Regional/perfusion analgesia

WILLIAM G. RONK, CRNA
Cooperstown, New York

The author provides a general review of the application for regional/perfusion analgesia, along with a review of the local analgesic drugs' effects on the body. He also highlights the drugs of preference used at the hospital with which he is associated.

Regional/perfusion analgesia involves the injection of a local analgesic solution into the vein of a limb which has been made ischemic by a tourniquet. Such analgesia is most useful for operations on the arms but can be used in the leg. The following is a review of this method of analgesia, including a general review of local analgesic drug effects and methods of treatment.

At our hospital,* we prefer the use of the two analgesic drugs referred to in this article, namely: lignocaine (lidocaine, Xylocaine®) and bupivacaine (Marcaine®) as the most effective for perfusion analgesia.

The intravenous block form of analgesia is a very useful tool for the anesthetist in both elective and emergency procedures. Utilized with proper technique, this block affords good analgesia with a relatively bloodless operative field. The patient is awake, with control of all vital reflexes, and thus, is less in danger than would generally be true of a general anesthetic.

The technique can be employed for repair of lacerations, reduction of closed fractures (especially in children), and more electively, for release of trigger finger, ganglia, and nevi excisions.

General considerations with respect to regional analgesia¹

Local analgesics retard or stop the propagation of nerve impulses and have a stabilizing effect on the cell membranes of nerve-fibers. With the exception of cocaine (a vasoconstrictor) and lignocaine (having no effect on vessels), local analgesic drugs are vasodilators. Vasoconstricting drugs like adrenaline are added to prolong action and delay absorption of the local drug. Detoxification of local analgesic drugs occurs in the liver and the speed of destruction is a measure of the drug's toxicity. Therefore, liver disease may increase the toxicity. Some drugs are excreted by the kidneys, with serum cholinesterase having an action in the aromatic esters.

Nerve-fibers vary in their susceptibility to the local analgesic solution in an inverse ratio to the size of the cross-section of the fiber; that is, small fibers are more susceptible than larger fibers. The site of action of local analgesic drugs is the surface membrane of cells of excitable tissues. Local analgesics affect not only nerve-fibers but all types of excitable tissues, including smooth and

*The Mary Imogene Bassett Hospital in Cooperstown, New York, an affiliate of the Columbia University Medical Center in New York City.
striated muscle, probably by interfering with cation fluxes across the muscle cell membrane.

Local analgesic drugs are lipoid-soluble bases which act by penetrating lipoprotein cell membranes in the non-ionized state. The blocking quality of a local analgesic drug depends on its: (1) potency, (2) latency (time between injection and maximum effect), (3) duration of action, and (4) regression time (time between commencement and completion of pain appreciation).

Signs of toxicity are due to: (1) special sensitivity of the patient to the drug and (2) a high blood concentration of the drug from any cause.

The factors influencing toxicity are: (1) quantity of solution, (2) concentration of the drug, (3) presence or absence of adrenaline, (4) vascularity of the site of injection, (5) rate of absorption of the drug, (6) rate of destruction of the drug, (7) hypersensitivity of the patient, and (8) age, physical status, and weight of the patient. Toxic signs are not always related to the dosage of the drug used.

Specific untoward effects and respective treatments

Central nervous system: Convulsive-respiratory failure. Central stimulation is followed by depression, restlessness, hysterical behavior, vertigo, tremors, convulsions, and then respiratory failure. The treatment includes: (1) artificial ventilation with air or oxygen, and (2) intravenous injection of thiopental or succinylcholine to control convulsions. Intravenous diazepam (Valium®) may also be useful.

Cardiovascular system: Hypotension. Acute collapse—primary cardiac failure—is followed by feeble pulses with bradycardia, pallor, anxiety, sweating, and hypotension. This form is due to rapid absorption of the drug before it has time to reach the brain. The treatment is to elevate the legs, give oxygen, provide a rapid intravenous infusion to raise blood pressure, and invoke cardiac massage if required.

Respiratory depression. This may progress to apnea from medullary depression.

Allergic phenomena, though rare, may take the form of bronchospasm or urticaria. The treatment consists of an injection of adrenaline, administration of hydrocortisone, and oxygen therapy as needed.

Reactions to vasoconstrictor drugs may include pallor, anxiety, palpitations, tachycardia, hypertension, and tachypnea.

Premedication prior to block

Adequate premedication of patients is essential for the successful administration of a local analgesia. A barbiturate is useful to reduce anxiety and possible toxic effects from the drugs employed. Diazepam can be an excellent choice. Some anesthetists find that neurolept drugs (like Innovar® in small incremental doses) allay anxiety and apprehension, and afford additional analgesia via the fentanyl component. Premedication is relative to the individual patient and should be given accordingly.

Administration of an intravenous perfusion block

The following is a step by step primer of how to administer an IV perfusion block.

Step 1. Prepare an analgesic solution of either .25% bupivicaine or .25% lignocaine. Use .5% of either solution; mix 20 cc of normal saline with 20 cc of the drug, thus preparing a total of 40 cc of .25% analgesic solution.

Step 2. Explain fully to your patient the procedure and assure him or her that the only sensations felt will be those of warmth and tingling. No pain will be felt; and only occasionally, the sensation of pressure will occur.

Step 3. Apply preferably a double-cuff tourniquet, being sure that adequate Webril cotton is applied first under the tourniquet.

Step 4. Insert either a butterfly (19, 21, 23 gauge) or an angiocath (18,
20, 23 gauge) into the vein on the dorsum of the hand or foot. Secure the end to the skin.

Step 5. Elevate the limb to ensure adequate blood drainage and then apply an Esmarch bandage up to and including the cuff. Inflate the cuff that is uppermost to occlude arterial blood from entering the limb. An efficient arterial tourniquet does not completely isolate the limb because of collateral circulation through bones. Now, remove the Esmarch bandage, being cautious not to dislodge your intravenous needle or catheter.

Step 6. Inject 40 cc of analgesic solution slowly for the arm. The amount of solution for the leg may vary from 40 cc to 60 cc, dependent upon its size.

Note the onset of the patient’s sensations of warmth, tingling, and muscle paralysis. Your block is now effective and the procedure can now begin. Occasionally, the patient will experience pain in the shoulder or at the cuff site. This is readily relieved by inflating the second cuff over the anesthesized area and then deflating the first cuff. Caution in this step is required to prevent the flowing of the agent into the main stream of blood circulation, thus losing all analgesia.

At the completion of the surgical procedure, the cuff is deflated; and the sensations and muscle tone return in a few minutes. Toxic effects are not a problem but their possibility should always be borne in mind. Signs of toxicity on releasing of the tourniquet include: drowsiness, bradycardia, hypotension, and EKG abnormalities.

Pharmacology of lignocaine and bupivacaine

Lignocaine (Xylocaine or lidocaine) is a basic amide, synthesized by Lofgren and Ludquist in 1943 in Sweden. Its effects come on quicker and last longer than those of procaine. It seems to spread over a wider field than equal volumes of other analgesic drugs.

The metabolism of lignocaine can give rise to the formation of methemoglobin, with cyanosis being rare. It is not a vasodilator nor does it interfere with the vasoconstricting action of adrenaline. It has a cerebral effect, causing drowsiness and amnesia. It is metabolized by the microsomes in the liver and is excreted renally.

Bupivacaine (Marcaine) is an amide-linked drug, synthesized by Ekstram and his colleagues in 1957. It is reputed to be four times as potent as lignocaine, so that roughly .5% solution is equal to 2.0% lignocaine. It causes sensory blockage, more efficiently than motor blockage. The margin of safety with bupivacaine appears to be wider than with lignocaine.

Duration of effect is between 5 and 16 hours, the longest of any local analgesic known. This may be related more to its binding to nerve tissue rather than to its overall retention in the body. Adrenaline does not greatly prolong its effect.

REFERENCES

Author
William G. Ronk, CRNA, attended Dutchess Community College, Poughkeepsie, New York. He is a graduate of Hudson River State Hospital School of Nursing in Poughkeepsie, New York and the United Hospitals of Newark School of Nurse Anesthesia, Newark, New Jersey. Since September, 1975, he has been a staff anesthetist at Mary Imogene Bassett Hospital, Cooperstown, New York, affiliated with Columbia University of New York.