, Laroxyl, Tryptanol) and clomipramine (Anafranil, Clopress), are often used for pain control or sleep rather than depression.

However, patients taking these medications may experience an exaggerated hypertensive response if local anaesthetic solutions containing adrenaline are inadvertently injected intravascularly. Therefore, care must be taken when administering local anaesthetics to these patients.

(d) Selective Serotonin Reuptake Inhibitors (SSRIs) and Serotonin Norepinephrine Reuptake Inhibitors (SNRIs)

These are commonly prescribed for sadness, stress, depression, anxiety, and mood disorders, and are some of the most prescribed drugs in New Zealand. An estimated 350,000 New Zealanders are on SSRIs.

Examples of SSRIs include Citalopram, Escitalopram, Fluoxetine (Prozac), and Sertraline, while Venlafaxine is an SNRI.

Fortunately, although there are theoretical issues and interactions with these medications, they can largely be ignored. Patients should take their medications as they normally would, and dental treatment can proceed as normal.

(e) Cardiovascular Drugs

Antihypertensive drugs are a large and frequently changing group of medications. See New Zealand formulary for an up-to-date list.

Classes include:

- **Peripherally acting ACE inhibitors** like captopril (Capoten), enalapril (Renitec), and lisinopril (Prinivil, Zestril).
- **Alpha-adrenoceptor blocking agents** such as prazosin (Minipress, Hyprosin, Pratsiol), which is also used for prostatic hypertrophy.
- **Beta-adrenoceptor blocking agents** like propanolol (Inderal, Angilol, Cardinol) and metoprolol (Betaloc, Lopresor, Mycol).
- **Angiotensin receptor blockers** like candesartan and losartan.
- **Calcium blocking agents** like amlodipine (Norvasc).

- **Diuretics** like frusemide, bendroflumethiazide, hydrochlorothiazide, and indapamide.
- There are many other drugs and combinations available, e.g. Losartan and hydrochlorothiazide (a combination) or Clonidine (Catapres, Dixavit), which is now mainly used in pain control to decrease sympathic outflow from the brain.

All of these drugs lower blood pressure, as does midazolam. This is generally beneficial, but care must be taken when sitting up patients taking antihypertensive medications, as many of these drugs can cause postural hypotension.

Beta blockers and clonidine reduce the effects of the sympathetic nervous system, making patients less anxious and easier to sedate. Beta blockers have even been used to reduce nerves before exams.

Cardiac glycosides like digoxin (Lanoxin) are traditionally used to treat atrial fibrillation, but have become increasingly rare because dosing can be tricky. These drugs are mainly seen in older patients, and care must be taken when administering local anaesthetics containing adrenaline, as this may trigger arrhythmias.

(f) Anti-psychotics

Anti-psychotics are powerful psychiatric drugs prescribed for schizophrenia and bipolar disorder.

Some, such as Quetiapine and Mirtazapine, are also used in lower doses to treat anxiety and help sleep.

These drugs can be categorised into newer "Atypical" types, including Amsulpride, Clozapine, Olanzapine, Quetiapine, and Risperidone, among others.

Older versions, such as Depixol, Haloperidol, and Chlorpromazine, also exist.

The patient's condition is more important than the anti-psychotic medication though. If they are stable, not hallucinating, and not agitated, proceed with sedation cautiously while staying alert for any unexpected response.

However, if the patient is unstable, actively hallucinating, or agitated, it is not the appropriate time for sedation.

While it may be tempting to simply refuse treatment, people with mental health issues often require sedation and may have poor dental health. The best answer is to avoid sedation in these cases until you have gained sufficient experience.

(g) Stimulants/Drugs of Abuse

Examples are: amphetamines, methamphetamine, methylphenidate (Ritalin), cocaine.

Aside from methylphenidate, most of these drugs have been removed from the legal market due to limited therapeutic

benefits. However, illegal production and distribution persist.

These drugs are often abused, and patients do not usually disclose their usage. You should be vigilant and tactfully question suspected patients, which will often uncover a history of drug abuse.

Sedating patients who take stimulants can be challenging due to brain stimulation. A 24-hour cessation often helps, but inexperienced practitioners should exercise extreme caution.

Patients using prescribed or illicit stimulant drugs can be challenging to sedate due to their brain's heightened stimulation. Ceasing drug use for 24 hours typically helps, but these patients may still present difficulties and should be approached cautiously by experienced practitioners and avoided by inexperienced practitioners.

(h) Anticonvulsants

Examples are: diphenylhydantoin (Dilantin), valproate (Epilim), clonazepam (Rivotril), carbamazepine (Tegretol), phenobarbitone (Gardenal), ethosuximide (Zarontin), acetazolamide (Diamox), clobazam (Frisium), diazepam (Valium), clormethiazole (Hemineurin), primidone (Mysoline).

Anticonvulsants like Dilantin are commonly used to manage epilepsy. Sedatives and anaesthetics like midazolam are not contraindicated, but anxiety before treatment may trigger a seizure.

(i) Corticosteroids

Refer to *3(c) Adrenal Insufficiency* under Patient Notes in Chapter 3.

(j) Alcohol, Cannabis and Other Drugs of Abuse

Alcohol and cannabis are the most prevalent drugs of abuse in New Zealand. Heavy users may be resistant to sedation due to their brain's adaptation to chemical impairment.

Avoid treatment if recent usage is suspected, as drug interactions may occur.

Examples of interactions...

- Alcohol combined with benzodiazepines or other CNS depressants may cause respiratory depression.
- Cannabis combined with benzodiazepines or other CNS depressants may cause restlessness, disorientation, treatment difficulty, or delayed recovery.
- Cocaine combined with psychoactive and sedative/ anaesthetic agents may lead to agitation and confusion.

Note: Patients with a history of drug abuse may also be more resistant to sedation drugs.

CHAPTER 4: DRUG ADMINISTRATION METHODS

When it comes to premedication and sedation, oral methods of drug administration are not always the best choice. This is because factors such as gastric absorption rates, individual tolerance, age, habits, medical history, and degree of pre-operative apprehension can all affect how well the medication works.

In adults, oral premedication can be particularly unpredictable, especially for those who are very anxious and need effective premedication the most. In some cases, the stress of an impending dental operation can delay the absorption of the drug until the least desirable time, such as during recovery from IV sedation or a general anaesthetic.

Another disadvantage of oral premedication is that patients need to be supervised from the moment they take the drugs and accompanied to and from the dental practice.

Intradermal, subcutaneous, and intramuscular premedication methods are more reliable than oral premedication, but they are relatively slow to take effect and are often painful to some degree.

Additionally, all of these methods share the same disadvantage as oral premedication in that dosage must be determined arbitrarily, which can result in under-dosage or overdosage.

On the other hand, the intravenous route is highly effective in dentistry because it creates a predictable and effective level of medication, especially for ambulatory patients (patients who are able to move around).

Advantages of the I.V. Route

1. **Accuracy.** The intravenous route allows for precise dosage adjustments based on the patient's reaction to the medication, reducing the risk of over dosage.
2. **Rapid Drug Action.** The full effect of an intravenously administered drug is seen almost immediately, and at most within a few minutes (latency).
3. **Relatively Painless.** With careful venipuncture technique and the use of sharp, sterile, fine gauge disposable catheters and surface obtundent on the venipuncture site, the administration of intravenous drugs should be painless.
4. **Increments.** Additional doses of the drug can be easily administered as needed ("titration") to maintain optimum drug action during treatment, facilitated by the use of an indwelling cannula.

5. **Supervision.** Direct supervision of patients during the peak action of the drug is possible, which is not always possible with other routes of administration, such as oral.

6. **Faster Recovery.** Intravenous administration results in the peak drug effect being seen at the beginning of treatment. This effect falls off during the operation more quickly than drugs administered through other routes, which results in a shorter duration of after-effects.

7. **Effective in Emergencies.** The intravenous route is usually the best option for handling emergencies, such as acute adrenal insufficiency or acute allergic reactions.

Disadvantages of the I.V. Route

1. **Limited Usefulness in Young, Uncooperative Children.** The intravenous route may not be the best option for young, uncooperative children. Nitrous oxide sedation, oral sedation, or even general anaesthesia may be better options.

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

3. **Systemic Complications.** Overdosage may occur if safe intravenous administration rules are not followed, leading to potential systemic complications.

Precautions

1. **Intravenous procedures seem simple,** but they actually require a high level of responsibility and care from the operator and support staff. Overdosage can easily occur, so you must never use arbitrary "rule of thumb" methods for estimating the dosage. Instead, it's important to consider factors such as drug concentration, speed of injection, age, habits (alcohol, smoking, drugs), medical history, and degree of anxiety.

   Always closely observe the patient during induction and use incremental doses.

   For safe IV administration, use dilute solutions when possible, a slow titrated rate of injection, and carefully monitor the patient.

2. **Staff.** Skilled, responsible, and dedicated support staff are crucial for the safe and efficient operation of an intravenous practice.

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

4. **Post-operative Care.** Adequate recovery facilities must be provided. Ensure that patients remain in the dental surgery until collected by a responsible person, and are supervised at all times during recovery.

It is essential that you undergo training in IV sedation procedures and pharmacology, as well as gain experience under supervision, before performing such procedures on patients.

While patients may readily accept these methods, their use can only be justified if they meet or exceed the same safety factor and treatment standards as other commonly used methods. If these criteria cannot be met, IV sedation should not be used in dentistry.

However, experience shows us that skilled and conscientious dental practitioners who are trained in the proper use of IV sedation can achieve both safety and high treatment standards.

Moreover, using IV sedation offers benefits beyond reducing anxiety and stress for both patients and dentists. Complete treatments can often be performed in one extended appointment, rather than spread out over multiple shorter ones. The traditional piecemeal approach is not only inefficient for dentists, but also inconvenient for patients with limited time.

Another significant benefit is the reduction of emotional and physiological stress in patients.

Principles for safe practice of mild to moderate intravenous sedation in dentistry

1. Moderate intravenous sedation is a drug-induced sedated state. Consciousness is reduced, but patients are capable of responding to verbal instructions, either on their own or in response to gentle touch.

    - The airway remains unobstructed (patent) without requiring any interventions to help them breathe.
    - Natural breathing is typically enough to keep them comfortable and safe.
    - The sedation generally does not result in any major cardiovascular changes, i.e. heart rate and blood pressure remain stable during sedation.

2. The techniques used must maintain a **wide margin of safety** to prevent the patient from entering a state of unresponsiveness, such as deep sedation or general anaesthesia. This can be achieved by taking the following factors into consideration:

    - Titration, or gradually adjusting the dose to achieve a safe target concentration, is critical for safe sedation.
    - Patients have varying responses to sedation drugs, so there is no universal correct dose; dosages must be personalised to each individual patient.

- The effects of sedative and pain-relieving drugs can be affected by the stimulus of the procedure being performed.

3. Sedation exists on a continuum, and it is not always possible to predict where a specific patient will fall on this continuum (as shown in Figure 1 below). Therefore, you must be prepared to rescue patients if their level of sedation becomes deeper than intended.

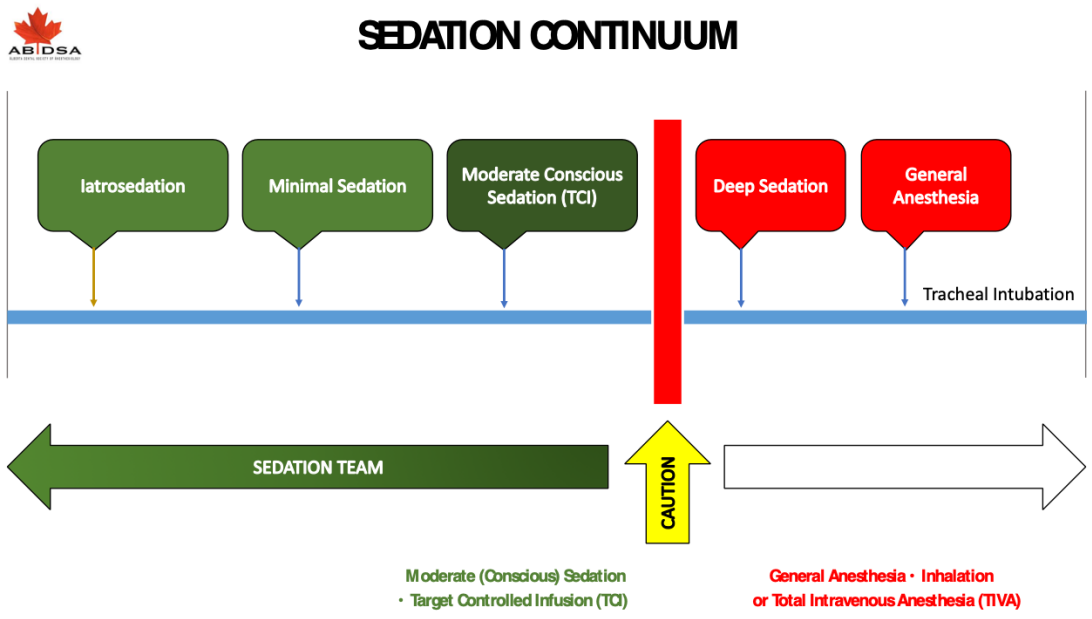


Figure 1. Diagram demonstrating the Sedation Continuum and where TCI-sedation and TCI-TIVA play a role in sedation and anaesthesia. (6)

4. Local anaesthetics are commonly used to manage pain during dental procedures. These drugs work by blocking nerve signals in the area, which reduces or eliminates pain sensation.

5. The risks associated with procedural sedation are directly proportional to the depth of sedation. While sedation is not without risk, the level of risk depends on various factors such as

the depth of sedation, the patient's age and health status, and the competence and skillset of the sedationist and their team.

In Summary

The goal is to achieve a minimal to moderate level of sedation. The amount of sedation needed to achieve this varies based on a number of factors, including:

- Individual biological differences in response to sedation
- Length and type of dental procedure being performed
- Patient age and overall health status.

To ensure safe sedation, it's important to tailor the sedation level to each patient's unique needs and closely monitor their response throughout the procedure.

REQUIREMENTS FOR IDEAL SEDATION

Requirements for the Ideal Sedative Agent for Dentistry

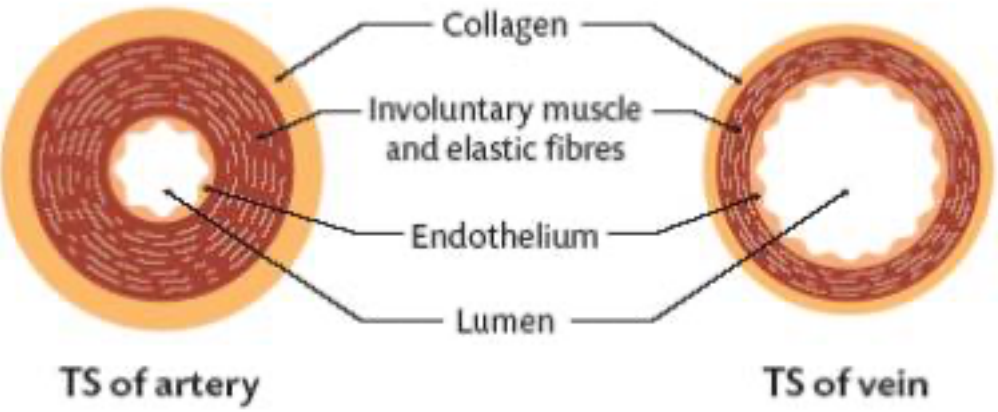
1. **Safety.** The ideal sedative agent should have low toxicity, wide therapeutic safety margins, reliable and predictable actions, and ensure a rapid and complete recovery following treatment.
2. **Selectivity.** It should have anxiolytic properties to reduce anxiety while maintaining verbal contact. It should also provide amnesia for unpleasant procedures, muscle relaxation (especially for masticatory muscles during long procedures), and analgesia (although this is less important when supplemented with local anaesthesia/analgesia).
3. **Freedom from side effects.** The ideal sedative agent should not cause local side effects such as thrombophlebitis or systemic side effects such as respiratory and/or circulatory depression.
4. **Freedom from drug interactions.** It should not interact with other drugs administered to or taken by the patient.
5. **Simplicity of administration.** The sedative agent should be easy to deliver.
6. **High degree of patient acceptance.** It should be well-tolerated and have a high degree of patient acceptance.
7. **Improved treatment quality.** The ideal sedative agent should allow for a higher quality of treatment compared to other means.

CANNULATION

Cannulation Technique

To ensure a safe and successful cannulation procedure, follow these steps:

1. Lie your patient down in a comfortable supine position.
2. Sit close to them.
3. Support their arm on a stable surface like a table arm board or your knee, and immobilise their wrist to prevent movement.
4. Inspect and palpate the vein to select a suitable site for cannulation.
5. Occlude the vein to stop venous flow using a tourniquet.
6. Clean the skin over the vein with an alcohol swab, if desired.
7. Estimate the centre of the lumen and immobilise the vein.



8. Anchor the vein with your non-dominant hand from below by gently pulling on the skin distal to the insertion site.
9. Enter the vein at a 30° angle, aligned with the vein. Once in, withdraw the needle and leave the cannula in place.
10. Place a valve or cap on the end of the cannula.
11. Release the tourniquet and and remove it from the arm.
12. Check if you are in the vein by injecting a few drops of solution or a few millilitres of saline. It should inject easily.
13. Inject the solution at a slow rate, with close observation of the patient throughout.
14. At the conclusion of the treatment, extract the cannula with one quick movement.
15. Apply pressure on the vein for at least 30 seconds with a sterile swab to prevent haematoma formation.

View the "**IV Cannulation NZSSD**" YouTube video to see a visual demonstration of venipuncture with cannula:
https://www.youtube.com/watch?v=0Q8Lf0HB3Ks

Preventing and Managing Complications of Cannulation

Antecubital Fossa Cannulation

When inserting a cannula into a vein in the antecubital fossa (the crease of the elbow), the following complications can arise:

1. Accidental entry into the brachial artery or its superficial ulnar branch. To avoid this, it is recommended to avoid the median basilica vein. Instead, use the cephalic or median cephalic vein and always palpate first before cannulation to ensure proper placement.
2. Potential damage to the median nerve.

Disadvantages of Dorsum of Hand Cannulation

Although the back of the hand or wrist is commonly used for cannulation on the operating table in medical general anaesthesia, it is not as suitable in the dental chair.

1. The skin on the dorsum of the hand is tougher, making entry more difficult.
2. Veins on the dorsum of the hand are usually smaller, which increases the risk of thrombophlebitis.
3. Veins on the dorsum of the hand are poorly supported by connective tissue and tend to slide away from the needle.

4. In older patients, veins on the dorsum of the hand can be thickened, with a narrowed lumen.
5. The risk of haematomas is greatly increased due to the above factors.
6. Haematomas in the dorsum of the hand cannot be easily concealed by the patient, unlike those in the antecubital fossa. This could potentially have a damaging effect on the dentist's professional reputation.

Overall, the antecubital fossa is a more suitable and easier-to-immobilise option for cannulation during dental procedures.

Complications of Cannulation

Cannulation can lead to complications, mainly due to faulty injection techniques. These complications can cause issues ranging from inconvenience to severe disability to the patient, damage to the dentist's reputation, and in some cases may even result in litigation.

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

   Cause: Leakage of blood into the surrounding tissue.

   Prevention: Immediate pressure on the vein after removing the needle or after a failed venipuncture. Using the antecubital fossa instead of the back of the hand can help prevent haematomas.

Treatment: Haematomas can be treated with local heat packs, pressure, massage, and time.

2. **Extravenous Injection**

   Cause: The cannula is partially or completely outside the vein.

   Prevention: Always ensure that the cannula is completely within the vein before injecting. Check the cannula's location frequently.

   Treatment: A small extravenous deposit of solution (one or two drops) will usually disperse and be absorbed without harm if the site is immediately massaged.

   Injection of large amounts of solution extravenously is considered negligent. However, in some cases, one or two drops of solution may be unavoidable, such as when dealing with very small veins or when the needle is only partially inserted (allowing positive aspiration but only partial injection into the vein).

3. **Intra-Arterial Injection**

   Intra-arterial injection is a serious complication that can cause irreparable harm to the patient and must be avoided at all times. It is usually considered negligent, particularly if more than a drop or two is injected.

   Cause: The cannula is inserted into an artery instead of the vein.

Prevention: Before applying a tourniquet, carefully examine and palpate the vein to select a suitable site for cannulation.

Avoid areas where arteries are known to be present, and watch for the aberrant superficial ulnar branch of the brachial artery.

Always aspirate to check for the presence of blood in the syringe, which indicates that the needle is in a blood vessel.

Examine the colour of the aspirated blood to ensure it is the expected colour (usually bright red for arterial blood and darker red for venous blood) and check for pulsation, which would indicate that the cannula is in an artery.

Always start by injecting a very small initial test dose.

If the patient complains of pain that spreads down their arm from the forearm to the hand and fingers, the injection should be stopped immediately. This type of pain should be distinguished from pain that spreads up the arm, which can be caused by an irritant or a cold solution.

Never inject if there is any doubt about the location of the cannula.

These are important safety measures to ensure that the medication is delivered to the correct location and to prevent complications such as an intra-arterial injection.

Symptoms: Severe burning pain radiating down the arm, blanching of the skin, and weakness or absence of the radial pulse (a late symptom indicating thrombosis).

Treatment: Leave the cannula in the artery. Inject up to 10 ml of 1% lignocaine to help relieve pain, dilute the drug injected, and relieve arterial spasm, then remove the cannula.

If pain persists or there is doubt about the prognosis, the patient must be hospitalised for anticoagulant therapy and possible brachial plexus or stellate ganglion block.

Full notes must be sent including the time of injection, site of injection, drug(s) used, dosages and solution percentages, and any treatment given.

No sedation or dental treatment should be performed following suspected intra-arterial injection, as symptoms could be masked, leading to irreversible damage.

4. **Injection into a Nerve**

Cause: Inserting or probing with a needle in an area where significant nerves are present, such as the medial nerve in the inner aspect of the antecubital fossa. Sensory or motor paralysis may result. This risk is increased when using larger gauge or long bevel needles.

Prevention: Whenever possible, only choose visible, superficial veins in the outer aspect of the antecubital fossa. This is particularly important for those who are inexperienced in venipuncture.

Treatment: If pain persists, refer the patient for medical consultation if necessary.

5. Thrombophlebitis

Cause: Inflammation and clotting of the vein (thrombophlebitis) may be caused by a solution that irritates the vein wall or is injected into the vein wall. It may also result from damage to the vein wall during cannulation or insertion of the cannula, or prolonged use of an IV cannula.

Specific Causes:

(a) **Irritant solutions**: Dilute irritant solutions whenever possible, inject them slowly into large veins, and flush with normal saline after injection.

(b) **Large needles**: They pose a greater risk of damaging small veins. Similarly, the use of an IV cannula can also increase the risk.

(c) **Smaller veins**: They are more susceptible to damage during cannulation, leading to haematoma or perivascular leakage.

(d) **Restricted venous return**: Tight sleeves or any condition leading to venous stasis may cause thrombosis formation. Instruct patients to wear loose clothing.

If you are unsure that you have achieved a successful cannulation with good venous patency, consider using a 1-5 ml saline flush to check.

CHAPTER 5: PHARMACOLOGY

Benzodiazepine Pharmacology

Benzodiazepines are a class of drugs that act as central nervous system (CNS) depressants.

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

Presentation

Midazolam is available as 7.5 mg tablets, and ampoules for injection (5 mg/5 ml and 15 mg/3 ml).

Benzodiazepine Pharmacology

The 1,4-benzodiazepine nucleus is the basis of a large number of benzodiazepine derivatives, some of which have typical agonist properties such as anticonvulsant, muscle relaxant, hypnotic,

sedative, and tranquillising effects, while others do not (known as "antagonist" benzodiazepine derivatives).

This text will cover only two benzodiazepines: midazolam (Hypnovel) and flumazenil (Anexate).

The clinically useful benzodiazepine agonists are 1,4-benzodiazepines, most of which 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. A physiological pH of 7.4 maintains the closed ring structure and may enhance lipid solubility.

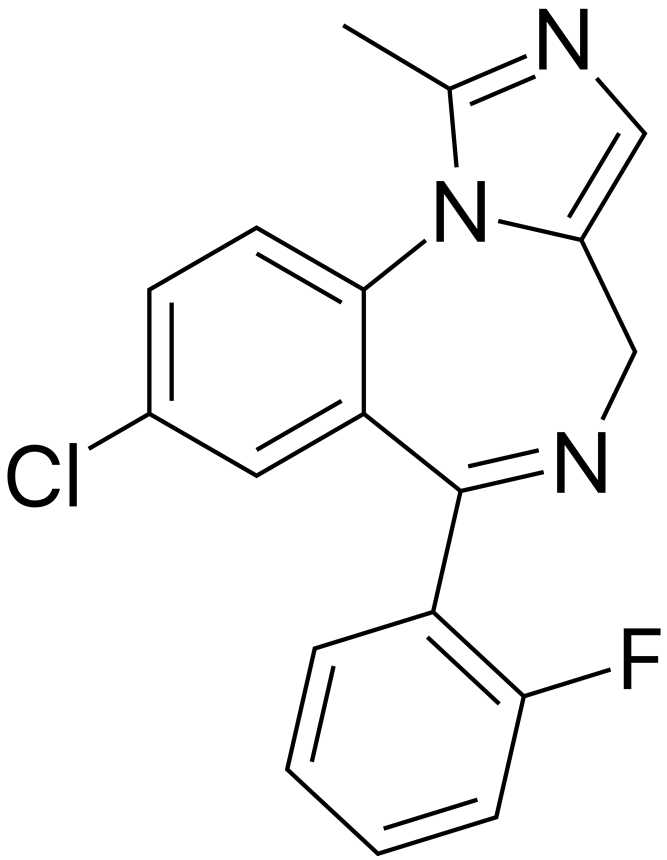
In an acidic aqueous solution, midazolam is water-soluble, and the parenteral formulation therefore does not contain organic solvents (such as propylene glycol in early Diazepam preparations) and is instead buffered to an acidic pH of 3.5.

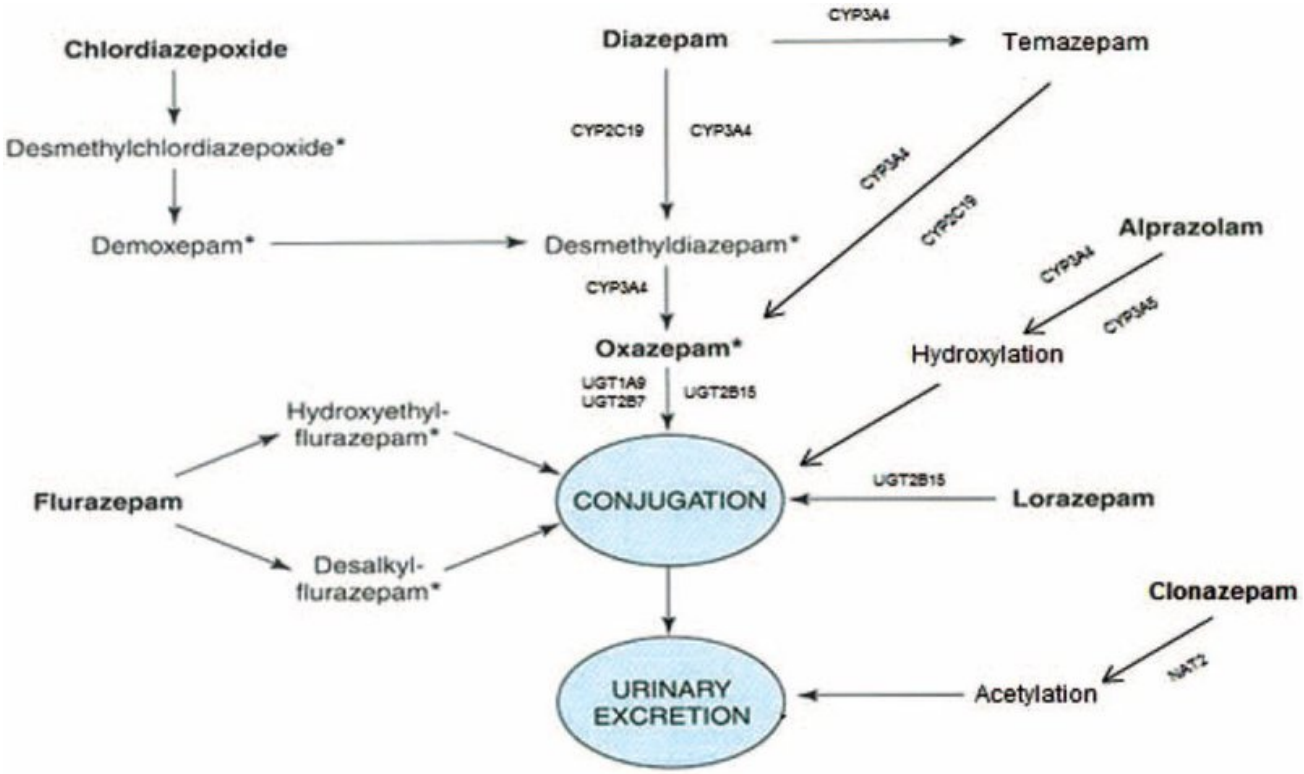
Once midazolam enters the body, the pH rapidly increases to 7.4 and the solubility properties of the drug change.

At physiological pH, midazolam becomes highly lipid-soluble, which increases the rate at which it enters the brain and the rate of onset following IV administration.

Midazolam is the most lipid-soluble benzodiazepine currently available. Its water solubility results in a low incidence of injection pain and venous thrombosis.

Understanding the pharmacology of benzodiazepines is essential for the safe and effective use of these drugs in dental practice.





Mechanism of Action

Gamma-aminobutyric acid (GABA) is a neurotransmitter that plays an important role in inhibiting brain activity, while glycine is a major inhibitory neurotransmitter in the spinal cord and brain stem.

Agonist benzodiazepines enhance GABAergic neurotransmission, resulting in sedation and anticonvulsant effects. In contrast, glycine-mimetic effects in the spinal cord and brain stem are responsible for anxiolysis and muscle relaxation.

N.B. A substance is GABAergic if it produces its effects via interactions with the GABA system, such as by stimulating or blocking neurotransmission. A GABAergic or GABAnergic agent is any chemical that modifies the effects of GABA in the body or brain.

Specific receptors for benzodiazepines were first discovered in the central nervous system in 1977. These receptors are most concentrated in the cerebral cortex, followed by the hypothalamus, cerebellum, corpus striatum, and medulla.

The identification of these receptors (binding sites) has allowed for the development of selective antagonists, such as flumazenil (Anexate), which bind to the benzodiazepine receptor and prevent benzodiazepine agonist drugs from occupying receptor sites.

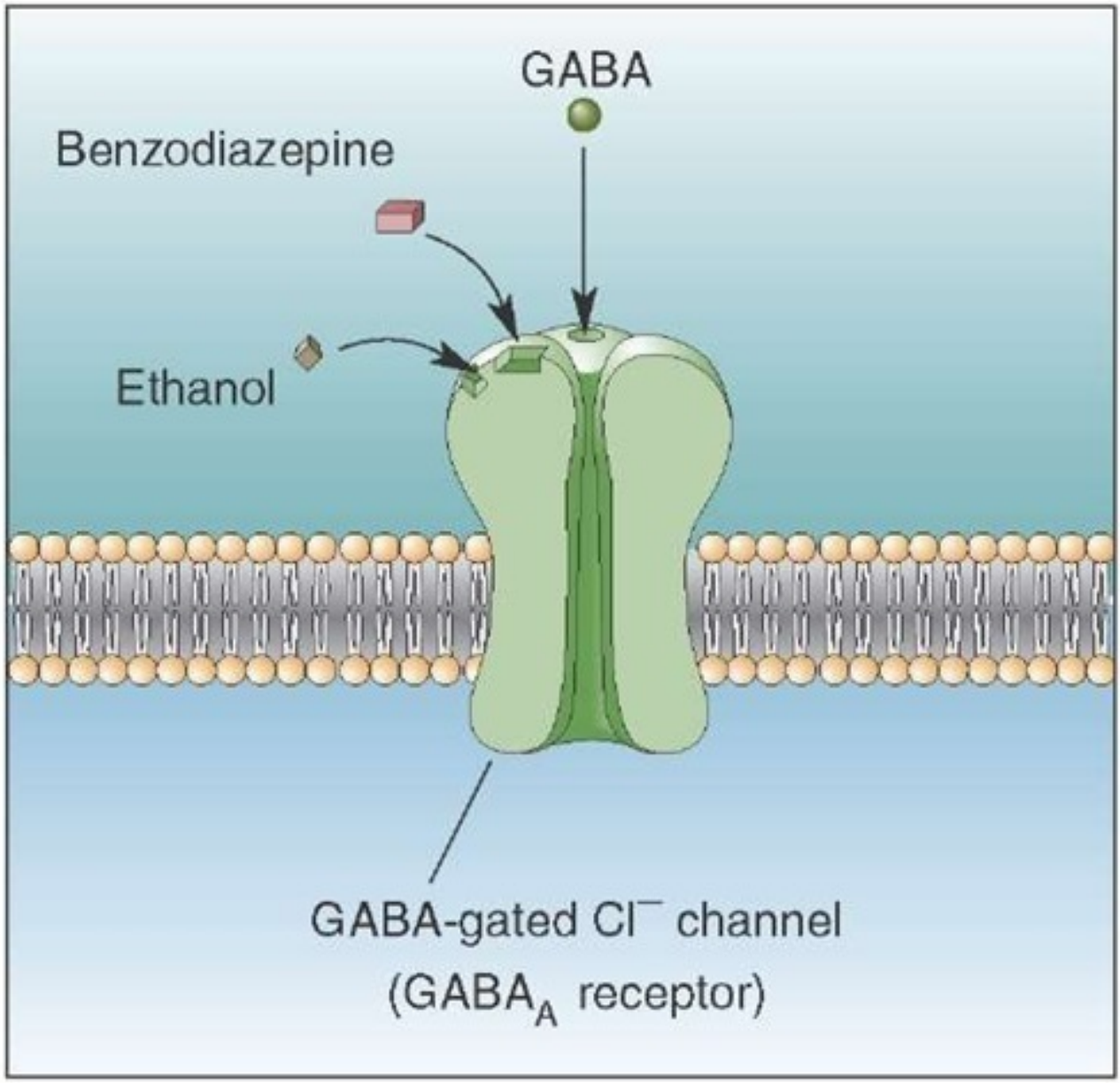
The regional distribution of these benzodiazepine receptors corresponds to the distribution of GABA receptors, suggesting that benzodiazepines enhance the inhibitory effects of GABA.

The relative receptor-binding affinities of benzodiazepines correlate to some extent with their potency.

In addition to the agonist and antagonist benzodiazepine molecules that bind to brain benzodiazepine receptor sites, a third group of anxiogenic benzodiazepine receptor compounds exist. These compounds, such as naturally occurring D-carbolines, can block the effects of benzodiazepine agonists and produce anxiety reactions, pro-convulsant activity, and seizures when administered alone.

These compounds, which are not benzodiazepines but can bind to benzodiazepine receptors, have been termed "inverse agonists". They have the opposite effect of benzodiazepine agonists, causing anxiety instead of reducing it.

These compounds are involved in regulating or modulating the physiological and psychological responses to anxiety in the body. In other words, they may have a role in how the body responds to stress and anxiety, and can affect the intensity or duration of these responses.



Pharmacological Action

CNS

- **Anxiolytic:** At **low doses**, the benzodiazepines are **anxiolytic**.

  - Sedation and induction of sleep.

- **Hypnotic:** At higher doses (**benzodiazepines**) produce **hypnosis** (artificially-produced sleep).
- **Muscle relaxation (skeletal):** By increasing **Pre-synaptic inhibition** in the spinal cord.
- **Anticonvulsant effects:** Several of the benzodiazepines have anticonvulsant activity and are used to treat **epilepsy and other seizure disorders**.
- **Anterograde amnesia**.

Peripheral

- Neuromuscular blockade (high dose)
- Coronary vasodilation (IV)

MIDAZOLAM PHARMACODYNAMICS

1. Central Nervous System

Midazolam acts on interneuronal transmission in the central nervous system, producing skeletal muscle relaxation by inhibiting polysynaptic spinal reflexes, and sedation by inhibiting the limbic system - the area of the cortex believed to regulate emotional influences on cortical activity.

The limbic system is associated with anxiety states, and because of its strong anxiolytic activity, midazolam is widely used to treat anxiety states, insomnia, and premedication.

Midazolam selectively inhibits the activity of the limbic system, which includes the amygdala, hippocampus, and cingulated gyrus. The amygdala may be the site of action of midazolam, where it breaks the patterns of neuronal activity responsible for anxiety.

A drug that selectively inhibits the activity of the limbic system and thus emotional response to external stimuli (anxiety, fear, rage, etc) also has obvious potential for managing anxious and apprehensive dental patients.

The highly selective action of midazolam is of great clinical importance. Instead of general CNS depression, such as that produced by the barbiturates, midazolam mainly acts on 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, generalised CNS depression (coma) can occur.

Midazolam does not possess analgesic properties.

Midazolam does not have pain-relieving properties. However, it does have an important and useful feature of causing anterograde amnesia. Although a patient's perception is not affected, their ability to consolidate new memories is significantly impaired. Even when the amnesia is mild, patients generally report that their dental procedure was much more tolerable than it would have been otherwise.

The degree and duration of midazolam-induced amnesia varies widely between patients and increases with dosage.

When administered intravenously, midazolam usually results in about 10 minutes of good amnesia, which covers the administration of local injections. This is followed by up to an hour or more of impaired memory function. Therefore, it is important to remember that even when a patient appears to be awake and alert after treatment, they may still be experiencing amnesia for some time.

2. Cardiovascular System

When used in small doses for minimal to moderate sedation, midazolam typically does not cause significant changes in the cardiovascular system, except for a reduction in heart rate and blood pressure due to the reduction in anxiety. At low doses, any

anti-arrhythmic effect is likely due to the reduction in anxiety rather than a direct effect of the medication.

3. Respiratory

At anaesthetic doses, benzodiazepines cause dose-dependent respiratory depression, which can lead to apnoea. However, small titrated intravenous doses typically used for premedication and sedation do not significantly affect the natural ventilatory response to carbon dioxide *(how the body responds to increased levels of CO_2 in the blood by increasing the rate and depth of breathing, in order to eliminate excess CO_2 from the body)* in healthy individuals.

Nevertheless, there is considerable variability in patients' sensitivity to the effects of benzodiazepines, including respiratory depression, so patients must be monitored closely.

Benzodiazepines do not cause bronchoconstriction, but in patients with chronic obstructive pulmonary disease or in the elderly, the respiratory depressant effects of midazolam may be more pronounced and prolonged than in healthy patients. Therefore, special attention should be paid to monitoring respiratory function in these patient populations.

You should also be aware of the potential for partial or complete upper airway obstruction caused by benzodiazepine-induced muscle relaxation, with little direct depression of the respiratory centre. To mitigate this risk, you must take care to ensure proper neck and chin support and position the patient to maintain a patent airway throughout treatment.

Note: The above applies to the use of midazolam as a sole agent. When midazolam is used in combination with respiratory depressants, such as opioids, the risks of respiratory depression, apnoea, and hypoxaemia are increased due to potential synergy effects.

This combination should not be used routinely in dental surgeries without experience and additional training due to their inherent risks. There are numerous reports of morbidity and mortality in the literature, particularly with midazolam/opiate combinations.

Similarly, the use of premedication, especially with opioids, should be carefully monitored, and its use is generally not necessary or desirable before most dental procedures.

In most cases, the need for premedication can be avoided with good communication skills, a good chair-side manner, and a compassionate approach to patient care.

4. **Muscle Relaxation**

The muscle relaxant properties of benzodiazepines have several important implications for dentistry.

The benzodiazepine action on polysynaptic reflexes diminishes gag reflexes by interfering with interneuronal transmission. However, monosynaptic reflexes (such as the knee-jerk) and muscle stimulation through its motor nerve are unaffected.

Additionally, benzodiazepines may be useful for some patients with dystonic and athetoid types of cerebral palsy.

The halfway ptosis (droopy eyelids), also known as the "Verrill sign," should not be used as most patients are relaxed, amnesic, and cooperative at dose levels well below those required to produce ptosis. Instead, use the patient's verbal acknowledgement of feeling more relaxed, and the physical evidence of such relaxation.

MIDAZOLAM PHARMACOKINETICS

Midazolam is metabolised in the liver through hydroxylation, producing two major metabolites: 1-hydroxymidazolam and 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.

Due to its high lipid solubility, midazolam rapidly crosses the blood-brain and placental barriers to access benzodiazepine receptors in the central nervous system (CNS). After intravenous administration of 5 mg of midazolam, concentrations decline bi-exponentially, with a distribution half-life of 30 minutes and an elimination half-life of 1-4 hours. The total clearance is about 50% of hepatic blood flow.

Oral midazolam undergoes substantial first-pass hepatic extraction, so about 50% of the drug does not reach the systemic circulation.

Midazolam has a relatively large volume of distribution, short elimination half-life, and 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 unlikely to occur following repeated doses of midazolam due to its high clearance and rapid redistribution.

Elderly males have an increased elimination half-life and reduced clearance of midazolam.

In obesity, the volume of distribution is increased, leading to a prolonged elimination half-life with no change in clearance. Therefore, dose reduction is necessary. On a per-kilo basis, a 120 kg person does not need twice as much as a 60 kg person but will likely 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.

Midazolam should be used with great care in patients with neuromuscular conditions, such as myasthenia gravis. They already have or can have weak muscles, and midazolam can worsen this condition, leading to dangerous respiratory consequences.

CLINICAL SIGNIFICANCE OF THE PHARMACODYNAMICS

After intravenous administration, midazolam takes 1-3 minutes to take effect ("latency"), and there is a wide range of individual responses to the drug.

Most adult patients require a dose between 1-8 mg to achieve sedation, relaxation, and cooperation. However, age and current drug therapy are factors that require dose adjustment.

Elderly patients are often more sensitive to drugs and may require doses as low as 0.25-1 mg. Therefore, slow injection and close patient monitoring are essential during administration, a process known as titration.

Important Note: Midazolam is a specific anxiolytic drug that should alleviate anxiety and induce amnesia before other signs of sedation, such as drowsiness or ptosis, become evident. The patient need not necessarily appear "sleepy."

The duration of clinical action varies but sedation typically lasts up to 30 minutes. Many patients can tolerate an extension beyond this period without distress, even if they appear fully recovered.

However, midazolam has a half-life of 2-4 hours, and there may be a considerable time following apparent recovery before the drug is completely eliminated from the body. Patients should therefore be taken home and supervised after treatment, even if they appear fully

recovered, due to the possibility of prolonged sedative and amnesic after-effects in isolated cases.

The maximum recommended dose of midazolam is 10 mg.

Paediatric dosage should be titrated at 0.05-1 mg/kg, bearing in mind that the dose response is poorly related to weight.

As midazolam can affect psychomotor function for several hours, patients must not operate machinery, drive, or undertake important tasks or business matters on the same day as treatment.

Alcohol consumption must also be avoided as it can have a synergistic effect with midazolam.

MIDAZOLAM SUMMARY

Presentation	Aqueous solution. 5 mg/5 ml amps (We advise you to stock and use only one concentration of Midazolam to avoid confusion about the dose being given. Use 5 mg/5 ml amps for titration with 1mg/1ml to remove confusion about the administered dose.)
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 & CV depression	Minimal at sedative dosages. More likely with rapid administration (especially respiratory depression) or combined with other CNS drugs (hypoxaemia).

FLUMAZENIL PHARMACOLOGY

Flumazenil (Anexate), an imidazodiazepine derivative, is structurally similar to midazolam but does not possess the same agonist properties as other clinically used benzodiazepines.

When administered alone, even in large doses, it has no clinical effects. However, when combined with an agonist benzodiazepine, flumazenil counteracts the central nervous system effects of sedation, amnesia, anxiolysis, muscle relaxation, ataxia, and anticonvulsant properties.

Flumazenil specifically targets the benzodiazepine receptor and does not antagonise (counteract) the effects of barbiturates, opiates, or ethanol.

After intravenous administration, flumazenil takes effect rapidly (within 5 minutes), but its duration is relatively short (about 1-3 hours) due to its short elimination half-life of approximately 60 minutes.

When given to patients sedated with benzodiazepines, flumazenil does not cause significant cardio-respiratory changes, except for reversing any depression.

The total dose required to reverse benzodiazepine sedation is approximately 0.4 mg. An initial dose of 0.1 mg is administered intravenously, followed by 0.1 mg increments up to a maximum of

1.0 mg. There is no need to flush the IV line, as midazolam is fully compatible with flumazenil.

Although patients may seem fully alert after flumazenil administration, ataxia may persist and become noticeable when they attempt to stand. Due to the pharmacokinetics of flumazenil, re-sedation is possible after reversing midazolam sedation, so patients should not be discharged prematurely. However, significant re-sedation is unlikely in healthy individuals receiving midazolam doses up to 10 mg.

Comparison of Pharmacokinetics

	Midazolam	**Flumazenil**
Protein binding:	96%	40%
Volume of distribution:	1.1-1.7 L/kg	0.63 L/kg
Plasma clearance:	265 ml/min	690 ml/min
Distribution half-life:	30 minutes	5 minutes
Elimination half-life:	2 hours	1 hour

Advantages of Flumazenil:

1. Serves as an emergency treatment for patients who are overly sedated.
2. Counteracts adverse reactions to benzodiazepine agonists, such as paradoxical excitation.
3. Speeds up recovery in specific cases, e.g. elderly patients who recovery slowly.
4. Interruption of sedation in certain situations, e.g. during the surgical placement of prosthetics, endoscopy, or specific neurosurgical procedures (e.g., trigeminal nerve ablation).
5. Reversal of sedation at end of procedure, although this is debated. See "*Midazolam - Is Antagonism Justified?*" below for more information.

Flumazenil

Presentation:

IV 0.5 mg/5 ml, 1 mg/10 ml

Disposition:

Flumazenil is broken down by the liver into inactive glycuronides. The resulting metabolite does not affect the pharmacokinetics of midazolam. Flumazenil and midazolam do not have any impact on each other's effectiveness.

Toxicity, Precautions, Contraindications:

Vein tolerance as good as midazolam. Contraindicated in BZ addiction as may precipitate sudden withdrawal 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."

Flumazenil has similar vein tolerance as midazolam. However, it is not recommended for patients with benzodiazepine addiction, as it may lead to sudden withdrawal symptoms.

It is also not recommended to use Flumazenil to sedate patients you would not normally sedate (due to age, psychological makeup or medical history) merely because an antidote is available.

Zopiclone, also sold under the brand name "Imovane," is a hypnosedative drug that belongs to the cyclopyrrolone derivatives

family. Although it is not a benzodiazepine, it acts on the benzodiazepine receptor and can be reversed by flumazenil.

Chronic Benzodiazepine Habituation:
Patients who have been taking benzodiazepine medication in high doses for a long time may develop a tolerance to the recommended sedation doses of benzodiazepines. It can be difficult to sedate such patients, even with large doses.

If these patients suddenly stop taking benzodiazepines, they can experience acute withdrawal symptoms such as convulsions and extreme anxiety. As a result, Flumazenil is contraindicated in such patients. This also applies to zopiclone abuse.

MIDAZOLAM: IS ANTAGONISM JUSTIFIED?

> *"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."*
>
> *SAAD Digest VOL. 7 NO. 4 OCTOBER 1988*

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

Elimination Half-Life and Displacement of Midazolam:
Flumazenil has a much shorter elimination half-life than midazolam in healthy adults, and even more so in elderly or metabolically impaired patients.

Flumazenil displaces midazolam from its receptor sites without affecting its elimination from the body. This means that after the flumazenil is eliminated, the remaining midazolam that has not been metabolised could still be active and cause re-sedation in patients after they have been discharged. This effect is dose-related, but will always be a risk.

Dangers of Increasing Midazolam Dosage:
If the sedative effect is less than desired, there may be a temptation to increase the midazolam dosage to an excessive level, then using flumazenil to reverse the sedation.

This is dangerous because midazolam causes dose-related cardio-respiratory depression, and over-dosage has resulted in increased morbidity and mortality. It is likely that the adverse effects of over-dosage follow an exponential curve.

Midazolam dosage levels must therefore be carefully titrated against a patient's responses, and never exceed the recommended maximum dose level.

Ethical Issues:
Using flumazenil to discharge patients sooner than they would naturally recover is unethical and potentially hazardous, particularly since patients may be discharged to an unknown situation. The risk of re-sedation presents an unacceptable hazard to the patient.

And subjecting a patient, for no good therapeutic reason, to a drug with a drastic effect on their metabolism is morally questionable.

Conclusion:
Although flumazenil has been widely advocated for reversing the action of benzodiazepines, its use poses significant pharmacological and ethical challenges. Careful titration of midazolam against the patient's response, and not exceeding the recommended maximum dose level, is a safer approach.

CHAPTER 6:
IV SEDATION TECHNIQUE

Intravenous Sedation Technique

1. **Preparation of midazolam for injection:**

   - Using a 5 ml disposable syringe, draw up the contents of a single 5 mg (5 ml) ampoule of midazolam.
   - Label the syringe. A black marker pen works well.
   - Record the batch number and expiry date.
   - Note: For dental sedation purposes, it is recommended that only the midazolam preparation containing 5 mg/5 ml be used.
   - 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. This higher dilution has the advantage of providing greater control of dosage during administration.

2. **Supine position:**

   - To ensure patient comfort and safety during sedation, seat them in a chair and then lower them to the supine position.

- We recommend keeping a blanket nearby to help prevent heat loss during long appointments.
- At this time, take initial measurements of the patient's oxygen saturation, blood pressure, and pulse.
- Note: Using the supine position is routine during sedation procedures to help prevent possible low blood pressure and decreased oxygenation to the brain.
- Although midazolam has relatively few negative effects on the cardiovascular system, the manufacturer recommends keeping patients in the supine position for up to one hour following IV administration to reduce the risk of postural hypotension (low blood pressure upon standing). Fortunately, supine is the default position for dentistry procedures.

3. **Tourniquet:**
   - Apply a tourniquet or similar device to the patient's arm and select a large vein in the anticubital fossa for cannulation.
   - It's best if the vein is located in the outer (radial) aspect to avoid the risk of injecting into the brachial artery or an aberrant ulnar branch of the artery, which can be present in up to 18% of patients and can be detected through observation and palpation.
4. **Cannulation:**
   - Use an alcohol wipe to swab the site.

- Verify entry into the vein by observing blood in the flash chamber of the needle and cannula.
- Once verified, stabilise the needle and fully advance the cannula into the vein, ensuring its position by observing a secondary flash of blood up the cannula.
- Then, release the tourniquet.
- Next, remove the cap from the back of the needle and carefully withdraw the needle from the cannula, and fit the cap onto the rear of the cannula.
- Secure the cannula in place with a Tegaderm.

5. **Confirmation:**
   - To confirm successful cannulation you can flush the cannula with 1-2 ml of saline through the ventral port. The same port is used to administer midazolam.
6. **Minimise noise and distractions:**
   - To ensure a successful procedure, it is important to create a peaceful environment free from any unnecessary noise or distractions.
   - Additionally, you can offer your patient reassurance and suggestions to help induce relaxation.

View the "**IV Cannulation NZSSD**" YouTube video to see a visual demonstration of venipuncture with cannula: https://www.youtube.com/watch?v=0Q8Lf0HB3Ks

7. Signs and symptoms of drug effect:

- When administering midazolam, patients may first experience relaxation, drowsiness, and difficulty focusing. They may also exhibit obvious loss of anxiety, slurred speech, muscular relaxation, and lateral nystagmus (a condition in which the eyes make rapid, repetitive, uncontrolled movements).
- While ptosis, as mentioned earlier, was once considered a useful indicator of optimal dosage, it is not a reliable sign and may not occur until dosages approaching overdosage are reached.
- It's important to note that active conversation between dental staff and the patient during induction may prolong sedation. Maintaining a calm, quiet and caring chair-side environment throughout the procedure, with no unnecessary engagement with the patient, will facilitate better sedation.
- The typical midazolam dosage is between 4-10 mg, with an average of around 7 mg. However, dosage must be adjusted based on factors such as age, medical history, and current drug therapy.
- Some patients, particularly the elderly, may be sensitive to benzodiazepines and experience a marked reaction to as little as 0.5-1 mg midazolam. The titration rate must be markedly reduced with these patients.

- It is important to adhere to a maximum dose of 10 mg to avoid undesirable side effects and prolonged recovery, except in rare circumstances.

8. **Local anaesthetic injections:**

- Local anaesthetic injections should be administered with care, using an aspirating syringe to reduce the risk of intravascular injection.

- To avoid toxicity and vasoconstrictor side effects, it is important to avoid using large volumes of local anaesthetic.

- Prilocaine (Citanest) with the synthetic vasoconstrictor felypressin (Octapressin) may be a better option than adrenaline and noradrenaline, as it produces fewer cardiovascular effects. However, although it has less efficacy and may increase the risk of methaemoglobinaemia (abnormally elevated methemoglobin - a form of haemoglobin - in the blood).

- Research indicates that even small amounts of adrenaline can have cardiovascular effects. The American Dental Association recommends a maximum allowable dose of 0.2 mg adrenaline for healthy adults (equivalent to 10 cartridges of 2% Lignocaine with adrenaline 1:100,000 or 5 cartridges of 1:50,000).

- Patients at cardiovascular risk should receive no more than 0.04 mg adrenaline (equivalent to 2 cartridges of 2%

Lignocaine with adrenaline 1:100,000 or 1 cartridge of 1:50,000).

- It is important to maintain a gentle and caring approach when administering local anaesthetic, even with the use of midazolam to provide sedation and amnesia. Any sudden painful stimulus can cause a calm and sedated patient to become agitated.
- Distraction can help reinforce amnesia with midazolam and should be used when appropriate.

9. **Commence dental treatment:**

- Once good local anaesthesia has been achieved, dental treatment can commence, assisted by mouth props, suction, and nurse assistance as necessary.
- It is important to monitor all variables throughout the procedure, including blood pressure, pulse rate, oxygen saturation, respiratory rate, CO_2 levels, drug dosages, administration time, and oxygen level supplied.
- Record these variables at regular intervals, which may vary depending on the stability of the patient, and take them at 5-, 10- or 15-minute intervals. Set the monitor to do this as required.
- Document a final set of readings at the conclusion of treatment.

- Make sure to record the total amount of midazolam used and the amount discarded.
- Discard any unused midazolam from the syringe into a paper towel and dispose of it in the waste bin.

CONTRAINDICATIONS TO IV SEDATION

1. Inadequate training and experience of the administrator and support staff.
2. Inadequate equipment and facilities, including emergency equipment and recovery facilities.
3. Absence of suitable veins.
4. Failure to arrange a suitable escort following completion of treatment.
5. Failure to obtain informed consent from the patient (or the parents of a juvenile).
6. Uncooperative patients who refuse to obey pre- and post-operative instructions regarding food, driving, alcohol, etc.
7. Medical contraindications include poor-risk patients requiring hospitalisation, and doubtful medical history.
8. If in doubt, always consult the patient's doctor and never treat the patient.
9. Watch for illegal drug consumption and recent alcohol consumption.
10. Pregnancy, especially during the first trimester, is also a contraindication.

Relative Contraindications

1. Young children with small veins and poor cooperation.
2. Poor cooperation during induction.
3. Cases where local anaesthesia may not be effective, such as acute abscess.
4. Patients with a history of difficult behaviour under sedation may require general anaesthesia for effective management. Generally, this only applies to a small percentage of patients, mainly children.

MONITORING

Mandatory monitoring requires you to record the patient's vital signs:

- Pulse rate,
- Blood pressure,
- Oxygen saturation,
- Respiratory rate, and
- CO_2 levels.

Careful attention to the airway and respiration, and to clinical signs such as eye movements and chest rise and fall is essential.

Mechanical monitoring is particularly important when deeper sedation is used.

When using midazolam, it is important to only administer enough to achieve minimal or moderate sedation, allowing the patient to remain conscious and responsive to the operator. However, continuous monitoring of the patient's physiological parameters is still necessary to ensure their safety.

Pulse oximetry provides a delayed indication of hypoxaemia of around 1½ minutes and is not enough to detect apnoea. In contrast, capnography can detect apnoea within a few seconds, allowing early intervention before any harm occurs.

Capnography provides much earlier detection of respiratory depression and by intervening immediately when the capnograph shows apnoea occurring, it is unlikely that the pulse oximeter will alarm during the procedure.

The principles for monitoring sedated patients are outlined in the DCNZ practice standard for sedation.

A trained observer must **continuously** monitor the patient from the time of drug administration until recovery.

While the dentist is performing the procedure, some monitoring tasks should be delegated to a trained assistant who should also keep a monitoring record.

Sedated patients are at risk of obstructing or losing their airway, so surgery design and work practice should ensure that they are **never left unattended**. A patient with an obstructed airway can become hypoxic and suffer severe harm within minutes.

Blood pressure recordings before and during treatment are crucial for detecting undiagnosed hypertension and other conditions, and may make it easier to diagnose inadvertent intravascular adrenaline injection, angina, etc.

The patient's airway, respiratory movement and level of consciousness must be closely monitored. This is necessary to detect any signs of paradoxical movements indicating upper airway

obstruction, or prolonged expiration that is often seen in acute asthma.

For midazolam sedation, the patient should be able to hear and respond to the operator throughout the procedure. This allows for instructions to be given, such as taking deep breaths if necessary.

PULSE OXIMETRY

All practitioners who use intravenous sedation must be familiar with pulse oximetry.

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

Hypoxic brain damage can occur due to cyanosis, changes in blood pressure, heart rate, or cardiac arrhythmias. Therefore, instruments that measure oxygenation are essential.

Traditional monitors that measure pulse, blood pressure and ECG, can quickly detect circulatory alterations, but dangerous changes in oxygenation are not evident until the patient is seriously affected.

Risk factors that may contribute to hypoxaemia include poor lung or heart function, or sleep apnoea.

History

- The spectrophotometer was invented in 1860. This device measures the absorption of different substances at different wavelengths.
- It was discovered that saturated and unsaturated haemoglobin (Hb) transmitted light differently. Oxyhaemoglobin (HBO_2) is more transparent to red light than reduced Hb.
- The first oximeters were used in aviation research during World War II.

- By 1950, pulse oximeters were available, but they had a limitation: they could not differentiate between arterial and venous blood.
- This changed in 1950 when the Japanese developed the concept of two-wavelength pulse oximeters, which solved this problem.
- Today, some oximeters can measure many wavelengths, allowing them to measure all four forms of haemoglobin and determine fractional saturation.

Haemoglobin is a protein molecule that transports oxygen to tissues. Haemoglobin and oxygen can bind and unbind easily, allowing haemoglobin to pick up and release oxygen as needed to meet metabolic demands.

The pulse oximeter measures the ratio of oxygen bound to Hb (HbO_2) to the amount of Hb available for binding (functional haemoglobin).

Problems and Limitations

- The pulse oximeter assumes that only arterial blood pulsates.
- The three major sources of artefacts that 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 significantly interfere with oximeter function. This can be minimised by covering the sensors with an opaque

shield. Radiofrequency interference from diathermy can also introduce artefacts.

- **Perfusion:** Reduced finger perfusion due to hypotension, hypothermia, or vasoconstrictor drugs will reduce the accuracy of the readings. Extremes in vasodilation result in signal loss. Monitoring of gravely ill patients is not reliable.
- **Patient motion:** Can cause a high AC to DC signal ratio, which can interfere with pulse oximetry readings. Manufacturers have tried different approaches to overcome this problem, such as increasing the signal-averaging time. This involves averaging measurements taken over a longer period of time to reduce the effect of intermittent artefacts, but this results in a slower response time to sudden changes in oxygen saturation (SaO_2).
- **Coughing:** Can produce increased venous pressure waves that exceed arterial pulsation, leading to transient artefacts.
- **Altered haemoglobin:** Carboxy Hb (HbCO), which is haemoglobin bound to carbon monoxide, and methemoglobin (MetHb) can affect oximeter accuracy. HbCO and MetHb both raise the SpO_2 above actual SaO_2.
  - **SaO_2 vs SpO_2:** Oxygen saturation can be assessed by measuring SaO_2 or SpO_2. SaO_2 is the oxygen saturation of arterial blood, while SpO_2 is the oxygen saturation as detected by the pulse oximeter.
- The level of MetHb in normal people is usually less than 1%, but it can be increased by certain medications.

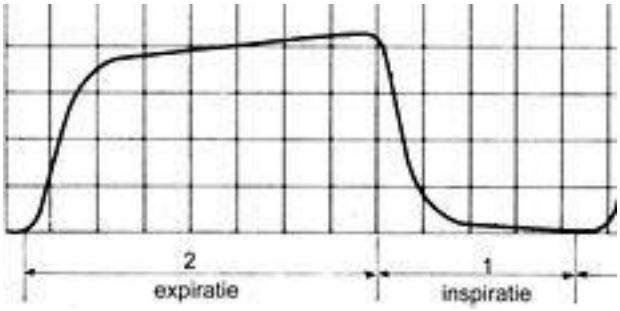
- The level of HbCO in non-smokers is typically less than 2%.
- **Heavy smokers** may have 10% HbCO. For example, the oximeter may show SpO_2 as 97%, even though the actual SaO_2 is only 90%.
- **Dyes:** Medical dyes (like methylene blue or indocyanine green) and some dark nail polishes (especially blue or dark false nails) can cause the displayed SpO_2 value to read lower than actual SaO_2. To fix this, turn the pulse oximeter sideways to avoid the nail.
- **Bilirubin** does not affect or interfere with the accuracy of oximeters.
- **Anaemia:** Anaemic patients may have inadequate amounts of Hb to meet their metabolic needs, but the pulse oximeter can still detect sufficient changes in saturation values. Thus, the patient may have a SaO_2 reading of up to 100%.

For dental sedation, the pulse oximeter sensor should be placed on a finger well clear of the operative site. Any finger can be used, but usually the index or ring finger is preferred.

If the sensor is placed on the same side as the blood pressure cuff, interference may occur when the automated cuff inflates to take readings. Once the blood pressure reading is complete, the pulse oximeter readings will return to normal. Therefore, placing the sensor on the other side is preferred.

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.)

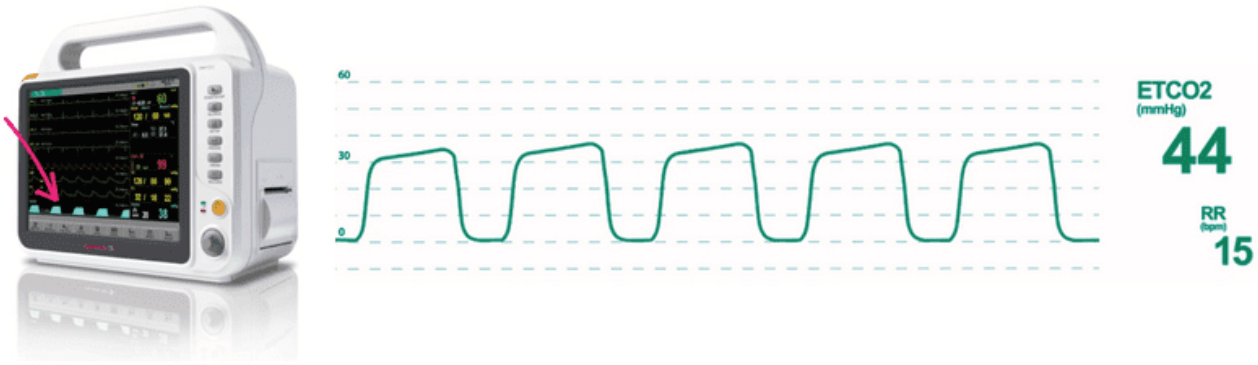


Typical capnogram. Expiration phase on the left, inspiration on the right.

Capnography refers to the use of a capnograph to measure the levels of carbon dioxide (CO_2) in a patient's exhaled breath over time. It is commonly used during anaesthesia and sedation.

A **capnograph** is a medical device that measures the concentration and partial pressure of carbon dioxide in exhaled air.

The results are presented as a graph of expiratory CO_2 measured in millimetres of mercury (mmHg) plotted against time. This graph is known as a **capnogram**.



In capnography, "concentration" and "partial pressure" refer to two different ways of expressing the amount of carbon dioxide in respiratory gases.

- **Concentration** is the amount of carbon dioxide in the gas expressed as a percentage of the total volume of the gas. The typical carbon dioxide concentration in an exhaled breath is about 4-5% by volume.
- **Partial pressure** is the pressure exerted by the carbon dioxide molecules in the gas, expressed in millimetres of mercury (mmHg). It is proportional to the concentration of CO_2, but also depends on the temperature and the total pressure of the gas mixture. Generally, under normal physiologic conditions, the value of the partial pressure of carbon dioxide (PCO_2) ranges between 35 to 45 mmHg.

Both concentration and partial pressure of carbon dioxide can be measured and displayed as a graph over time. Partial pressure is often used as a more precise indicator of the amount of CO_2 in the blood since it accounts for factors like ventilation and blood flow.

Capnographs directly monitor the concentration and partial pressure of inhaled and exhaled CO_2, and indirectly monitor the CO_2 concentration in arterial blood.

Healthy individuals typically have a very small difference between arterial blood and expired gas CO_2 concentrations. However, in individuals with lung disease or certain congenital heart conditions

(known as cyanotic lesions), the difference between arterial blood and expired gas CO_2 partial pressures can increase and even exceed 7mmHg.

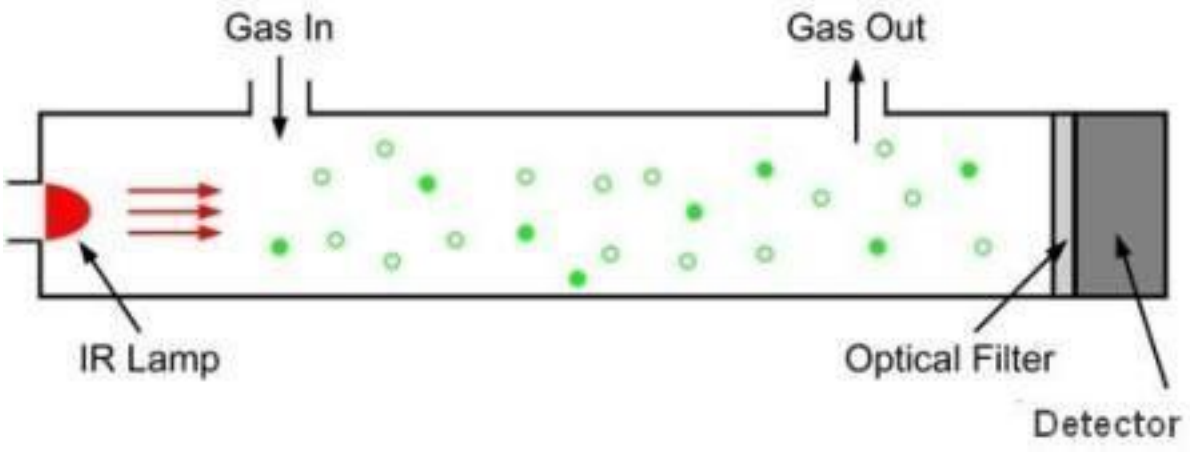
Capnograph vs Capnometer

- Capnographs continuously analyse and record the CO_2 concentration in respiratory gas and provide the measurement data in a timeline graph known as the capnography waveform. This waveform gives experienced practitioners detailed insights into the patient's condition and the effectiveness of their breathing over specific timelines.
- Earlier versions were considered a capnometer. Capnometers measure the CO_2 in respiratory gas without a continuous written record or waveform (i.e. analysis alone).

How does a capnograph work?

Capnographs work on the principle that CO_2 absorbs infrared radiation. A beam of infrared light is passed across the gas sample before hitting a sensor.

If CO_2 is present in the gas, it will cause a reduction in the amount of light reaching the sensor/detector. This change in light intensity will modify 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.

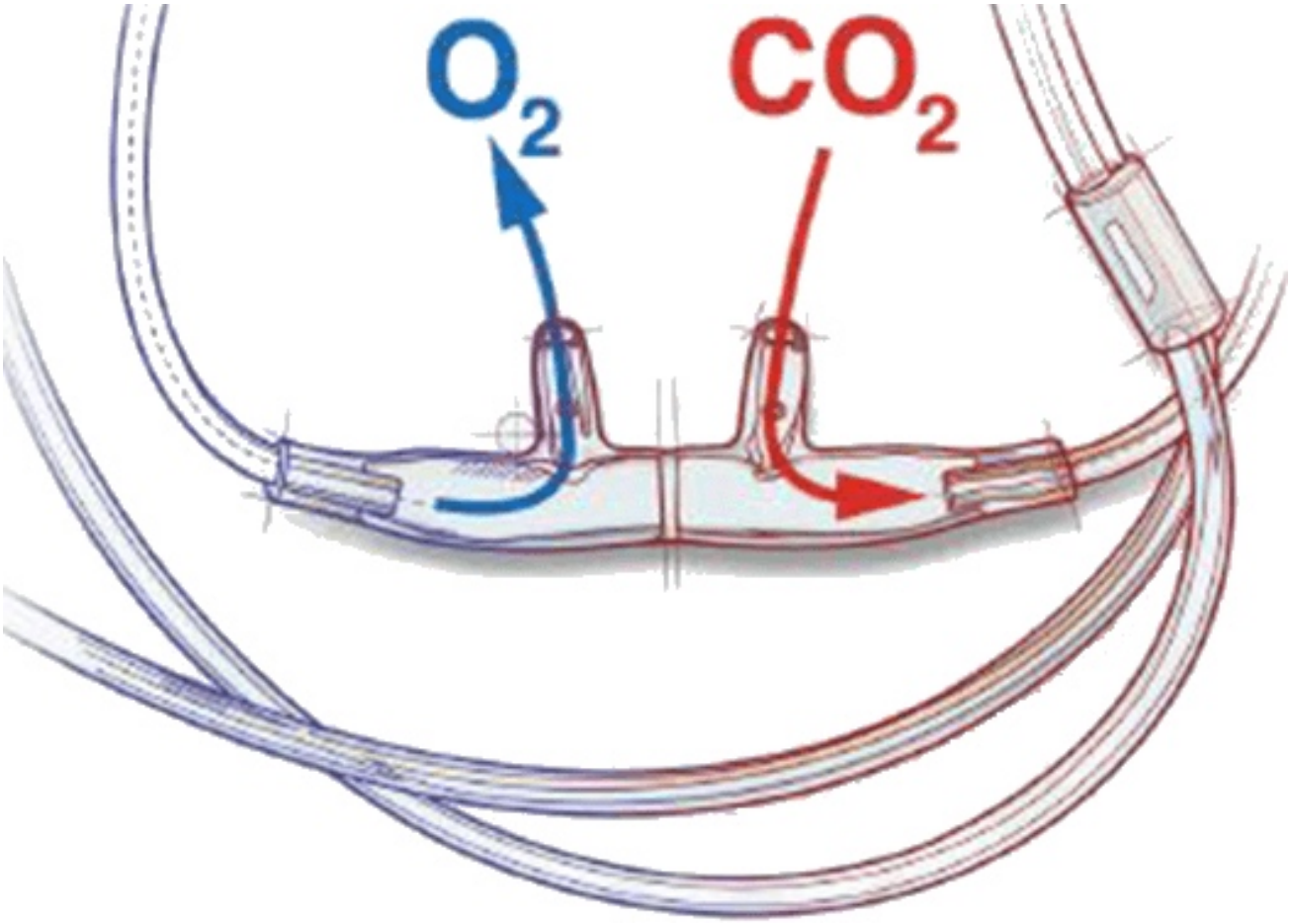
The shape of the capnogram curve can be affected by obstructive conditions such as bronchitis, emphysema, and asthma, which affect the mixing of gases within the lung.

However, conditions such as pulmonary embolism and congenital heart disease, which affect perfusion of the lung, do not alter the shape of the capnogram, but have a great impact on the relationship between expired CO_2 and arterial blood CO_2.

Capnography can also measure carbon dioxide production, which is an indicator of metabolism. Increased CO_2 production is observed during fever and shivering, while decreased production is seen during anaesthesia and hypothermia.

Use in sedation

When administering sedation during dental procedures, CO_2 levels are usually monitored using nasal prongs that also deliver oxygen.



Capnography directly measures the elimination of CO_2 by the lungs, and indirectly measures the production of CO_2 by the tissues and its transport to the lungs.

Compared to relying solely on clinical judgment, capnography is more effective at detecting early signs of respiratory distress, such as hypoventilation and apnoea, which may be caused by oversedation or airway obstruction. This allows prompt intervention to prevent harm to the patient.

During sedation procedures, capnography provides more useful information about ventilation frequency and regularity than pulse oximetry.

Capnography is a fast and reliable way to identify life-threatening conditions such as tracheal tube malposition, undetected ventilatory failure, circulatory failure, and faulty breathing circuits.

Capnography helps to prevent potentially irreversible patient injury by providing an early warning sign of slowed or stopped breathing. Therefore, it has become an essential tool for ensuring the safety of sedated patients.

Remember:

1. Almost all serious complications in sedation are due to respiratory depression.
2. Early detection is key to preventing and addressing respiratory distress.

BLOOD PRESSURE MONITORING

Blood Pressure (BP) is the amount of force that blood exerts against the walls of an artery. It is measured in millimetres of mercury (mm Hg). BP is affected by various factors, such as the force of the heart's contraction, the volume of blood pumped per heart beat, and the ease of blood flow through the vessels.

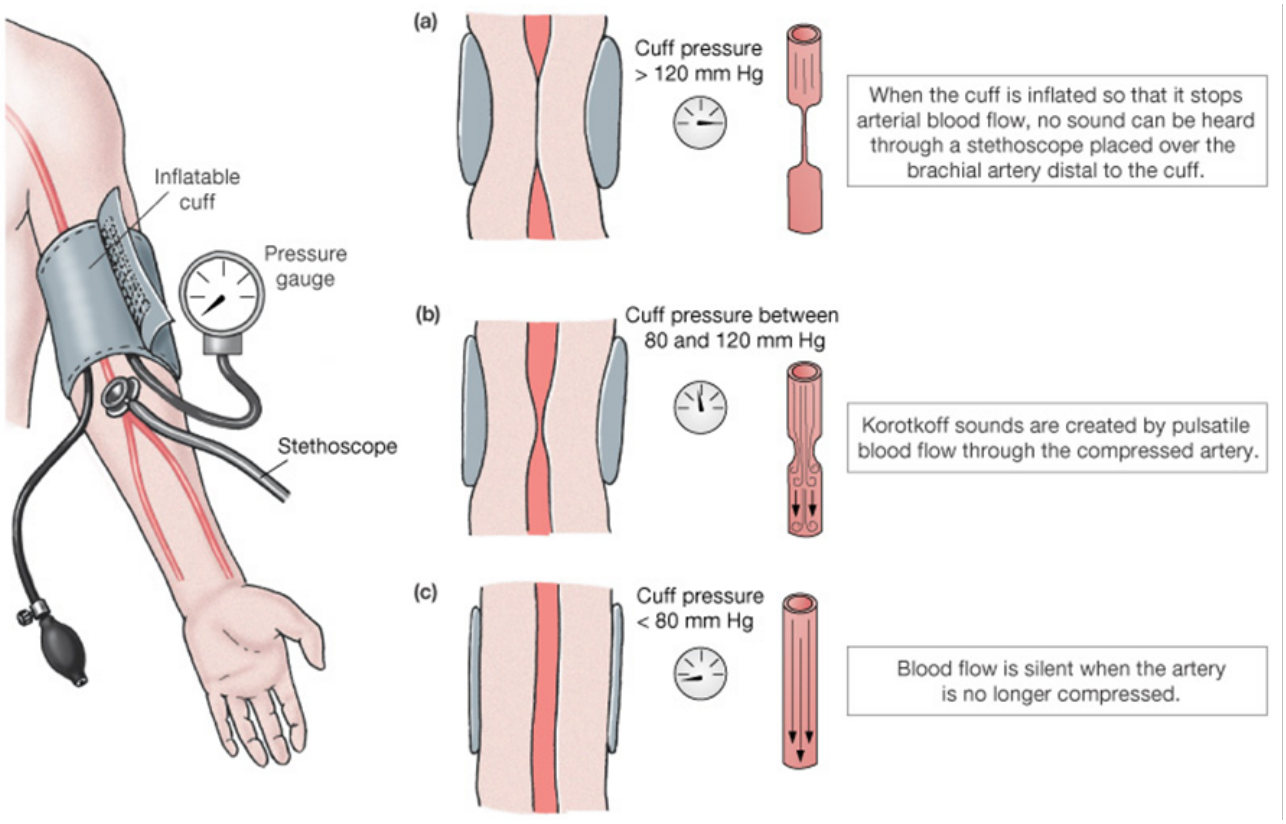
There are two components of blood pressure, and both are measured. The **systolic level**, which is the highest pressure, occurs when the heart contracts. The **diastolic level**, which is the lowest pressure, occurs when the heart is relaxed. These two levels are written as systolic/diastolic.

A normal BP range is 100/60 to 150/90 mmHg. However, BP can be influenced not only by age, sex, and blood volume but also by factors such as emotions, pain, exercise, body size, and medication.

History of Blood Pressure Monitoring

- In the 18th Century, Rev. Hales first measured BP by inserting a tube into the artery of a horse and observed the blood level rising 9 feet.
- In the 19th Century, Italian Physiologist Sicipone Rocci invented the **sphygmomanometer** (sfig-mo-ma-Nom'eter) for measuring BP, but it could only measure systolic pressure.
- Russian Surgeon Dr. Korotkoff developed a technique for measuring both systolic and diastolic pressures. He discovered

that with the aid of a stethoscope, if a cuff was pumped up and slowly released, it was possible to hear different sounds as the pressure dropped, which indicated different phases of the heartbeat.



The 5 phases of Korotkoff sounds

Phase 1: Sharp tapping. This is the first sound heard as cuff pressure is released. The first appearance of faint, repetitive, sharp tapping sounds that gradually increase in intensity for at least two consecutive beats provides the **systolic pressure reading**.

Phase 2: Soft swishing/whooshing. A brief period follows during which the sounds soften and acquire a swishing/whooshing quality when the blood starts flowing through blood vessels as the cuff is deflated.

Phase 3: Crisp, intense thumping (softer than phase 1). Intense thumping sounds that are softer than phase 1 are heard as the blood flows through the artery, but the cuff pressure is still inflated to occlude flow during diastole.

Phase 4: Blowing. A softer and muffled blowing sound that fades as the cuff pressure is released. The change from the thump of phase 3 to the muffled sound of phase 4 is known as the first diastolic reading.

Phase 5: Silence. When the cuff pressure is released enough to allow normal blood flow, all sounds disappear completely, and there is silence. This is known as the second diastolic reading and is recorded as the **diastolic pressure reading**.

The second and third Korotkoff sounds have no known clinical significance.

Equipment

There are three types of sphygmomanometer:

- Mercury sphygmomanometer
- Aneroid sphygmomanometer
- Electronic sphygmomanometer

Blood pressure is usually taken on the arm, measuring the brachial artery. Both mercury and aneroid sphygmomanometers require clinical skills and the use of a stethoscope to listen for sounds (known as auscultatory technique). Palpation methods are also possible.

Components

- All sphygmomanometers have a **pressure cuff**.
- Mercury and aneroid sphygmomanometers also have a **measuring scale**.
- Electronic sphygmomanometers are automatic and therefore do not require a measuring scale. Once the cuff is applied in the correct position, the pulse and blood pressure are automatically calculated and displayed by the unit.

Semi-automated sphygmomanometers operate based on different principles, including:

- Detection of Korotkoff sounds, or
- Detection of arterial blood flow through ultrasound, or
- Phase-shift method, which measures pressure changes between two segments of a double cuff, or
- Oscillographic detection of arterial pulsation using a double arm cuff, or
- Tonometry, which utilises a force-sensitive transducer over a superficial artery. This is the most common method used today.

Blood Pressure Measurement Technique

Please note: This section is included for interest only. For dental sedation you will be using automated non-invasive blood pressure devices.

Here is a general technique for measuring blood pressure:

- Use well-maintained equipment.
- Have the patient's bare arm exposed and relaxed, ensuring no tight clothing.
- Place the cuff around the arm and make sure the arm is supported and level with the 4th intercostal space at the sternum (heart level).
- Palpate the brachial artery and place the stethoscope over it, held firmly in place. Inflate the cuff rapidly.
- When the pulse sound disappears, increase the pressure by 30mmHg above this level. The point at which the pulse first disappears is around the systolic pressure level (a guide as to when to expect the first sound).
- Slowly let the pressure out of the cuff at a rate of 2-3mmHg per heartbeat/second. The appearance of sound indicates systolic pressure, and the disappearance of sound indicates diastolic pressure. Record these values to the nearest 2mmHg.

During the initial examination, measure blood pressure in both arms. If there is a difference of more than 10mmHg, use the arm with the highest pressure.

When recording blood pressure, it is helpful to record additional information such as the arm used, the position of the patient, and any unusual circumstances such as anxiety.

When measuring blood pressure in children, use a smaller bladder size (cuff) and a paediatric diaphragm on the stethoscope. Alternative methods may be required, such as measuring the leg.

Elderly patients may have medical conditions that influence blood pressure, such as hypertension (high systolic pressure). Blood pressure regulating mechanisms in the elderly are less effective, and some may be susceptible to a fall in blood pressure when standing (postural hypertension).

Many elderly patients are on antihypertensive drugs. In these cases the leg can be used, with the popliteal fossa/artery (the area behind the knee) being a good location to measure blood pressure.

Sources of Errors

Faulty equipment:

- The mercury meniscus is not at the zero-calibration level when the device is at zero pressure.
- Dirty scale.
- Air vent blockage slows mercury flow.
- Cuff bladder not applied correctly or not adequately maintained (perished rubber).
- Cracked or non-airtight tubing.

Observer:

- Inadequate clinical skills.
- Observer biases: rounding off figures, e.g. rounding down for a fit person and rounding up for an obese person.
- Digit preference: preference for terminal digits 0 & 5 even though 5mmHg does not appear on many scales.
- Incorrect viewing angle. The scale should be viewed from a distance of no more than 1 metre, and vertically level with the meniscus, to see the real meniscus.
- Rushed method. If the pressure is released too quickly, this leads to underestimation of the systolic pressure and overestimation of the diastolic pressure. It takes 5 minutes to measure BP correctly.

Patient:

- Circadian rhythm. BP varies greatly within a day, typically rising a few hours before waking up in the morning, peaking in the mid-morning to early afternoon, and then declining in the late afternoon and evening. This daily pattern of blood pressure variation is believed to be influenced by a complex interplay of various factors, including the body's production of hormones like cortisol and adrenaline, the sleep-wake cycle, physical activity, and even diet.
- Obesity (actual). Actual obesity can lead to an increase in blood pressure due to factors such as increased blood volume, increased cardiac output, increased peripheral resistance, and activation of the renin-angiotensin-aldosterone system. This increase in blood pressure can also lead to hypertension.
- Obesity (artefact). Artefact obesity can affect blood pressure measurement due to the size of the patient's arm, i.e. the arm circumference may be too large to fit the cuff properly, leading to inaccurate readings. This is particularly true for manual sphygmomanometers, where the width of the cuff needs to match the arm circumference. An oversized cuff may give falsely low blood pressure readings, while an undersized cuff may give falsely high readings. Using an appropriately sized cuff is essential for accurate BP measurement in obese patients.
- Arrhythmia. During arrhythmia, the heart may not pump blood efficiently, resulting in changes in blood flow and stroke

volume. The heart may not fill properly, leading to a decrease in preload and stroke volume. Alternatively, the heart may not contract effectively, leading to a decrease in contractility and stroke volume. Measuring BP during arrhythmia can therefore be challenging, as the normal pattern of blood flow and pressure may be disrupted. The accuracy of BP measurements may be affected by the timing of the measurement, the type of arrhythmia, and the individual patient's response to the arrhythmia. In some cases, multiple measurements or more advanced monitoring methods may be needed to accurately assess blood pressure in such patients.

- Postural position. Normal people have no difference in BP, provided the arm is supported at heart level. Vertical displacement of the arm increases hydrostatic pressure as the arm is dropped and leads to an error as large as 10mmHg. If the arm is unsupported, then muscles are working and this increases diastolic pressure by up to 10%.
- Other factors. Things such as exercise, meals, smoking, alcohol, temperature, bladder distension and pain also influence blood pressure.

Repeated measures of BP (inflations) can cause venous congestion. Deflate the cuff between readings and leave 15 seconds between successive measures.

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

CHAPTER 7: PRACTICE ORGANISATION

The administration of IV sedation requires a collaborative effort from a team, with each member having clearly defined roles, duties, and responsibilities. These should be documented as a protocol and shared with all team members.

It is essential to have written protocols for both routine care and emergency situations. Departing from standard procedures increases the likelihood of problems and difficulties. If you choose to deviate from your routines, make sure you understand the reason for doing so and acknowledge the risk-benefit ratio.

You will need to create a specific protocol for your individual situation and practice. It is your responsibility to ensure that your staff is adequately trained or arrange for their training. Also, ensure that your premises, facilities, and equipment comply with current guidelines.

Facilities And Equipment

DCNZ and NZDA policy documents on sedation for dental procedures define the necessary facilities and equipment, including:

1. A facility that is appropriately sized, staffed, and equipped to handle cardio-pulmonary emergencies.
2. A chair that can easily be tilted to a horizontal position.
3. Sufficient uncluttered floor space to allow for CPR if necessary.
4. Equipment suitable for measuring the patient's blood pressure.
5. Adequate suction and room lighting.
6. A supply of oxygen and suitable devices for administering oxygen to a spontaneously breathing patient.
7. A means of inflating the lungs with oxygen, such as a range of laryngeal airways and a self-inflating bag suitable for artificial ventilation.
8. Appropriate drugs for CPR and a range of intravenous equipment.
9. Capnography and pulse oximetry to measure carbon dioxide and oxygen saturation levels.

Specialised Equipment

- Tourniquet
- 10 ml syringes
- Filtered drawing up needles (18 gauge)
- Steriwipe disinfectant patches
- Tegaderm

- Intravenous cannulae
- Mouth props
- Mouth packing devices, rubber dam, Isolight
- Tray for intravenous equipment
- Sedation drugs and their reversal drugs
- Nasal prongs for supplemental oxygen and capnography

Routine Care

Receptionist's Responsibilities

- Provide the patient with a medical history form.
- Provide the patient with information forms on sedation and pre-operative instructions.
- Check that the appropriate forms have been signed and that the patient understands the requirements for fasting, transportation, and the need for an escort.
- Upon the patient's arrival for sedation, verify compliance with the pre-operative instructions.

Dental Assistant Responsibilities

Before the First Patient of the Day:

- Check oxygen cylinders/oxygen concentrator, suction and lighting to ensure they are working properly.

During Patient Care:

- Prepare all equipment and materials needed by the dentist for the procedure.
- Escort the patient to the surgery, and verify and record compliance with pre-operative instructions.
- Remove partial dentures, if necessary.
- Provide the patient with protective glasses and a blanket for comfort.
- Place nasal prongs and check connections for supplemental oxygen and capnography.
- Monitor the patient's blood pressure, pulse, oxygen saturation, carbon dioxide level, and respiratory rate throughout the procedure, recording these parameters at specified intervals.
- Assist the dentist with clinical tasks, but especially responsible for maintaining the airway with suction.
- Assist the patient to the recovery area. **Do not leave any sedated patient unattended** until the clinician responsible for the sedation has discharged the patient into the care of an escort.

After Patient Care:

- Discharge the patient only after authorisation from the dentist and provide the escort with written and verbal post-operative instructions.

- Safely dispose of drugs and contaminated intravenous equipment.
- Routinely check emergency equipment and drugs monthly, taking note of the expiry dates of drugs.

Dentist's Responsibilities

- The dentist is responsible for overseeing the entire team and their activities.
- It is the dentist's responsibility to ensure that the correct drug, at the correct dosage, is administered appropriately.
- Ensure the dental staff receive appropriate training.
- Ensure that a written protocol of responsibilities and duties for all team members is developed and provided to each person.
- The dentist is also responsible for providing written pre-operative instructions, post-operative instructions, medical history, consent forms, and monitoring/record of sedation forms to the patient.
- Some of these tasks may be delegated to other responsible staff members.

CHAPTER 8: EMERGENCIES IN THE DENTAL SURGERY

Emergencies are, by definition, unexpected and extremely stressful. Please download and review the following two documents from NZDA and DCNZ:

NZDA

- Code of Practice: Medical emergencies in dental practice »

  https://www.nzda.org.nz/assets/files/Standards__Guidelines/Codes_of_Practice/CoP_Medical_emergencies_in_dental_practice.pdf

DCNZ

- Medical Emergencies Practice Standard »

  https://www.dcnz.org.nz/assets/Uploads/Practice-standards/Medical-Emergencies-practice-standard.pdf

These two documents outline the necessary requirements for emergencies in the dental surgery.

It is important to be familiar with and comply with these documents for both **general dentistry and sedation**.

Please review them before the course.

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.

Intravenous sedation is a straightforward and safe technique that can enhance the dental experience for many patients.

By following guidelines, using common sense, and heeding the advice of experienced professionals, you can implement IV sedation with confidence.

Good luck!

APPENDIX

Intravenous Sedation Information Sheets

On the following pages you will find formatted information sheets for patients that you can use in your dental practice:

1. What is Intravenous Sedation?
2. Pre-Operative Instructions
3. Post-Operative Instructions

WHAT IS INTRAVENOUS SEDATION?

Intravenous sedation is a modern technique used in dentistry to help patients overcome their fear of dental treatment and make it a comfortable experience.

This technique is suitable for most patients, but if you have any health conditions or are taking medication, be sure to inform your dentist so that the sedation can be adjusted to suit your needs.

A small amount of sedative will be injected into a vein in your arm. The injection is painless, and the sedative takes effect quickly. You'll become very relaxed and drowsy, allowing the dentist to perform the necessary treatment with comfort for you. Local anaesthetic injections will also be given once the sedative has taken effect.

You will not be fully unconsciousness ("put to sleep"), which requires general anaesthetic with an anaesthetist in an operating theatre. You may, however, feel like you've been asleep for most of the treatment due to the amnesia (no memory) effect of the sedation drugs, even though you will be conscious and relaxed the whole time.

To prepare for your appointment, you must not eat any food for 6 hours, but you can drink water up until 2 hours before.

Your dentist will provide you with detailed pre- and post-operative instructions.

After the treatment, you will rest for a short while before your escort takes you home.

Although you may feel like you're back to normal, it takes your body several hours to eliminate all the sedative. That's why you must have someone to take you home and stay with you for 2 to 3 hours.

Intravenous sedation is a pleasant way to undergo dental treatment. Your dentist will be happy to explain the procedure in more detail.

PRE-OPERATIVE INSTRUCTIONS

- Before your appointment, please let your dentist know if you are taking any drugs, particularly sleeping pills, tranquillisers, or cortisone preparations.
- Do not eat any food for 6 hours prior to your appointment time, but drinking water is allowed up until 2 hours before.
- Please avoid wearing tight clothing, especially sleeves that cannot be easily drawn up past the elbow.
- Dress warmly for your comfort.
- It's best to remove your contact lenses before the appointment.
- Visit the restroom before entering the surgery.

Additional Instructions

- Before accepting a sedation appointment, you must agree:
  - Not to drive a vehicle or operate machinery on the same day, after the sedation.
  - Not to undertake responsible or important business matters.
  - Not to drink any alcohol, until the next day.
- You must be escorted home and looked after by another person.

Your appointment time has been reserved for you.

Please carefully review the instructions overleaf before the day of your sedation appointment.

If you are unable to attend, please give sufficient advance notice to reschedule.

If you develop any COVID or upper respiratory track symptoms, please call to discuss or reschedule.

POST-OPERATIVE INSTRUCTIONS

Give these instructions to the person taking the patient home.

Instructions to Patient

- You must NOT drive a vehicle or operate machinery today.
- You must NOT drink alcohol today.
- You should NOT undertake any responsible/important business matters today.

Instructions to Person accompanying Patient

This person has had dental treatment carried out with intravenous sedation. The sedative drugs may produce drowsiness for several hours, particularly after long appointments. You are therefore requested to:

1. Take the patient home and stay with them for 2-3 hours, especially if their appointment was a long one.
2. Make sure they comply with the "Instructions to Patient" above.