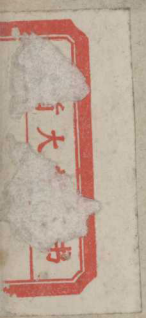
F. L. Jenkner

Peripheral Nerve Block

Pharmacologic - By Local Anesthesia
Electric - By Transdermal Stimulation



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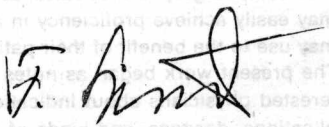
Introduction

Fifty years ago surgeons often performed various operations in conduction anesthesia or local anesthesia in the hope of circumventing pulmonary complications. The preparation for the operation was the task of an assistant who had to know and carry out the diverse local anesthetic procedures and who was responsible for their effectiveness. In this way a large number of physicians learned to carry out nerve blocks, which they also applied more and more outside their operative duties.

The introduction of modern general anesthesia led almost to the disappearance of the special techniques of nerve blocking at the former "classic" places of their teaching. Therefore it is a great merit of my former associate F. L. Jenkner to recall the art of nerve blocking, be it for diagnostic or therapeutic purposes. He presents the many possibilities as an addition to the therapeutic armamentarium and explains their indications, bases, and techniques.

I do not think that one needs to be a specialist to carry out these useful techniques; but one has to have an understanding of certain topographic-anatomic situations, and one needs to know the rules of the procedure to obviate the risks that are always inherent in any disruption of the integrity of the integument of the human body. Thus by presenting all the mentioned aspects a renaissance of good though old practices may be initiated in the light of new achievements.



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Preface

An understanding of the perception of pain impulses and of their conduction and processing to the sensation of pain provides the basis for what is generally called treatment of painful conditions. The most rational method of this therapy is to remove the causes of pain, i. e., of the noxious agents or conditions leading to pain. This, however, is not always possible. Therefore methods of interruption of pain conduction (so-called conduction anesthesia or nerve blocking) are gaining importance. Destructive surgical approaches for lasting interruption of conduction pathways or dorsal column stimulation by implanted electrodes should be considered only as a last resort. A third approach is the modification of personal engagement in pain perception, which may be achieved by either psychotherapy, psychopharmacological agents, or psychosurgery. The method most commonly applied for relieving pain is the use of analgesics that raise the threshold of pain.

Regional anesthesia came into being in 1848 when Koller demonstrated the effect of cocaine to ophthalmologists. Infiltration then became possible by the invention of the injection needle by Alexander Wood (1853). Difficult problems of general or regional measures for the treatment of pain should be handled by anesthetists. However, conditions of pain that allow one to obtain a painless state by nerve blocking are so frequent that other physicians are frequently confronted by them. Specialists in internal medicine, surgery, orthopedics, as well as general practitioners – with some dexterity – may easily achieve proficiency in a number of simple blocking procedures, which they may use to the benefit of their patients.

The present work began as notes to a lecture series. It is hoped that it will inform interested physicians about indications, techniques, evaluation of effects, possible complications, dosages, and kinds of local anesthetics as well as the duration of some of the more frequently used nerve blocks by means of sketches and concise discussions. This material is presented in such a way as to allow the reader to obtain a maximum of information in a minimum of time. The excellent responses, mainly from general practitioners, prompted the presentation of this monograph in the English language, as suggested by Springer-Verlag New York. In this edition a small chapter on transdermal stimulation has been added, because this most modern method promises to become of great importance in the relief of pain.

F. L. Jenkner

Vienna, 1977

Contents

General Section

Application of Nerve Blocks

In spite of the great strides made by general anesthesia to date, conduction anesthesia has gained increasing importance in temporary relief of pain itself as well as in enabling surgeons to carry out some minor procedures that must be done immediately or are performed more easily this way (e. g., in cases of dislocated shoulder). If blocking a nerve is accompanied by positive effects on other organs, thereby improving the condition of a patient (e. g., spreading of solution to stellate ganglion in brachial plexus block and thereby possibly improving an existing hypoxic cardiac state), it is preferred to a general anesthetic. If metabolic disorders are present (such as diabetes), routine intubation anesthesia carries a certain risk. Such risks may not exist if the (simple) required procedure is carried out under a nerve block. It should, however, be recalled that nerve blocking should not be performed in children under about 10 to 12 years of age. Also neurasthenics, neurotics, and depressive patients (these latter presenting possible symptoms of organic diseases as signs of their psychic disorder) do not allow one to expect good results from therapeutic blocks. The majority of pathologic states that may be ameliorated by nerve blocking are presented together with the respective blocking procedure. Some indications are not generally accepted but are included because of the experience of the author. One basic principle is imperative and generally accepted: Only the smallest amount of anesthetic in a solution of the lowest possible concentration of which a certain effect is to be expected should be used.

Premedication

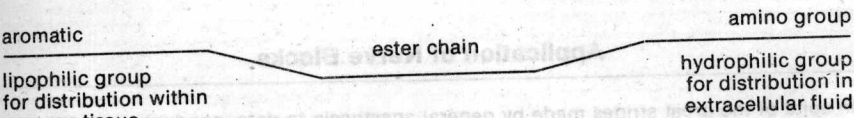
Some practitioners always use premedication. This author, however, is of the opinion that normal adults do not require any premedication for diagnostic, prognostic, or therapeutic blocking. All drugs used for premedication (such as nembutal, dilantin, lytic cocktail, or the like) are apt to narrow the sensorium of a patient to such an extent that they prevent an exact verbalization of paresthesias or tingling sensations by the patient, thereby masking the immediate effect of a blockade. Only with nerve blocking for surgical procedures is premedication (including atropine) advised. Even here, patients in shock do not require premedication. If children have to be blocked, they should be premedicated. Geriatric patients may be given small doses of a mild neuroleptic, but in general they also do not require premedication at all. A so-called light premedication is not at all recommended. Instead, it should be stated that careful psychologic handling of the patient and cultivation of a good patient-doctor relationship always renders premedication unnecessary.

Mode of Action of Local Anesthetics

Understanding the action of local anesthetics as means for interruption of nerve conduction needs only a brief and very fragmentary knowledge of physiology and pharmacology. Those more interested in the problem are referred to the extensive literature on the subject. Here, only the essentials will be sketched briefly.

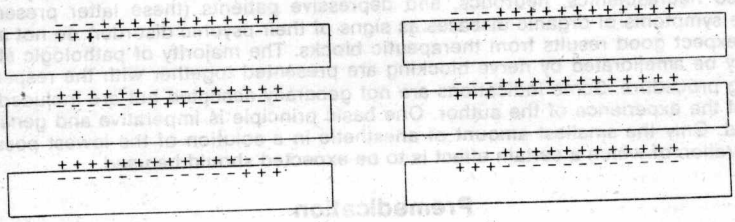
The subjective sensation of pain is possible only with a conscious subject and with intact conduction pathways from the (adequate) stimulus in the periphery to the central organ, i. e., the brain. Every interruption of these pathways, wherever it occurs, prevents the sensation of pain. The effect of local anesthetics consists in a temporary interruption of these pathways. The time limit of the effect gives the duration of the effect, whereas the interval from application (= injection) to the beginning of the effect constitutes the time of onset (or latency).

All local anesthetics are available as aqueous solutions of their salts. They must be carried to the nerves by extracellular fluids and therefore they must have a hydrophilic group. On the other hand, they need to be fat soluble to enter a nerve, which means that they must also have a lipophilic group in their structure. Consequently, local anesthetics have in principle the following chemical structure:



From the salts, tissue alkali liberates the free base which enters the nerve cell and stabilizes its membrane to potassium loss; the nerve remains in a state of rest, i. e., polarized. Depolarization, a characteristic feature of nerve conduction, may not occur. Sodium chloride and carbon dioxide are the two other substances split off the salt by tissue alkali. The more acid a salt is, the faster the base is liberated. Lengthening the ester chain lowers basicity, lowers solubility, and increases toxicity and relative potency.

Schematic of membrane potentials



In conduction

In interruption of conduction

Onset and duration of the interruption of conduction depends on the time required for the local anesthetic to penetrate all fibers of a given nerve. Small fibers will be interrupted sooner than larger ones. Therefore nerves conducting vasospastic impulses will be interrupted almost immediately, followed by those conducting pain impulses. Then sensory and last motor fibers are interrupted. It may be mentioned that this sequence of events is just the reverse of the sequence in the event of pressure on a nerve: Here motor fibers suffer first, followed by sensation, pain, and vasomotricity.
To influence pain, the blocking of a nerve must be carried out between the stimulus producing pain and the central site of processing to the conscious sensation of pain. This means that, in the case of projected pain, the site of stimulation (e. g., an intervertebral foramen) must be found; only if the block is carried out central to this location will the pain disappear. Any procedure at a place peripheral to the stimulus is absolutely ineffective.

Choosing the Anesthetic

There is available a whole series of local anesthetics, a small selection of which is given in the table on page 6. However large this number seems, one may safely limit the application of these to only two or three out of the selection of four given below. These are described with all the details necessary for their proper use. Properties required for optimal effects are fast onset, great depth, good penetration, long duration of action, total reversibility, and no tissue toxicity. There should also exist good chemical stability. All these properties usually are compared to procaine, which is given the therapeutic potency and toxicity of 1 simply because from 1907 to 1947 no other local anesthetic could compare with it. This historic reason is the only one for citing it here, because today, procaine should never be used for nerve blocking.

Procaine hydrochloride (Novocaine) was synthesized in 1907 and was the leading local anesthetic up to 1947; the maximum single dose without adrenaline is 500 mg (with

adrenaline it is 1000 mg). Duration of action is 45 min, beginning 5 to 10 min after injection. Partially because of this short action, but more because it is an ester and immediately upon injection is split by esterases (which are always present in blood and tissues) and one of the split products is *para*-aminobenzoic acid, an allergene, it is not wise to use it for nerve blocking.

Lidocaine hydrochloride (Xylocaine, Lignocaine) was synthesized in 1943 by Löfgren. The pH of a 2 percent solution is 6.9. Lidocaine diffuses into tissue four times better than procaine. Its toxicity is 2, its potency is 4; its cumulative toxicity is 6 mg/kp of body weight. The maximum single dose without adrenaline is 200 mg, with adrenaline it is 400 mg. A 0.8 percent solution suffices for synaptic blocking of sympathetic fibers; for analgesia a 1 percent solution and for muscular relaxation a 2 percent solution is required. Duration of action without adrenaline: 2 percent, 60 min; 1 percent, 50 min; with adrenaline: 2 percent, 135 min; 1 percent, 80 min; latency 2 min.

Mepivacaine hydrochloride (Carbocaine) was synthesized 1953 by Ekenstam. The pH of a 2 percent solution is 6.8 to 6.9. Toxicity without adrenaline is 1.5 to 2; with adrenaline it is 0.5. Its cumulative toxicity is 8 mg/kp of body weight. Its potency is 4. The maximum single dose without (with) adrenaline is 300 (500) mg. Duration of action without (with) adrenaline at 2 percent is 100 (135) min, at 1 percent is 70 (90) min. It has greater affinity to nervous tissue than lidocaine. It has no vasodilatatory property and therefore is resorbed much more slowly; it is to be preferred where adrenaline is contraindicated, as in geriatric patients. Its latency is somewhat shorter than lidocaine (37). Duration of action without adrenaline is 40 percent longer than lidocaine, with adrenaline it equals that of lidocaine.

Prilocaine hydrochloride (Xylonest) was synthesized in 1960 by Wielding. The pH of a 2 percent solution is 4.6. Its toxicitiy is 1.5 to 4, its potency is 4. The maximum single dose without adrenaline is 400 mg; with adrenaline it is 600 mg. Cumulative toxicity is 7 mg/kp of body weight, but may lead to methemoglobinemia. For muscular relaxation a 2 percent solution is required. Duration and latency is as for lidocaine. It has less vasodilatatory effect than lidocaine, in this respect more resembling mepivacine. The addition of adrenaline only barely prolongs the action. It is contraindicated in cases of insufficient O_2 levels in the blood.

Bupivacaine hydrochloride (Marcaine) was synthesized in 1947 by Ekenstam. The pH of a 0.5 percent solution is 6.3 to 6.6. Its toxicity is 8, its potency is 16. It is less cumulating than lidocaine or mepivacaine. The maximum single dose without (with) adrenaline is 150 mg (2.5 mg/kg body weight within 3 hr). Analgesia is obtained with a 0.25 percent solution, muscular relaxation with a 0.5 percent solution. Duration of action without (with) adrenaline at 0.5 percent is 300 (400) min; at 0.25 percent it is 200 (300) min. With adrenaline, the 0.5 percent solution may have a duration of up to 1500 min and longer, if the area injected is not very vascular. On the average, duration is three times longer and twice as deep as with mepivacaine. This substance represents a true improvement for anesthetists looking for an extremely long acting local anesthetic.

Note: Data on kind and quantity of local anesthetic to be used for a certain blocking procedure should be regarded under the criteria of the substances just given. It is not possible to mention all drugs with every nerve block. The physician carrying out the procedure will have to make his selection accordingly. One should, however, recall that solutions of higher concentration are resorbed faster than equal amounts of a drug as a weaker solution. Therefore, the single maximum doses given are for average concentrations; in lower concentrations, they may be up to 25 percent higher, in the highest concentrations up to 20 percent less. When using various concentrations this should be remembered. If a block is used for a diagnostic procedure, a very short-acting and fast-acting drug is preferable. For therapeutic blocks, long-acting local anesthetics should be preferred.
Local anesthetics should be used in the form of ampoules only on which name and batch number are imprinted. This is to assure errors from, for example, injecting al-

Comparison of Several Local Anesthetics

Generic name	Proprietary name	Relative analgesic potency	Relative toxicity	Single maximum dose in mg (adult, 70 kp) without adrenaline	Single maximum dose in mg (adult, 70 kp) with adrenaline	Type	Time for onset in min	duration with adrenaline	duration without adrenaline	Vasodilatation
Procaine	Novocaine	1	1	500	1.000	E	5–10	–45	60	Yes
Tetracaine	Pantocaine	10–15	10	20	100	E	10	60	–90	Yes
Lidocaine	Xylocaine	4	2	200	500	A	–2	60	–180	Yes
Prilocaine	Xylonest	4	1,5	400	600	A	–2	60	–120	Some
Mepivacaine	Carbocaine	4	2	300	500	A	1	100	–180	No
Bupivacaine	Marcaine	10	4	150		A	2–5	300	–900	No
Butanilicaine	Hostacaine	4	2	*)	*)	A	–2	60	–150	Yes
Tolycaine	Baycaine	*)	*)	250	600	E+A	2–5	60	–90	Yes

E = ester: In solution of limited stability, procaine has limited capacity for penetration. It is split by (most probably) pseudocholinesterase in *p*-aminobenzoic acid, which acts as an allergene and may cause sensitization. The latter causes allergic side effects, which are known for this type of local anesthetic.
A = amide type: Allergic reactions are unknown for this type of local anesthetic. They are not split in tissue nor are they metabolized in the liver. Single maximum dose is given for normal pharmacokinesis and excretory conditions. Therefore, in case of severe renal insufficiency and liver damage these drugs should not be given in full single maximum dose.
*) No data.

cohol instead of local anesthetic. This does not apply only to use in an office procedure. Today all needles and syringes should be used only once and then discarded. In this way sharpness of needles and cleanliness and sterility are assured and the possibility of provoking hepatitis is obviated. Unfortunately, for some blocks longer needles than those available in disposable packs are required. 3.6 and 8 cm needles do not cause a problem. It is at 10, 12, and 15 cm that it is still necessary to clean and resterilize needles. For practical purposes it is advised that after sterilization each needle be put in a separate vial with the point in a cotton pellet at the bottom of the vial. The sterile vial is then closed by a stopper. After use, the unsterile needle is inserted into the vial the other way around to distinguish clearly sterile from used needles.

Additions to Local Anesthetics

With the exception of mepivacaine and bupivacaine, all local anesthetic substances have a more or less vasoconstricting property. Therefore the action is not so very long and lends itself to prolongation by various additions. This has been done for a long time.

In general, vasoconstricting agents are used for the purpose. We prefer not to use vasoconstricting agents not only because adrenaline is apt to cause undue side effects, but also because such agents may affect a patient's blood pressure in an adverse manner. This statement is stressed in spite of the fact that relevant ready mixtures are available commercially. If one desires to use these mixtures, as ear, nose and throat specialists usually do, an optimal concentration of adrenaline is 1:200,000 in the solution; higher concentrations do not increase vasoconstriction or prolong time of action, but only increase undesirable side effects. Where adrenaline causes sensitivity reactions, *n*-adrenaline may be preferred, which almost never has such actions.

Other possibilities for prolonging the action of a local anesthetic twice to fivefold is to admix an equal amount of "periston N," a substance with very high molecular weight, to the solution with higest concentration (lidocaine 2 percent, bupivacaine 0.5 percent). This mixture, however, is not available commercially and should be prepared just prior to injection. Its components are available commercially, and this combination has found relatively wide application. Prolongation of action to several weeks or longer is obtained by admixing ammonia sulfate (20 percent solution) with equal parts of a local anesthetic in high concentration. This most effective mixture then contains half the concentration of the local anesthetic and 10 percent ammonia sulfate. It is said to have a duration of action of up to 4 to 6 weeks. Others, however, have been unable to duplicate this observation. Side effects, as seen with phenol or 96 percent alcohol, in the form of necroses have not been reported with the ammonia sulfate mixture. However, because the 20% solution of ammonia sulfate has to be prepared magistraliter, the mixture has not gained wide acceptance.

Pain Conduction and Pain Projection

The projection of the subjective sensation of pain originating in an organ, is quite specific for that organ and is directed to a specific area of the body: One speaks of pain projection. Knowledge of this projection is certainly reflected in the experience of older physicians, and is frequently spoken of as "clinical vision." However, knowledge about the correlation of somato-segmentary innervation of organs as well as autonomic (sympathetic) correlation of segmental nature to these organs or vessels is most essential for the achievement of therapeutic effects via blocking the respective somatic or sympathetic nerves.

There follows a tabular survey correlating some of the more important organs and their somatic and sympathetic segmentary innervations. This table is not intended to be complete; but it represents an attempt to provide assistance to the less experienced with hints to those segments the blocking of which may give good results in relieving pain or other conditions amenable by nerve blocks.

Naturally, projection of pain is a much more complicated process than can be indicated here. In addition to the primary region where pain originates, the specific area to which pain is projected from the primary area is just as important for the patient and just as distressing or characteristic, as these characteristics of pain projection are an important indication for the experienced pointing to the painful organ or area of body. The table presented, however, should not seduce one to simply relieve all pain in a certain region by nerve blocking and forget about the basic process underlying the pain. It should rather help to bridge the time gap between finding the reasons for pain and the beginning of rational therapy by definite measures when these are possible. Or it should help to alleviate pain by multiple blocks of nerves in those unfortunate patients suffering from malignancies that are no longer operable or that are no longer amenable to treatment. This last use is especially common in some countries and is gaining reputation in others. In these cases nerve blocking combines relief of pain of sometimes long duration with preservation of mental power and undisturbed sensorium (quite contrary to the action of narcotics, which are being used much too frequently and freely). Blocks may be repeated several times, and in these cases should be given interchangeably as somatic and sympathetic blocks of the respective segments. This is because not all pain is mediated via somatic nerves but vascular pain impulses sometimes originate in vascular spasticity and the latter is positively influenced by sympathetic blocking. These alternative blockades have gained much favor in treatment of pain of malignancy.

In giving segmentary correlation of various organs, it should be noted that various authors have divergent opinions, especially concerning autonomic (sympathetic) fibers. The range presented here coincides with the segments agreed upon by most authors. If one disregards the extreme segments, given in brackets, and adheres to blocking the median of the given segments, positive results of the interruption of pathways may certainly be expected. If the epidural segmental approach is used, the spread of solution should also be calculated to give interruption in additional segments next to the one injected.

Survey of Pain Projection

Organ	Pain projection	Pain conduction via segmental nerves	Origin of preganglionic sympatic fibers
Head and neck			
Meninges	Scalp	Nucl. sens. N. V.* IX, X u. XII	Th 1–Th 2 (3)
Eye	Eye socket and forehead	Nucl. sens. N. V. (first branch)	Th 1– Th 3 (4)
Tear glands	Eye socket	Nucl. tract. solit. N. VII u. IX	Th 1, Th 2
Parotid gland	Parotid region	Nucl. tract. solit. N. V via N. VII + IX	Th 1, Th 2
Salivatory glands	Submandibular region	Nucl. tract. solit., N. ling. via N. VII and geniculate ganglion	Th 1, Th 2
Thyroid gland	Ventral part of neck	C 2–C 4 Th 1, Th 2	Th 1, Th 2
Larynx	Throat and ventral part of neck	N. laryng. sup. ganglion	Th 2–Th 7
Thorax			
Trachea, bronchi	About the sternum	Th2–Th 7	Th 2–Th 7
Lung parenchyma	Insensitive for pain	Insensitive for pain	Th 2–Th 7
Parietal pleura in area of: Shoulder	Shoulder	C 3–C 5	
Supraclavicular	Supraclavicular (brachial plexus)	C 8–Th 1	
Intercostal	Intercostal nerves	Th 1uTh 12	
Heart	Precordium and left (right) arm	Th 1–Th 4 (5)	Th 1–Th 4 (5)
Thoracic aorta	Upper half of thorax and neck	Th 1–Th 5 (6)	Th 1uTh 5
Abdominal aorta	Lower half of thorax and abdomen	Th 6uTh 12	Th 6–L 2
Esophagus Upper half Lower half	Mid-sternum	Th 5–Th 8	Th 2–Th 5 Th 5–Th 8

via stellate ganglion

* Roman numerals refer to cranial nerves.

Abdomen	Stomach	Epigastric and interscapular region	Th (6) 7, Th 8 (9)	Th (5) 6–Th 10 (11)
	Liver and gall bladder	Right hypochondrium	Th (5) 6–Th 8 (9) and phrenic nerve	Th 6–Th 11 right
	Pancreas	Epigastrium, lower part of sternum, midline on back in area of 10th and 11th rib	Th (5) 6–Th 10 (11) and vagus nerve celiac ganglion	Th 5–Th 11 left
	Spleen	Left hypogastric area	Th 6–Th 8	Th 6–Th 8
	Small intestine: Duodenum	Epigastrium and	Th (5) 6–Th 7 (8*)	Th 6–Th 11
	Jejunum and ileum	Umbilical area	Th 9–Th 11	
	Large intestine: Cecum and ascending colon	Suprapubic area	Th 9–Th 11	Th 8–Th 11 right
	Appendix	Right lower quadrant	Th 10–Th 11 (–L 1)	Th 8–Th 11 right
	Descending colon and sigma	Deep pelvic area and anus	L 1 and L 2 S 2–S 4	Th 11–Th 12 L 1–L 4 left
	Adrenal gland	None	None	Th 6–L 2 unilateral
	Kidney	Hip and groin	Th 10–L 2	Th 10–L 1 (2) unilateral
	Ureter	From back to groin	Th 11–L 2	Th 11–L 1 (2) unilateral
	Urinary bladder: Fundus	Suprapubic area	Th 11–L 1	L 1–L 2 (hypogastric nerve)
	Neck	Perineal and anal region	S 2–S 4	
	Testicles	Testes	Th 10	Th 10–L 1
	Prostate gland	Perineal area and lower back	Th 10, Th 11 S 2–S 4	Th 10–L 1
	Ovaries and tubes	Both lower quadrants	Th 10	Th 6–L 2
	Uterus	Perineum, lower pelvic area	Th 10–L 1, S 2–S 4	Th 6–L 2
	Female external genitalia	Perineum and local	S 2–S 4	L 1–L 2
Extremities	Blood vessels, sweat glands, hair follicles etc. of:			
	Upper extremity	Local on skin	C 5–Th 1	Th 2–Th 8 (9)
	Trunk	Local on skin	Th 1–Th 12	Th 1–Th 12
	Lower extremity	Local an skin	L 4–S 3	Th 10–L 3

* Oral half right, aboral half left and vagus nerve.