The Relaxed Patient

A Manual of Sedative Techniques

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Sharon, Thank You

Preface

This text is intended as an introduction to sedative management for practitioners in all fields of dentistry and presupposes no prior knowledge of these sedative modalities. Recognizing that more is required than a listing of suitable drugs, we have included clinically tested background on patient evaluation, treatment planning, monitoring of the patient's momentary status, modes of action of the various drugs and regimens, armamentarium and treatment of complications as well as basic cardiovascular and respiratory phenomena. (The inherent differences between sedation and general anesthesia are highlighted by a chapter on general anesthesia included to provide background and perspective rather than for implementation. General anesthetic management can be learned only by experience with a large number of cases in a supervised clinical situation.)

Following the progression outlined in the text, it should be possible for the general practitioner of dentistry as well as the specialist to start conservatively utilizing less critical techniques and to advance to more complex management using more potent methods and drugs for more profound effect. Because of the safety and simplicity of nitrous oxide analgesia, this modality serves as an ideal introduction to treatment of patients with altered consciousness, and has been so used for many years in our practice. Each technique presented is one in which the patient remains awake. Anesthetically speaking, this means that certain protective reflexes remain operative and with careful usage, safety should not be compromised. Gradually, using other agents and other routes of administration, the dentist can graduate to the use of a balanced medication utilizing combinations of methods and drugs, securing benefits here and avoiding disadvantages there.

Sedation techniques should be taught in the dental colleges and teaching hospitals where supervised clinical experience can be gained. Short courses in various sedative techniques are available in some areas independently of the dental schools. Ideally, in order to derive the greatest benefit from this text, it should be used to supplement such formal training. It is expected, however, that it would be used most often as the nucleus of a self-study program and, sensibly followed, should serve quite adequately in this regard.

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The drugs here presented for use are relatively safe ones when used as directed (and allowing for some leeway), and were carefully selected as being unlikely to produce harmful effects. But because any drug can cause harm in certain situations or with certain patients, warnings against such possibilities are included. All reliable publications describing the use of drugs contain such listings of potential hazards.

Acknowledgements

Preparation of a text requires more than the desire to do so. It is imperative that the author possess a knowledge of the field and for this I am indebted to Joseph Turbin, M.D. for his tutelage, encouragement and assistance.

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In her various roles as sounding board, photographic model and typist, my wife, Sharon, has left her imprint indelibly upon this text. A task as large as preparation of a text requires many long hours of estrangement from one's family and these lost moments can never be recaptured. Only such an exceptional mate could have subordinated her understandable frustration with such a situation for the eventual benefits.

As an amateur author I could not have created this product without the guidance and cooperation of Mr. J. Stuart Freeman, Jr., Editor, Dental Books, J. B. Lippincott Company.

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1 Introduction

It is a relatively simple matter to numb any area of the mouth, and in most instances the effect can be reliably determined by objective signs; nevertheless, the dentist is often confronted by patients whom he cannot treat with comfort. Although actual somatic pain may have been eliminated, its psychological component has not.

Because of fear, emotional instability or various handicapping conditions, many patients are inadvertently denied optimal dental care. The treatment they do receive often falls short of meeting total dental needs and long-range maintenance of the oral apparatus in a condition of health.

Some persons are fortunate to have access to specialists who treat them with general anesthesia. However, the cost is high, such practitioners are unavailable in most locales, and the impersonal relationship inherent in infrequent treatment by a specialist militates against motivating these patients to accept dental care more willingly in the future.

The ideal situation would certainly be for treatment to be rendered by a dentist who has rapport with the patient on a continuing basis. Even if altered consciousness is mandatory, the patient should be familiar with the practitioner who would be better able to train and motivate him to accept dental treatment performed in a more normal fashion. A condition could be created that would predispose him to the best care and to periodic recall maintenance, the doctor's retention of the patient and attainment of optimal dental health.

Successful management of such patients requires an altered sensorium—sedation if you will—to lower their level of reflex irritability, thus allaying apprehension, promoting calm and facilitating subjective acceptance of the absence of pain. The number of persons for whom such methods fail is exceedingly small.

Consider also the patient who, through determination and perseverance, can allow himself to submit to dental treatment but whose limits are narrow. There are many procedures we perform today that require prolonged sessions whether they are part of an overall rehabilitative regimen or a single long appointment for some definitive procedure. These persons need help if they are to submit to this type of management. Unaided, they succumb to their nervousness, making care difficult and fatiguing for the dentist.

With techniques of modern medicine, there is a host of modalities available to dentists to further this aim. To begin a discussion of this nature, some definitions are helpful.

Simply stated, a *sedative* is a drug or agent that reduces functional activity of the central nervous system, producing a state of lessened excitation, agitation or reflex irritability, and depressed metabolic rate. A *hypnotic* produces loss of consciousness or a state of sleep. An *analgesic* relieves pain or lessens perception of pain sensation without loss of consciousness.

An *amnesic* obliterates memory. An *anesthetic* produces localized or total absence of sensation.

In clinical application, however, further detail and distinction are essential to comprehend these concepts of altered sensorium. Perhaps the key to understanding the ramifications in our dental situation can best be described as a function of the patient's awareness of his surroundings.

A well-sedated patient is somnolent in the absence of external stimuli but will react if they are applied. The so-called "protective reflexes" of the pharynx and larynx are operative; response to pain is present, although altered.

However, if the patient is maintained in a general anesthetic state, he displays no response to external stimuli and exhibits total lack of protective reflexes. Conduct of a case is vastly different, because vital functions, in the absence of sustaining mechanisms, must be assiduously monitored and managed.

Sedative medication requires concomitant use of analgesic or local anesthetic drugs. General anesthesia does not.

With sedation, amnesia is variable. It is usually present to some degree, but the amount varies from patient to patient and from one session to another for a given pharmacological regimen. Naturally, different agents or combinations of agents produce different results. With general anesthesia, amnesia is total.

In our dental situation where the operator usually functions both as surgeon and as anesthesiologist, sedative management is patently more expeditious. The simpler maintenance requires less attention and distracts less from the concentration necessitated by the operative procedures.

2 Respiratory Anatomy and Physiology

Respiration provides oxygen for bodily function and eliminates the carbon dioxide waste production from the organism. The sequence includes the transport of atmospheric air to the lungs where oxygen diffuses into the bloodstream and is carried to the tissues for use in metabolic processes. Carbon dioxide, the main waste product of cellular metabolism, travels the reverse route from the tissues to the lungs for diffusion out of the blood and transportation back to the ambient atmosphere.

NORMAL FUNCTION

External Respiration

Conducting Portion

The airways of external respiration are, in effect, a connecting system of tubes that serve as a communication between the atmosphere and the lung alveoli, the site of actual gaseous exchange. The upper air passages include the nasal and oral cavities, the pharynx, the larynx, a cartilaginous structure which is divided by the vocal cords and leads to the trachea, a large, cartilage-reinforced tube passing down the neck anterior to the esophagus. The trachea divides into right and left major bronchi which, in turn, divide into many smaller bronchioles with fine lumens and thin walls, each leading to a single atrium with a cluster of alveolar sacs, the actual site of gaseous diffusion into the lung capillaries.

No diffusion occurs in this system of passages from the nose (or mouth) to the bronchioles and for this reason it is termed the *anatomic dead space*. The contained gases do no more than move in and out with each inspiratory or expiratory effort. Since the capacity of the system, approximating 1 ml. per pound of body weight or about 150 ml. for the average adult, does not change and the depth of respiration may vary; the resultant efficiency of inspiratory effort is subject to some variation. The first 150 ml. of air reaching the lung alveolar sacs comprises not fresh air but, rather, the contents of the passages with atmospheric air being drawn in only to the extent that the tidal volume exceeds the volume of the dead space.

Ventilating Portion

The diffusion portion of external respiration functions in the exchange of gases between the alveoli and the blood of the pulmonary circulation. The anatomy of this, the actual functional unit of the lung, consists of numerous lung alveoli, each supplied by a single bronchiole and separated from its investing capillary network by two thin endothelial membranes (the wall of the alveolus and the wall of each capillary).

Oxygen diffuses from the alveolus into the blood and carbon dioxide diffuses in the opposite direction according to pressure gradients determined by the partial pressures of the gases on each side of the membranes, the solu-

bility of the gases in blood and the physical properties of the membrane. Carbon dioxide is approximately 25 times more soluble in blood than oxygen and may diffuse more rapidly even in the presence of a lower pressure gradient. Disease processes in the lungs may alter the diffusion potential of the membrane. Pulmonary edema, for example, may lessen the amount of alveolar space available for gases and slow diffusion.

The term *physiological dead space* expands the concept of anatomic dead space to include all the air contained in alveoli that is not actually in contact with the epithelial walls. *Anesthetic dead space* goes one step further to include the dead space of the anesthetic equipment.

Internal Respiration

Transportation Portion

The transportation portion of the internal respiration carries the gases in the blood to the tissue cells. The pulmonary capillaries are oxygenated in the lungs and then introduce this oxygenated blood into the systemic vascular system. Carbon dioxide produced by cellular metabolism is returned by venous blood to the right atrium, then to the right ventricle and back to the lungs to be reconverted to arterial blood.

Most of the oxygen is transported by the blood combined chemically with hemoglobin in the form of oxyhemoglobin with only a small amount being carried in physical solution in the plasma.

Hemoglobin, a blood protein in the red cells, will combine with oxygen under certain conditions and release it under others according to the reversible reaction:

$$O_2 + Hb \rightleftharpoons HbO_2$$

The capability for oxygen saturation of hemoglobin varies as the oxygen partial pressure in the blood. The high oxygen concentration in the perialveolar capillaries promotes the tendency to combination with hemoglobin rather than release, as contrasted with the situation in the tissues where the relatively low oxygen tension favors its liberation.

Carbon dioxide is carried in the blood mainly as sodium bicarbonate in the plasma, but lesser amounts are transported either combined with hemoglobin in the form of carbaminohemoglobin, as carbonic acid in physical solution in plasma, or as potassium bicarbonate in the red blood cells. Normally, as much as 85 percent of the carbon dioxide is carried in the plasma.

Since venous pulmonary blood is characterized by an oxygen tension lower than that in alveolar air and a carbon dioxide tension which is higher, the pressure gradients favor diffusion of oxygen into the blood and of carbon dioxide out of the blood and into the alveoli for elimination. Since carbon dioxide in solution in the blood increases its acidity ($CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3-$) through presence of hydrogen ions (carbonic acid) its diffusion out of the blood lowers acidity. The loss of carbon dioxide and lowered acidity enhances the affinity of hemoglobin for oxygen at the alveolar level.

Intracellular Respiration

Intracellular respiration involves the exchange of gases between the blood and the tissue cells. The culmination of the entire respiratory process is the delivery of oxygen to the cells and the removal of carbon dioxide. As elsewhere, the rate of interchange is regulated by the partial pressure difference of oxygen and carbon dioxide in the cells and blood. As tissue cells are more

active, they deplete the local oxygen supply and accumulate carbon dioxide in the area. The reduced cellular oxygen tension causes more efficient withdrawal from the blood, and the higher cellular carbon dioxide tension favors more rapid diffusion into the blood.

INSPIRATORY AND EXPIRATORY DYNAMICS

Air enters and leaves the respiratory conduits as a result of variations in the internal pressure of the lungs caused by transient rhythmic alterations in the dimensions of the thoracic cavity. The chest cavity enlarges through outward and upward movement of the ribs on their cartilaginous connections and by the downward movement of the diaphragm. If these structures are mechanically unimpeded, free of disease and the airway is patent, changes in thoracic size will result in pressure variations within the lungs that will be equalized by inflow or outflow of air. In the normal resting phase between inspiration and expiration the lungs remain suspended within the thorax in a slightly stretched elastic condition, since some intrapleural, negative pressure always remains. If the chest wall is ruptured (pneumothorax) this slight residual negative pressure is lost, the lungs collapse because of inherent elastic recoil, and no air can be drawn in.

During a normal inspiratory sequence the thorax expands from downward movement of the diaphragm and an outward and upward movement of the rib cage which results from contraction of the intercostal muscles. Since the extrapulmonary areas of the thorax have no communication with the outside, no air can enter and thoracic cage expansion is accompanied by increased negative pressure. The lungs, surrounded by negative pressure, expand, thereby transiently increasing the intrapulmonic negative pressure until air rushes in (assuming a patent airway) to equalize internal and external pressures.

During expiration the sequence is reversed. Relaxation of the intercostal muscles and diaphragm causes the thoracic dimensions to become smaller. Increased intrathoracic pressure forces gas out of the lungs until, again, internal and external pressures are equalized.

Inspiration results from an active muscular effort. Expiration is a normally passive process brought about by the inherent elastic recoil of the thoracic structures but, in forced expiration, it can result from active contraction of the intercostal muscles.

Total lung capacity, in the average young adult male, approximates 6000 ml. but only about 450 to 500 ml., the *tidal volume,* moves with each usual inspiration or expiration. There are reserves so that more air can be brought into the lungs after the usual inspiratory effort, more can be breathed out after normal expiration. Even after maximal forced expiratory effort, about 1200 ml. of residual air remains.

In the normal conscious adult the respiratory rate falls in the range of 14 to 18 per minute while for children in the 60-pound range it is from about 22 to 26 per minute. Tidal volume in the same child would be about half the adult value of 450 to 500 ml.

REGULATION

The stimulus initiating respiration comes from the inspiratory portion of the respiratory center in the medulla. The expiratory portion of the same center acts in concert with the pneumotaxic center in the pons to inhibit the inspiratory stimulus, imparting a rhythmic pattern to respiration. Efferent impulses elicited by various reflex phenomena also cause cessation of inspiration and allow expiration to occur passively.

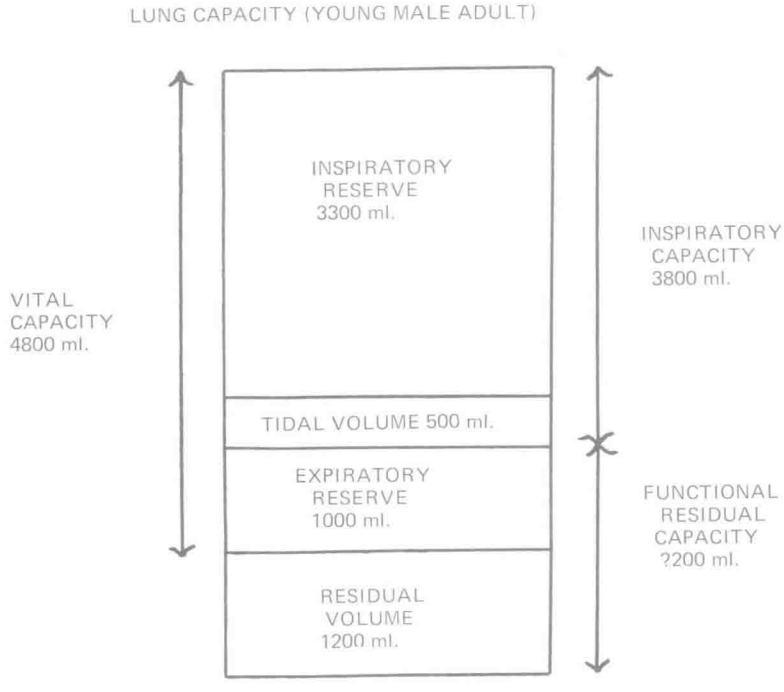

FIG. 1. Diagram showing the relative portions of the total lung capacity which are involved with each ventilatory exchange and those portions available as inspiratory and expiratory reserves.

The respiratory center acts as a clearinghouse, sorting out the various bits of information made available to it by afferent nerve impulses and directing appropriate responses through efferent nerve stimuli to effector organs. Basically, the phrenic nerve stimulates diaphragmatic activity and the intercostal nerves innervate the intercostal muscles and also some contributing abdominal muscles.

When the lungs are distended, stretch receptors in the alveoli are stimulated and the resulting afferent impulses to the brain, travelling along the vagus nerves, act to inhibit further inspiratory effort and allow expiration to occur. This reflex, the *Hering-Breuer reflex,* complements the activity of the pneumotaxic center but is independent of it.

When oxygen is depleted in the blood, chemoreceptors located in the carotid and aortic bodies send respiratory stimulating impulses (primarily related to rate) to the respiratory center by way of the vagus and carotid sinus nerves. The *carotid and aortic body reflexes* (chemoreflexes) are activated by oxygen lack rather than by carbon dioxide accumulation (unlike the respiratory center) and may account for as much as 30 percent of normal respiratory urge. Oxygen lack, if it is severe enough, can render the inspiratory center inactive and, in this event, the chemoreflexes may remain as the sole stimulation of respiratory

activity. Some of the barbiturates increase the sensitivity of this mechanism but nitrous oxide has no appreciable altering effect.

The *carotid and aortic sinus reflexes* (pressor-reflexes), activated by pressure on these structures, inhibit respiratory activity. The afferent nerves are the glossopharyngeal and vagus and efferent impulses travel along the vagi. Other neural mechanisms, arising anywhere in the body, may reflexly excite the respiratory center. These *peripheral or proprioceptive reflexes* may cause such effects as quickening of the rate due to powerful emotional experiences, increase depth of respiration during rigorous exercise, or cause a sudden gasp after perception of an unexpected severe pain.

The chemical makeup of the blood can affect the various neural determinants of respiratory function. Lowered carbon dioxide levels, by increasing the alkalinity of blood, activates chemoreceptors to augment both rate and amplitude of breathing.

The respiratory center, contrary to what might be expected, is only slightly affected by oxygen tension in the blood. Increased oxygen tension does not significantly slow respiration even though the need for further ventilation is lessened. Oxygen lack, particularly if the deficit is great, depresses the respiratory center. Carbon dioxide accumulation lowers the threshold of the respiratory center to reflex stimuli.

Voluntary Control of respiration, suspending or modifying the usual pattern, is possible, and without this potential one would be unable to talk, whistle, hum, blow, breathe deeply or hold his breath. Limits are imposed on this capability by chemical considerations but practice and training widen the range of voluntary control even as disease entities may narrow it. During consciously controlled respiration, impulses arriving at the respiratory center emanate from the motor cortex.

ANESTHETIC CONSIDERATIONS

Various premedicant agents and general anesthetics have the potential to depress the respiratory center and to alter the respiratory pattern. The patient must be carefully monitored for assurance that oxygenation is adequate.

Many of the classic signs of anesthesia relate to modifications of respiratory activity resulting from depression of the respiratory center by anesthetic agents. This is in addition to possible depression of the respiratory center caused by any hypoxia that may exist. Being less sensitive to carbon dioxide-induced stimulation, the depressed respiratory center transmits fewer and feebler impulses to the diaphragm and intercostal muscles with the possible result of decreased pulmonary ventilation. Insufficient ventilation can further result in hypoxemia and oxygen deprivation at the cellular level. If ventilation is seriously reduced (unlikely during sedative management) it should be assisted or augmented by manual compression of the reservoir bag.

Induction with inhalation agents depends on adequate alveolar concentration of the gas or vapor. If respiration is depressed, as may happen with overzealous premedication or sedation, unassisted breathing may be too shallow for uptake of sufficient agent to provide the desired hypnotic effect.

Certain physical factors affecting ventilation may also prevent adequate inhalation of agent. The trunk must be in such a position so as not to impede movement of the rib cage necessary for thoracic enlargement. The dead space of the anesthetic equipment should not be so great that, when added to existing physiological dead space, a lessened