AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
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Local and Regional Anesthesia

Emergency physicians frequently perform procedures that may be painful, and the mastery of local and regional anesthetic techniques is vital to ensure a painless experience for the patient. Using a case-based format, the various types of anesthetic agents available and optimal techniques for their use in various parts of the body will be discussed.

- Explain the techniques for local and regional anesthesia on various parts of the body excluding the face.
- Discuss the pharmacologic properties, advantages, and disadvantages of various local anesthetic agents, including topical agents such as EMLA, TAC, and LET.
- Discuss recent advances in local and regional anesthesia.

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FACULTY

Michael F Murphy, MD, FRCPC (Anesthesia), FRCPC (Emergency Medicine)

Associate Professor, Emergency Medicine and Anesthesia, Dalhousie University; Executive Director, Emergency Health Services, Province of Nova Scotia, Halifax, Nova Scotia, Canada

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Local Anesthetic Agents

Michael F. Murphy, MD, FRCPC*

Local anesthetics are agents that reversibly block nerve conduction when applied to nervous tissue (central and peripheral) in an appropriate manner and concentration.[5, 8] Permanent neural damage may result from inappropriate technique. It is this "reversibility" that makes these agents so useful in clinical practice.

The purpose of this article is to present those essential features of local anesthetics that the emergency physician must be aware of to allow safe clinical practice.

HISTORY

Although general anesthesia was discovered in the 1840s in America, it was not until 40 years later that the local anesthetic properties of cocaine were discovered by Niemann in Europe.' This alkaloid, derived from the leaves of *Erythroxylon* coca, an Andean shrub, had been used for centuries by the inhabitants of the Peruvian Andes to produce a sense of well-being. Niemann noted that it had a bitter test and, remarkably, made the tongue numb. Von Anrep in 1880 discovered its usefulness in producing anesthesia when injected subcutaneously.[5]

Credit for the introduction of cocaine into clinical use in the 1880s as a local anesthetic usually is attributed to Segmund Freud and Karl Koller.[5] Freud used the central effects of the drug to wean a colleague from morphine, in the process producing a cocaine addict, an affliction from which Freud himself is reputed to have suffered. Koller used the drug to produce topical anesthesia of the eye. By 1885, Coming had used cocaine to produce spinal anesthesia in dogs.[5]

The toxicity of cocaine led to the synthesis of procaine in 1905, a popular agent in clinical practice to this day. Following the introduction of procaine (Novocaine), numerous compounds of similar chemical structure were developed, including tetracaine (Pontocaine) and chloroprocaine (Nes-

'Department of Emergency Medicine. Victoria General Hospital, Halifax. Nova Scotia, Canada

acaine). All of these agents are "ester" derivatives of para-aminobenzoic acid.

In 1943 Lofgren synthesized lidocaine (Xylocaine), an amide derivative of diethylaminoacetic acid.[1] Additional "amide" derivatives introduced since then include many commonly used agents such as mepivicaine (Carbocaine), bupivicaine (Marcaine), prilocaine (Citanest), and etidocaine (Duranest).

PHARMACOLOGY OF LOCAL ANESTHETIC AGENTS

The development of modern local anesthetic agents has been predicated on several tenets. The agent should

1. Be nonirritating to tissues
2. Not produce damage to nervous tissue
3. Have limited systemic toxicity at usual clinical doses
4. Have a finite duration of action
5. Be acceptable from a cost perspective

Agents in clinical use today meet these requirements. The emergency physician should understand the pharmacology of those agents to permit rational and safe clinical practice.

As has been mentioned, local anesthetics can be divided into two groups, amides and esters:

Amides	*Esters*
Lidocaine (Xylocaine)	Procaine (Novocaine)
Bupivicaine (Marcaine)	Chloroprocaine (Nesacainc)
Mepivicaine (Carbocaine)	Tetracaine (Pontocaine)
Prilocaine (Citanest)	
Etidocaine (Duranest)	

The practicing clinician is less interested in the specifics of the pharmacology of the agent than in the implications for clinical use. This is generally presented as the "clinical properties" of the agent and includes potency, duration of local anesthesia, and speed of onset.' The physicochemical properties that define these clinical properties are lipid solubility, protein binding, and pKa.[1]

Potency

Nerve membranes are essentially a lipoprotein matrix. Local anesthetic agents that have a high degree of lipid solubility (e.g., tetracaine, etidocaine) are thus of greater potency than are those of lesser solubility (e.g., mepivicaine, procaine; Table 1). In fact, lipid solubility appeal-s to be the prime determinant of local anesthetic potency.'

Duration of Anesthesia

Local anesthetic agents produce blockade by occluding sodium channels in the axon membrane. Binding of the agent occurs with a protein receptor in the sodium, channel.[1] Thus, those agents possessing a high

Table 1. *Physiochemical and Biologic Properties of Local Anesthetic Agents*

AGENT	LIPID SOLUBILITY	POTENCY	PROTEIN BINDING (%)	AVERAGE DURATION (MIN)	PKa (25°C)	ONSET (MIN)	MAXIMUM DOSE (MG/KG)
Esters							
Procaine	Low	1	6	30–44 (40)	8.9	18	7
Chloroprocaine	Low	1	—	30–60 (45)	8.7	5	8
Tetracaine	High	8	76	180–360 (200)	8.5	15	1.5
Amides							
Bupivicaine	High	8	96	180–600 (200)	8.1	10	3
Lidocaine	Medium	2	64	60–120 (100)	7.9	5	4
Prilocaine	Medium	2	55	60–120 (100)	7.9	5	5
Etidocaine	High	6	94	180–420 (200)	7.7	3	3
Mepivicaine	Low	2	78	90–150 (100)	7.6	3	4

Data from Savarese JJ, Covino BG: In Miller RD (ed): Anesthesia. New York, Churchill Livingstone, 1986, pp 985–1013; and *from* Tucker GT: Pharmacokinetics of local anesthetics. Br J Anesth 58:717–731, 1986; with permission.

affinity for protein (e.g., tetracaine and bupivicaine) have a longer duration of action than those less tightly bound (e.g., procaine).

Speed of Onset

The speed of onset of block is related directly to how quickly the drug is able to diffuse to and across the nerve sheath and membrane to get to the protein receptor in the sodium channel. This in turn is related to the concentration gradient (e.g., 1 per cent vs 2 per cent solution) developed within the tissues between the site of injection and the site of action. and the amount of drug in the nonionized form. One should recall that it is this nonionized form that diffuses quickly through tissues and membranes.

Local anesthetic agents are weak bases and thus are proton acceptors. As such, they tend to become positively charged as the pH of the environment falls, impeding movement through tissues and delaying the onset of blockade.

The pKa of a substance is that pH at which 50 per cent of the agent is ionized and 50 per cent is nonionized. Among the local anesthetics, pKas vary from 7.6 (mepivicaine) to 8.9 (procaine). Thus, at physiologic pH (7.4) procaine is almost totally charged (protonated) and very slow in onset, whereas mepivicaine is less than 65 per cent protonated and has a very rapid onset.

Table 1 relates the physicochemical and biologic properties of local anesthetic agents and should be consulted to clarify those properties important to the clinician.

Biotransformation and Elimination

As with any drug, the balance between rate of absorption and rate of destruction influences the toxicity of local anesthetic agents. The physician must be familiar with dosing limits and route ofelimination to avoid toxicity.

Ester-type local anesthetic agents are degraded very rapidly by liver and plasma esterases, including plasma cholinesterase. This occurs rapidly

and completely and limits the potential systemic toxicity of the drug. Amides, on the other hand, depend mainly on hepatic microsomal degradation, which is a slower process, thus increasing the possibility of toxicity.

Dosage

Table 1 lists maximal dosage guidelines on a milligram per kilogram basis for commonly used local anesthetic agents. In the practice of emergency medicine, the clinician rarely approaches these limits; however, clinical scenarios leading to potential toxicity include multiple intercostal nerve blocks for rib fractures, topical and infiltration anesthesia for extensive "road rash"; and in pediatrics, where diligence in dosage is mandatory.

Additives

The addition of epinephrine to local anesthetic solutions (I: 100,000 = 10 μg per ml; 1:200,000 = 5 μg per ml) reduces blood flow to the area of infiltration owing to the alpha effect of the epinephrine. There are three related effects:

Reduced blood loss
Prolonged duration of the block
Reduced plasma drug levels and toxicity

The implication of this last effect is that a larger total dose of agent may be administered when epinephrine is added (about 25 per cen t increase). Additionally, an increase in duration of action approaches 50 per cent for the short- and intermediate-acting drugs, but is much less apparent for those drugs already possessing a long duration of actions (e.g., bupivicaine, tetracaine, and etidocaine).

Contraindications to the use of epinephrine are essentially those in which vascular compromise is a possibility such as appendages (e.g., finger, penis, ear, etc.) and Rap-type lacerations. As always, significant doses of epinephrine should be used with caution, if at all, in patients with hypertensive and ischemic cardiovascular disease or pheochromocytomas.

Another common additive to multidose preparations of local anesthetic solutions is methylparaben. An ester of parahydroxybenzoic acid, this agent has antibacterial and antifungal properties useful in preserving the sterility of multidose vials.' The small concentration of methylparaben leads to no direct systemic toxicity.' However, methylparaben and its breakdown products are felt to be responsible for some allergic reactions attributed to local anesthetic agents. Thus, persons relating a history of allergy to local anesthetic solution, and in need of local anesthetic, should not be administered solutions containing methylparaben. Allergy will be discussed later in this article.

NEUROPHARMACOLOGY OF BLOCKAGE

Local anesthetic agents reversibly block transmission in all elements of the peripheral nervous system. It is useful for the practicing emergency medicine physician to have some appreciation of the relationship between

Table 2. *Classification of Peripheral Nerve Fibers*

FIBER CLASS	FIBER SUBCLASS	MYELIN	A) LOCATION B) FUNCTION
A	Alpha	+ + +	a) Afferent/efferent muscles b) Motor/proprioception
	Beta	+ + +	a) Afferent/efferent muscles b) Motor/proprioception
	Gamma	+ +	a) Efferent—muscle spindles b) Muscle tone
	Delta	+	a) Afferent sensory b) Pain, temperature, touch
B		+	a) Preganglionic sympathetic b) Autonomic function
C		—	a) Afferent sensory b) Pain, temperature, touch

the anatomy of the peripheral nerve and the ability of the drug to block transmission along it.

The peripheral nervous system includes the roots, rami, and branches of cranial and spinal nerves, as well as elements of the autonomic nervous system that accompany them.[5]

A mixed peripheral nerve contains multiple smaller bundles of nerve fibers called *fascicles*. *Endoneurium* binds these fibers together within a fascicle, and *perineurium* surrounds each fascicle. Fasciculi grouped together in a peripheral nerve are in turn wrapped in epineurium. These connective tissue elements organize and protect nerve fibers and provide a conduit for lymphatics and blood vessels. At the same time, such structural elements impede the diffusion of local anesthetic agents to their sites of action.

Individual nerve fibers vary in their degree of myelinization, speed of conduction, and susceptibility to blockade. A-alpha and A-beta fibers are heavily myelinated, rapidly conducting fibers subserving motor and proprioceptive functions (Table 2). This degree of myelinization leads to an inability of weak local anesthetic solutions to produce motor blockade. The persistence of a sensation of pressure at the operative site, but not pain, is therefore explained.

A-delta and C fibers are the least myelinated fibers, subserving pain, temperature, and touch. These fibers are easily blocked by local anesthetic agents, explaining the rapidity of onset of sensory blockade seen clinically.

B fibers, comprising preganglionic sympathetic fibers, are lightly myelinated and of intermediate sensitivity. Postganglionic sympathetic fibers are of the C class.

One is now able to explain what is observed clinically following the block of a peripheral nerve. Namely, that sympathetic block (vasodilation) is followed quickly by sensory blockade, with variable blockade of motor function and proprioception.

TOXICITY

Adverse reactions to local anesthetic agents are uncommon in the day-to-day practice of emergency medicine owing to two factors: (1) allergy to

local anesthetics is rare; and (2) dosage limits are rarely approached. Nevertheless, the emergency physician must be able to identify the "at-risk" situation, as well as the signs of systemic toxicity should they occur.

Local anesthetics and accompanying preservatives are nontoxic to neural tissue in available preparations. However, direct damage to nerves is possible during blockade should nerve fibers be lacerated by the needle, or if the agent is injected under pressure into the nerves, leading to pressure necrosis of nerve fibers. It is therefore recommended that paresthesias not be elicited by "needling" the nerve, and one should aim to deposit the local anesthetic *near* the nerve, not *in* it.

Systemic toxicity in everyday practice is rare. Psychophysiologically mediated vasovagal reactions, although unrelated to the drug, are probably the most common "systemic" reactions occurring during the administration of local anesthetic agents. In fact, when interviewed closely, most patients professing a local anesthetic "allergy" al-c found to have suffered a vasovagal episode.

The systemic toxicity of local anesthetic agents may become manifest if the agent is injected directly intravenously or if it is rapidly absorbed from peripheral sites ("cumulative toxicity").[1] The major manifestations of systemic toxicity relate to the CNS and the cardiovascular system.

The initial symptoms and signs of CNS toxicity include tinnitus, drowsiness, lightheadedness, and tingling of the lips. This progresses to a "thick tongue" and slurred speech and ultimately to tonic-clonic seizures. Should these events follow direct intravenous injection they are usually self-limited and benign if handled appropriately. However, significant CNS toxicity resulting from a depot injection (e.g., extensive local infiltration) of local anesthetic solution may not be so self-limited or benign and require active intervention to halt seizures and maintain ventilation.

By and large, the cardiovascular system is four to seven times less susceptible to the toxic effects of local anesthetic agents than the CNS.[6]

Individual agents affect the heart in varying degrees, although due to similar mechanisms: a direct depressant action on cardiac and vascular smooth muscle, and on the cardiac conduction system.[6] Direct cardiovascular depression is secondary to the effects of local anesthetic agents on ionic conductance in these tissues. Rhythm and conduction disturbances include bradycardia, asystole, and ventricular tachycardia.[4] Varying degrees of heart block, direct myocardial depression, and pump failure also are seen.

When approaching the limits of toxicity of a particular agent, one must be aware of potential adverse drug/drug interactions. The cardiovascular toxicity of local anesthetic agents may be increased by beta-blocking drugs,[7] and perhaps calcium channel blockers.

Although vasovagal reactions and systemic reactions to the adrenaline (e.g., palpatations) contained in local anesthetic solutions may be relatively common, hypersensitivity reactions are distinctly uncommon.[2,4]

A true allergic reaction is characterized by urticaria, edema, bronchospasm, and hypotension. Esters are more allergenic than amides and most case reports in the literature deal with this class. Proven allergy to members of the amide class is very rare.[7]

The investigation of a reported "allergic" reaction typically involves an exhaustive history followed by intradermal testing (1 mm bleb) of dilute solutions (1:10,000) of the implicated agent, in an area where resuscitation equipment is immediately available. Unfortunately, the problem ordinarily is presented to the emergency medicine physician at the time a local anesthetic agent is needed to care for an injury. Postponement of the procedure to clarify the alleged allergy is not possible. Usually, the simplest course to follow is to use an agent of the class to which allergy has not been identified. Of course, this assumes the clinician is able to ascertain which class was initially implicated. Additionally, in such a patient, solutions with added preservative and vasoconstrictor should not be used.

Finally, the issue of amide local anesthetics and malignant hyperthermia (MH) needs to be addressed. Early literature on this topic recommended that patients with MH and their close relatives not be given amide local anesthetics because of the possibility of triggering the disease. This statement has not withstood scientific investigation, and amides are felt to be safe in these groups. However, old convictions die hard, and MH-susceptible persons continue to be suspicious of amide local anesthetics. If confronted, discretion being the better part of valor, the selection of an ester agent serves to allay such fears.

CLINICAL USE

The use of local anesthetic agents in the Emergency Department is an indispensable aspect of practice in the treatment of surgical disease. Although the anesthesiologist is generally considered to be the "expert" when it comes to local anesthetics, and has as his or her domain the performance of *major* regional anesthesia (e.g., epidural and spinal), the emergency physician performs blocks of a lesser magnitude with a far greater frequency.

Several general terms are used to describe the type of regional anesthesia administered?

1. Local infiltration: the usual method of injecting local anesthetic solution subcutaneously
2. Field *block:* the infiltration of a wall of local anesthetic to surround an operative site
3. *Nerve* block: regional anesthesia created by the injection of local anesthetic agent near a nerve/or nerves supplying a particular area
4. *Topical anesthesia:* this type is self-explanatory.

Thus, the first decision to be made by the emergency physician is the type of block to be produced. Local infiltration, either through or alongside a wound, is the usual method used to allow the care of smaller lesions. More extensive areas are often amenable to field or nerve block techniques, saving time and reducing the amount of agent used. Typically, a field or block is most useful for the incision and drainage of cutaneous or subcuta-

neous abscesses, and the repair of complex lacerations, in which the anatomic distortion produced by local infiltration is detrimental.

The other major consideration is agent selection. It is impractical, because of the large number of agents available, for the emergency physician to maintain facility and safety with all of them. It would seem reasonable to become familiar with a short-acting agent (e.g., lidocaine), a long-acting agent (e.g., bupivicaine), and an agent of the ester class (e.g., procaine), should amides be contraindicated. Such an armamentarium should handle the breadth of clinical problems facing the practicing emergency physician.

SUMMARY

The rational selection and safe use of local anesthetic solutions is of paramount importance to the practice of emergency medicine. Such decisions are based on a sound knowledge of the pharmacology and toxicity of those agents one uses clinically in day to day practice. In addition, such information explains what one actually sees clinically following the injection of a local anesthetic solution, and, more importantly, what one ought to expect.

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Department of Emergency Medicine
Victoria General Hospital
1278 Tower Road
Halifax, Nova Scotia
Canada B3H 2Y9

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Regional Anesthesia In The Emergency Department

Michael F. Murphy, MD, FRCPC*

The use of local anesthetic agents is an essential aspect of practice in emergency medicine. Most cases involve local infiltration of local anesthetic agent to facilitate wound cleansing and closure. There are situations, however, in which local infiltration is a less than optimal option and the clinican considers a regional technique or, in some cases perhaps, a general anesthetic.

The advantages of regional anesthesia over local infiltration of an agent are governed by the situation. A regional anesthetic technique may be chosen where local infiltration is very painful (e.g., abscesses), very extensive (approaching dosage limits for the agent), or may distort a complex wound complicating closure. Some regional blocks (e.g., intercostal and mandibular blocks) are useful in acute pain management.

The purpose of this article is to acquaint the reader with the field of regional anesthesia and detail specific blocks useful in the practice of emergency medicine.

DEFINITIONS

Anesthesia is generally divided into two broad areas: general anesthesia and local anesthesia. Local anesthesia can he subdivided into *major* regional anesthesia (epidural and spinal), and additional subsets of local anesthesia:

1. Topical anesthesia
2. Local infiltration anesthesia
3. Field block anesthesia
4. Nerve block anesthesia

A field block is produced when a wall of local anesthetic solution is infiltrated subcutaneously around the horder of an operative field. A common example is the incision and drainage of a cutaneous abscess, in which injection of solution into the abscess is painful and ineffective, while the subcutaneous injection circumfercntially allows adequate anesthesia. The rationale for

*Department of Emergency Medicine, Victoria General Hospital, Halifax, Nova Scotia

this block lies in the fact that usually the branches of sensory nerves run parallel to the skin surface in the subcutaneous tissue. The pinna of the ear is another example of a structure anesthetised easily by a field block.

In a nerve block, regional anesthesia is created by the injection of local anesthesia near the nerves supplying that area. It goes without saying, therefore, that successful blockade depends on a thorough knowledge of surface and neural anatomy. Such knowledge is critical to the accurate placement of needle and solution in the production of a block. Often the knowledge of surrounding structures helps to prevent the complications associated with a particular block (e.g., intercostal nerve block and the risk of pneumothorax).

Approach to the Patient

As with all procedures, patient consent and cooperation is essential to a successful block. The physician should therefore explain to the patient the indication and rationale for the procedure. The patient should be made aware that the injection of local anesthetic will produce a burning or stinging sensation, which will be minimized by a slow speed of injection.

Most importantly, potential complications such as nerve damage should be described and discussed as necessary. They al-e covered in this article when the individual blocks are discussed.

Indications

The indications for regional anesthesia are as many and varied as the operative procedures that require them. Regional anesthesia permits the painless management of a particular disorder.

Contraindications

Although rare, an absolute contraindication would be an allergy to a particular local anesthetic agent. This can be circumvented by using an agent of the other class of local anesthetic agents. Infection in the area of the block (i.e., the needle must pass through infected tissue) contraindicates the technique for obvious reasons. Coagulation disorders (PT greater than 1.5 times control; platelets less than 50,000) may contraindicate blocks of major nerve trunks where accidental arterial puncture is a risk, but applies especially in spinal, epidural and caudal anesthesia.

A previous neurologic deficit in the area to be blocked constitutes a relative contraindication to *nerve* block anesthesia. The risk is that of a possible enhancement of the preexisting disability due to damage to the nerve by the block itself. The consequence is likely more a medico-legal concern than a practical one.

Finally, poor patient acceptance or cooperation, or a physician's unfamiliarity with a block, constitute relative contraindications.

TECHNIQUE

Preparation and Supplies

With the exception of intravenous regional anesthesia ("Bier block"), all other blocks require no additional equipment to that required for simple infiltration of local anesthetic solution.

Preparation and injection of the solution should follow aseptic technique, as with any injection. The author recommends an iodine cleansing solution for the skin and the use of sterile gloves (as much for the physician's protection as the patients'). For most simple blocks, drapes are not necessary. Recapping of needles is to be avoided as a matter of principle to eliminate the attendant risk of accidental needle sticks.

I prefer to use a 10 ml syringe for most blocks. The local anesthetic solution is drawn up with an 18-gauge needle, a skin wheal raised with a 25- or 27-gauge needle, and a 22-gauge needle used to inject the solution. This larger gauge allows for easier injection and reduces the risk of breakage seen with the smaller gauge needle.

Resuscitation equipment should be available should some catastrophe occur. This includes oxygen, bag-valve mask, suction apparatus, airways, laryngoscopes, endotracheal tubes, and adjuvant medications as needed.

Specifics of Performance

Patient discomfort is minimized by the slow injection of local anesthetic solution, avoidance of sensitive structures such as periosteum and a gentle, reassuring demeanor.

The emergency physician, in addition to a sound knowledge of anatomy, will use three techniques to allow accurate needle placement in producing a nerve block[4]:

1. Elicitation of paresthesias—when a nerve is struck by a needle, paresthesias in the peripheral distribution of that nerve will occur. The patient may experience considerable discomfort and surprise, and in a reflex move away from operator, may fracture a needle or damage the nerve.

Additionally, the sharp bevelled needle may lacerate nerve fibers potentially producing a neural deficit. For these reasons the author discourages the deliberate elicitiation of paresthesias, relying instead on sound anatomical knowledge and experience.

2. Approximation to bony landmarks—bony landmarks serve as keystones in relating deep to surface anatomy and are used extensively to aid in the location of nervous structures for blockade.

3. Approximation to arteries-in many areas of the body, significant nervous structures run in conjunction with palpable arteries (e.g., femoral and ulnar nerves). The relationship of nerve and artery is constant and precise and allows accurate deposition of local anesthetic solution. Such proximity, however, mandates caution in performance, and the elimination of epinephrine from the anesthetic solution.

An intravenous line should be established prior to a procedure (I) that includes intravenous regional anesthesia, (2) may approach a toxic dose of local anesthetic agent; (3) where adjuvant intravenous medications may be needed.

COMPLICATIONS

Specific complications will vary with the particular block being done and will be highlighted at the time those blocks are discussed. However, certain complications are common to all techniques and include

1. Needle breakage
2. Needle damage to nerves, vessels, viscera or other structures (e.g., pleura)

3. Unintentional blockade of additional nerves (e.g., optic)
4. Ecchymosis and hematoma
5. Local infection
6. Systemic toxicity of the agent or its additives, including allergy
7. Prolonged block (idiosyncratic)

SPECIFIC BLOCKS

Several blocks (field and nerve) are extremely useful in the emergency setting. The list is by no means complete, although those blocks most useful to the practicing physician are included.

I. Head and Neck
   a) Ear
   b) Supraorbital nerve block
   c) Infraorbital nerve block
   d) Mental nerve block
   e) Mandibular nerve block
   f) Posterior superior alveolar block
II. Intercostal Nerve Block
III. Upper Extremity
   a) Median nerve block at the wrist
   b) Ulnar nerve block at the wrist
   c) Radial nerve block at the wrist
   d) Metacarpal block
IV. Lower Extremity
   a) Anterior ankle block
   b) Posterior ankle block

Head and Neck

a. Ear: This block is particularly useful in repairing complex lacerations of the pinna of the ear and is essentially a field block (Fig. 1). The sensory supply of the pinna is derived from branches of the lesser occipital, greater auricular, and auriculotemporal nerves that travel subcutaneously to the ear. Islands of unanesthetized skin may remain surrounding the external auditory meatus and auditory canal due to supply by the auricular branch of the vagus.

The skin is prepared and a wheal raised with a 25-gauge needle 1 cm caudad to the base of the lobe. A 2%gauge, 1% inch needle is advanced subcutaneously anterior and posterior to the ear and local anesthetic solution injected as the needle is withdrawn. This procedure is mntinued around the circumference of the ear with due caution in the vicinitv of the temporal artery.

The use of a "donut" in positioning the patient's head may facilitate stability for injection. Local anesthetic solutions containing epinephrine should not be used for obvious reasons.

b. Supraorbital Nerve Block: This block facilitates complex or extensive repairs of the forehead as far back as the lambdoidal suture (Fig. 2). It is especially useful in the cleaning and repair of "road" and "glass" rash of the forehead.

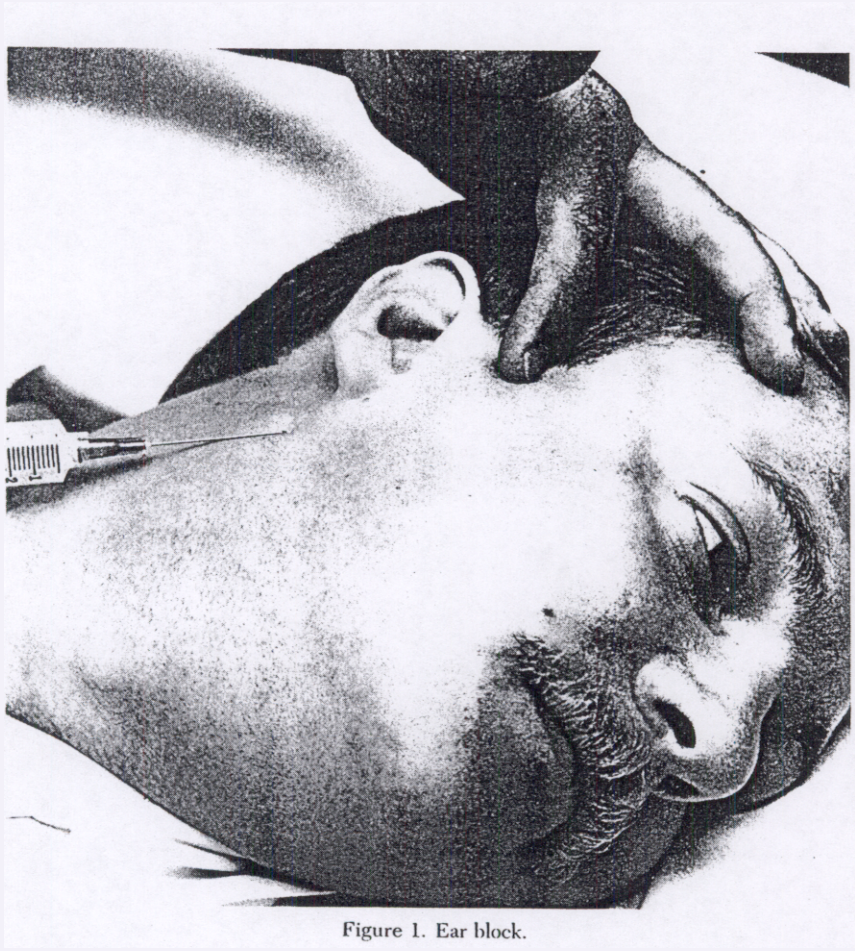


Figure 1. Ear block.

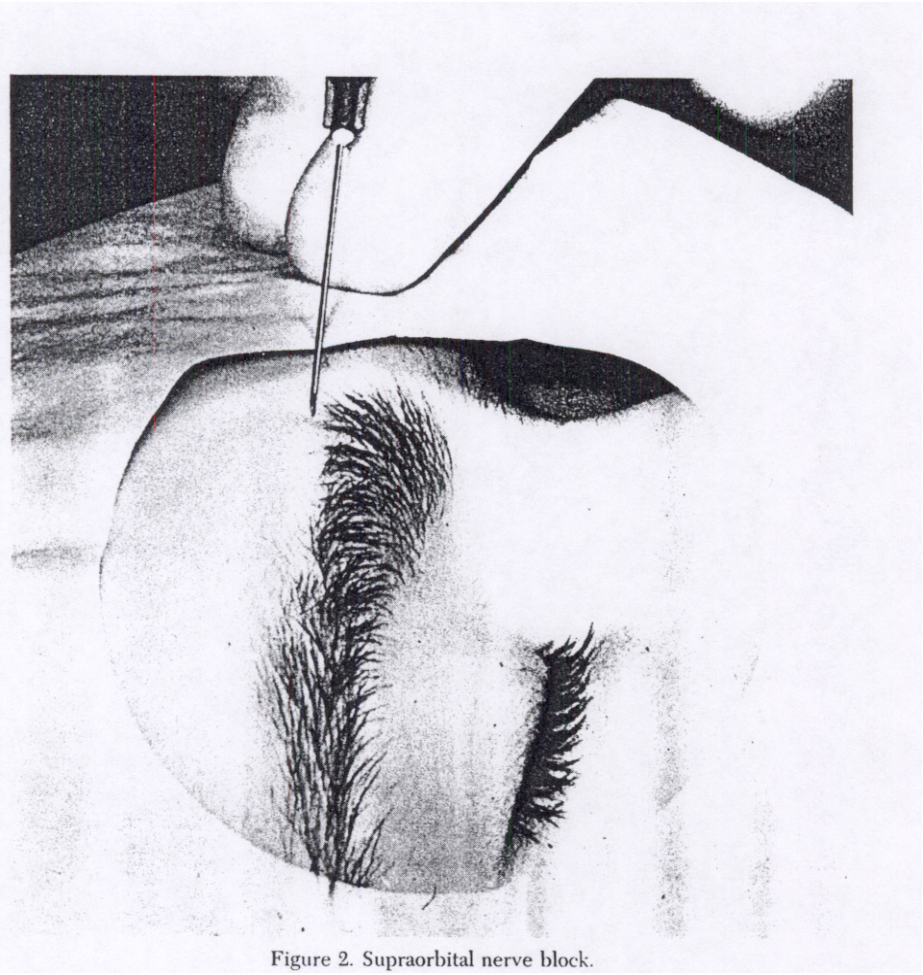


Figure 2. Supraorbital nerve block.

A skin wheal is raised in the midline at the medial limit of the eyebrow. A 22-gauge needle is advanced subcutaneously beneath the eyebrow and local anesthetic agent injected as the needle is withdrawn. Five to ten milliliters of solution may be used on each side. The needle is reinserted at the lateral limit of the first injection and the procedure repeated to block lateral branches. Both supraorbital and supratrachlear nerves are blocked by the method.

Open skull fractures in the area may contraindicate the block because cranial and bulbar palsies may follow injection of local anesthetic agent into the subarachnoid space. The careless operator or an uncooperative patient may predispose to needle injury of the eye. Additionally, inadvertant orbital injection may lead to temporary blindness should the second cranial nerve be blocked.

c. Infraorbital Nerve: Repairs of the lower lid, side of the nose, the upper lip and its mucous membrane are possible with this block. Dental pain from upper incisors and cuspids also may be alleviated.

The infraorbital foramen is palpable slightly nasal of the midline and 0.5 to 1 cm below the inferior orbital rim (Fig. 3). Although intraoral or

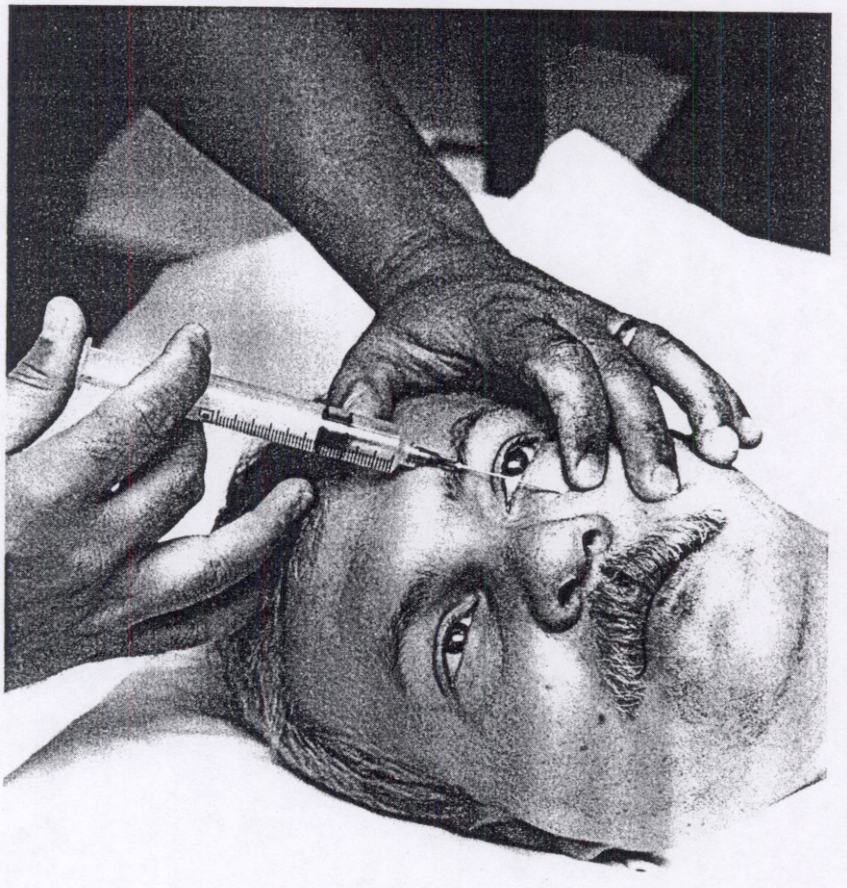


Figure 3. Infraorbital nerve block.

extraoral approaches may be used, the simpler extraoral route will be described. The author prefers to approach the foramen by directing the needle through a wheal in a caudad direction, standing at the head of the patient. The foramen is palpated with a finger of one hand and the needle guided to the orifice. The needle should not be inserted into the canal because pressure injury to the nerve may occur with injection. Two to five milliliters of the local anesthetic agent is injected slowly at this site and 1 additional milliliter as the needle is withdrawn.

Intraorbital block with oculoparesis or temporary blindness may occur but is exceedingly rare with this approach. Ocular injury by the needle may occur in the case of an uncooperative patient or careless operator.

d. Mental Nerve Block: Repairs of the lower lip, its mucosa, and the chin are made possible by this block.

The mental foramen lies midway between the upper and lower borders of the mandible in line with the second premolar and is easily palpable extraorally (Fig. 4). In the difficult patient, the position can be approximated by recalling that the supraorbital notch, infraorbital foramen, and mental foramen lie on a straight line that passes vertically through the pupil and the corner of the mouth (Fig. 5). The nerve may be approached either

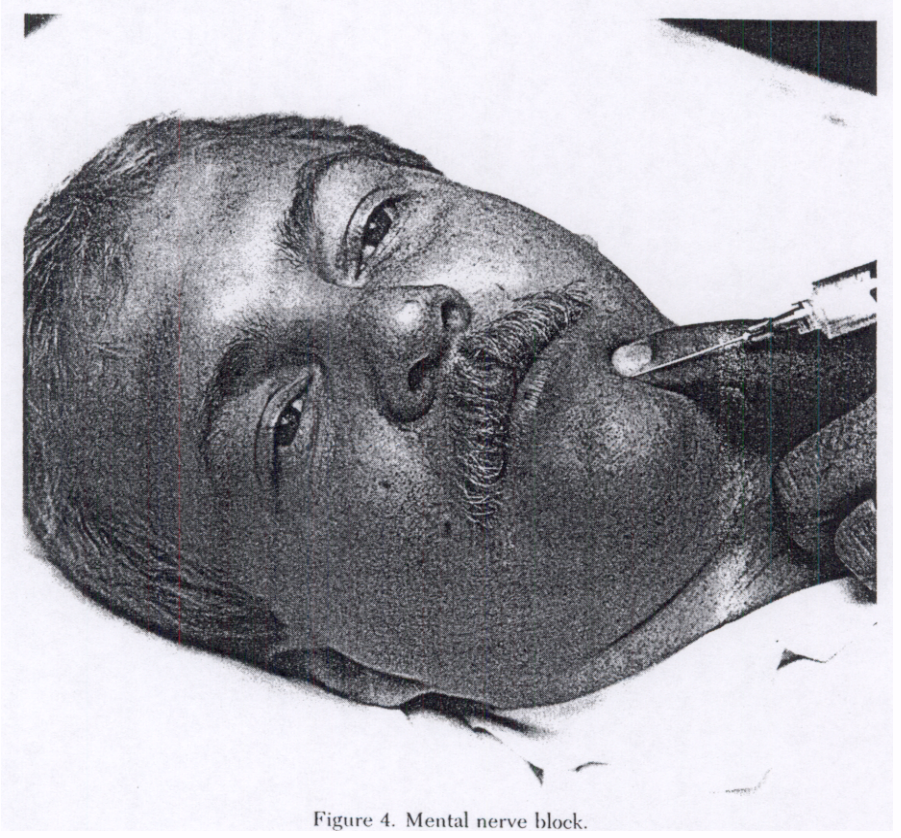


Figure 4. Mental nerve block.

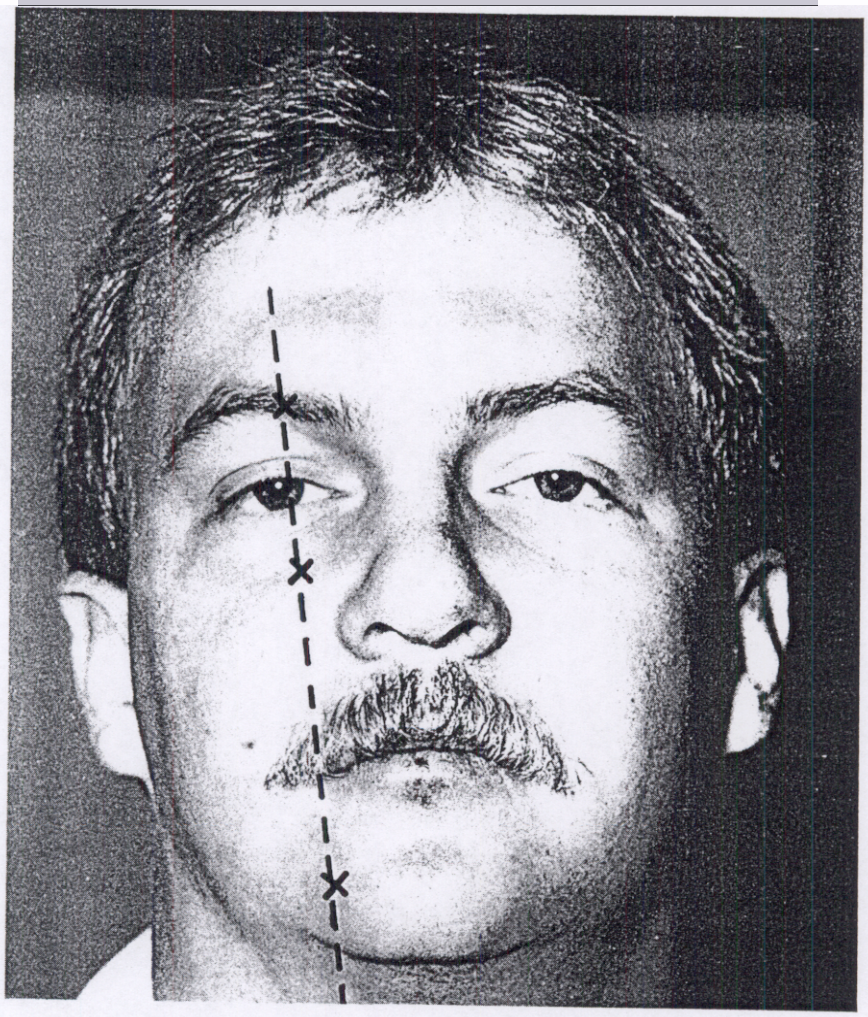


Figure 5. Diagram showing the position of the supraorbital notch, infraorbital foramen, and mental foramen. See text for details.

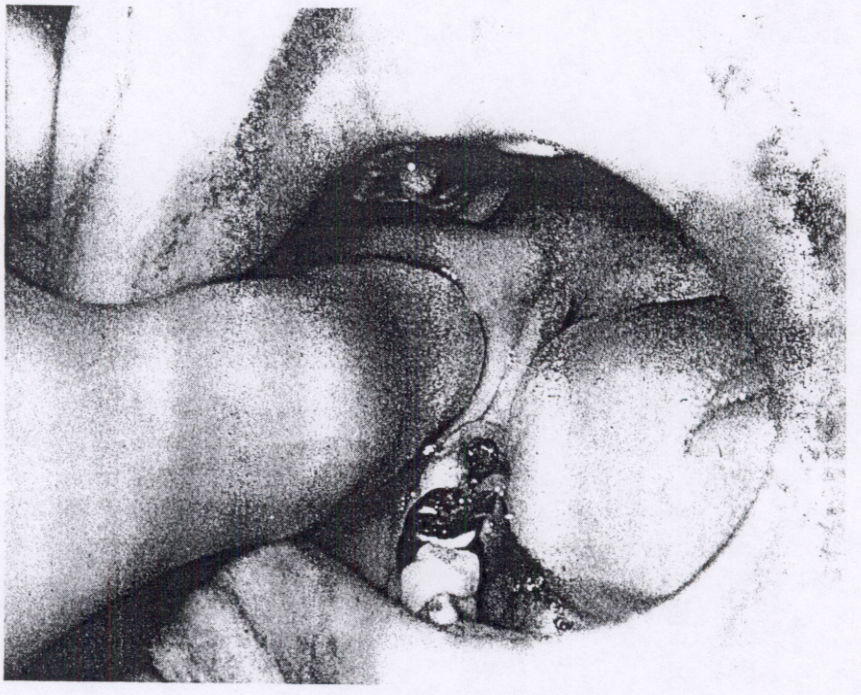


Figure 6. Mandibular nerve block.

intra- or extraorally as preferred by the operator. Some authors recommend entering the mental canal with the needle directed anteriorly. Injecting in the canal may produce pressure injury to the nerve, however.

Three to five milliliters of solution is used with an additional two ml injected as the needle is withdrawn.

e. Mandibular Nerve Block: This block is useful in the repair of lacerations involving the anterior two-thirds of the tongue or to alleviate pain from mandibular dentition. The latter indication is particularly useful if malingering or substance abuse is suspected. The patient is seated facing the physician with the head stabilized posteriorly to avoid its being withdrawn.

The mandibular nerve divides into several branches below the foramen ovale. The inferior alveolar branch and the lingual nerve descend on the medial side of the ramus of the mandible, and the inferior alveolar nerve enters the mandibalar foramen just above the last molar to course through the substance of the mandible.

To block the right side, the anterior border of the ramus is palpated with the left thumb (Fig. 6). The needle is advanced over the dorsum of the thumb to the oral mucosa and then to bone about 1 cm above the last molar (Fig. 7). The needle and syringe are then rotated in a horizontal plane to the opposite side of the mouth, maintaining contact with bone (Fig. 8). Five to ten milliliters of solution are deposited, following aspiration.

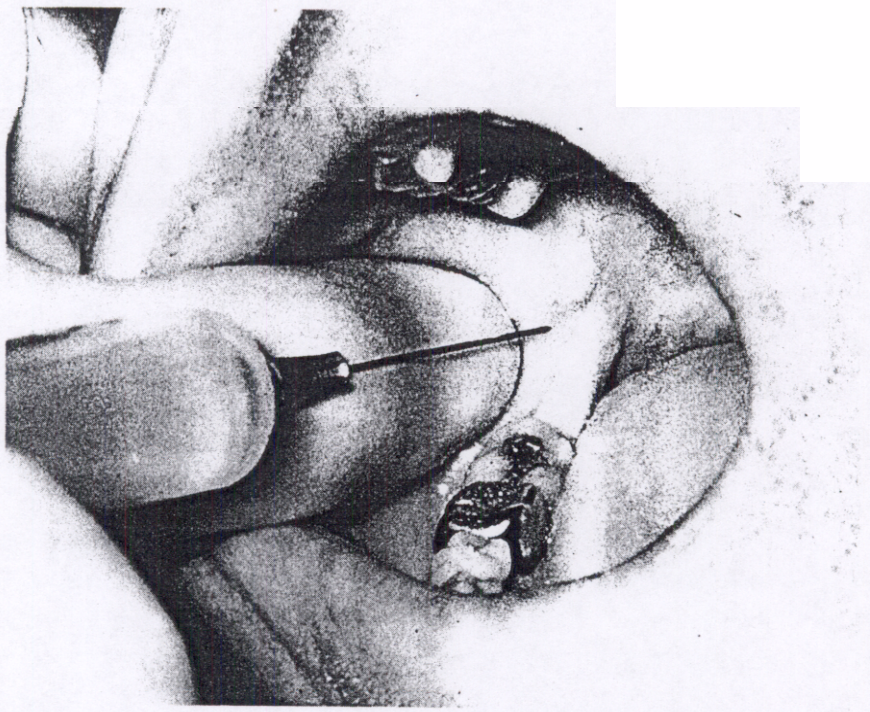


Figure 7. Mandibular nerve block. The needle is advanced to bone about 1 cm above the last molar.

The patient should be warned that anesthesia of tongue and lip may produce difficulty with speech, mastication, and swallowing. Damage to the tongue and buccal mucosa may also occur due to the loss of protective sensation.

f. Posterior Superior Alveolar Block: The upper molar teeth are supplied by the posterior superior alveolar branches of the maxillary nerve. Blockade of these nerves is useful in the emergency department in the management of dental pain, particularly if substance abuse is suspected. The remainder of the maxillary teeth are supplied by branches of the infraorbital nerve after it emerges from the foramen, and they are thus blocked by the infraorbital nerve block.

A 25-gauge ⅝ inch needle on a 10-ml syringe, armed with 6 ml of local anesthetic solution, is used to perform, this block. The needle is then bent at a forty-five degree angle where it meets the hub (Fig. 10). This needle is then inserted to the hilt just distal to the second molar directed superiorly and slightly posteriorly along the maxilla (Fig. 10). Following careful aspiration 4 to 6 ml of solution is *slowly* injected.

The performance of this block necessitates placement of the needle and injection of solution into the pterygopalatine fossa to block the posterior superior alevcolar nerve. Owing to the rich vascularity of this area, gentle

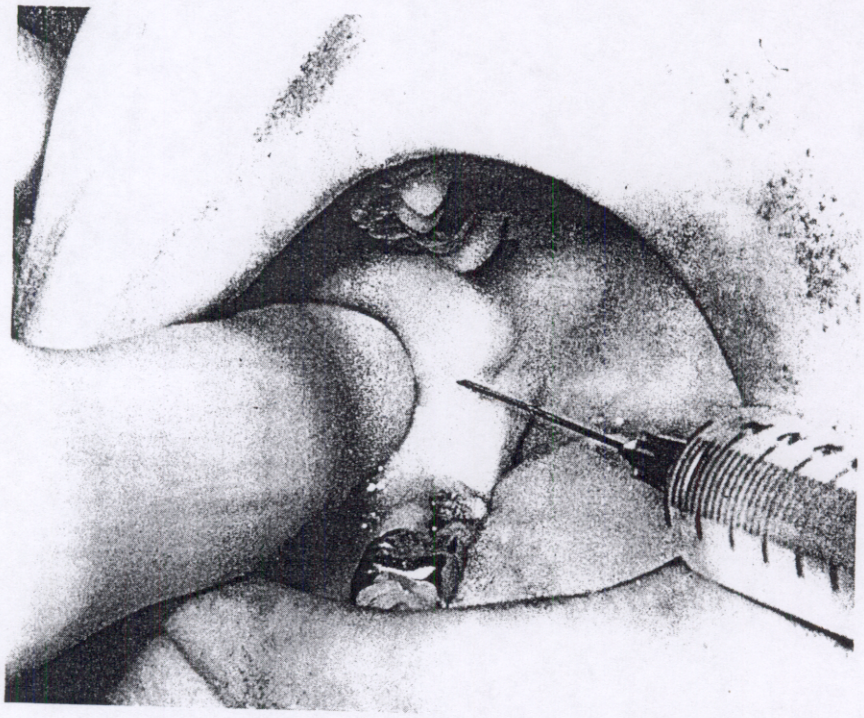


Figure 8. Needle and syringe are rotated in a horizontal plane to the opposite side of the mouth, maintaining contact with bone.

technique is advocated to avoid hematoma formation. Careful aspiration will avoid a direct intravascular injection.

Intercostal Nerve Block

The management of pain due to rib fracture is the usual emergency room indication for intercostal nerve blockade. One usually notes pain relief far exceeding the anticipated duration of action of the local anesthetic agent used and this usually is attributed to an interruption of the pain–spasm–pain cycle. Patient comfort, ventilation, and pulmonary toilet enjoy significant improvement with this block.

Ordinarily, the patient is seated straddling a chair, leaning slightly forward on the chairback, with the arm on the affected side raised above the head with the elbow flexed, allowing the forearm to rest on the head (Fig. 11). This allows maximal exposure of the rib angles to palpation, as well as rotation of the scapula superiorly as far as possible, exposing a maximal number of ribs. Even with this optimal positioning blocks higher than the fifth rib are not usually possible. The intercostal neurovascular bundles run in grooves deep to the inferior border of the rib and are oriented vein–artery–nerve, superiorly to inferiorly.

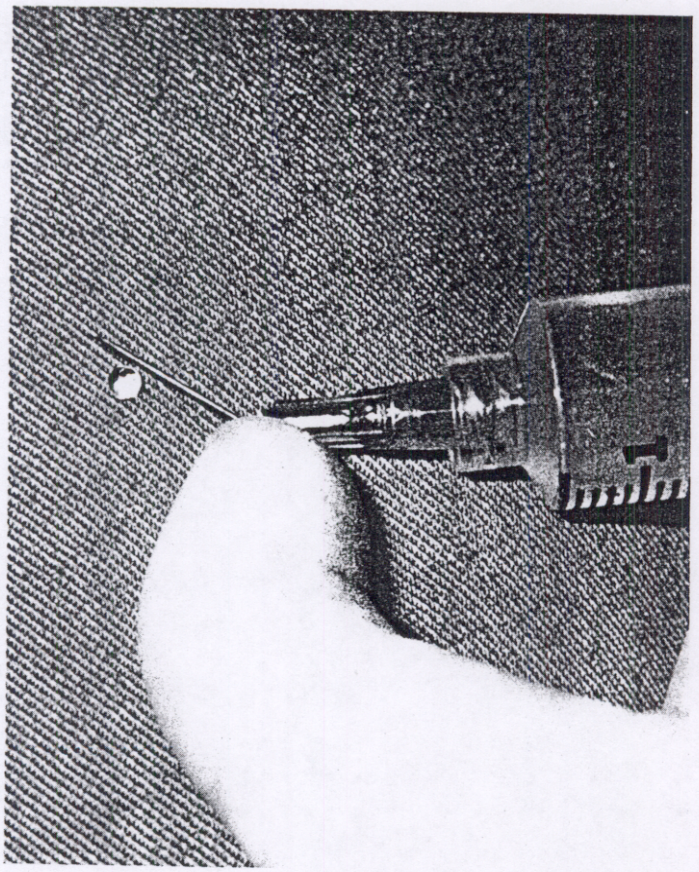


Figure 9.

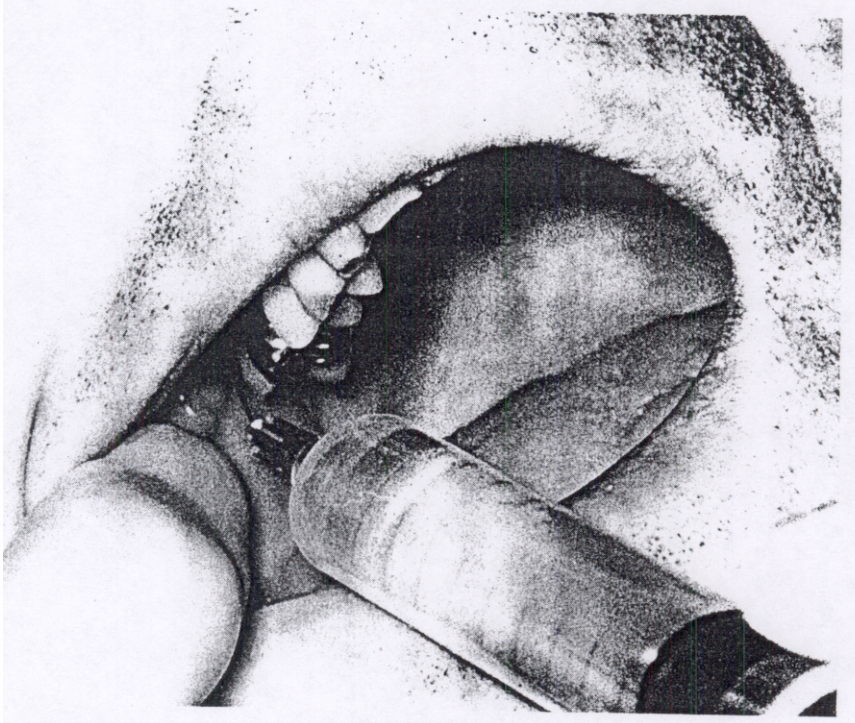


Figure 10.

The skin is prepared and skin wheals are raised at the inferior aspects of the angles of the ribs to be blocked with a 25-gauge needle. While the inferior border of the ribs is palpated, a 22-gauge 1½ inch needle is advanced to the rib and "walked" off the inferior aspect of the rib (Fig. 12). Often a popping sensation or loss of resistance is felt as the neurovascular space is entered. Aspiration for blood or air is followed by the injection of 3 to 5 ml of local anesthetic solution.

Often, several ribs may be blocked at a time. In this case, attention must be paid to the total dosage of local anesthetic agent used to avoid systemic toxicity. Pneumothorax is an uncommon complication of a carefully performed block but must be addressed with the patient prior to the procedure. If the patient is to be treated as an outpatient, I recommend an expiratory chest x-ray 1 hour after the block to rule out an occult pneumothorax.

Upper Extremity

a. Median Nerve Block at the Wrist: This block is useful for extensive repairs in the distribution of the median nerve in the hand, especially when coupled with radial or ulnar blocks. The median nerve enters the palm at the wrist through the carpal tunnel. At the proximal end of the tunnel the

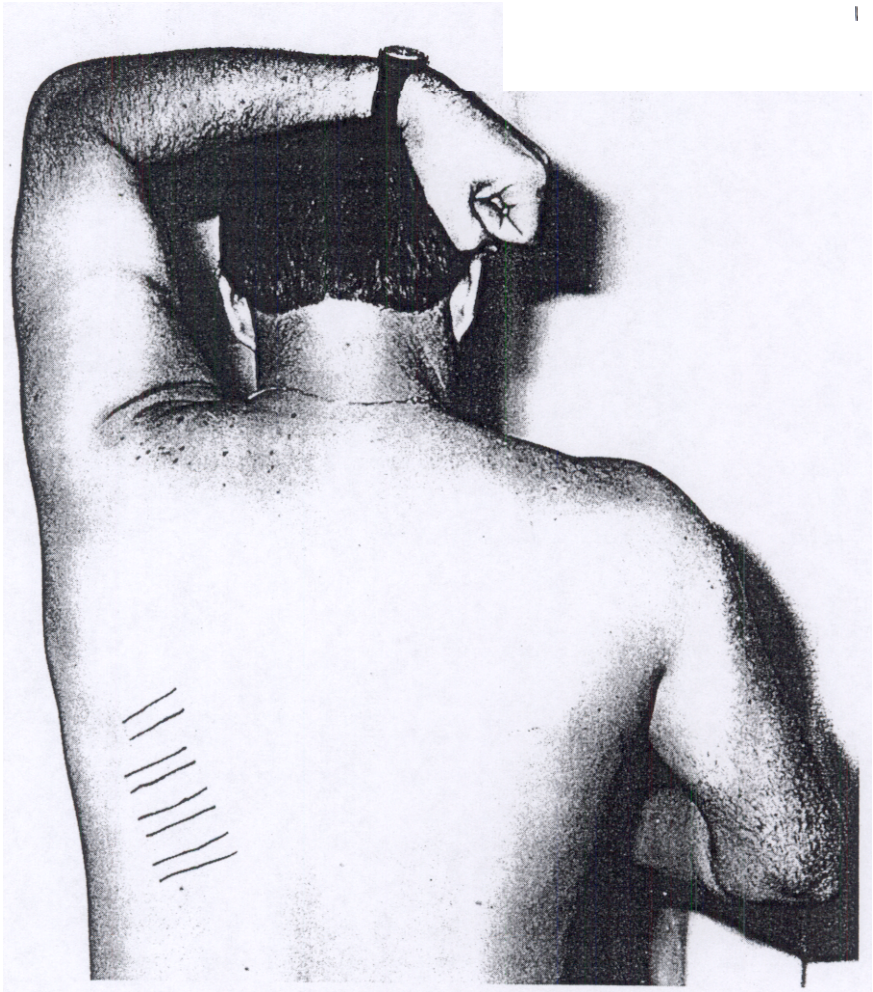


Figure 11. Position for intercostal nerve block.

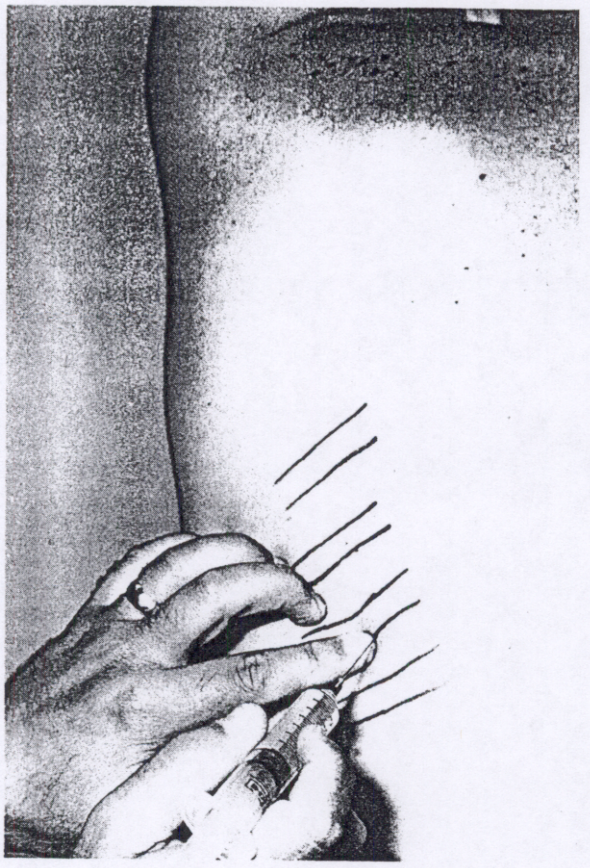


Figure 12. Intercostal nerve block.

location of the nerve is easily identified since it lies between flexor carpi radialis and palmaris longus tendons.

The skin is prepared and a skin wheal is raised with a E-gauge needle between these two tendons at the level of the proximal wrist crease (ulnar styloid). This should be just proximal to the proximal limit of the carpal tunnel. A 22-gauge 1% inch needle is advanced to a point just deep to palmaris longus tendon and 5 ml of solution injected slowly (Fig. 13).

Should paresthesias be elicited, the needle should be withdrawn slightly to avoid nerve fiber damage or intraneural injection. Carpal tunnel syndrome is a relative contraindiction, for obvious reasons.

II. Ulnar Nerve Block at the Wrist: The indications, as for the median nerve, are repairs in the distribution of this nerve. The ulnar nerve at the wrist lies between the flexor carpi ulnaris tendon and the ulnar artery.

A skin wheal is raised at the level of the proximal wrist crease (ulnar styloid) between the tendon and the artery. The 22-gauge needle is advanced to a depth of 1 cm and 5 ml of solution is injected after careful

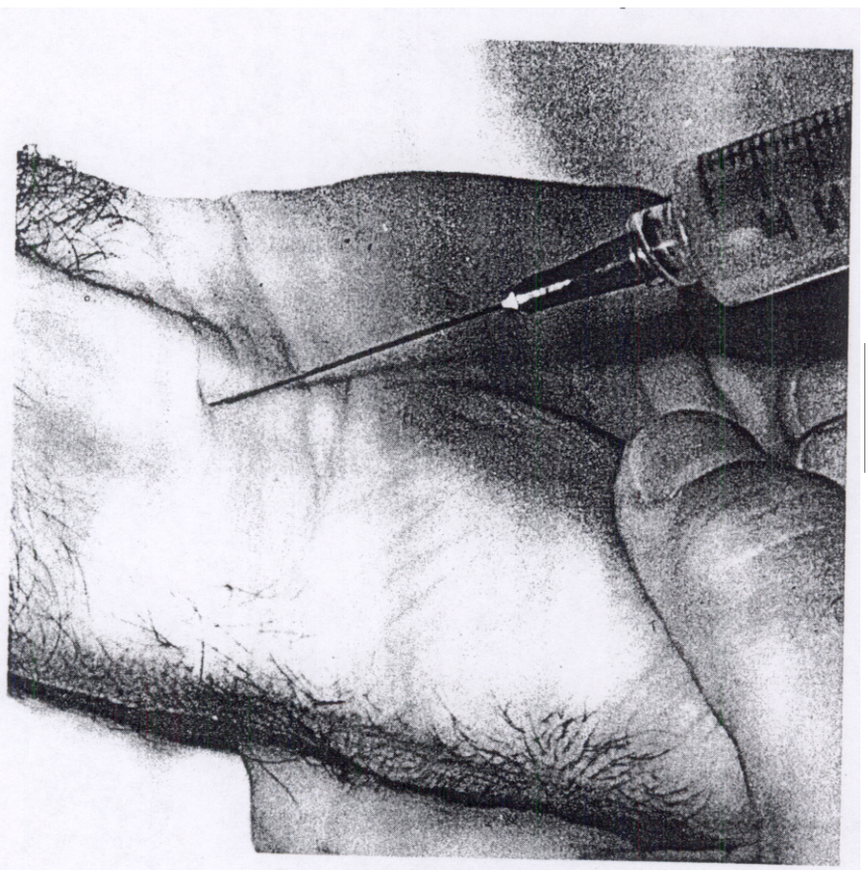


Figure 13. Median nerve block.

aspiration (Fig. 14).[3, 4] The dorsal cutaneous branch of the ulnar nerve arises approximately 5 cm proximal to the wrist and courses distally to supply the dorsum of the hand. It can be anesthetized by subcutaneous infiltration over the dorsal and ulnar aspect of the wrist at the level of the ulnar styloid (Fig. 15). A modified Allen's test may be used to verify collateral arterial supply to the hand but is not reliable. Epinephrine-containing local anesthetic solutions should be avoided due to the proximity to the ulnar artery.

c. Radial Nerve Block at the Wrist: The radial nerve subserves sensation to a limited and variable portion of the dorsum of the hand. These sensory branches emerge from beneath the tendon of brachioradialis and lie subcutaneously at the wrist.

The subcutaneous infiltration of local anesthetic agent over the dorsoradial aspects of the wrist will block the sensory branches of the radial nerve at this level (Fig. 16).

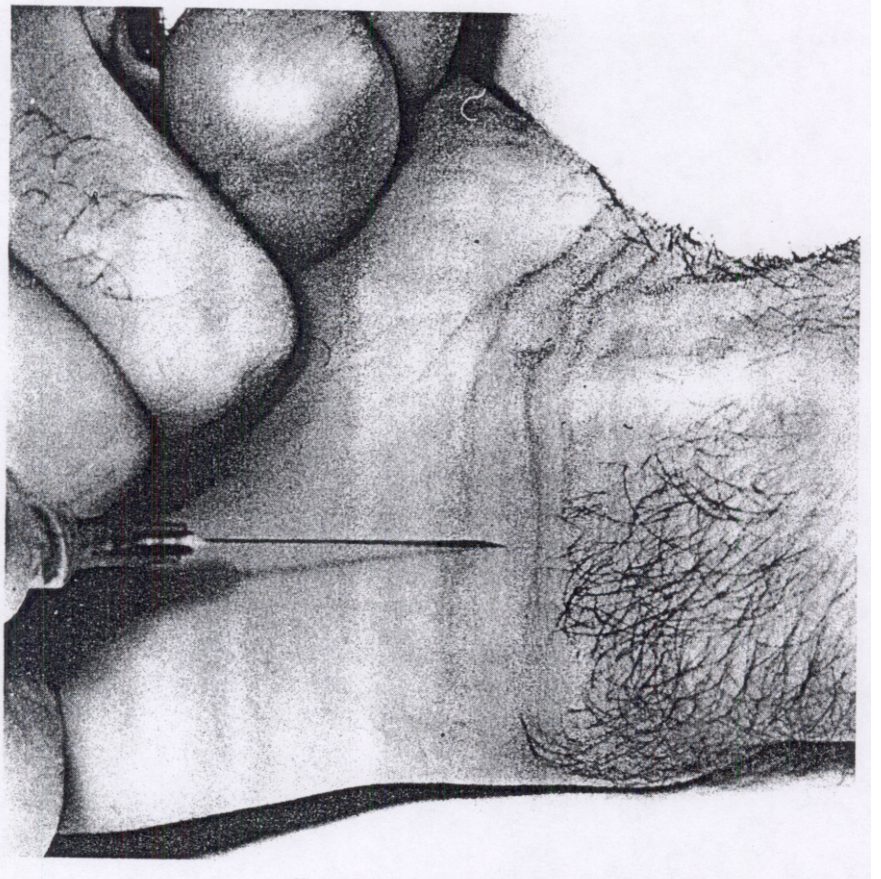


Figure 14. Ulnar nerve block.

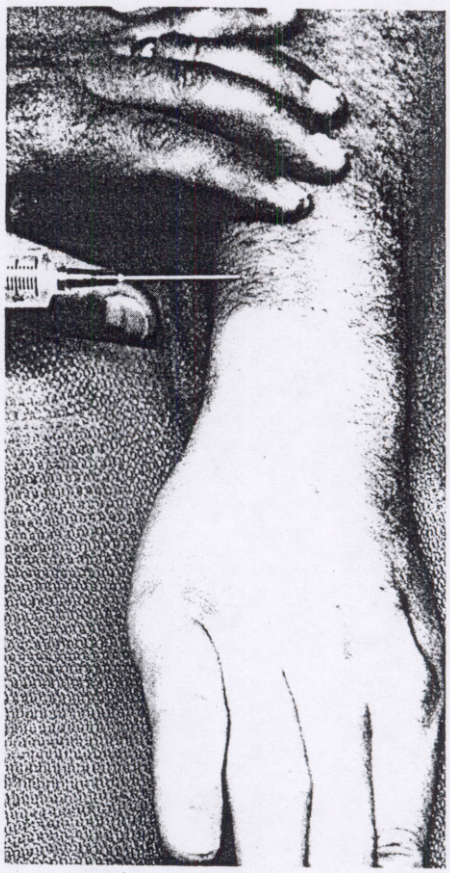


Figure 15. Dorsal cutaneous branch of the ulnar nerve block.

d. Metacarpal Block: This block is indicated for repairs and reductions involving any of the fingers. I consider metacarpal blockade preferable to digital block because of the vascular supply of the digits. At the level of the webspace the radial and ulnar digital arteries are "end" arteries. However, at the level of the metacarpal heads (distal palmar crease) the superficial and deep palmar arterial arches send independent contributions, anastomosing to form the digital arteries. A" injury to four arteries is less likely than to two, favoring metacarpal over digital blockade. However, not all authors agree. [1, 2]

Four nerves enter each linger: dorsal and palmar, radial and ulnar. The operator initially holds the patient's hand palm up slightly hyperextending the metacarpal joint of the digit to be blocked. At 25-gauge, ⅝ inch needle is inserted in the midline of the skin crease at the base of the finger to travel subcutaneously, parallelled to tho palm in the direction the digital nerve should take. The needle is advanced to the hilt, placing it at the level of the distal palmar crease. Aspiration is performed, and 3 ml of solution is deposited. The needle is withdrawn to skin (but not out) and

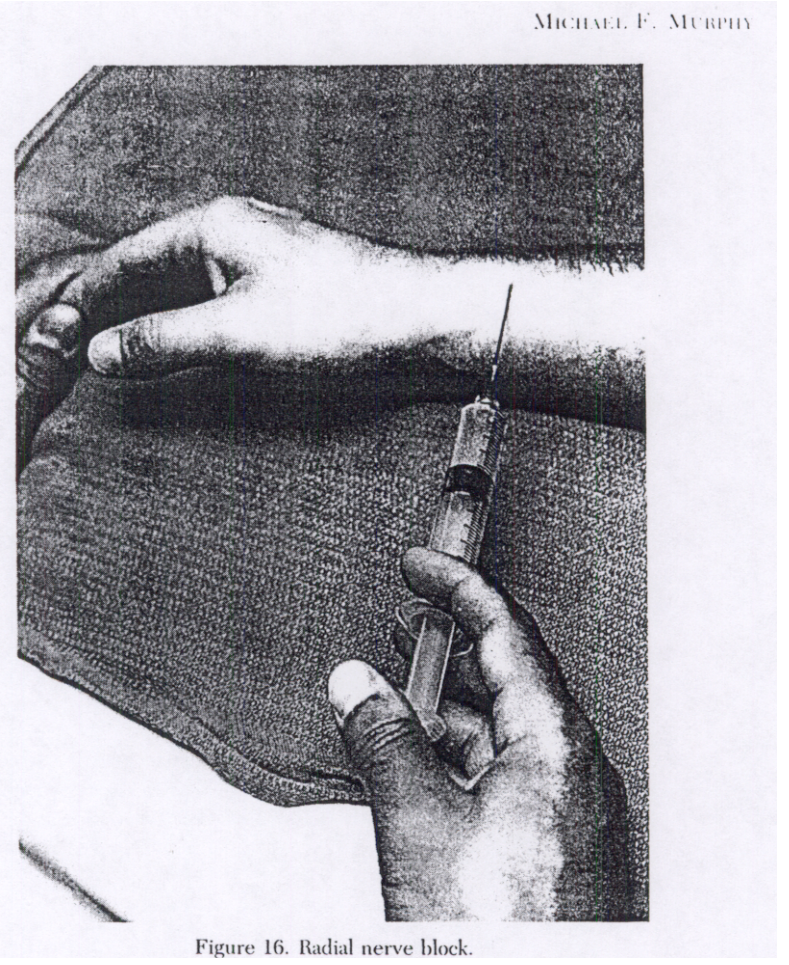


Figure 16. Radial nerve block.

redirected towards the opposite volar digital nerve in like fashion (Fig. 17). If the lesion to be repaired extends dorsally, the relevant digital nerves can be blocked in addition by the subcutaneous infiltration of solution over the metacarpal joint of the appropriate digit (Fig. 18). Local aoesthctic solutions containing epinephrine should not be used in blocks of end organs.

Lower Extremity

a. Anterior Ankle Block: This block is useful for operative procedures, repairs, and reductions involving the dorsum of the foot.

Three nerves supply sensation to the dorsum of the foot. The superficial peroneal nerve has several branches that course subcutaneously anterior to the lateral malleolus. The deep peroneal travels with the anterior tibial artery, between and deep to extensor hallucis longus and tibialis anterior tendons at the level of the malleoli. The saphenous nerve I uns subcutaneously with the long saphenous vein anterior to the medial malleolus. Owing

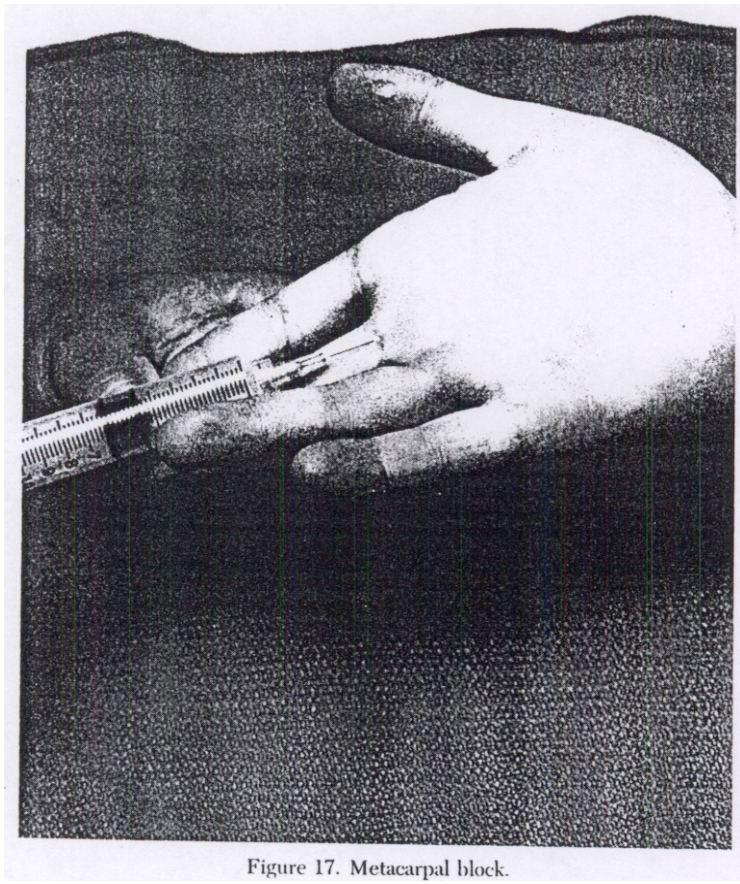


Figure 17. Metacarpal block.

to the unprcdictibility of the territories supplied by these three nerves, the anterior ankle block blocks all three.

Prepare the area of the anterior ankle with the patient lying supine and the ankle slightly plantar flexed. Identify the groove between the extensor hallucis longus and tibialis anterior tendons at a point parallel to the top of the medial malleolus, and raise a skin wheal (Fig. 19). Use a 10 ml syringe full of local anesthetic agent, and a 22-gauge 3½ inch spinal needle to enter the skin in a perpendicular direction. Advance to a point deep to the tendons and slowly inject 5 ml of agent. Withdraw the needle to, but not out of skin, and advance subcutaneously to the top of the medial malleolus (Fig. 20). Aspirate and deposit 5 ml of agent in this area. Reload

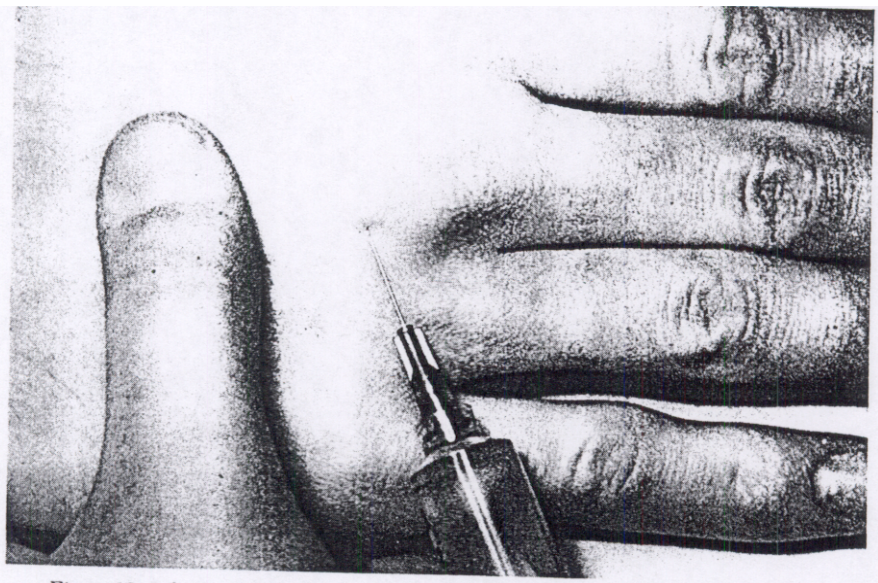


Figure 18. Subcutaneous infiltration of solution over the metacarpal joint of the appropriate digit.

the syringe with 10 ml of solution and inject 5 ml as the needle is withdrawn to the skin, but not completely out. Redirect the needle laterally and subcutaneously to the top of the lateral malleolus (Fig. 21). As the needle is withdrawn, deposit the remaining 5 ml of solution.

Collateral circulation to the foot by the posterior tibial artery should be noted before the block is performed.

b. Posterior Ankle Block: This block is particularly useful in operative procedures and repairs on the sole of the foot.

Two nerves supply the sole of the foot. The tibial nerve is a branch of the sciatic nerve and at the ankle runs behind the medial malleolus just posterior to the palpable posterior tibial artery. The sural nerve is subcutaneous in location, behind the lateral malleolus with the short saphenous vein.

This block requires the patient to lie prone with the foot extending beyond the end of the stretcher and held slightly dorsiflexed. Two skin wheals are elevated on either side of the Achilles tendon with a 25-gauge needle level with the top of the malleoli. On the medial side the operator palpates the posterior tibial artery and advances a 22-gauge 1½ inch needle towards the pulse. Five milliliters of solution is deposited here and an additional five milliliters as the needle is withdrawn (Fig. 22). The lateral side then is blocked, advancing the needle subcutaneously to the top of the lateral malleolus and injecting 5 ml as the needle is withdrawn.

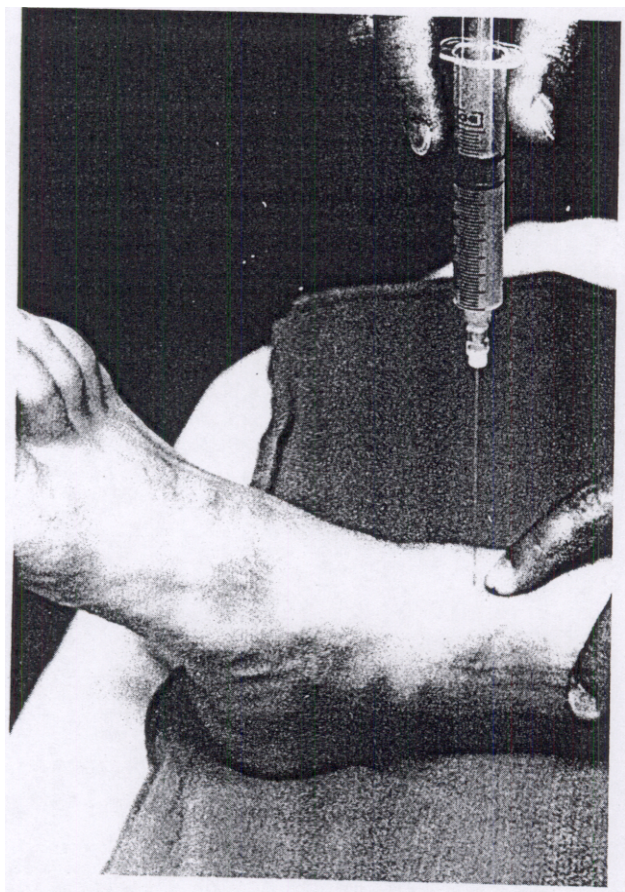


Figure 19. Anterior ankle block. Deep peroneal nerve.

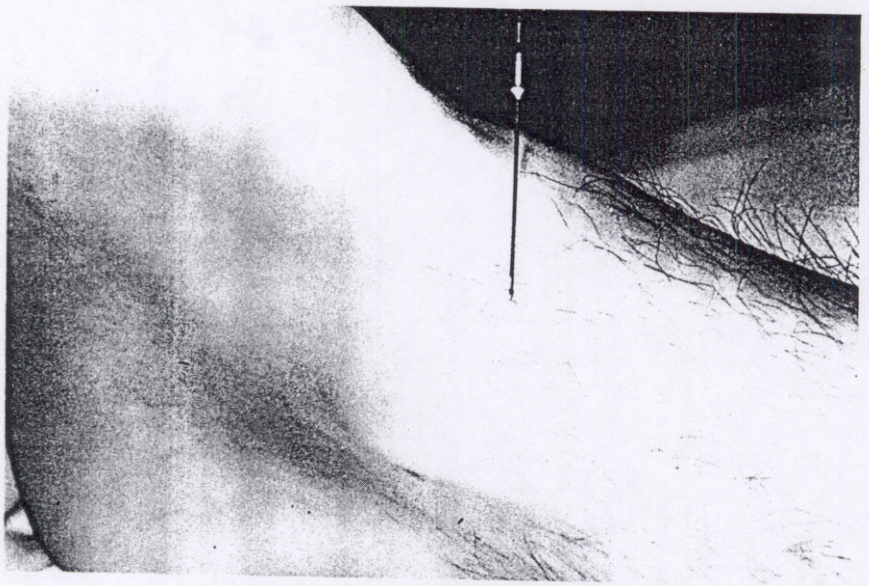
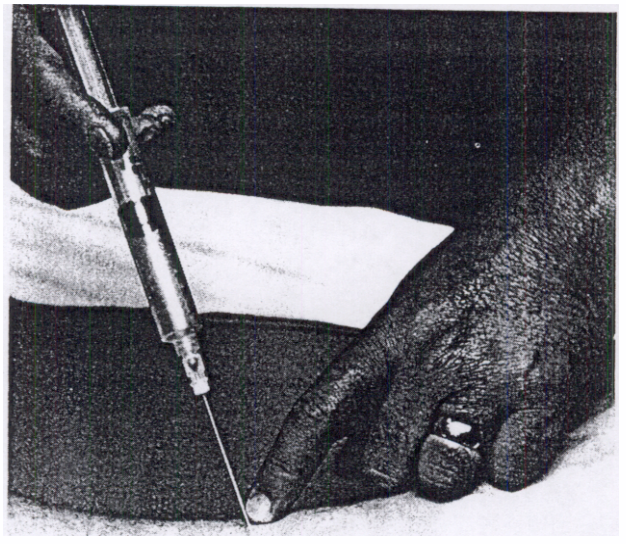


Figure 20. Anterior ankle block. Saphenous nerve.



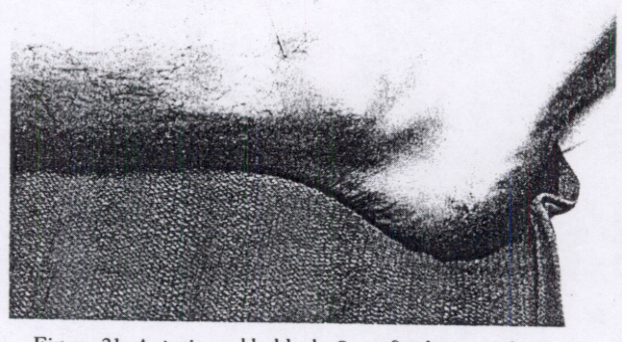


Figure 21. Anterior ankle block. Superficial peroneal nerve.

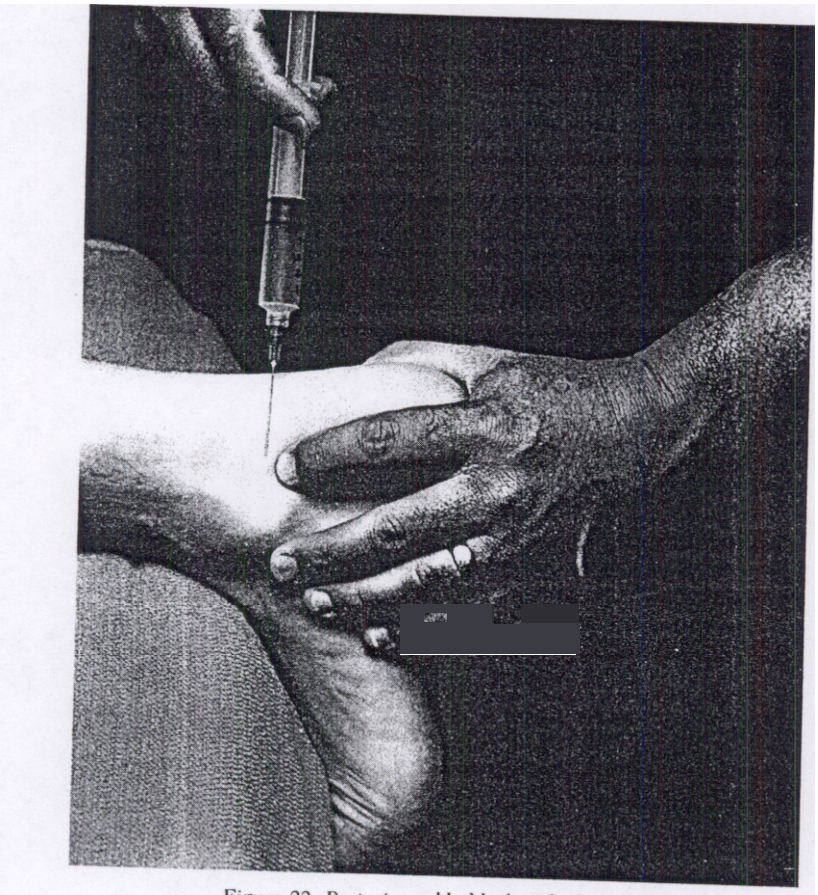


Figure 22. Posterior ankle block. Tibial nerve.

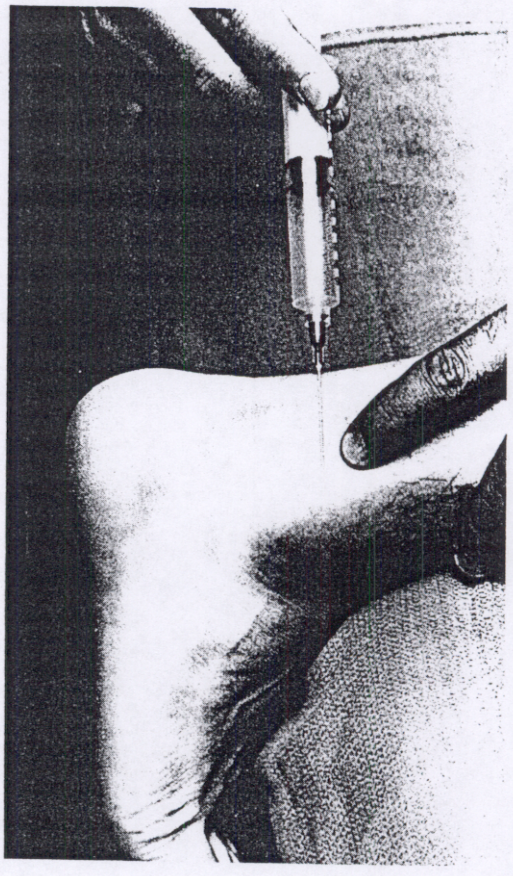


Figure 23. Posterior ankle block. Sural nerve.

Collateral circulation to the foot by the dorsalis pedis pulse should be noted prior to performing this block.

In the performance of any block the operator should always keep two points in mind:

1. Use sufficient quantities of local anesthetic, of sufficient concentration to allow a reasonable chance of successful blockade.
2. Wait sufficient time to allow blockade to occur. Agents such as bupivicaine tend to have a relatively slow onset time (10–30 min), whereas others such as lidocaine may be somewhat more rapid (5–15 min).

SUMMARY

Relatively few emergency physicians are aware of the spectrum of regional anesthesia and the advantages it has to offer in the day-to-day practice of the specialty. Understanding the types of block and the principles that apply to neural blockade are only a beginning in the appropriate use

of blockade techniques. A detailed knowledge of anatomy is essential to successful and safe practice; however, only repeated performance of the blocks will lead to predictable success!

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Department of Emergency Medicine
Victoria General Hospital
1278 Tower Road
Halifax, Nova Scotia
Canada B3H 2Y9