Locally, I treated five separate areas differently. One area I curetted and made no applications; another I curetted and then applied a 4 per cent. ointment of potassium iodid in vaselin; a third area was curetted and a mixture of equal parts of tincture of iodine and guaiacol applied; to the fourth area I applied pure carbolic acid, and to the fifth a mixture containing equal parts of iodine and carbolic acid. The fourth and fifth areas were not curetted.

The disease showed improvement in all of the areas treated. The area treated by curetting and applying iodine and guaiacol did better than any of the others. The least improvement was observed in the areas treated by pure carbolic acid, and by curetting followed by the potassium iodid ointment.

ACUTE ANTERIOR POLIOMYELITIS IN A YOUTH OF 18 YEARS. REMARKS ON THE SENSORY SYMPTOMS.

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A. M., aged 18 years 2 months, was taken with a febrile attack Oct. 13, 1900, accompanied by much pain in the back and thighs, and followed by acute paralysis of the lower limbs.

The patient, an intelligent young man, gives the following account of the attack: On the day he first took to bed he went to school as usual in the morning, although feeling poorly. He was too ill to remain at school, but walked home, a distance of one mile, and went to bed very ill. The next two days he was very restless, suffering especially with pain in the loins and thighs; he was so sensitive to touch that he dreaded to have anyone take hold of him, and it made him sore and tired to lie, and he was in and out of bed frequently. Finally, he could not get up and realized that his lower limbs were paralyzed. Thus, he went to bed Friday after returning from school, and on Sunday he was paralyzed.

He is confident that this painful stage lasted eight to ten days. He instances various collateral data which show that his statements are accurate. After the severe pain there still remained about a week during which moving and touching the paralyzed members, especially the left, caused considerable pain.

During the fourth week after going to bed he discovered that slight voluntary movements were present in the right leg, but none in the left. He did not sit up until the sixth week and then his back was so weak that he could not remain up long.

During the fever stage it was necessary to use a catheter for a day or so, and the bowels were constipated.

Dr. C. A. Anthony, Fredericktown, Mo., the attending physician, observed the case with much interest, remembering the details very accurately for the reason that they were very confusing to him for some days. His account of the onset and the great amount of hyperesthesia and pain in the back and paralyzed members conformed exactly to the statements previously obtained from the patient. No sensory tests were made at the time.

I first examined the patient Jan. 25, 1901, more than three months after the attack. I found a paraplegia of the lower extremities, very little movement in either of them, the right some better and improving faster than the left; but even the right leg had to be lifted in or

out of bed or over the left. He could only get to a chair from the bed and back again with effectual assistance from a strong person.

Dr. M. W. Hoge kindly assisted me in making routine sensory and electric tests. We found all the muscular groups of both extremities more or less atrophied. The most intense atrophy was in the left thigh; apparently, the best preserved group was the calf of the right leg. The flexors of the left knee and foot were becoming contracted, and there was also a slight contracting tendency apparent in the toes of the right foot. Electrical tests showed a reaction of degeneration, or a partial reaction of degeneration, in all the atrophied groups, the right calf and some of the right thigh muscles alone giving normal reaction. The skin of the feet and legs was cold, clammy, livid; improving in appearance from below upward and better on the right side than on the left. Careful sensory tests (touch, pressure, pain, temperature, etc.), showed slight obtundity, but not more than would be accounted for by the bad trophic state of the skin. There were no dissociate sensory symptoms, except possible pain and temperature sense were a trifle more dulled in the feet than tactile. The general sensation was improved from below upward, its impairment corresponding exactly with the nutritive impairment of the skin. There was no muscle tenderness or hyperesthesia anywhere.

This patient presents another interesting pathologic condition, which probably has no etiological relation to the myelitis, but which we may record as follows: March 31, 1899, he was accidentally shot through the chest, the ball entering to the left of the sternum about the third rib and coming out behind the right clavicle. There is an aneurysm of the arch of the aorta as a result of this injury which causes him no annoyance. He made a rapid recovery after the accident and seemed perfectly well and strong until the time of the paralytic attack which was eighteen months later.

At this writing, eight months after the attack, the patient is hobbling about on crutches, the right leg having improved considerably but the left very slightly.

On account of this patient's age there was an unusual opportunity to obtain an account of the subjective sensory symptoms in a severe attack of acute anterior poliomyelitis. I believe, as do others, that the importance of the sensory symptoms has been somewhat overlooked, and more especially in some of the text-book discussions of the disease. Without attempting an extended consideration of the question I simply wish at the present time to embrace this opportunity of calling attention to it.

Dana¹ thus defines the disease: "Anterior poliomyelitis is a disease of the spinal cord characterized by motor paralysis of rapid onset, followed by muscular wasting, without sensory symptoms. It occurs at all ages but vastly oftener in infancy; hence it is often called infantile palsy."

And Starr² thus: "Anterior poliomyelitis is an acute disease observed most commonly among children, but occasionally in adults, characterized by sudden complete loss of power in one or more limbs, followed by wasting of the muscles paralyzed, and by interference of the growth of the parts, but not attended with any sensory disturbances."

Below are quotations from a number of writers whose texts I have picked from my shelves at random:

Hirt:³ Vague pains in the limbs.

Struempell:⁴ The child complains of headache and sometimes of pain in the loins and limbs.

Rosenthal:⁵ Sensibility is sometimes exaggerated at the outset but at a later period it becomes normal.

Bruns and Windscheid:⁶ Sensation is always preserved, as far as this can be determined in little children. The bladder may be affected in the initial stage, although, of course, this is a difficult matter to determine in very young children.

Oppenheim:⁷ There is no pain generally; rarely it may be so acute as to simulate acute rheumatism. The muscles may be sensitive to pressure. If the pain of the muscles and nerves is considerable some neuritis is probably present.

M. Allen Starr:⁸ It is generally observed that children cry a good deal during the period of onset, and some of those who are able to complain say that they suffer from pain in the back and in the affected limbs. This pain may remain for some weeks. . . . Retention of urine has been noticed for a few days. . . . There is no complaint of numbness or of paresthesia and there is never any loss of sensation; but the limbs are sometimes painful upon any movement, especially in the joints.

Dana:⁹ After the general disturbance subsides there may be some pain in the back and limbs for a few days, and in rare cases the bladder is involved so that there is retention of urine.

Gray:¹⁰ Other reflex phenomena as variable as the fever itself may accompany it, such as delirium, somnolence, convulsions, with or without loss of consciousness, muscular twitchings, vomiting, headache, temporary retention of urine, pain in the loins and in the limbs that are subsequently paralyzed, or general pains. Children of four or five years may be able to locate the pain, while younger ones will only evidence it by crying when they are touched or lifted.

Collins:¹¹ Usually accompanied in children by manifestations of irritation of the central sensory sphere, such as vomiting, convulsion, flightiness, irritability, and a serious disinclination to be handled, as if pain were caused thereby.

After the general phenomena of inflammation subside, there are no sensory disturbances, subjective or objective, although enduring pains in the extremities have occasionally been noted. When such symptoms occur they indicate an involvement by the inflammatory process of the posterior cornua and the root fibers passing therein.

Ross:¹² Sensory disorders are almost entirely absent during the whole course of the disease. At the outset patients may complain of pains and various paresthesiæ, but these symptoms are of short duration. A certain degree of cutaneous hyperesthesia, or rather hyperalgesia, has been described as being present during the febrile stage, but this tenderness to touch probably depends on tenderness of deeper structures, such as rheumatic inflammation of joints. The cutaneous sensibility is sometimes blunted in the paralyzed extremities in old standing cases, but this probably depends on underlying nutritive and vascular changes.

Church and Peterson:¹³ Most writers state that there is a complaint of pain in the afflicted members only rarely, but that, as a rule, sensation in all its phases is entirely normal. It is probable, however, that early dysesthesia, owing to the usual infantile age of the patient and a lack of careful search for such difficulty, has been frequently overlooked. In some considerable number of the cases handling of the affected limbs during the initial fever provoked outcries which were not elicited by similar manipulations of the other members. It is likely that more attention in this direction will show localized hypersensitiveness, or some kindred state, to be usually present and of diagnostic importance. Indeed, complaints of pain and of formication have been generally noted in older children and in adults, lending, perhaps, undue weight to the usual supposition that such cases are not of a true spinal type. . . .

The adult form is in no way different from the infantile cases excepting the variations due to complete growth having been attained. In anomalous cases the onset of the disease is insidious and the course may be subacute. After infectious diseases one or more limbs may be found useless, flaccid and atrophic, declaring the antecedent myelitis. Occasionally the onset is marked by pains of a severe cutting character, which are frequently attributed to neuritis or to rheumatism, but may be due to irritation of the sensory pathway in the cord

by the location of the myelitic focus backward, involving the neck of the posterior horn.

Peterson:¹⁴ Pain referred to the limbs or back and sometimes even muscular tenderness is apt to be present. The differential diagnosis between peripheral neuritis and poliomyelitis is often difficult in adults, and sometimes indeed, impossible, owing to the occasional prominence of sensory symptoms.

Of the texts above cited some maintain one feature of the sensory phenomena and some another until with them all before us the relative importance of these disturbances may be made out. Only one work gives the subject adequate consideration, namely, Church and Peterson's. My individual experience leads me to recognize the force of the statements therein contained.

In a communication¹⁵ written in 1888, I dwelt briefly upon the sensory symptoms of an interesting case therein reported. Before, and more especially since then, I have been interested in remarking the sensory phenomena of cases of poliomyelitis coming under my own observation, and in those reported by others. I think there is no question that hyperesthesia and pain are both usually present and that they are also usually proportionate to the intensity of the invasion.

In the communication just alluded to I explained the sensory symptoms, as had others, by assuming that there was produced an irritation of the posterior areas of the cord by the overflow of the inflammatory process, whose point of most intense activity was, for some reason which we could then not explain, always in the anterior horns. Collins, Church and Peterson, as herein cited, and others, seem to find this explanation satisfactory. On the other hand some lean to the opinion expressed above by Oppenheim, that pronounced or important sensory symptoms mean a concomitant neuritis. This latter is probably often a hasty supposition, as Church and Peterson intimate. The sensory symptoms of these myelitic attacks are too ephemeral and intense in their duration for even a mild neuritis. They sometimes reach their full intensity within a few hours and are gone within a few days, leaving all forms of sensation quite normal; and secondly, their distribution is not the usual one of neuritis. They infest the back and the proximal portion of the extremities and are not heralded by paresthesias in the hands and feet.

From the above it is evident that these phenomena are the source of some confusion. This is due to the fact that as yet they have probably not been sufficiently studied. The complete absence in typical anterior poliomyelitis of final or permanent sensory disturbances has led us to overlook the significance of those which occur in the onset. For example, in each of the definitions cited above it is affirmed that there are no sensory symptoms attending the disease. This arises probably in the eagerness of these writers to define anterior poliomyelitis as a distinct clinical and pathologic entity, instead of merging it too much in the descriptions of other forms of myelitis, as some clinicians have done. Failing to mention these symptoms at all in a definition under the circumstances would be better than to affirm their absence.

The diagnostic importance of this study is thus suggested by Church and Peterson: "The differential diagnosis between peripheral neuritis and poliomyelitis is often difficult in adults and sometimes indeed, impossible, owing to the occasional prominence of sensory symptoms." With a little pruning this sentence conveys the gist of the discussion. I would say that the sensory symptoms are more than occasionally prominent, and that, while the greatest difficulties of diagnosis need

not arise very often, they do occur frequently enough and are important enough to demand increasing attention. In considering these difficulties we must keep in mind the following possibilities:

1. Cases of acute anterior poliomyelitis with no noticeable sensory symptoms. These are rare, at least in adults and in children old enough to express themselves.

2. Cases of acute anterior poliomyelitis with severe pain and hyperesthesia in the paralytic members in the onset and lasting for a variable time. These cases are also rare.

3. Cases of acute anterior poliomyelitis in which the sensory disturbances fall between the extremes of numbers 1 and 2. These are the usual cases.

4. Cases of multiple neuritis with unusual sensory symptoms and distribution.

5. Cases of so-called "multiple neuritis of the motor type," with little or no sensory symptoms.

6. Cases in which multiple neuritis and poliomyelitis are associated. Such cases have been pathologically demonstrated. They are difficult to diagnose; and certainly are not frequent. They are caused by some widespread and intense toxic process, producing grave general symptoms.

7. Miscellaneous conditions, as acute ascending paralysis, "family periodic paralysis," myesthesia, hysteria, etc., which, in the differentiation under consideration, will hardly create confusion when carefully studied.

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SOME CLINICAL ASPECTS OF CHEMISTRY.

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Even the most superficial observer of the expansion of the medicine of to-day might accept without much question the claim of chemistry to be the first of the pure sciences which form the foundation of medicine, for the potent influence of its leaven may be perceived throughout the whole medical mass. Clinical laboratories are growing in favor and influence; publishers have produced a superabundance of text-books which purport to "make clinical chemistry easy"; medical journals accept at sight articles on almost any chemical subject, some of scientific value, some of practical value, some of no value.

At a recent large medical congress so much time was spent on the chemical side of internal medicine that one not interested in that subject must have had to exert unusual efforts to look interested and knowing. What did he care what the intermediate products of sugar metabolism are, or whether the body can recognize the difference between a "right-handed" and a

"left-handed" pentose with the same empiric formula? Of what "practical value" is it to discover an extra ferment in the stomach? Does the "practical man" care to know how a hen synthesizes uric acid?

Neurologists now examine urine with greater interest and demand that we find there the cause of a neuritis. Oculists and dermatologists awake to the fact that the whole body is one and that the chemistry of this whole may throw light on the organ in which they are interested. Students are required to take chemical courses before beginning to study medicine and it is a professor of anatomy who suggests the addition of another year of pure chemistry to the already long list of requirements of one school.

But chemical education does not emanate alone from medical schools, medical journals and medical congresses; every practitioner gets a free course through the mails. He is bombarded with pamphlets giving a detailed account of some recent "Arbeit" in chemistry, and advertising a new food or new preparation of some well-known substance, or some brand-new drug which is the tangible and expensive embodiment of the above-mentioned "Arbeit." The whole practical medical world, in fact, is studying chemistry.

But it is not alone medical men who are turning chemists for the pathologists are also studying chemistry—their hemolysins, bacteriohemagglutinins, anti-hemagglutinins, toxophores, heptophores, complementophilic groups and intermediary bodies are now or soon will be playthings of "pathologic chemists."

The physiologic laboratories have also felt the leaven. The kymograph is run down, the plethysmograph leaks, the nonpolarizable electrodes almost spark, burettes, test tubes and beakers cover the tables, for the physiologists are, many of them, studying chemistry. Here the recently-called professor of physiology is a pure chemist; there almost the whole physiologic staff is working on chemical problems. The journals which publish the mechanical side are termed by a pure physiologist "uninteresting," journals of physiologic chemistry grow in number and circulation and the physiologic chemical laboratories are admitted by eminent clinical men to be the centers from which the medicine of to-day is progressing.

The growth of the physiologic chemical laboratory is of the utmost importance to clinical medicine, for now the clinical laboratory will be provided with methods which are of practical use to the clinical man, and with men well enough trained to use them. Clinical chemistry is no new subject. Urinary examination has long been recognized as of the greatest importance, but the work has not been done by men especially fitted and the methods in use were many of them poor. Nevertheless, and in fact because of this, there has grown a very extensive literature of clinical chemistry and to increase this has been one of the earmarks of a "very scientific practitioner." The advent of physiologic chemistry, the occupation of the field by thoroughly-trained men, has sifted and weeded this literature to a surprising degree. Instead of a long list of diseases in which the xanthin bodies are increased or diminished we now read that the method used was so inaccurate that "our knowledge of the relations of xanthins to pathologic processes is as defective as it was years ago"; the alkalinity of the blood is not arraigned as often as a few years ago, although we still suspect it is an offender, but can not prove it; phosphaturia is now a name only. Compare a text-book of clinical chemistry of this year with one

* Read before the Clinical Section of the Medical and Chirurgical Faculty of the State of Maryland.