

CHAPTER 27

Complications

There are a number of complications that can arise when the IV route of drug administration is used. Most, fortunately, are relatively benign and easily managed. Some, however, may be quite a bit more significant and can lead to morbidity and death.

The complications associated with IV drug administration are divided into four groups: those associated with venipuncture, localized complications related to drug administration, general drug-related problems, and specific drug complications. These are outlined below:

- Venipuncture complications
 - Nonrunning IV infusion
 - Venospasm
 - Hematoma
 - Infiltration
 - Local venous complications
 - Air embolism
 - Overhydration
- Local complications
 - Extravascular drug administration
 - Intraarterial drug administration
 - Local venous complications
- General drug-related complications
 - Nausea and vomiting
 - Localized allergy
 - Respiratory depression
 - Emergence delirium
 - Laryngospasm
- Specific drug complications
 - Diazepam
 - Local venous complications
 - Emergence delirium
 - Recurrence of amnesia
 - Oversedation
 - Pentobarbital
 - Oversedation
 - Respiratory depression

- Promethazine
 - Oversedation
 - Extrapyramidal reactions
- Narcotics
 - Nausea and vomiting
 - Respiratory depression
 - Rigid chest
- Scopolamine
 - Emergence delirium

VENIPUNCTURE COMPLICATIONS

Nonrunning IV infusion

One of the most commonly occurring complications of venipuncture and IV sedation is the nonrunning or very slowly running IV infusion. Once a venipuncture has been successfully completed the tourniquet is removed and the IV drip opened. During injection of drugs the drip rate should be quite rapid; at other times the rate should be slow. Some of the causes of a nonrunning or slowly running IV infusion are listed below:

IV infusion bag too close to the heart level. Gravity forces the solution from the IV bag down to the patient. The greater the difference in height between the bag and the patient's heart, the more rapid is the flow of solution. A simple experiment will demonstrate this:

The IV bag is held high above the patient's body and the rate of flow is checked. With the rate adjusting knob opened fully the drip should be quite rapid. The bag is gradually lowered toward the level of the patient's heart. The rate of flow of the drip will decrease until when held at the patient's heart level the flow ceases entirely. As the bag is lowered below the level of the patient's heart, blood returns into the tubing.

This situation might arise when the dental chair is placed low to the floor at the start of a proce-

ture and is elevated at a later time. Lowering the chair or elevating the bag of IV infusion solution will correct the situation.

Bevel of needle against wall of vein. It was recommended that the bevel of the needle be facing upward during venipuncture so as to make entry into the skin as atraumatic as possible. Following entry into the skin, the needle was laid down and advanced into the vein. At this point the tourniquet was removed and the infusion started. If the drip rate was rapid until the needle was taped into position and then slowed considerably, it is quite possible that in taping the needle into position the bevel was lifted and lies against the wall of the vein. This would prevent or retard the flow of fluid from the IV drip into the patient.

To determine if this is the cause of the slow or nonrunning drip, the needle is carefully uptaped and the wings gently lifted. This will lower the bevel off the wall of the vein. If the drip rate increases when this is done, then the protective cap from the scalp vein needle (Fig. 27-1) or a 2 × 2 inch gauze square is carefully placed under the wings of the needle and the needle is retaped.

Tourniquet left on arm. This is an embarrass-

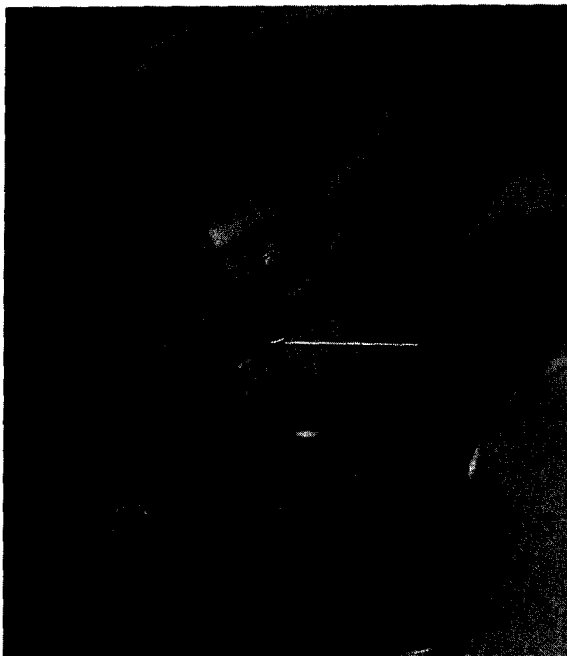


Fig. 27-1. Placing the protective sheath of the needle (or gauze) beneath the wings of the needle (arrow) will increase the rate of IV infusion if the bevel is pressing against the vein wall.

ing, but not uncommon cause of a nonrunning IV drip following successful venipuncture. The tourniquet may have simply been covered by a sleeve that has inched down. Following the return of blood into the IV tubing the doctor or assistant opens up the control knob to start the IV drip. It is noted that the drip is not flowing and that the blood does not leave the IV tubing. It is usually noted that more blood appears to be entering the IV tubing (as it is forced from the vein into the tubing). Once excessive blood is noticed in the tubing, simply removing the tourniquet will alleviate the problem.

Infiltration. Following successful entry into the vein the needle has become dislodged during the securing of the needle. The doctor and assistant, unaware of this development, open the rate knob, but little or no solution flows. If no solution is flowing, the first three causes of nonrunning IVs should be checked for. If the drip rate is extremely slow and cannot be increased, look at the site where the needle tip is located under the patient's skin. If the needle has left the vein and fluid is still flowing, a small colorless swelling will develop at this site. This is termed an *infiltration*.

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In all cases in which an IV drip that was previously running well has either slowed or stopped running entirely, the needle *should not* be removed from the vein until it has definitely been determined that the needle is no longer within the vein. The following procedure should be followed to determine the cause of the slow or nonrunning IV drip:

1. Open drip rate knob
2. Elevate bottle. Does flow rate increase?
3. Place bottle below heart level. Does blood return?
4. Check IV site. Is tissue swelling? Does tissue at needle site feel cooler than surrounding tissues? (See Infiltration, p. 390.)
5. Elevate wings of needle. Does flow improve?

Venospasm

Venospasm is a protective mechanism in which the vein responds to stimulation from the needle by constricting. As the needle approaches the vein, the vein appears to disappear or collapse. Venospasm is occasionally accompanied by a burning sensation in the immediate area. This burning sensation resolves without treatment. Venospasm may occur before or after entry of the

needle into the vein, securing of the needle, and starting of the IV drip.

Prevention. There is no way to prevent venospasm.

Recognition. Venospasm is identified by the disappearance of a vein while attempting venipuncture. A burning sensation may or may not occur.

Management. *The needle should not be removed from the skin, for the vein has not yet been entered or damaged. The needle is pulled back slightly (1 to 2 mm) and heat is applied to the vein site in an attempt to dilate the vessel. When the vein reappears the venipuncture may be reattempted.*

The sensation of burning will occur with several other complications and one noncomplication. The IV administration of diazepam will occasionally produce a sensation of warmth or burning; however, this sensation will travel *up* the patient's arm as the drug passes through the veins. Intraarterial injection of a drug produces a burning sensation or pain travelling *down* the arm toward the fingers. Extravascular injection of a drug produces a burning sensation at the site of injection that *remains* at the site. The injection of meperidine occasionally produces the release of histamine and a burning or itching sensation along the path of the vein. Venospasm occurs more frequently in apprehensive patients, presumably caused by their higher levels of circulating catecholamines.

Hematoma

Infiltration is the most common complication of venipuncture. It is the extravasation of blood into the interstitial space around the blood vessel. The presence of a volume of blood in this space leads to localized swelling and discoloration.

When a venipuncture is properly carried out, the needle itself acts as an obturator, sealing the hole within the vein made by the needle. In some patients, particularly older patients in whom vein walls are less elastic, leakage of blood around the needle may occur during the IV procedure although the needle is still within the vein.

Hematoma may occur at 2 times during the procedure. First, it may develop during the attempted venipuncture if the vessel is damaged. This is not always preventable. The second cause of hematoma is usually preventable. In this situation the IV procedure has been completed and the needle removed from the patient. Improper

pressure or inadequate time of pressure on the venipuncture site can lead to a hematoma.

Recognition. A hematoma is a painless, bluish discoloration of the skin at the site of the needle. It develops during venipuncture attempt or at the conclusion of the IV procedure.

Prevention. It is not always possible to prevent a hematoma during venipuncture attempt, although careful adherence to recommended techniques will minimize its occurrence. Hematoma developing after the procedure can be prevented by the application of firm pressure for a minimum of 5 to 6 minutes. The technique illustrated in Fig. 27-2 of placing gauze over the venipuncture site in the antecubital fossa and having the patient flex the arm *does not* provide adequate pressure to prevent hematoma.

Management. When hematoma develops during attempted venipuncture the swelling will increase rapidly because the tourniquet is still on the patient's arm. Immediate management consists of the following:

1. Remove the tourniquet to decrease blood flow.
2. Remove the needle.



Fig. 27-2. Placing gauze over the injection site and bending the elbow does *not* provide adequate pressure and frequently results in hematoma formation.

3. Apply firm pressure with sterile gauze for 5 to 6 minutes.
4. If the site is tender, ice may be applied in the first few postoperative hours. Ice acts as a vasoconstrictor and as an analgesic.

When hematoma develops following removal of the IV needle, the immediate management consists solely of direct pressure with gauze and ice. Subsequent management of either form of hematoma can best be described as tincture of time. It will require approximately 7 to 10 days for the subcutaneous blood to be resorbed by the body. There is nothing that can be done to speed this process up. Should the patient complain of discomfort or soreness (more likely if the hematoma is located in a joint), he can be advised to use moist heat on the area for 20 minutes every hour. Heat should *not* be used within the first 4 hours after the hematoma occurs for it acts as a vasodilator and might produce further bleeding.

Infiltration

Infiltration is similar to a hematoma in that a fluid is being deposited into the tissues surrounding a blood vessel. In fact, a hematoma is actually the infiltration of blood outside of a blood vessel. Extravascular injection of a drug is an infiltration of drug outside of a blood vessel. *Infiltration* is defined as a painless, colorless swelling that develops at the site of the needle tip when the IV infusion is started.

In this situation we are discussing the deposition of the IV infusion solution into the tissues surrounding the blood vessel. The infiltration discussed here differs from the hematoma in that the swelling that develops will not occur until the IV drip is turned on, whereas the hematoma occurs as soon as the vein is damaged.

In the continuous IV infusion technique that I recommend, if infiltration does occur, it will only be a solution such as 5% dextrose and water or normal saline, which will not produce any tissue irritation or damage. In contrast, in those IV procedures in which a drug is injected directly into a blood vessel, it is much more likely that the drug will produce tissue damage if deposited outside the blood vessel.

Prevention. Infiltration can be prevented by careful venipuncture technique and by not starting the IV drip or injecting drugs until it is certain that the needle tip lies within the lumen of the vein. Checking for this is quite easy. The flash bulb on the IV tubing may be squeezed and blood

will return into the tubing when the pressure is released, or the IV bag may be held below the level of the patient's heart.

Cause. Movement of the needle either while it is being secured or through movement of the patient's arm during the sedation procedure may cause infiltration. The most common causes of a needle becoming dislodged are (1) attempting to thread a needle too far into the vein and (2) carelessness during taping of the needle.

Recognition. Infiltration is a painless, colorless swelling occurring around the tip of the needle when the IV drip is started. The tissue around the needle tip will be elevated and the skin at this site will feel cooler than skin at a distance away from this site. This is because the infusate is at room temperature (72° F), not body temperature.

Management. The IV infusion must be immediately stopped and the needle removed from that site. A 2 × 2 inch gauze is placed at the site and pressure applied for 5 to 6 minutes. The pressure will stop any bleeding as well as spread out any solution within the tissue. This solution will be resorbed into the cardiovascular system.

Localized venous complications

Localized venous complications can develop following IV sedation procedures. There are many factors responsible for their development, and indeed there are a number of different forms that venous complications take. Trauma to the vein wall produced by the needle is a possible cause of this problem. In the dental outpatient environment, the most likely cause of venous complications is chemical irritation produced by the drug being administered, primarily diazepam. Localized venous complications will be discussed further under local complications (p. 394).

Air embolism

Air embolism is a possible, though extremely unlikely, complication of IV sedation. It is best avoided by using a technique that is free of air: eliminating air bubbles from the syringe of medication(s) and from the IV tubing prior to the start of the procedure and periodically observing the IV infusion bag to prevent its becoming emptied.

In the highly likely event that one or more small bubbles of air enter into the venous circulation, they will be absorbed quite rapidly by the blood and produce no problem. Not all air bubbles can be removed from the IV tubing or syringe and it is quite probable that they will enter

into the venous circulation of the patient. The patient, sighting the air bubble slowly moving down the IV tubing toward his arm, may become quite agitated, thinking that as little as one bubble of air is lethal. Fortunately, this is not so. A rule of thumb in the hospital environment is that a patient can tolerate up to 1 ml/kg of body weight of air in the peripheral venous circulation without adverse effect.

The average IV administration set will hold approximately 13 ml of air. When it is considered that 15 drops of solution (or air) equals 1 ml, then it can be seen that the chances of introducing large volumes of air into the patient's circulatory system are quite low. A 50 kg (110 lb) patient can tolerate 50 ml of air. This is equivalent to 750 drops of air from an adult IV administration set.

When managing small children air embolism is a more significant problem, as their bodies cannot tolerate large volumes of air. A 30 lb child (13 kg) is at greater risk of this complication than the larger patient.

Should air embolism occur, management is based on the attempt to prevent this air from entering into the cerebral and pulmonary circulation. This is accomplished by positioning the patient in the dental chair lying on the left side (prevents entry into pulmonary circulation) and in a head down position (prevents entry into cerebral circulation).

Overhydration

Overhydration of the patient is not a very common problem during IV sedative procedures in the dental office. The two most likely candidates for overhydration, however, are the patient with congestive heart failure and the child. Signs of overhydration include pulmonary edema, respiratory difficulty, and an increase in the heart rate and blood pressure. These are also the signs and symptoms occasionally noted in a patient with congestive heart failure.

A rule of thumb for replacement of fluid in a patient is that the initial dose of IV solution administered is equal to 1.5 times the number of hours a patient has gone without food times the patient's weight in kilograms. This is the volume of fluid in milliliters required to replace the fluid deficit created by the patient's taking nothing by mouth. If a patient had taken nothing by mouth for 6 hours prior to coming to the office, the initial volume of milliliters of IV solution administered would be 9 times the patient's body weight

in kilograms. The maintenance dose of IV solution is 3 ml/kg. The problem of underhydration is not significant in the usual outpatient environment.

When administering IV medications to pediatric patients, it is recommended that a pediatric infusion set be employed. This set, which permits 60 drops per milliliter instead of the usual 15, allows for a more careful administration of fluids to the younger, smaller patient or to the adult with serious congestive heart failure. In most instances, these two classes of patients are not good candidates for elective IV sedation procedures.

LOCAL COMPLICATIONS

Extravascular drug administration

When a drug is injected into the subcutaneous tissues instead of a blood vessel, three problems may develop:

1. Pain
2. Delayed absorption of the drug
3. Tissue damage

The pain occurs at the site of the needle tip under the skin and tends to remain localized. This distinguishes the extravascular injection from intraarterial and IV injections where a burning sensation will radiate either peripherally or centrally. The patient will complain of discomfort as the drug is being injected.

A potentially greater problem is delayed absorption of the drug into the cardiovascular system, especially if larger volumes have been deposited into the tissues.

Another potential problem is damage to the tissues into which the drug has been deposited. Some of the drugs used intravenously are quite irritating to the tissues. This is especially true for diazepam and pentobarbital. The initial reaction in the tissues is for arteriolar and capillary constriction, which decreases the blood supply to the region. If the vascular constriction is prolonged or if the chemical is irritating enough, necrosis and sloughing of tissue may occur.

Causes. There are two causes of extravascular drug administration. The first is the needle coming out of the vein. This usually leads to immediate formation of a hematoma that is quickly recognized. No drug is usually injected at this time. The second cause is the needle entering the vein and then being pushed through the other side as the doctor attempts to advance it further into the vein. Blood will have returned into the tubing as the needle entered the vein originally, thereby

giving the impression that the needle is still in the vein. However, with removal of the tourniquet it is unlikely that the blood will leave the tubing, as is normally the case, because the lumen of the needle is no longer in the vein but in subcutaneous tissue. On occasion the blood will reenter the patient and the IV infusion will be running even though the needle tip has left the vein. This will occur if the IV solution is quite high over the patient's heart or if the patient's skin and underlying soft tissues are not "firm."

The continuous IV drip technique really minimizes the possibility of extravascular injection of a drug because an infiltration of infusate produces a swelling immediately. Secondly, prior to administration of a drug it is recommended that the patency of the vein be rechecked by squeezing the flash bulb or holding the IV bag below the level of the patient's heart. Despite these precautions, a subtle movement of a patient's wrist or elbow just after this check but just before drug administration can produce this complication. The administration of a 0.2 ml test dose of a drug is a means of catching this occurrence before a larger, potentially more damaging bolus of drug has been deposited.

Recognition. As the chemical, especially diazepam or pentobarbital, is injected extravascularly, the patient will complain of a more intense pain that occurs at the site of the needle tip and does not migrate up or down the arm. In addition, as a volume of solution is injected into the patient, the tissue at the site of the needle tip will swell if the solution is being forced into the subcutaneous tissues. If the chemical is irritating, the skin overlying the swollen tissue will become ischemic as the blood vessels in the tissue constrict in response to the irritation. A second possible reaction is for the tissues to become erythematous as a result of inflammation.

Management. The two major problems to be managed are the possible delayed absorption of the drug and its effect on the patient and the possible damage to the tissues at the site of injection. Management initially consists of removing the needle and applying pressure to the site of the injection to (1) stop the bleeding and (2) disperse the solution deposited under the skin. If less than 1 or 2 ml of drug has been deposited extravascularly, these steps are all that are required for effective management.

In the highly unlikely situation that larger volumes of solution have been deposited extravascu-

larly and the overlying tissue is swollen and ischemic, two problems must be addressed: (1) tissue damage and (2) delayed sedation. Pressure alone may not prove adequate to spread the solution and drug management may be required. The drug of choice is 1% procaine, a local anesthetic with profound vasodilating properties. Several milliliters of procaine can be infiltrated into the affected tissues, using a single puncture point and a "fan-type" injection. A second benefit of the procaine will be to eliminate any discomfort.

Management of the possible delayed onset of sedation produced by the slow absorption of the chemical must be managed with basic life support procedures: maintaining the airway, ventilation, and circulation.

In the conscious patient receiving medication via a continuous IV infusion, it is highly unlikely that a large volume of drug will ever be administered extravascularly. If the doctor is titrating drugs at the recommended rate of 1 ml per minute, it will become obvious well within a minute that the needle is not in the vein. Injection of the drug must be immediately stopped and the IV rechecked.

Intraarterial injection

The most significant of the localized complications of IV sedation is the intraarterial injection of a drug. There are numerous reasons why this serious complication occurs only infrequently. However, when it does occur, immediate and vigorous therapy is indicated in order to prevent tissue damage, gangrene, and amputation. The drugs that are injected into an artery produce irritation of the wall of the artery as the drug is carried peripherally. As the artery narrows, the chemical is in contact with the wall of the artery to a greater degree. The immediate response of the artery to this insult is to constrict. Arterial constriction, especially if it occurs in one of the larger arteries of the upper limb, as is quite likely in this situation, will compromise the circulation to all, or a large portion, of the tissue distal to the injection site.

Prevention. Prevention is the most important feature in this discussion of intraarterial injections. Fortunately, it is rather difficult to accidentally enter into an artery and even more difficult to accidentally administer a drug into the artery. There are many signs and symptoms to alert the doctor and assistant to the fact that the needle is not within a vein.

INTRAARTERIAL INJECTION

Prevention

Palpate vessel prior to placing tourniquet
Avoid anatomically risky areas (e.g., median antecubital fossa)

Recognition

Intense pain during venipuncture attempt
Bright cherry red blood
Pulsating flow of blood in IV tubing when tourniquet is removed
Intense pain radiating down arm toward fingers as drug is injected
Loss of color in limb
Loss of warmth in limb
Loss or weakening of radial pulse in limb

1. The vessel is palpated prior to venipuncture. Arteries conduct a pulse that can be palpated *before* the tourniquet is placed on the patient's arm. Once the tourniquet is in place the artery may not pulsate and the doctor may mistake this vessel for a vein. This is especially likely to happen on the medial aspect of the antecubital fossa where the brachial artery is somewhat superficial.

2. As the needle approaches the artery the vessel will go into spasm. This is much more vigorous than venospasm and is associated with a more intense burning sensation. For this reason alone it is usually very difficult to accidentally (and on many occasions even purposefully) enter into an artery.

3. If the needle enters the artery, blood will return into the IV tubing. With the tourniquet in place the return of blood will be similar to that seen in venipuncture; however, the *color* of the blood is different. Arterial blood is a brighter cherry red, whereas venous blood is a darker maroon color.

4. On removal of the tourniquet a significant difference is noted. Venous blood leaves the IV tubing when the tourniquet is released and the IV drip started because of the relatively low venous blood pressure. Arterial blood, on the other hand, with a much higher blood pressure (e.g., 120/80), will remain in the IV tubing and demonstrate a pulsatile flow with every contraction of the heart.

To this point, nothing damaging has been done to the patient or the artery. If the intraarterial puncture is noted at this time, the needle is carefully removed and firm pressure exerted over the

site for at least 10 minutes. If, however, the drug is injected into the artery at this time, problems develop rapidly.

Recognition. There are a number of signs and symptoms of intraarterial injection:

1. The patient will complain of a severe pain that radiates distally from the site of injection of the drug toward the hand and fingers.

2. The radial pulse should be checked for. Absence of pulse indicates that the problem is quite severe and that immediate management is essential. Presence of the radial pulse, even though it may be quite weak, indicates that at least some volume of arterial blood is entering the hand and fingers. A serious problem may still exist, but it is not as acute.

3. The skin color of the affected hand should be compared with that of the opposite hand. Lack of blood flow into the affected limb will produce a loss of normal skin color.

4. Both limbs should be felt to determine temperature. The flow of blood to the hand provides warmth. When blood flow to the limb is compromised that limb will feel cooler than the opposite limb with normal blood flow.

The major cause of injury from intraarterial injection is chemical endarteritis that results in thrombosis and ischemia. Crystals of the drug precipitate as a result of the change in pH, and this leads to further occlusion of vessels. The result of this ranges from small areas of gangrene to the loss of fingers or a limb (Fig. 27-3).

Management. Management of intraarterial injection is best achieved by the following steps:

1. Leave the needle in place. Do not remove the intraarterial needle that has been accidentally placed. It provides an avenue for introduction of the drug used in management of this situation.

2. Administer procaine. Slowly inject 1% procaine, to a volume of between 2 and 10 ml, into the artery. Procaine serves four functions at this time:

- Anesthetic to decrease pain
- Vasodilator
- pH about 5, counterbalance for drugs with alkaline pH (pentobarbital)
- Diluent

Procaine frequently breaks the arterial spasm, which is noted by a return of color and warmth to the limb, as well as a return of a pulse wave equal in strength to that of the opposite limb.

3. Hospitalize the patient. All patients having had accidental intraarterial drug administration



Fig. 27-3. A, Gangrenous fingers secondary to intraarterial drug administration. B, Gangrene of forearm, hand, and fingers.

should be seen in the emergency room of a hospital where a vascular surgeon or anesthesiologist will be consulted. The dentist should accompany the patient to the hospital so that the physicians can be advised of the drug(s) administered intraarterially and the treatment rendered. Additional treatment may also be deemed necessary. Additional treatment may consist of a sympathetic nerve block such as the stellate ganglion or brachial plexus block. Where indicated, general anesthesia or surgical endarterectomy may be needed. Heparinization may be employed, if needed, to prevent further thrombosis. If all modalities of treatment fail to return an effective blood flow to the limb, amputation of gangrenous parts may be required.

The intraarterial injection of a drug is a serious complication that should not occur if the basic concepts recommended in earlier chapters of this section are adhered to. In the unlikely situation that it does occur, management as described above is recommended, followed by accurate record keeping and contacting the doctor's insurance carrier immediately.

Local venous complications

Following a successful IV sedation procedure, the patient is discharged and goes home. The patient may feel fine through the next day only to find 2 days after the procedure that the hand in which the needle and drug were placed is swollen, red, hot, and painful.

The general category of local venous complica-

tions is being used here because of the multiple names given to the situation being discussed.

Phlebitis is the inflammation of a vein.

Thrombophlebitis is a condition in which inflammation of the vein wall has preceded the formation of a thrombus.

Phlebothrombosis is the presence of a clot within a vein, unassociated with inflammation of the wall of the vein.

Gelfman and Driscoll have done several prospective and retrospective studies into the problem of local venous complications. Criteria that they established for identification of these entities were:

Thrombophlebitis—pain, induration, and a delay in onset of these symptoms

Phlebothrombosis—a condition of venous thrombosis without inflammation; occurs much more immediately, and pain is not a prominent feature.

It appears that the primary problem that develops following IV sedation is thrombophlebitis. Clinical features of thrombophlebitis are

Edema

Inflammation

Tenderness

Delayed onset: 24 to 48 hours, may develop after up to a week

Causes of thrombophlebitis. Anything that produces either mechanical or chemical irritation of a vein is capable of inducing thrombophlebitis. Among the factors involved in the development of thrombophlebitis are the following:

pH of the infusion liquid
 Components of the infusate
 pH of the drug(s)
 Duration of the IV infusion
 Mechanical factors:
 Bevel and dullness of needle
 Technique of venipuncture
 Improper fixation of needle
 Size of needle in relation to vein lumen
 Type of needle (metal vs. plastic catheter)
 Presence of infection or disease
 Age and sex of the patient
 Site of venipuncture

Solutions, be they infusions or drugs, that have pH values at either end of the spectrum are associated with a greater incidence of venous complications.

Some of the drugs that are injected intravenously have vehicles, such as propylene glycol and alcohol, that are quite irritating to vein walls. Diazepam is an example of such a drug. It was mentioned earlier in this section that some patients experience pain on the administration of IV diazepam. Gelfman and Driscoll reported that in patients experiencing such discomfort on injection, the incidence of phlebothrombosis, but not thrombophlebitis, was increased.

The duration of the infusion is not as great a concern in outpatient sedation as it is within the hospital, where an IV infusion may be necessary for days at a time. Common practice within hospitals today is for an IV team to change the site of the infusion every other day, thereby minimizing the risk of local venous complications.

Improper technique, use of dull needles (highly unlikely with disposable needles), and improper fixation of the needle are mechanical causes of irritation. A needle that is not well secured will continually produce irritation within the walls of the vein.

Placement of a very large needle within the lumen of a smaller vein will potentially produce greater irritation and an increased risk of thrombophlebitis. As recommended in this section, the 21-gauge needle will not impinge on the walls of any vein within the upper limb.

The site of venipuncture is also a factor. Venipuncture of the femoral or saphenous veins of the leg is associated with a higher incidence of thrombophlebitis and thromboembolism. There are significantly fewer complications with the superficial veins of the arm and the dorsum of the hand. Within the upper limb, there are differences in the incidence of thrombophlebitis. Nordell et al.

(1972) reported 5 cases of thrombophlebitis in 52 patients. The following is a breakdown of the site of venipuncture and incidence of thrombophlebitis:

Site	Number of venipunctures	Number of cases of thrombophlebitis	Percentage of cases of thrombophlebitis
Hand, wrist	26	3	11.5
Forearm	15	2	13.33
Antecubital fossa	11	0	0

Other studies have demonstrated similar statistics. Chambiras (1972) found a twofold increase in incidence of venous complications in the hand when compared to the antecubital fossa.

Prevention. It is not always possible to prevent local venous irritation when one of the factors responsible for its development is mechanical irritation produced by the venipuncture or the needle being used. Fortunately the superficial veins of the upper limb are less likely to produce serious postinjection complications than are the veins of the leg. Prevention is based on

Use of sharp, sterile needles

Atraumatic, sterile venipuncture technique

Securing the needle firmly in position

Injecting IV drugs slowly into a rapid infusion

Dilution of IV drugs whenever possible

When dilution is not possible (diazepam), use of larger veins (antecubital fossa and forearm)

Recognition. The patient will usually be asymptomatic for 1 or more days after the IV procedure. The inflammatory process requires approximately 24 to 48 hours to fully develop and at that time the patient contacts the office complaining of soreness, (possibly) swelling, redness of the area, and (possibly) warmth.

Management. Management of any localized venous complication requires that the patient return to the office for evaluation. The doctor should examine the patient to determine the nature and extent of the situation. All findings are recorded in the patient's chart and the patient is examined regularly until the situation resolves. The key to successful management is patient cooperation and satisfaction.

Management of thrombophlebitis includes:

1. Activity of the limb must be limited through the use, if possible, of a sling.
2. The affected limb must be elevated when possible.
3. Moist heat must be applied for 20 minutes three to four times a day.

4. Should thrombophlebitis occur in a joint (elbow or wrist), immobilization is more difficult but should still be attempted. Constant movement of the affected area leads, in some patients, to discomfort. Management of this is through the administration of antiinflammatory analgesics such as aspirin, every 4 to 6 hours as needed for pain.
5. Anticoagulants and antibiotics are not part of the usual therapy and will not be prescribed unless the situation worsens. At this point, however, the patient will have been referred to a physician (vascular surgeon) for definitive management.

In the usual course of events, the acute phase, involving tenderness, swelling, and discomfort, resolves within a few days, gradually leading to the chronic phase in which the discomfort is gone but the vein remains hard and knotty. This may occur at the site of the venipuncture or anywhere along the path of this vein and its tributaries. The extent of these lumps and bumps will subside over time. Treatment of choice during this phase is tincture of time.

The patient is seen in the office on a less and less frequent basis as the situation resolves. Records are kept of the findings at each visit. In most cases full resolution occurs within 3 to 4 weeks, although cases have been reported in which patients have suffered lingering tenderness for over 3 years (wrist vein).

In the unlikely event that fever or malaise develops, consultation with a vascular surgeon (the person most familiar with management of this complication) is recommended. In any situation in which a patient is being referred for medical (or dental) consultation it is recommended that the referring dentist speak with the physician (discussing management of the case) before sending the patient. Another indication for referral to a physician is patient dissatisfaction. If the patient expresses doubt over the dentist's handling of the problem (after all, this is no longer a dental problem), immediate consultation with a physician is recommended.

Management of phlebothrombosis, which is a small painless nodule located at the site of venipuncture, is essentially the same as that described for thrombophlebitis:

1. Immobilization of the affected limb
2. Moist heat applied to area
3. Tincture of time

Virtually all cases of local venous complications

resolve within a short period of time without residual effects.

GENERAL DRUG-RELATED COMPLICATIONS

In this section we will discuss those systemic reactions brought about through the administration of IV drugs, as opposed to those localized complications previously described.

Nausea and vomiting

The incidence of nausea and vomiting associated with IV sedative procedures is extremely low. However, the potential does exist for some of the medications being administered to produce this problem. Among the drugs recommended for use via the IV route, the narcotics are most likely to induce nausea and vomiting. Promethazine and scopolamine, which possess antiemetic properties, are among those drugs least likely to produce nausea and vomiting.

Causes of nausea and vomiting

1. Narcotic administration
2. Hypoxia
3. Swallowing blood

The potential problem is not in the development of nausea, but in the act of vomiting, especially in the sedated patient. The patient who vomits lying in a dental chair in the supine position with the face upward faces the possibility of aspirating vomitus into the trachea or suffocating on the vomitus. The fact that a patient has received a CNS depressant medication only increases this risk because their protective reflexes may be depressed.

As mentioned above, it is the general category of narcotics in which the incidence of vomiting is greatest. The production of nausea and vomiting related to narcotic usage is a dose-related response. The greater the dose of the narcotic, the higher the incidence of nausea and vomiting. In the dosages recommended in Chapters 25 and 26, the incidence of vomiting has proved to be virtually zero. When I first began to use nalbuphine clinically, it was employed in doses greater than those currently recommended. Five of the first ten patients receiving nalbuphine either became nauseous or vomited later that same day. Decreasing the dosage of the drug has eliminated this as a significant problem.

The incidence of vomiting following narcotic administration is increased in ambulatory patients compared to hospitalized, nonambulatory patients. Patients receiving very large doses of nar-

cotics during general anesthesia do not have a high incidence of postoperative vomiting, whereas patients undergoing ambulatory surgery and receiving significantly lower doses of narcotics have a greater incidence of vomiting. This increased rate is related to the more frequent changing of body position that occurs in the ambulatory patient. Unfortunately this is a fact of life with which we must live. Happily, however, when narcotic dosages are kept within the limits recommended herein, the incidence of this complication is extremely low.

Management of nausea. Should nausea develop in the dental office the patient can be placed back in the dental chair and administered O₂. This alone usually leads to recovery. Hypoxia is a very frequent cause of nausea. Since recommending that nasal O₂ be administered routinely during IV sedation we have seen a virtual absence of nausea in our patient population.

If nausea develops when the patient returns home after the procedure, the postoperative instructions recommend that the patient lie down for awhile and, if available, drink a cola beverage, as this may settle the stomach.

Management of vomiting. Should nausea progress to vomiting when the patient is at home, the most important thing for the patient to remember is that he should not be lying on his back, as the possibility of aspiration is greater in this position. This is one of the reasons for strict adherence to the recommendation that the patient be able to manage himself at home before being discharged from the office.

If vomiting should occur while the patient is still undergoing treatment (a situation that has only occurred once in over 2000 cases at USC) the airway must immediately be cleared of all dental equipment. The patient's head is then turned to the side (away from the operator) so that the vomitus pools on one side of the mouth leaving a patent airway. Suction is then applied so that the remaining material is then removed.

Localized allergy

It is not uncommon for a patient receiving IV medications to mention that the skin at the injection site is itching. There may be a localized or diffuse reddening of the tissues. Several possible explanations are available.

The narcotic agonist meperidine produces the localized release of histamine. As the drug enters and travels up the vein toward the heart, a red



Fig. 27-4. Meperidine-induced localized histamine release along path of vein (*dark lines on wrist*).

line following the course of the vein may be observed (Fig. 27-4). The patient may mention that it is itching. Histamine release is a normal pharmacological property of meperidine and does not represent allergy. Within 5 minutes this response will resolve by itself.

Less frequently it will be observed that the skin around the site of the needle is diffusely erythematous. The word *blotchy* may appropriately be used to describe its appearance. The site may burn or be quite itchy. The reaction is localized to the immediate area but is not as localized as the meperidine-induced reaction. The most frequently observed cause of this type of localized allergic response is the adhesive on the tape used to secure the needle. Many persons are allergic to adhesive tape. The erythematous reaction will appear to be located around the tape. Management of this situation requires the IV administration of an antihistamine, either diphenhydramine 50 mg or chlorpheniramine 10 mg. The use of hypoaller-

genic tape on this patient will prevent recurrence.

If the allergic response appears to be more directly related to the administration of a drug, treatment need be more vigorous and immediate. The allergic reaction will be traveling up the patient's arm and may soon involve the entire body: the skin and respiratory and cardiovascular systems. Management of this potentially more serious reaction involves placement of a tourniquet high on the patient's arm as soon as the reaction is noted. This will prevent or at least slow the development of a generalized reaction. If the reaction is still limited to the limb, the administration of an antihistamine is recommended; however, if the reaction has become more generalized, 0.3 to 0.5 ml of a 1:1000 epinephrine solution is administered intravenously, followed by antihistamine administration.

Prevention of allergic reactions is greatly preferred to their management. Prevention is based on a careful pretreatment discussion of the patient's prior allergic history and response to the drugs being administered. The patient should be questioned about any previous reactions to adhesive tape. In addition, the 0.2 ml test dose recommended for all drugs will aid in determining if allergy is present or not. Although the administration of a dose as small as 0.2 ml of an allergen can, in some cases, induce anaphylactic reactions, in most circumstances the reaction will be less severe and management can follow that mentioned above.

Respiratory depression

Morbidity and mortality from IV sedation has usually been related to the presence of respiratory depression that was undetected and led to respiratory arrest, cardiac arrhythmias, and cardiac arrest.

All of the sedative drugs being discussed in this book are respiratory depressants. All are capable, in some doses and in some patients, of producing respiratory arrest. Respiratory depression is a more significant problem with certain groups of drugs, primarily the narcotic agonists and the barbiturates.

Respiratory depression occurring after drug administration is a dose-related response. Smaller doses of the drug produce little or no respiratory depression *in the average patient*; however, increasing the dosage of the drug increases its CNS depressant effects and leads ultimately to respiratory depression.

Respiratory depression may occur following extremely low doses of any drug, if the patient lies on the hyperresponding slope of the bell-shaped curve. Unfortunately there is little that can be done prior to drug administration to determine this fact. The patient can be questioned as to his usual response to drugs, such as analgesics and tranquilizers (e.g., oral diazepam). A patient who mentions falling asleep in response to one 2 mg Valium tablet should alert the doctor to the possibility of hyperresponsiveness. The 0.2 mg test dose, followed by a wait of 30 seconds before administering additional drug is another means of discovering a patient's sensitivity to drugs. One other means of accounting for variations in patient responses to drugs is titration. Always titrate drugs to clinical effect, if titration is possible. If this is done, oversedation and respiratory depression following IV drug administration should not occur, or will do so only infrequently. The drug doses recommended in Chapters 25 and 26 will provide effective sedation with little or no respiratory depression, in the typical patient.

The two categories of drug most likely to produce respiratory depression are the narcotics and barbiturates. Narcotic-induced respiratory depression is characterized by a decrease in the rate of breathing. The rate, normally between 14 to 18 breaths per minute, may fall to 5 or 6 per minute. Barbiturate-induced respiratory depression may be characterized by a more rapid but shallow respiratory effort than that seen with narcotics.

Management of respiratory depression. The initial steps in management of respiratory depression are universal, regardless of the cause of the problem. These are the steps of basic life support.

1. If not already in the supine position, the patient is placed in this position immediately. The patient's feet are elevated slightly (10 to 15 degrees) to aid in return of venous blood to the heart.

2. Probably the most important step in basic life support is the maintenance of a patent airway. This is accomplished through the head-tilt, neck lift or head-tilt, chin lift procedures (Fig. 27-5, A and B). Proper performance of this step will elevate the tongue from the hypopharynx and provide an adequate airway.

3. The doctor or assistant performing the previous step places his or her ear about 1 inch from the patient's mouth and nose, looking at the patient's chest, while listening and feeling for air exiting the victim's mouth and nose and watching

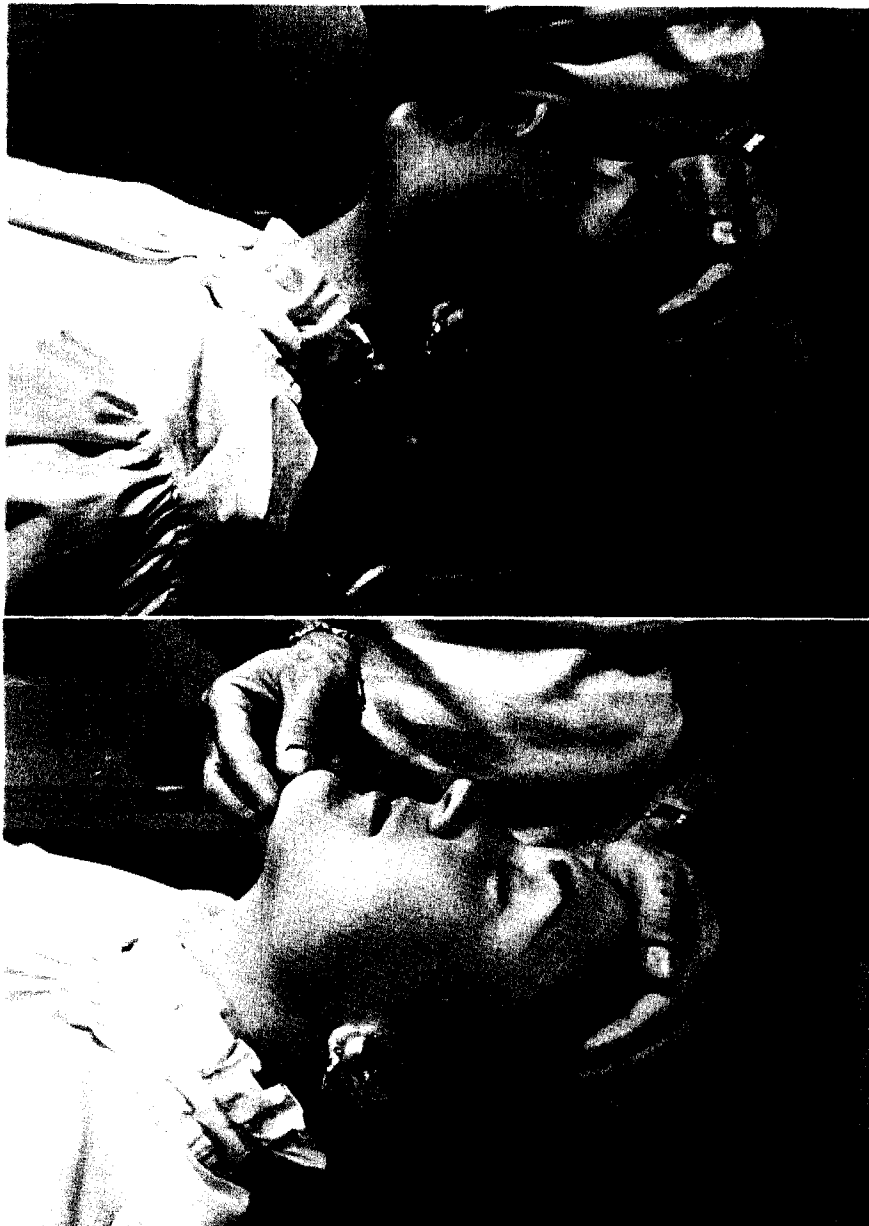


Fig. 27-5. A, Airway maintenance employing head-tilt, neck lift. **B,** Airway maintenance employing head-tilt, chin lift.



Fig. 27-6. Look, listen, and feel while checking for respiration and airway patency.

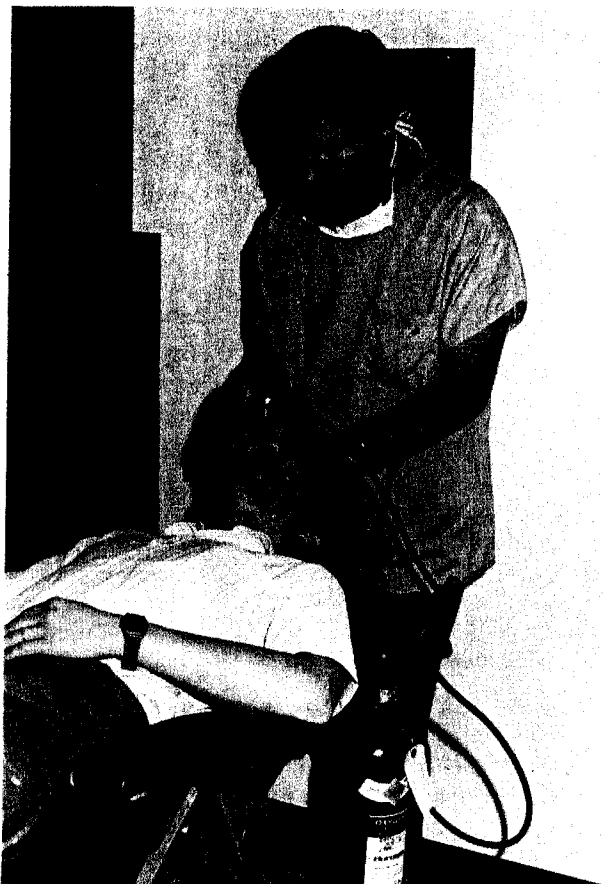


Fig. 27-7. Assisted or controlled ventilation using a positive pressure demand device.

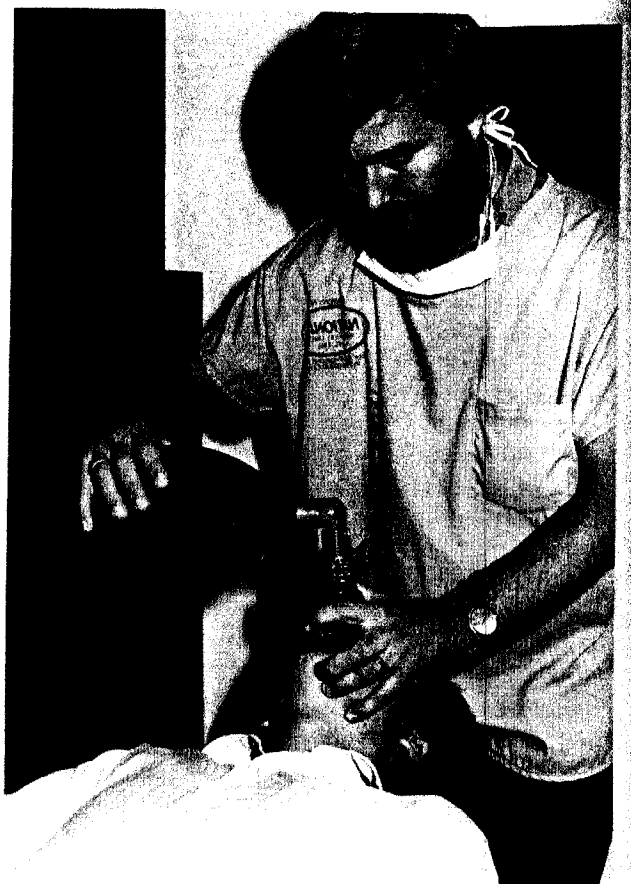


Fig. 27-8. Assisted or controlled ventilation using a self-inflating bag-valve-mask device.

the victim's chest for signs of spontaneous ventilatory efforts (Fig. 27-6).

4. In the highly likely event that the patient is breathing spontaneously but the rate and depth are depressed, the rescuer will assist or control the victim's breathing. Using a positive pressure O₂ device (Robertshaw or Elder Valve) (Fig. 27-7) or a self-inflating bag-valve-mask device (Ambu bag) (Fig. 27-8), the rescuer will inflate the victim's lungs every time a respiration is attempted. If the victim's respiratory rate is less than 8 per minute, the rescuer will increase the rate of breathing by interposing a controlled ventilation between each of the spontaneous attempts by the patient.

5. The carotid pulse is checked to determine the status of the cardiovascular system. If respiratory depression is recognized early, the carotid pulse will still be strong and regular.

6. If the respiratory depression has occurred following the administration of a narcotic to the patient (either as a sole drug or as one of a combination), a narcotic antagonist should be administered to the patient.

Naloxone is currently the drug of choice for reversing narcotic-induced respiratory depression. Nalbuphine has been employed with great success; however, more study of this drug is required before it can be recommended for this function. Naloxone should be diluted from its original 0.4 mg/ml concentration by adding 3 ml of diluent (5% dextrose and water, normal saline), thus producing a 0.1 mg/ml concentration for injection. The patient is continually observed for signs of increased respiration while 0.1 mg (1 ml) is slowly administered every minute. In most cases less than 0.4 mg naloxone will be required to reverse respiratory depression. In the not too distant past, naloxone was administered in 0.4 mg increments. It was found, however, that larger doses of naloxone also antagonize the analgesic properties of the narcotic. If the patient has undergone a painful procedure, the removal of the analgesic actions of the narcotic leads to an acute onset of pain, which is a significant stimulus to the heart and cardiovascular system. This has led to life-threatening emergencies in patients with prior histories of cardiovascular disease. Slow titration of 0.1 mg per minute minimizes this reaction. During the time between the administration of naloxone and its onset of action, the steps of basic life support must be continued.

7. With the return of more rapid and deep respiration the patient begins to look and feel considerably better. He may be unaware of what has

transpired for he has been deeply sedated during this time. Dental treatment may be halted, but the patient should not be permitted to leave the office at this time. Naloxone is a rapid-acting respiratory depressant; however, its duration of action is fairly short. It is therefore possible, though unlikely, for respiratory depression to develop again, approximately 30 minutes after the initial dose of naloxone was given. With the use of the shorter-acting narcotics, fentanyl and alphaprodine, this is unlikely to occur. When meperidine, pentazocine, morphine, and butorphanol are administered, the likelihood of this occurring increases. The patient should remain in the office, in a monitored recovery area, for at least 1 hour after the administration of naloxone.

Following the initial dose of naloxone and the recovery of the patient, it is recommended by many persons that 0.4 mg naloxone be administered intramuscularly. The duration of clinical action of IM naloxone is considerably longer than that of IV naloxone, thereby minimizing the risk of a return of respiratory depression.

Respiratory depression that is not produced by narcotics is not reversible with naloxone administration. The barbiturates are the group most likely to produce this problem. Management of nonnarcotic-induced respiratory depression is based on maintenance of the steps of basic life support—airway, breathing, and circulation—until the blood level of the offending drug has been lowered, through redistribution and metabolism, to the point that breathing is no longer depressed. There are no effective antidotal agents for the barbiturates.

Respiratory depression induced by diazepam and midazolam is extremely uncommon. Management is based on basic life support measures as described above. Research studies have indicated that there may be some beneficial effect in administering naloxone or physostigmine or both to these patients. However, until considerably more research proves the utility of these drugs conclusively, their administration in diazepam-induced oversedation or respiratory depression cannot be recommended.

Emergence delirium

A phenomenon known as emergence delirium has been reported following the administration of many CNS depressants, as well as some adjunctive drugs commonly employed intravenously in sedative procedures. The patient's response is one of transient delirium, hallucination, anxiety, or rage

that develops at some time during or immediately after the sedative procedure. Very often the response is associated with recall of an upsetting event in the patient's life. Minichetti and Milles reported a case of a 27-year-old patient receiving 7.5 mg IV diazepam, in addition to meperidine, atropine, N₂O-O₂, and local anesthesia, for extraction of several teeth. The patient felt quite comfortable and sedated. Following the administration of an additional 2.5 mg diazepam later in the procedure, the patient became progressively more excited. His eyes closed, he began crying, and he would not respond to verbal commands. Attempts were made to communicate with the patient but he would not respond to questioning. He became hyperexcitable and began thrashing about in the dental chair. Removal of the N₂O and administration of 100% O₂ did not resolve the situation, nor did administration of 0.4 mg naloxone. He continued to hallucinate for about 20 minutes, exhibiting rage and anxiety. He recovered gradually, calmed down, and stopped crying, responding to his name. When questioned later about the reaction he said that he had dreamt about an unpleasant experience he had in Vietnam.

The drugs most likely to produce emergence delirium are scopolamine and diazepam. Other benzodiazepines, such as lorazepam, have been reported to produce emergence delirium too. Experience with midazolam is too incomplete to permit any definitive statement; however, it is probable that being a benzodiazepine, midazolam is capable of producing this reaction. Scopolamine, however, is far and away the drug most likely to produce emergence delirium.

The reactions associated with emergence delirium are thought to be manifestations of the central anticholinergic syndrome (CAS). The CAS includes such paradoxical reactions as acute hyperactivity, anxiety, delirium, hallucinations, and recent memory impairment. In its more severe form the CAS produces apnea, medullary paralysis, coma, and death, though these reactions are extremely rare. The dose of scopolamine or benzodiazepine required to produce the CAS is extremely small; it is not a dose-related phenomenon; therefore the doctor administering IV anticholinergics or benzodiazepines must be aware of the CAS, its prevention, and its management.

Prevention. The incidence of the CAS and emergence delirium is considerably lower in patients between the ages of 6 and 65 years. The use

of scopolamine is not recommended in patients under 6 and over 65 years of age for this reason. Other anticholinergics, atropine and glycopyrrolate, that are less likely to produce CAS are recommended in these patients. Fortunately the indication for IV sedation in these two groups of patients is not great. Slow injection of drugs and use of minimal doses may aid in minimizing these reactions.

Management. Management of the usual form of emergence delirium, in which the patient may exhibit dreaming and appears uncomfortable, but does not respond to verbal questioning, takes two forms.

1. Symptomatic management. Monitoring of the patient, assurance of a patent airway and adequate blood supply to the brain (both usually no problem at all), and prevention of injury to the patient are the goals of treatment. Given appropriate time the reaction will terminate and the patient will open his eyes and be able to respond to commands and questions. In one case of emergence delirium that I witnessed, the patient had received scopolamine as a part of the Jorgensen technique and for almost 5 hours continued to dream and make uncoordinated movements in the chair. She was unresponsive to questioning but had a very adequate airway and her vital signs were slightly elevated over baseline. Approximately 5 hours after the administration of the scopolamine, the patient opened her eyes and was able to respond to commands.

2. Physostigmine administration. The second manner of managing emergence delirium is through the administration of physostigmine, a reversible cholinesterase. IV administration of physostigmine rapidly reverses emergence delirium and the CAS.

The dose of physostigmine for reversal of emergence delirium is 1.0 mg for the 70 kg adult and 0.5 mg for the child, administered intravenously. One milligram may be administered per minute until the reaction is terminated or a maximum dose of 4 mg is reached. Because physostigmine is metabolized within 30 minutes, the patient must be monitored closely to be certain that signs and symptoms do not recur.

Laryngospasm

Laryngospasm is a protective reflex of the body. In the fully conscious (non-CNS-depressed) patient, foreign objects are kept out of the airway (trachea) by the swallowing reflex, the epiglottis,

and the cough reflex. As a patient becomes more and more CNS depressed through the administration of drugs, these protective reflexes are depressed to a greater degree. In light stages of sedation, as observed with N_2O-O_2 , IV diazepam, midazolam, the Jorgensen technique, and IV promethazine, there is but little impairment of these reflexes. Foreign material such as water and scraps of dental materials will be easily removed by the patient as he spits or swallows. Aspiration is not a common occurrence with these techniques of sedation. However, as the depth of sedation is increased through the addition of other drugs or increased doses of the same drugs, the protective reflexes are depressed to a greater degree.

In stage two, ultralight general anesthesia, foreign material present in the area of the larynx will stimulate the tissues to provide a protective reflex in which the vocal cords adduct and attempt to seal off the trachea from foreign debris. Though this is truly a protective reflex it is obvious that this reflex will also prevent the passage of air into and out of the trachea and lungs (Fig. 27-9).

Laryngospasm will not occur in stage one of anesthesia, which we have called sedation or analgesia, because the other protective reflexes are still intact. It is only when the patient enters the grey zone between stage one and stage two that laryngospasm will occur.

Recognition. Recognition of laryngospasm is based on the presence or absence of sounds. A partial laryngospasm is identified by a high-pitched crowing type sound, produced as air is forced out through the partially closed vocal folds. A complete laryngospasm is identified by the absence of sound, an ominous "sound" indeed.

The patient will also be attempting to breathe against this partially or completely closed airway. Respiratory efforts will be exaggerated, expansion of the chest greater than usual, and accessory muscles of respiration used. Substernal and intercostal soft tissue retraction may be evidenced. This is the drawing in of the soft tissues overlying the spaces between the ribs and sternum, as the chest expands and the intrathoracic pressure becomes quite negative. Retraction is a sign of a partially or totally obstructed airway.

Management

1. The first step in managing a laryngospasm is to remove the offending material from the patient's airway. A large diameter suction tip or a tonsil suction is placed into the pharynx to re-

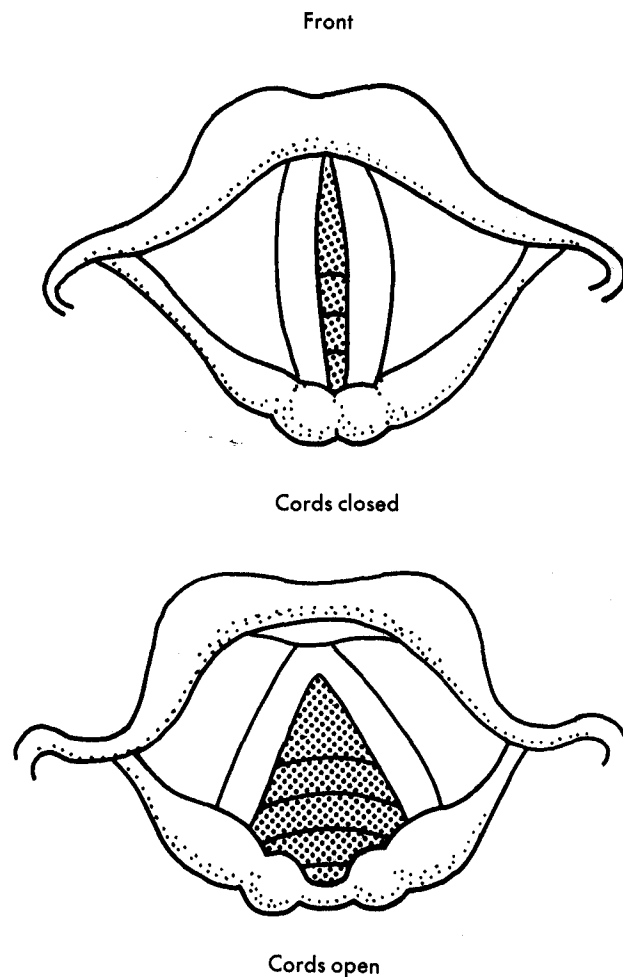


Fig. 27-9. View of vocal cords. Top: cords are adducted (closed) thus preventing foreign material and air from entering trachea. Bottom: cords abducted. (From Shibel, E.M., and Moser, K.M.: *Respiratory emergencies*, St. Louis, 1977, The C.V. Mosby Co.)

move any material it finds. This step alone will break the spasm in most cases.

2. Positive pressure O_2 is administered, either via the self-inflating bag-valve-mask device or by the positive pressure demand valve. Very often it is possible to break the spasm by forcing O_2 past the vocal cords. Steps 1 and 2 are highly recommended.

3. The administration of drugs to terminate laryngospasm is never recommended unless the doctor is well trained in anesthesiology and in management of the apneic patient. The drug of choice in this situation is succinylcholine, a short-acting depolarizing muscle relaxant. It is administered in a concentration of 20 mg/ml. An IV

dose of 20 to 40 mg is usually adequate to break the spasm by paralyzing the muscles of respiration. At this point, however, the patient is no longer breathing and the doctor is responsible for instituting controlled ventilation for the period of 3 to 4 minutes until the typical patient resumes spontaneous ventilation.

4. If no drugs were administered to the patient during the laryngospasm, the level of CO₂ in the blood would increase, the level of consciousness decrease, and the spasm break spontaneously. Though this technique is acceptable, it is *not* recommended for the untrained doctor.

Laryngospasm should never develop if lighter levels of sedation are maintained and if the airway is kept free of debris, water, and saliva

SPECIFIC DRUG COMPLICATIONS

Diazepam

The most commonly seen complications associated with diazepam administration are

1. Local venous complications
2. Emergence delirium
3. Recurrences of amnesia
4. Oversedation

Local venous complications and emergence delirium have previously been discussed. The recurrence of amnesia following diazepam administration has only occurred twice in my experience; however, it was this phenomenon that caused me to decrease the recommended dose of diazepam used for sedation.

Two patients received diazepam, one a dose of 45 mg and the other a dose of 38 mg, and achieved clinically ideal sedation that lasted for the usual 45 minutes. Recovery was normal, the patients appearing unsedated after 1 hour. It was later reported that for the first 24 hours after they left the clinic (these cases were done at different times and on different days) their recovery was normal. However, in both cases, approximately 24 hours later the patients experienced a relapse of amnesia. One patient had driven to work, parked his car, and entered his office building when suddenly he did not remember where he was, how he had gotten there, or what day it was. Within a few minutes the patient's memory returned. No further relapses occurred. The same type of response occurred in the second patient. I have not heard of similar responses developing in patients who have received less than 30 mg of diazepam at a single treatment, thus the recommendation that this dose not be exceeded as a total for one treatment session.

Oversedation is not likely to develop with diazepam if the drug is titrated at the recommended rate of 5 mg per minute. However, if a patient becomes oversedated, management is to ensure a patent airway and ventilation. Within a few minutes redistribution of diazepam will lead to a lightening of the level of sedation and increased responsiveness.

Studies have indicated that naloxone and physostigmine administered intravenously may be capable of reversing diazepam oversedation. More research is required in this area.

Pentobarbital

The most common problems associated with pentobarbital administration are oversedation and respiratory depression. Management of both of these situations requires implementation of the steps of basic life support: airway, breathing, and circulation. There are no antidotal drugs that can be administered to reverse barbiturate oversedation or respiratory depression. Unfortunately, because of the prolonged duration of action of pentobarbital, it may take 30 minutes to an hour for the patient to become considerably more responsive.

Promethazine

Promethazine-related complications include oversedation and extrapyramidal reactions. Oversedation is managed through basic life support. There is no effective antidote for promethazine-induced oversedation or respiratory depression. Extrapyramidal reactions, though quite rare, do develop following promethazine administration. Four types of reaction are identified: akathisia (motor restlessness), acute dystonias, parkinsonism, and tardive dyskinesias. These are described in Chapter 8. Management is the administration of IV diphenhydramine, 50 mg for the adult and 25 mg for the child.

Narcotics

The major side effects of narcotic administration are nausea and vomiting, respiratory depression, and rigid chest. The first two complications are discussed earlier in this chapter.

Rigid chest is an uncommon phenomenon that has been observed primarily after administration of fentanyl but can develop with any narcotic. It is most commonly seen when N₂O-O₂ has been administered concomitantly. In this situation the skeletal muscles of the thorax appear to be paralyzed and inflation of the chest is impossible. The

cause of rigid chest is unknown. The patient will be unable to breathe. Efforts to force air into the patient will prove futile as the chest will not expand. The chest has a firm boardlike feel during this reaction. Management of rigid chest involves:

1. The airway is supported and an attempt is made to force O₂ into the lungs.
2. IV succinylcholine 20 to 40 mg is administered (only recommended for those trained in anesthesiology).
3. Following the release of the rigid chest (caused by the actions of succinylcholine), the patient will be apneic for approximately 3 to 5 minutes during which time controlled ventilation is absolutely necessary.

Rigid chest has been observed in conjunction with the fentanyl and N₂O combination. Use of this combination is therefore not recommended in outpatient sedation procedures. The use of combinations of techniques will be discussed in Chapter 28.

Scopolamine

The major problem associated with scopolamine administration is emergence delirium, which has been discussed earlier.

SUMMARY

Complications do occur during IV sedation. Trieger and Malamed conducted independent surveys of doctors having completed their basic

Table 27-1. Percentage of doctors who have witnessed IV complications*

	Malamed (n = 114)		Trieger (n = 117)	
	no.	%	no.	%
Hematoma	39	34.2	32	27.0
Infiltration	38	33.3	41	35.0
Pain on injection	29	25.4	50	42.0
Hyperexcitement	29	25.4	11	9.0
Thrombophlebitis	28	24.5	24	20.0
Prolonged sedation	19	16.6	12	10.0
Vomiting	16	14.0	8	6.0
Hypotension	12	10.5	11	9.0
Apnea	1	0.8	0	0.0
Arterial injection	0	0.0	3	2.5

From Malamed, S.F.: *Anesth. Prog.* 28:158, 1981.

*The statistics illustrate the number of doctors who have seen the complication listed, not the number of times they have seen it occur. Therefore, a doctor may have seen one case of vomiting in 5000 IV procedures, but since 16 of the 114 reporting doctors reported at least 1 case of vomiting, it is listed as a 14% incidence.

IV sedation programs to determine which complications did in fact develop most often. The following chart presents their findings (Table 27-1).

BIBLIOGRAPHY

- Albo, D., Jr.: Effect of intra-arterial injection of barbiturates, *Am. J. Surg.* 120:676, 1970.
- Alderman, D.P.: Complications during general anesthesia. In Lichtiger, M., and Moya, F., editors: *Introduction to the practice of anesthesia*, Hagerstown, 1974, Harper & Row Publishers.
- Allen, G.D., Ricks, N.B., and Jorgensen, N.B.: The efficacy of the laryngeal reflex in conscious sedation, *J. Am. Dent. Assoc.* 94:901, 1977.
- Bidwai, A.V., Stanley, T.H., Roger, C., and Riet, E.K.: Reversal of diazepam induced postanesthetic somnolence with physostigmine, *Anesthesiology* 51:256, 1979.
- Blitt, C.D. and Petty, W.C.: Reversal of lorazepam delirium with physostigmine, *Anesth. Analg.* 54:607, 1975.
- Brandsetter, R.D., and Gotz, V.P.: Recurrence of intravenous-diazepam induced phlebitis from oral diazepam, *Drug Intell. Clin. Pharm.* 17:125, 1983.
- Braunstein, M.C.: Apnoea with maintenance of consciousness following intravenous diazepam, *Anesth. Analg.* 5:52, 1979.
- Brerton, R.B.: Incidence of complications from indwelling venous catheters, *Del. Med. J.* 41:1, 1969.
- Caldwell, C.B. and Gross, J.B.: Physostigmine reversal of midazolam-induced sedation, *Anesthesiology* 57:125, 1982.
- Campbell, R.L., Satterfield, S.D., Dionne, R.A., and Kelley, D.E.: Post anesthetic morbidity following fentanyl, diazepam and methohexital sedation, *Anesth. Prog.* 27:45, 1980.
- Chambiras, P.G.: Sedation in dentistry, intravenous diazepam, *Aust. Dent. J.* 17:17, 1972.
- Cunningham, E., and Korbon, G.A.: Venospasm preventing peripheral venous access, *Anesthesiology* 59:141, 1983.
- Dentist loses license in child death case. *Board Perspective: Calif. Board of Dent. Exam. Newsletter* 3:1, 1978.
- Donaldson, D., and Gibson, G.: Systemic complications with diazepam, *J. Oral Surg.* 49:126, 1980.
- Driscoll, E.J., Gelfman, S.S., Sweet, J.B., Butler, D.P., Wirdzek, P.R., and Medlin, T.: Thrombophlebitis after intravenous use of anesthesia and sedation: its incidence and natural history, *J. Oral Surg.* 37:809, 1979.
- Driscoll, E.J., Smilak, Z.H., Lightbody, P.M., and Fiorucci, R.D.: Sedation with intravenous diazepam, *J. Oral Surg.* 30:332, 1972.
- Ellis, F.R.: Malignant hyperpyrexia, *Anaesthesia* 28:245, 1973.
- Engler, H.S.: Gangrenous extremities resulting from intra-arterial injections, *Arch. Surg.* 94:644, 1967.
- Evaskus, D.S.: Apnea from improper administration of diazepam, *J. Oral Surg.* 35:875, 1977.
- Francis, J.G.: Intra-arterial methohexitone; injection into the central artery of the rabbit's ear, *Anaesthesia* 19:501, 1964.
- Gelfman, S.S., Dionne, R.A., and Driscoll, E.J.: Prospective study of venous complications following intravenous diazepam and in dental outpatients, *Anesth. Prog.* 28:126, 1981.
- Gelfman, S.S., and Driscoll, E.J.: Thrombophlebitis following intravenous anesthesia and sedation: an annotated literature review, *Anesth. Prog.* 24:194, 1977.
- Gold, M.I.: Treatment of bronchospasm during anesthesia, *Anesth. Analg.* 54:783, 1979.

- Goldsmith, D., and Trieger, N.: Accidental intra-arterial injection: a medical emergency, *Anesth. Prog.* **22**:180, 1975.
- Graham, C.W., Pagano, R.R., and Conner, J.T.: Pain and clinical thrombophlebitis following intravenous diazepam and lorazepam, *Anaesthesia* **33**:188, 1978.
- Graham, C.W., Pagano, R.R., and Katz, R.L.: Thrombophlebitis after intravenous diazepam—can it be prevented? *Anesth. Analg.* **56**:409, 1977.
- Grainger, J., and Marshall, K.: The Jorgensen technique revisited. A comparison with intravenous diazepam, *Dent. Anaesth. Sedat.* **10**:75, 1981.
- Greenblatt, D.J., and Koch-Weser, J.: Adverse reactions to intravenous diazepam: a report from the Boston Collaborative Drug Surveillance Program, *Am. J. Med. Sci.* **266**:261, 1973.
- Grower, M.F., Ayer, W.A., and Getter, L.: Solubility of injectable Valium in plasma and saline, *Anesth. Prog.* **23**:45, 1976.
- Hall, S.C., and Ovassapion, A.: Apnea after intravenous diazepam therapy, *JAMA* **238**:1052, 1977.
- Hayden, J., Jr.: The Jorgensen philosophy of intravenous sedation—a retrospective, *SAAD Dig.* **4**:257, 1981.
- Hegarty, J.E., and Dundee, J.W.: Sequelae after the intravenous injection of three benzodiazepines—diazepam, lorazepam, and flunitrazepam, *Brit. Med. J.* **2**(6099):1384, 1977.
- Hillman, J.D., McFall, W.T., Jr., and Gregg, J.M.: Intravenous conscious sedation in the periodontal patient, *J. Periodontol.* **52**:24, 1981.
- Jensen, S., Huttel, M.S., and Schou Olesen, A.: Venous complications after I.V. administration of Diazemuls (diazepam) and Dormicum (midazolam), *Br. J. Anaesth.* **53**:1083, 1981.
- Jusko, W.J., Gretch, M., and Gasset, R.: Precipitation of diazepam from intravenous preparations, *JAMA* **225**:176, 1973.
- Kortilla, K., and Linnoila, M.: Recovery after intravenous sedation. A comparison of clinical and paper and pencil tests used in assessing late effects of diazepam, *Anaesth.* **31**:724, 1976.
- Kortilla, K., and Linnoila, M.: Recovery and skills related to driving after intravenous sedation: dose-response relationship with diazepam, *Brit. J. Anaesth.* **47**:457, 1975.
- Langdon, D.E., Harlan, J.R., and Bailey, R.L.: Thrombophlebitis with diazepam used intravenously, *JAMA* **223**:184, 1973.
- Litchfield, N.B.: Complications of intravenous diazepam—allergy? *SAAD Digest* **4**:228, 1981.
- Litchfield, N.B.: Complications of intravenous diazepam: depression of respiration (an assessment of 16,000 cases), *Anesth. Prog.* **29**:11, 1981.
- Lopez, J.: Physostigmine reversal of diazepam-induced respiratory arrest: report of case, *J. Oral Surg.* **39**:539, 1981.
- Lynes, R.F.A., and Bisset, W.I.K.: Intra-arterial thiopentone: inadvertent injection through a cannula on the back of the hand, *Anaesthesia* **24**:257, 1969.
- Malamed, S.F.: Continuing education in intravenous sedation: part 2, complications and non-use, *Anesth. Prog.* **28**:158, 1981.
- Mariano, B.P., Roper, C.L., and Stapel, T.W.: Accidental migration of an intravenous catheter from the arm to the lung, *Radiology* **86**:736, 1966.
- Mark, L.C.: A lone case of gangrene following intraarterial thiopental 2.5%, *Anesthesiology* **59**:153, 1983.
- McVeigh, J.B.: General anesthetic emergencies and complications. In McCarthy, F.M., editor: *Emergencies in dental practice*, ed. 3, Philadelphia, 1979, W.B. Saunders Co.
- Miller, R., Arthur, A., and Stratigos, G.T.: Intraarterial injection of a barbiturate—a case report, *Anesth. Prog.* **23**:25, 1976.
- Minichetti, J., and Milles, M.: Hallucination and delirium reaction to intravenous diazepam administration: case report, *Anesth. Prog.* **29**:144, 1982.
- Mitchell, P.F.: Diazepam-associated thrombophlebitis: a review and discussion of possible prevention, *J. Am. Dent. Assoc.* **101**:492, 1980.
- Monteferrante, R.J.: Consultation report, *Anesth. Prog.* **27**:64, 1980.
- Nordell, K., Mogensen, L., Nyquist, O., and Orinius, E.: Thrombophlebitis following intravenous lidocaine infusion, *Acta Med. Scand.* **192**:263, 1972.
- Orkin, F.K. and Cooperman, L.H., editors: *Complications of anesthesiology*, Philadelphia, 1982, J.B. Lippincott Co.
- Padfield, A.: The local and systemic complications of intravenous diazepam, *SAAD Digest* **3**:241, 1978.
- Pallasch, T.J., and Gill, C.J.: Naloxone-associated morbidity and mortality, *Oral Surg.* **52**:602, 1981.
- Pandit, U.A., Kothary, S.P., Samra, S.K., Domino, E.F., and Pandit, S.K.: Physostigmine fails to reverse clinical, psychomotor, or EEG effects of lorazepam, *Anesth. Analg.* **62**:679, 1983.
- Pinder, R.M., Brogden, R.N., Speight, T.M., and Avery, G.S.: Dantrolene sodium: a review of its pharmacological properties and therapeutic efficacy in spasticity, *Drugs* **13**:3, 1977.
- Rosenberg, H.: Physostigmine reversal of sedative drugs, *JAMA* **229**:1168, 1974.
- Rosenberg, M.: Muscle rigidity with fentanyl: a case report, *Anesth. Prog.* **24**:50, 1977.
- Samuels, S.I.: Premedicating the dental patient, *SAAD Digest* **4**:252, 1981.
- Silver, D., and Harrington, M.P.: Thrombophlebitis: prevention, recognition, and management, *Primary Care* **4**:585, 1977.
- Sokoll, M.D., Hoyt, J.L., and Gergis, S.D.: Studies in muscle rigidity, nitrous oxide, and narcotic analgesic agents, *Anesth. Analg.* **51**:16, 1972.
- State charges dentist with negligence in three deaths, *Los Angeles Times*, Feb. 23, 1983.
- Stewart, R.C.: Respiratory depression with diazepam: potential complications and contraindications, *Anesth. Prog.* **25**:117, 1978.
- Swanson, J.T., and Aldrete, J.A.: Thrombophlebitis after intravenous infusion, *Rocky Mountain Med. J.* **66**:48, 1969.
- Thomas, E.T., Evers, W., Racz, G.B.: Postinfusion phlebitis, *Anesth. Analg.* **49**:150, 1970.
- Topazian, R.G.: Accidental intra-arterial injection: a hazard of intravenous medication, *J. Am. Dent. Assoc.* **81**:410, 1970.
- Trieger, N.T.: A comparative study of psychomotor effects of intravenous agents used in dentistry, *Oral Surg.* **30**:34, 1970.
- Trieger, N.T.: Teaching intravenous sedation: follow-up of 200 dentists, *Anesth. Prog.* **25**:154, 1978.
- Trieger, N.T.: *Pain Control*, Chicago, 1974, Quintessence.
- Venipuncture and venous cannulation, Chicago, 1971, Abbott Laboratories, Inc.