certainly belong to the same group, and we have classified them as proteus germs. It will be observed, however, that while 6d is motile, 6e is non-motile. 6d produces indol, while 6e does not; 6d coagulates milk more promptly than 6e. However, we do not lay a great deal of stress on this point, nor on the slightest differences seen in the potato cultures, except that 6d was colorless on potato, while 6e was plainly yellowish pink. So far as gas production is concerned, the two germs might be the same. Whatever the relation between 6d and 6e, both of these certainly differ from 6a. The latter does not liquefy, does not produce indol, does not coagulate milk, even after ninety-six hours, and does evolve a much larger amount of gas than either 6d or 6e; 6a, as will be seen from the table, we have classified as doubtful. This water would be condemned.

The differences between 7a and 7b are plainly seen. 7a is a non-liquefying, while 7b is a liquefying organism. 7a produces no indol, while 7b produces this substance in abundance. 7a neither coagulates nor produces acid in milk; 7b does both. 7a is a typical germ of the class, which in this laboratory we have designated under the head of *venenosus*. Its close resemblance to the typhoid bacillus is easily seen by a study of the table.

8a came from a spring which breaks from a hillside, the top of which is covered with houses, and there are many chances of pollution. This organism certainly belongs to the proteus group, and the water is not condemned.

14a and 14b were obtained from the heart's blood of animals inoculated from the public water supply of Petoskey, Mich. It will be seen that one of these does not liquefy gelatin, while the other does, and one produces no indol, while the other produces this substance. One coagulates milk more promptly than the other. So far as growths on potato and the evolution of gas are concerned, these organisms closely resemble each other. 14a is classed as doubtful, and 14b as proteus. The water is condemned on account of the presence of 14a.

20a, 20b and 20e were obtained from the heart's blood of animals inoculated with water from Battle Creek, Mich. 20e is quite typical of the group *venenosus*. 20b certainly belongs to the colon group, while 20a we have classed as doubtful. This water is condemned.

21a was from a test well sunk in searching for good water. The water was not condemned.

22a is what we have called *Bacillus venenosus brevis*. Under the microscope this short bacillus may easily be mistaken for a coccus, especially in stained specimens, but when examined in a hanging drop, it will be seen that it is a short motile bacillus. 22e is classed as doubtful.

9a came from a well near Ann Arbor, and was sent for examination because one man in the family using this water developed typhoid fever. It must be stated, however, that this man was several times away from home, and at least once, about two weeks before the development of the disease, spent twenty-four hours in a city where typhoid fever was prevalent.

10a and 10d are interesting. They were obtained from the heart's blood of the same animal. The water came from a well at a farmhouse in which there were two cases of typhoid fever, one of extreme severity and the other light in character. The only marked difference between 10a and 10d is that 10a produced acid in milk, while 10d did not. Neither coagulated milk.

11, 12 and 13 came from different samples of water. All these waters were sent from Petoskey. 11a came from a test well, while 12a and 13a came from the city supply. 11a was not condemned; 12a and 13a were condemned.

CONCLUSIONS.

1. Of 709 samples of drinking water sent by health officers and other physicians to the hygienic laboratory of the University of Michigan between Oct. 1, 1888, and Dec. 31, 1903, 213, or 30 per cent., contained toxicogenic germs.

2. Ten samples, or 1.4 per cent., contained no bacteria capable of growth at 38 C. Of these 10 samples, 4 were from deep springs, and 6 from the Great Lakes—4 from Lake Superior and 2 from Lake Huron.

3. Waters that contain no germs capable of growth at 38 C. or higher can not cause disease.

4. Waters that contain no toxicogenic germs are not condemned.

5. Waters that contain as their only toxicogenic organism a typical colon bacillus are not condemned. By a typical colon bacillus we mean one which is non-motile, produces indol abundantly, produces acid in milk, coagulates milk within twenty-four hours, and evolves more or less gas from glucose and lactose cultures.

6. Waters which contain as their only toxicogenic organism a typical proteus bacillus are not condemned. It must be remembered that other germs can not be excluded until after repeated plating of the cultures made from the heart's blood fails to show their presence.

7. Waters that contain any member of the *venenosus* group or any such germ as we have marked doubtful in the table given are condemned.

8. I have never found in drinking water a germ that responded to the Widal test.

9. Sixteen years of experience with this method of water analysis convinces me that the results given by it are in the majority of instances trustworthy.

The development of this method of water analysis and its application have been largely due to the skillful and conscientious work of the many assistants who, since 1888, have worked with me on this subject. I can at present do nothing more than mention their names. During the first three years I had the assistance of the late Dr. F. W. Brewer, who subsequently became a professor in the Agricultural College of Utah. Much credit is also due to Drs. Ives, McClintock, McClymonds and Cooley; and the method owes much to my more recent assistants, Drs. Carhart, Cooper and Williams.

A CASE OF ATROPHY OF HAND MUSCLES

WITH LOCALIZED SENSORY DISTURBANCES; POSSIBLE
EARLY SYRINGOMYELIA.*

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CINCINNATI.

The following case is reported because it possesses very interesting features:

History.—Miss L., aged 27, consulted me first, Feb. 27, 1903, sent by her family physician because of a localized wasting of the thumb. There is no neuropathic family taint, nor has any myopathic disease ever been present in the family so far as known. Two other sisters were examined carefully with

* Read at the Fifty-fourth Annual Session of the American Medical Association, in the Section on Nervous and Mental Diseases, and approved for publication by the Executive Committee: Drs. Richard Dewey, H. A. Tomlinson and F. W. Langdon.

negative results. The girl is unusually tall and very slender. While tallness is a family characteristic, she is the tallest of all. Tuberculosis has not existed in the family, and patient is also free therefrom.

Pain in the right arm has been noticed since her twelfth year. The arm has also seemed weaker at times than the other, but she has performed all the arduous duties of laundry and house work for many years. There is no history of the super-vention of the pain and weakness after any illness, acute or otherwise, which might be regarded as an etiologic factor. Trauma is not in evidence. The pain was never severe in character, but always more noticeable after any special muscular effort, or when heavy clothing was worn. Neither quivering nor twitching has ever been noted. During the last five years and up to the date of the institution of electric treatment by me cramps would appear in the fingers of the right hand. These cramps would cause a stiffening of the fingers, producing extension of the hand on the wrist and flexion of the distal phalanges. Occasionally only two fingers would stiffen in this way, sometimes the little and ring finger, at others the thumb and neighboring two. The cramps would come on several times daily for a few weeks, followed by free intervals of varying duration. There was never any tendency of the cramps to further extension to the arm, and, as said, since the beginning of electric treatment they have been absent.

About three to five weeks prior to February 27, 1903, she noticed for the first time a sinking in of the thumb. She was positive that the wasting must have occurred at about the time stated, certainly not at a period much earlier. It is, however, my impression that the atrophy did not come on so suddenly as she states, though her opinion on this point is quite firmly maintained.

Subjective sensory disturbances were never observed. At no time did she complain of any "numbness" or "tingling" or "coldness," and when questioned by me she did not recall any such symptom.

To sum up she came to me solely because of the atrophied muscle, for had she not discovered this the weakness and pain would have appeared of not much import.

Examination.—Patient is 6 feet 2 inches high, very slender, weight 146 pounds. The right arm shows no change, either in the muscles above or below the elbow, but along the outer border of the thumb there is a decided wasting. This is limited to that part of the thenar eminence occupied by the opponens, and the outer part of the abductor pollicis. The interossei and lumbricals are not involved, consequently there is neither deepening of the spatia interossei or the palm.

Movements of Thumb: Abduction and apposition are both performed with less power than on the left. The grip of the right hand is slightly less than the left. Flexion, extension and all other movements of the arm, wrist and hand are properly performed, but on the whole the strength is slightly diminished.

Electric Examination: Reactions are prompt with both currents to equal current strength in all arm and hand muscles, except in the case of the right musculus opponens and abductor.

Pollicis: In these two there is a marked quantitative change to both currents, and the response to both faradic and galvanic is slower than the normal type.

Sensation: No objective disturbance of any kind is present anywhere in the body, except along the ulnar border of the right arm. At the first examination I found a narrow area of sensory change beginning at the internal condyle, and extending in a straight path not over one-half inch wide at any point to the pisiform bone. In the upper third of this narrow area there was thermo-anesthesia; cold was often called warm, very hot was called warm, and occasionally even cold. Tested with the ether spray and cauterization the results were similar. In the upper half of this dysesthetic area, or band, there was a considerable degree of analgesia. Throughout the entire area there was a hypesthesia, and in several spots the size of a dime complete anesthesia. With the probability of incipient syringomyelia in view, careful tests for dysesthesia elsewhere were made, and as was the case with the motor disturbance, none was found other than as herein indicated. After two or three

weeks electrization, the thermo-anesthesia and analgesia became much less apparent, and indeed I may say that by repeated tests on different days I failed to elicit the marked departure from the normal type, so well seen when first examined. On one or two occasions, however, they would apparently recur, but in a more uncertain and negative manner than at first. On the other hand, the anesthesia, or at least the hypesthesia, was always manifest along the whole narrow band, but never above the elbow. There were no oculopupillary symptoms present. The reflexes were nowhere increased.

The salient features of this case then are: (a) Isolated atrophy of two thumb muscles, subacute in its beginning, showing, so far as the limited time of observation will permit any inference, no progressive tendency; (b) definitely localized dysesthesia.

The sensory disturbance present differs from the true dissociated sensory type of syringomyelia. In the latter, preservation of tactile sensibility is the rule, in this case hypesthesia prevails. It is true that even in syringomyelia there may be disturbance in the tactile element of sensibility. Roth found it in the proportion of 1 to 18, and considered it to indicate an invasion of the posterior tracts by the gliosis or cavity formation. When it occurs, therefore, we should expect it to be, and it

Suggested diagram of location of lesion. (After Buzzard.)

really is, a late and not an early symptom, being due to the extension of the syringomyelic process. I am unable to find the presence of marked anesthesia as an early symptom of syringomyelia anywhere recorded. I also find but little reference to early syringomyelia, and find no case of it with such slight unilateral muscle atrophy, and such strictly localized sensory change as shown in this case. It appears to me rather difficult to picture a gliosis or cavity formation even in the incipient stage—so situated as to occasion the lesion peculiar to my case. Syringomyelia may, therefore, be excluded.

This case comes under the class of uniradicular palsies, so well described by Buzzard in *Brain*, 1902, where the meager literature of the subject is also cited. Six cases are here detailed, all of which involved one or more of the roots of the brachial plexus; some of the fifth and sixth cervical, others the first dorsal, and in one case the lesion appears to have attacked the fifth cervical and first dorsal, i. e., the upper and lower roots of the plexus. There was thus produced a partial Erb, and a partial Klumpke. The root involved in my case is the first dorsal, and evidently in such a way as to exclude the sympathetic fibers for the pupil.

It is difficult to understand why the atrophy should be confined to the two superficial thumb or thenar muscles,

and why the little finger muscles at least should not also have been involved. The interossei, lumbricals and the long flexors of the hand are not exclusively supplied by the first dorsal, but receive their innervation in part from the eighth cervical. Kocher's table gives the eighth cervical complete control over the long finger flexor muscles, and the first dorsal, according to the views of the same author, controls all the small hand muscles. If we adopt this scheme of Kocher's, with which that of Bruns closely corresponds, but from which that of Edinger and Starr is at variance, we shall be able to understand why a lesion of the first dorsal root need not involve the long flexors of the fingers and hand.

It will be less easy to explain why the other small hand muscles should escape and the atrophy involve only the two superficial thumb muscles. It must be remembered, however, that it was stated that the whole right hand was weaker than the left, and although electric changes or atrophy could not be demonstrated in the hypothenar, interossei and lumbrical muscles, a certain degree of implication may still be present or may yet set in. The site of the lesion must be above the point where the ramus communicans of the sympathetic to the ganglion stellatum is given off, as no oculopupillary symptoms were present.

The absence of any increase of reflexes, so frequent in syringomyelia, may also serve to indicate that the cord itself is intact. The presence of sensory disturbance and the course of the affection warrant one, I think, in excluding atrophy of the Aran Duchenne type.

Now with reference to the sensory disturbance, the point is well made by Buzzard that there was no knowledge of any change in sensation on the part of the patients themselves, whereas in peripheral neuritis subjective sensory phenomena are the rule. Buzzard thinks that this absence of subjective sensation of numbness or tingling may prove a differential diagnostic point between root and peripheral neuritis. The character of the dysesthesia, furthermore, is allied to that experimentally produced by a severance of a single posterior root, and as has already been mentioned, different from that form seen in syringomyelia.

I quote from Buzzard the original words of Sherrington:

1. The disturbance of skin sensation produced in macacus by severance of spinal roots is to some extent a dissociative one.

2. In macacus the destruction of a single sensory spinal root, at least in the case of certain spinal nerves, entails in a limited skin area, much smaller than the total skinfield of the root, an abolition of heat sensitivity without abolition, but with some concomitant impairment of actual sensitivity.

3. In the skin of macacus the pain field and the heat field of a single sensory spinal root, at least in the case of certain spinal nerves, are each less extensive than is the "touch field" of the same root.

The changes in my case correspond closely to this description of Sherrington's, i. e., there was a small algesic and temperature field and a larger touch-field. The site of the lesion is in my case near the point of junction of the eighth cervical and first dorsal to form the outer cord of the brachial plexus, but attacking a single nerve only, viz., the first dorsal.

The lesion must be beyond the posterior root ganglion, as evidenced by the absence of herpes. The limitation of the atrophy to the two superficial thenar muscles, and, furthermore, the absence of any sensory changes in the zone of the first dorsal root above the elbow would seem to plead for a very small vascular lesion of some kind.

The prodromal period in my case, or rather the pre-atrophic stage in which the only symptoms were pain and weakness, was rather longer than in any of the cases mentioned in Buzzard's paper, but it is my impression that the atrophy was present for a much longer time than the patient stated.

As to the character of the lesion I have nothing to offer that would not be entirely speculative. The only improvement has been the absence of cramps (which I never saw) and the decrease of the pain. The sensory changes became less marked and more variable, but the atrophy remains unaltered.

DISCUSSION.

DR. H. T. PATRICK, Chicago—I would suggest that this case was possibly an example of the local atrophy which affects most frequently the hand muscles of persons who are engaged in certain occupations. It is usually accompanied by pain. The cases nearly always recover. They are generally seen in just the kind of people Dr. Wolfstein has described—tall, slender, poorly nourished individuals. In my experience, it occurs most often in seamstresses and in girls who work at book-binding, as well as in other occupations where there is a particular strain on a certain group of muscles. It is, perhaps, allied to some forms of writer's cramp or palsy. Very little can be found on the subject in the text-books. I do not offer this as a positive diagnosis in Dr. Wolfstein's case, but merely throw out the suggestion, as he did not mention this possibility in speaking of the diagnosis.

DR. WHARTON SINKLER, Philadelphia—The trouble was apparently of peripheral origin. I recently saw a stunted, undeveloped boy of fourteen, who worked hard at manual labor and was a heavy cigarette smoker. He got into a fight, and received a severe blow on the back of the hand. Quite a swelling developed, and subsequently there was a loss of power in the extensor muscles of the hand, with marked atrophy of the thenar and hypothenar eminences. In that case the trouble was undoubtedly of peripheral origin.

DR. ALBERT E. STERNE, Indianapolis—I also was impressed with the idea that the trouble in Dr. Wolfstein's case was in all likelihood due to the occupation of the woman. Dr. Patrick's statement that these patients usually recovered is not in accord with my experience. Still, recovery often occurs. Three years ago I noticed that the ball of the first finger of my right hand, as well as the thenar eminence, began to shrink. This was accompanied by muscular twitchings, which became so violent that I could scarcely hold a knife. I worried a good deal about it, as there was also some pain, and in addition to this I had a localized pain on the left side of the head. If I had seen such a case in my office, I would unquestionably have kept in view the possibility of a gross lesion in the brain. At the Atlantic City meeting I took occasion to speak to Dr. Patrick about it, and he advised me to spend a month at the seashore. I took his advice, and the twitching and pain disappeared. It has since recurred in different regions, but has not been so persistent. In this connection I would recall that in my own paper I referred to this local neurasthenia as being vascular, due to overcirculation and overexcitation, and I am inclined to think that the case reported by Dr. Wolfstein might belong to the same category.

DR. F. W. LANGDON, Cincinnati—I saw this case with Dr. Wolfstein. At first glance, I was inclined to believe that it was one of early syringomyelia, but the later symptoms made me more doubtful, and finally I practically excluded that idea. Dr. Wolfstein's explanation that the symptoms were due to a root lesion I regard as very probable, more so than the explanations offered by those who have taken part in the discussion. An occupation atrophy could hardly be conceived of that would affect merely one or two muscles. The distribution of the atrophy and the sensory symptoms would also exclude traumatism, to which reference was made by Dr. Sinkler. I see no reason why there should be these objective sensory disturbances as the result of overworking a few muscles. I

think the probabilities are in favor of the case being one of radicular palsy, and this view is strengthened by the cases of Buzzard referred to by the author. My experience with cases of muscular twitching has not been always as happy as that of Dr. Sterne in his own case. Some years ago I was consulted by a physician of 40, who had been overworked and somewhat worried. He complained of a loss of dexterity of the right hand, with twitching and numbness. After examining him, I advised him to spend a season at the seashore. This he did, and on his return his health was apparently restored. Shortly after he was found dead in his bathroom, death having been caused presumably by cerebral apoplexy. This shows that we can not always eliminate the presence of cerebral trouble, even if the local symptoms improve.

DR. D. I. WOLFSTEIN—The simple explanation of these symptoms naturally appeals to us, and we thought first of the existence of an occupation neurosis, but on account of the train of symptoms that developed and the peculiar sensory disturbances of the dissociated type, I believe that explanation of the case can be positively excluded. The woman's occupation was that of a general houseworker, and she did not spend much of her time at laundry work.

ACCIDENTAL PERFORATIONS OF THE UTERUS.*

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DES MOINES, IOWA.

Accidental perforations of the walls of the uterus by instruments in gynecologic practice and in efforts to produce abortion are probably more frequent than the reports of cases would appear to show. The circumstances under which these accidents occur are generally such as would lead them to be passed over and the results which sometimes follow are often no doubt assigned to other causes.

Dr. Brothers of New York has been able to collect sixty-six recorded cases of accidental perforations of the uterus, of which seventeen died and forty-nine recovered.

The use of the sound and the curette in gynecologic practice is probably the most frequent cause of this accident in legitimate practice. The facility with which the sound can be used in exploring the interior of the uterus and in repositing a retrodisplaced organ is no doubt responsible for some of these accidents.

There are no means by which we are able to measure the resistance which the uterine walls may offer to the passage of an instrument, or to determine the ease with which the walls may be penetrated. It is well recognized that when the tissues of the uterus are much softened by inflammation or by degenerative changes in connection with disease that a curette or sound will pass through into the abdomen with so much ease that the fact may not be at once recognized, and it may be sometimes assumed that the depth of the uterine cavity is much greater than the facts will warrant, or it may be thought that the sound has passed into a fallopian tube. It is possible that the instrument has been diverted into the tube, but the probabilities are very much greater that the abdominal cavity has been entered.

When the walls of the uterus are apparently healthy they may be penetrated with greater facility than is generally supposed. This fact will be illustrated by a case related in this paper. While I have never had the accident happen in my own practice, I have always been apprehensive of it, and have been governed by a degree of

care which has equaled that exercised in any work I do. There can be no doubt that examinations of the uterus by the sound are altogether more frequent than they should be. I am so certain that information as to the condition of the uterus can be better obtained by other means that I rarely use the instrument.

The curette is no doubt a very useful instrument in a certain class of chronic intra-uterine affections, and often indispensable, but at the same time a very dangerous one. I am quite sure that it is often used in an indiscriminate manner, and as a routine treatment in a great variety of uterine difficulties that could be better treated by other means with a greater prospect of permanent results.

The perforation of the uterine walls by the curette would not be so serious if it were not for the associated treatment which is so often regarded as essential when the curette is indicated. I refer to irrigations. If the uterus is perforated and irrigation is employed a considerable amount of infection material may be washed into the abdominal cavity and the chances of setting up a fatal peritonitis are great. The infection from the curette is serious enough, but when in addition to this the washings from the uterus are carried into the peritoneal cavity, the risk is greatly increased. It should be remembered that when the curette enters to an unexpected depth there is a great probability that the instrument has passed through the walls and the irrigation should be refrained from unless it can be absolutely demonstrated that no perforation has taken place.

Our attention is called from time to time to cases where simple curettage has been performed, followed by a fatal result. It may fairly be presumed that in some of these cases the uterus has been perforated and the peritoneal cavity infected without the knowledge of the operator. Indeed, we have known of cases where not only were the uterine walls perforated, but a considerable quantity of gauze was packed into the abdominal cavity through the opening in the uterus, which was only discovered on postmortem examination.

In April of this year a case came under observation which illustrates the ease with which a perforation of an apparently healthy uterine wall can be accomplished, and what needs to be done in cases of this kind.

CASE 1.—A woman about thirty years of age had acquired considerable skill and dexterity in producing abortion by introducing instruments into the uterus. Suspecting herself to be pregnant or of the possibility of being pregnant, she, about 4 o'clock in the afternoon, straightened out a hairpin, then bent the end over on the main stem to make a close loop so as to avoid the sharp end, which she thought might injure the uterus. She then lay down and introduced the loop end of the hairpin into the uterus, allowing the opposite end to project from the os so that she might remove it at pleasure. After resting in bed two hours she attempted to seize the pin with thumb and finger to remove it, but it slipped from her fingers. She attempted to seize it again, and again it slipped from her and disappeared within the uterus. She at once summoned her doctor, who attempted to secure it with a pair of uterine dressing forceps, but failed. The woman was now in great fear of the results, and her physician finally persuaded her to go to the hospital, where I saw her the next morning. She was so certain that the hairpin was in the uterus or the abdominal cavity that she was willing to submit herself to any operation that was deemed necessary to secure it. The patient was anesthetized, the cervix dilated and the uterine cavity explored with a large blunt curette, but nothing could be felt. The abdomen was opened and the uterus freely exposed. The organ was perfectly normal in size and appearance and free from any evidence of recent or old inflammation or

* Read at the Thirteenth Annual Meeting of the Western Surgical and Gynecological Association, held at Denver, Dec. 28-29, 1903.