what beyond Dr. John Fothergill,[44] who considered that the neuralgia was due to a "sharp, corrosive, cancerous acrimony"; but how much beyond, possibly a later generation alone can tell. Oliver Wendell Holmes,[45] it will be remembered, advocated most emphatically the malarial origin of the disease.

The Gasserian ganglia, which have been removed in the cases above recorded, have nearly all been subjected to examination by expert neuropathologists. Dr. Spiller[28] and Dr. Barker[11] have already published careful reports on the histologic findings of the first four of them in which they were so kind as to take an interest. The tissues from the later cases have been studied by Dr. Rusk, who feels that no lesion sufficiently characteristic to consider it typical of a particular disease is present. Numerous other ganglia, removed postmortem, have been studied as controls and exceptional opportunities for carrying out the investigation were offered by the character of the surgical material itself; several of the ganglia were from patients on whom no previous neurectomies had been performed so that cellular changes consequent on degenerations following these earlier operations led to no confusion in the histologic picture, as they so often do; furthermore, the tissues from the fatal case made it possible to compare a ganglion from the neuralgic with that from the supposedly sound side of the same individual.

The Gasserian ganglion, whether or not primarily so, in the end seemingly becomes affected, and clinical evidence goes to show that the process, whatever it be, is migratory, involving, as pointed out above, one after the other of the three groups of cells which represent the fused tripartite ganglion. The experiments which Dr. Barker and I carried out, though few in number, sufficed to show by the study of Nissl changes that the groups of cells are quite distinct, and thus it is that one cluster alone may become involved in an acute lesion like that of herpes zoster, the pathology of which Head and Campbell have done so much to clarify. The ganglion lesions which produce this segmental outcrop of herpes, particularly when occurring in individuals who have passed middle life, not uncommonly result in a vexatious so-called "postzoster neuralgia." I have seen several individuals suffering from this residual of zoster, two of them after a herpetic involvement of the ophthalmic division of the trigeminus. Though an acutely troublesome and intractable malady, it remains clinically quite distinct from true neuralgia; the pain is not paroxysmal in the same degree, and remains limited to the original field of vesiculation, showing no tendency in the case of the semilunar ganglion to migrate to the other divisions of this structure. Its severity at times may be great enough to demand surgical intervention, and here again are shown characteristics distinguishing it from the more common type of neuralgia, since peripheral operations in the postzoster form do not suffice even to give temporary relief, as I have learned to my sorrow through several failures; the posterior root ganglia must be removed.

The morbid lesion underlying true trigeminal neuralgia, unlike that of zoster, has so far eluded search and the histologic changes in the cells, the swelling and vacuolization, the chromatolysis, alterations in the tigroid, nuclear fragmentation and other departures from the normal which have been so painstakingly described are, as Coenen[46] has emphasized, due in large part, if not entirely, to the effects of earlier peripheral neurectomies and correspond histologically to the lesions found, for example, in dorsal root ganglia months after the amputation of a limb. The tissues from two of the cases in Lexer's series examined by Coenen showed no histologic abnormalities whatever; no peripheral operations had preceded the ganglion extirpation in these two cases. And with these findings those of Monari, Schwab and ourselves have been in almost exact accord.

Let us hope that some one with new histologic methods and possibly more extensive material may solve this pathologic riddle, for not until the lesion is known may we expect to discover its causal agent, and not until then may we have hopes of supplanting for surgical measures others less difficult, though they could hardly be more certain of affording immediate and permanent relief.

44. Fothergill: A Painful Affection of the Face, Medical Observations and Inquiries, vol. v, p. 129, London, 1773, Lettsom's edition of Fothergill's works.

45. Holmes: "The Nature and Treatment of Neuralgia," Boylston Prize Dissertation, 1836.

NORMAL SALT SOLUTION AND OTHER LOCAL ANALGESICS

IN THE OFFICE TREATMENT OF ANO-RECTAL DISEASES.*

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Recent discoveries and improved technic in the use of local analgesics have enabled us to treat as ambulatory cases many patients suffering from various forms of minor ano-rectal diseases whom we formerly sent to the hospital for treatment. This ofttimes obviates the necessity of putting aside all business and social obligations and of making a two or more weeks' stay in a hospital, with all its attendant anxieties and embarrassments. Such patients, with few exceptions, are now operated on at the office or at the patient's home. Before operating, if there are no contraindications, about the only question asked the patient is: "Did your bowels move to-day?" If so, and if an operation is indicated or necessary, and the patient so desires, the field is prepared and the operation is performed, either a normal salt solution or a eucain-adrenalin solution being used as the local analgesic.

In my first experiments some years ago I used cocain as a local anesthetic. In many instances the toxic effects of this drug were so alarming that I discontinued its use. Recognizing, however, the great advantages and utility that a satisfactory local analgesic would afford in treating many of these cases, I continued my investigations and experiments along these lines.

Recently, in selected cases, and quite satisfactorily too, I have used sterile water for this purpose. The use of water as an analgesic was suggested to me by Dr. Gant of New York, who has reported over 300 cases of various kinds operated on under the influence of this agent. He states, however, that the injection in some instances may cause considerable discomfort or even pain. I found this to be true, especially in those cases of acutely inflamed thrombotic piles and abscesses, or in which it was necessary to make the injections into the deeper tissues, and in cases in which the operation necessarily was prolonged.

46. H. Coenen: Mikroskopische Befunde am Ganglion Gasseri in den 15 Fällen von Lexer, Archiv für klin. Chir., 1902, vol. lxvii, p. 333.

* Read before the Chicago Medical Society, 1904.

In a cursory review of the literature, however, I find that the use of water as a local analgesic is not new. According to Dawbarn of New York,[1] Halsted used water hypodermically as a local anesthetic for small operations as long ago as in 1885. He found that it succeeded best when the water was injected into the skin, not beneath it. Dawbarn also made experiments with this agent on himself, assisted by Dr. F. A. Manning of New York.

Oscar Liebreich[2] found that distilled water produces the caustic as well as the anesthetic effect, and that those substances producing painful anesthesia cause dilatation rather than contraction of the blood vessels.

Schleich[3] noted that sterilized water, when injected hypodermically, will produce anesthesia in the region of edema. He tried the experiment on himself and on his assistants, and used it in removing a large carbuncle.

Sollmann of Cleveland[4] refers to local anesthesia produced by distension of the tissues with pure water and says that the edema is very painful.

Dr. T. C. Witherspoon, St. Louis,[5] frequently has used boiled water or air infiltration in opening abscesses.

Gould, in his recent "Dictionary on New Medical Terms," 1904, refers to water anesthesia as "anesthesia dolorous of Liebreich."

Heinze[6] of Dresden in his experimental investigations found that pure water alone is an intense irritant to the sensory nerves, and that anesthetic solutions diluted with it beyond a certain extent have the same effect, and render the injections painful. He also demonstrated that the addition of salt entirely obviated this source of irritation.

Hence, it occurred to me that some isotonic solution would be a better agent than water with which to produce pressure or mechanical analgesia when injected hypodermically. The nearest approach to this is sterilized urine or normal salt solution. The latter agent I have used very satisfactorily. The addition of salt to the water is not for the purpose of affecting analgesia, but to prevent the irritative symptoms, as it is desirable that the solution injected should give as little pain as possible and also that it should not be harmful to the tissues.

Should it be desirable, as it is in some instances, especially where the injections must be made into the deeper tissues and the operation necessarily prolonged to secure the specific action of some drug, as eucain or cocain, alone or in combination with the distension of the tissues, I then add a small quantity of eucain lactate and adrenalin chlorid to the salt solution. Adrenalin intensifies and prolongs the analgesia, and when the analgesic agent is used in combination with it, less of the agent is required to produce the full effect.[7]

SOLUTIONS.

To determine the potential effect of various solutions on normal tissues I had Dr. W. A. Evans of the Columbus laboratories make certain tests for me and incorporate herewith his report, which is as follows:

I have made use of two procedures. I have mixed the solution and freshly drawn blood, using the hanging drop proceeding and protecting the drop from the evaporation by the use of grease along the edge of the cover glass. In this way, I have been able to watch the behavior of the red blood cells mixed with this solution. In addition to this I have determined the freezing point of different solutions. I am not certain that a determination of the electrical conductivity of the solution would give any more information than is to be obtained from the two procedures already noted. I made a solution of adrenalin and eucain in a normal urine with a gravity of 1,020. I found that this froze at about 1.5 degrees. When blood was added to this mixture the corpuscles were promptly crenated. A mixture of eucain and adrenalin in from .6 per cent. to .75 per cent. salt solution seemed to be isotonic to red blood cells; on the one hand these cells did not crenate, on the other they did not lose their hemoglobin. These mixtures froze at about .5 degrees C. and at 9 degrees C. respectively. The average freezing point of blood is .56 degrees C.

It will thus be seen that these solutions are practically isotonic with blood and we would expect them to give the minimum amount of pain and the minimum disturbance of the tissues.

The following is one of the solutions that was submitted to the above tests and clinical experience proves that it answers well:

Beta-eucain lactate	2
Sodium chlorid	75
Adrenalin chlorid solution	65
Distilled water	100

This entire quantity of fluid, according to Barker of London, who has made valuable contributions to the literature on the subject of local analgesia, can be used with comparative impunity. He has used as much as 6 grains of beta-eucain hydrochlorate and 20 minims of adrenalin chlorid solution without noting any ill effects. Matas of New Orleans and others have used larger amounts. They are careful, however, not to allow more than 5 grains of the drug to remain in the system. To make the normal salt solution, put into an Erlenmeyer or Jena glass flask 3½ oz. (100 c.c.) of distilled water and 11.5 grains (.75 gm.) of chemically pure sodium chlorid. Boil for two or three minutes; when cooled to blood heat it is ready to use. To make the eucain-adrenalin solution add 3 gr. (.2 gm.) of beta-eucain lactate to the water at the same time the salt is added, and after boiling and cooling to the body temperature add 10 drops of adrenalin chlorid solution, and it is ready to use.

The cases in which I have successfully employed one or the other of these solutions to produce local analgesia include radical operations for protruding and non-protruding internal hemorrhoids; interno-external, thrombotic and cutaneous hemorrhoids; polypi, anal prolapse, fistula, fissure, ulcerations, abscesses, sacral dermoids, lipomas, condylomatas and secondary operations for colostomy.

TECHNIC.

The technic depends more or less on the nature of the operation to be performed. In internal hemorrhoids, the salt solution is injected directly into the tumor until the latter becomes white, when it is grasped with a pair of forceps and the mucosa carefully divided down to the submucosa; this tunic, which contains the principal blood vessels, is then ligated with a very fine linen ligature and the major portion cut off. As the lymphatics and circulating blood rapidly remove isotonic solutions from living tissues, the anesthesia is of short duration and the operation must be performed very quickly. In polypi, the salt solution is injected into the base of the pedicle, a ligature thrown around it and the tumor cut off. Interno-external piles are treated in the same way

1. Med. Rec., Nov. 14, 1891, p. 613.
2. "Note sur l'anesthesie locale," Compt. rend. Soc. de Biol., Paris, 1888, 8th ser., vol. v, p. 340.
3. Deutsche med. Zeitung, 1891, No. 66.
4. Pharmacology, 1901, p. 234.
5. Interstate Med. Jour., July, 1903.
6. Arch. f. pathol. Anat. und Phys., und Ztft. f. klin. Med. vol. cliii. No. 3.
7. H. Braun of Leipsic.

as the internal variety. In prolapsus ani the injection is made into the mucosa and deeper structures; sections of these tunics are then ligated and cut off.

In cutaneous hemorrhoids the skin is first anesthetized, then the central portion of the hemorrhoid; the tumor is then removed with a sharp knife. I say knife, because I have observed that scissors are more apt to cause pain than the scalpel. In all cases requiring deep or extensive operations the skin should be carefully and thoroughly anesthetized first, and then the deeper structures. Very fine needles are used for the skin, coarser ones for the subcutaneous tissues, and for the deeper structures still larger ones, which are blunt and have holes in the side instead of in the end, to avoid injury to vessels. It is important that the solution be warmed to the body temperature. Rapid injections should be avoided, as sudden distension of the tissues may be painful. For the same reason neither solution should be used when cold or too hot. Strange as it may seem, most patients complain of but little pain on the days after the operation. In fact, they have much less pain than after taking chloroform or ether, to say nothing of the suffering experienced by some in coming out from under the influence of the anesthetic, and the healing process seems to be much more rapid and satisfactory. This is probably because the resisting power of the patient has not been lowered with a general anesthetic, as it has been shown by George Rubin of Chicago[8] that the administration of ether and chloroform has a decidedly detrimental influence on the natural defenses against infection.

After hemorrhoidectomy a cone-shaped dressing covered with a piece of the thinnest rubber dam is applied to the anus, and over this the ordinary dressings, all being held in position by a snugly fitting T bandage. Should the patient complain of much pain, hot compresses or a hot sitz bath may be used and will usually give relief.

I have operated on about 75 patients under local analgesia. The aggregate amount of pain has been much less than in a similar class of cases operated on under general anesthesia. In some instances the pain was quite sharp for a few moments, but nothing in comparison with the pangs, anguish and writhings occasionally seen in patients after the ligature operation for hemorrhoids under general anesthesia. In one patient I had to abandon the operation. This was purely a case of "psychic pain." I do not believe that any local analgesia can control this nervous dread and fear experienced by some patients. A hypodermic of morphin, administered 20 to 30 minutes before operating, seems partially to control the pain in some cases. Many of these patients did not have any pain worth mentioning.

CONCLUSION.

Since practice and experience have demonstrated that effective and radical treatment in this class of diseases can be accomplished in the office or in the patient's home, and without his foregoing all business and social obligations, going to the hospital, taking an anesthetic, which always occasions more or less anxiety on the part of both the surgeon and patient, and remaining there for two or three weeks, I believe that another step in advance has been made in the treatment of selected cases in the practice of proctology.

103 State Street.

8. Trans. Chicago Path. Soc., March 14, 1904, vol. vi, No. 4.

THE APPLICATION OF LABORATORY METHODS TO THE DIAGNOSIS OF VARIOLA.

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The rapid increase of our knowledge of infectious diseases since the middle of the last century, together with the recent advances in laboratory methods, particularly in bacteriologic and histologic technic, has had a profound influence on the accuracy of medical and surgical diagnosis.

Tuberculosis, cholera, typhoid fever, anthrax, actinomycosis, diphtheria, bubonic plague, pneumonia, bacillary dysentery, and various forms of primary and secondary wound and other infections, among the bacterial diseases; trichinosis, pébrine, malaria, amebic dysentery, filariasis, trypanosomiasis, and various forms of helminthiasis, among the diseases caused by animal parasites, may be enumerated as examples of infectious diseases for the diagnosis of which laboratory methods are in constant use. In short it may be said that, as soon as the parasitic origin of a disease is established, the demonstration of the presence of the causal agent is used as the basis of exact diagnosis. Laboratory methods are also availed of in the diagnosis of certain affections whose etiology is still undetermined, as rabies, tumors and certain diseases of the blood-forming organs for instance. The methods in use for these purposes are (1) microscopic examinations (*a*) of coverslip preparations, (*b*) of sections of tissue; (2) cultures; (3) serum reactions; and (4) animal inoculations.

Recent advances in our knowledge of variola make it possible, it seems to me, to utilize two of these methods for the diagnosis of this disease.

Councilman, Magrath, Brinckerhoff,[1] and Tyzzer,[2] in 1903 and 1904, published a series of articles concerning the etiology of vaccinia and variola, in the skin lesions, of which they found certain bodies situated in the cytoplasm and nuclei of the epithelial cells, and which they describe as stages in the life cycle of a protozoan parasite, *cytorrhyctes variolæ*. These bodies, of various sizes, shapes and staining reactions, occur only in the cytoplasm in vaccinia and in both the cytoplasm and nuclei in variola.

Calkins,[3] using their variola material, has made out a very complex life cycle for this parasite, consisting of one cytoplasmic and three internuclear stages. He classes the organism among the microsporidia, a form of protozoa. More recently Howard and Perkins,[4] working on material from seventeen cases from the recent Cleveland variola epidemic, found the bodies as described by Councilman and his co-workers, and confirmed their work, with the exception of one of the intranuclear stages of Calkins, which they could not find. Howard and Perkins[4] added a second cytoplasmic stage, and according to them the life cycle of the organ-

1. Councilman, Magrath and Brinckerhoff: A Preliminary Communication on the Etiology of Variola, Jour. of Med. Research, 1903, vol. ix, p. 372. The Pathologic Anatomy and Histology of Variola, Ibid., 1904, vol. xi, p. 12. The Occurrence of *Cytorrhyctes Variolæ* (Guarnieri) in the Skin of the Monkey Inoculated with Variola Virus, Ibid., 1904, vol. xi, p. 173.

2. E. E. Tyzzer: The Etiology and Pathology of Vaccinia, Jour. of Med. Research, 1904, vol. xi, p. 180.

3. Gary N. Calkins: The Life Cycle of *Cytorrhyctes Variolæ*, Guarnieri, Jour. of Med. Research, 1904, vol. xi, p. 136.

4. Howard and Perkins: A Study of the Etiology of Variola, Jour. of Med. Research, 1904, vol. xii, 359.