The influence of trauma in the first case may be disputed, but it certainly cannot be denied, since the man never felt quite the same after it up to the time of the development of more serious symptoms. In the second case the symptoms may be referred definitely to the trauma.

In both cases syphilis must be regarded as extremely doubtful; the absolute denial in the first case, and the very doubtful nature of the sores in the second, render it highly improbable. In the second case, also, antisyphilitic remedies were of no avail.

Pantzer¹¹ has reported a case with somewhat similar symptoms, associated with fracture and displacement of the laminae, relieved completely by laminectomy. In the absence of any discoverable vertebral changes such a condition seems improbable in these cases.

The very gradual onset of the symptoms in the first case renders the diagnosis of spinal meningeal hemorrhage most improbable. The partial recovery and the greater severity of the earlier symptoms in the second case might suggest a hemorrhage, but we must bear in mind that the symptoms did not develop for over a week and that there were no pronounced root symptoms or symptoms of a focal lesion of the cord, and no sudden pain or muscular spasm. In both cases the symptoms suggest changes in the pyramidal tracts, and I am inclined to the belief that in both we have to do with a degenerative process in the cord itself. In the absence of more definite data, however, support for that belief must be sought in the record of other cases.

Schmaus, in the paper to which I have already referred, and in a subsequent communication,¹² reports certain cases where the autopsies showed changes similar to those produced experimentally in rabbits, foci of softening, swelling and degeneration of axis cylinders, degeneration of ganglion cells, fascicular degeneration, especially in the lateral tract and not secondary to other lesions, occasionally gliosis. In one case there was a hemorrhage which was apparently of recent date and was regarded as secondary to the softening. In the majority of cases the vertebral canal was intact. These changes in the lateral columns recall the degeneration found in the same region by Edes¹³ ten years ago. In a previous paper¹⁴ I have cited further evidence bearing upon this point.

Gowers¹⁵ reports a case which, according to Watson, must be a myth. "A lady was severely shaken in a railway collision. She seemed, immediately after the accident, to have suffered no injury, but in a few days paraplegia developed, and from its consequences she died six weeks after the accident. Throughout the dorsal region of the cord I found indications of subacute myelitis, chiefly in the white columns, varying in its extent in different regions, but in most parts considerable in the pyramidal tracts."

Gowers thinks that "in the face of such a case as this, it is superfluous to discuss the question whether or not the cord can suffer concussion." The evidence seems clear from such cases that we may have changes

in the cord, without hemorrhage, fracture or direct crushing, of gradual onset and producing grave results. I have omitted to cite cases presenting the symptoms of ordinary acute myelitis, coming on soon after an injury, where no evidence of fracture or crush of the cord can be obtained.

There is yet another class of cases which Page and Watson ignore, yet where, according to all the knowledge we have of nervous pathology we must hold that the injury has given rise to changes of the most gradual and insidious onset, which begin with the most minute alterations, probably not much greater than those found by Hodge in fatigued cells, where the symptoms do not develop until long after the injury, and where the progress is gradual yet persistent to a fatal termination. I refer to the cases of tabes and spinal progressive muscular atrophy of traumatic origin. Both are, of course, rare, but trauma may be the sole discoverable cause of tabes according to Erb, and traumatic muscular atrophy is probably more common. Not long ago I reported¹⁶ a case of progressive spinal muscular atrophy which gradually developed after a fall on the elbow. There may have been some neuritis at first; at any rate the arm was very painful, but the atrophy extended and bulbar symptoms finally developed. Such cases prove conclusively that the cord may be affected by injury where there is no gross lesion at the time and where the symptoms are not of immediate onset.

Finally, we have the cases which Page and Watson accept, where there is spinal hemorrhage, fracture of the vertebræ, crushing of the cord, etc., which I need not cite.

From the evidence thus collected it becomes clear that the position taken by Watson and Page is untenable. There are cases where the symptoms are in large part if not entirely due to disturbances of the spinal cord, and where the cerebral and psychical factors may be eliminated. Changes occur in the cord from injury where there is no fracture or dislocation of the vertebræ, no crushing of the cord, no spinal hemorrhage, and where the vertebral canal is intact. Such changes, furthermore, may be of gradual onset, and symptoms may not develop for some time after the injury. As we know that severe cases occur with marked lesions in the cord, post-mortem, it is only fair to suppose that milder cases may also occur where there are less marked lesions in the cord which either recover or persist, causing partial disability without a fatal termination.

PUERPERAL ECLAMPSIA: A REPORT OF THREE CASES.

BY H. D. ARNOLD, M.D., ROXBURY, MASS.

OF the three cases reported in this paper, the first occurred in my private practice, and the other two at the Boston Lying-in Hospital during my service as house-officer. I am indebted to Dr. C. M. Green, in whose service these two cases occurred, for permission to report them, thus adding materially to the interest of the paper.

All three of the patients were primiparæ. The first case is one of post-partum eclampsia, with recovery. The second case is one of ante-partem eclampsia occurring about the end of the sixth month of pregnancy.

¹⁶ P. C. Knapp: Hereditary and Traumatic Motor Tabes, Boston Medical and Surgical Journal, October 1, 1891.

¹¹ H. O. Pantzer: Two Cases of Laminectomy, New York Medical Journal, August 26, 1893.

¹² H. Schmaus: Zur Casuistik und Pathologischen Anatomie der Rückenmarks erschütterung, Archiv für klin. Chirurgie, xiii, 112, 1891.

¹³ R. T. Edes: The somewhat frequent Occurrence of Degeneration of the Postero-lateral Columns of the Spinal Cord in so-called Spinal Concussion, Boston Medical and Surgical Journal, September 21, 1882.

¹⁴ P. C. Knapp: Art. cit.

¹⁵ Gowers: A Manual of Diseases of the Nervous System, 2d ed., 1, 591.

Labor came on without interference, and the child was born alive, but lived only a few minutes, as it was premature. The mother made a good recovery. In the third case the patient was in labor when brought to the hospital. She had had sixteen convulsions already, and was in a very weak condition. A living child was delivered after the twentieth convulsion, and survived; but the mother died later, having had twenty-four convulsions in all. The chief interest in these cases centres in the treatment, which will be reported in detail.

In discussing the treatment of a disease like puerperal eclampsia, about the nature of which divergent views are held by the profession, it seems important that the view of the physician treating a case should be clearly understood. The more generally accepted view is that the eclampsia is due to the failure of the kidneys to perform their function. Owing to this failure, there remains in the circulation some substance, otherwise eliminated, which acts as a poison—its manifestations coming through the nervous system. Pregnancy is in some way the cause of this failure of elimination by the kidneys. Thus much is common to all views of eclampsia, except for those rarer cases (if they exist) where eclampsia is due to reflex irritation. If such cases do exist, and the kidneys are fulfilling their whole function of elimination, it would be conducive to clear reasoning to have these rare cases set apart in a class by themselves, so as not to obscure the consideration of the common form of eclampsia.

When we come to the question of the nature of this substance which acts as a poison, and of the way in which pregnancy causes renal inadequacy, we touch the points on which authorities differ. The view that the substance which acts as a poison is a normal product of the cell-life of the individual, perhaps some form of leucomaine, and that in a healthy person it is only the adequate elimination by the kidneys which prevents the constant accumulation of this poison, seems to me the most probable hypothesis in view of recent investigations. In like manner it seems probable that the pregnant uterus affects the kidneys through pressure upon the ureters. But these views do not carry in my mind the same weight as the fact of the existence of some poisonous substance in the blood as a cause of the convulsions. That is the essential fact, from a clinical standpoint. Puerperal eclampsia is a toxæmia. What the poison is, and how it gets there, are matters of minor importance compared with the problem of getting rid of it. I emphasize this, because I think in some cases too much attention in treatment is paid to the uterus and kidneys, and the treatment of the general condition is not prompt or energetic enough. The removal of the child may give no immediate relief where such relief is necessary, and the woman may die before we get her kidneys into working order if we depend chiefly upon them for elimination.

How shall we get rid of the poison? Obviously we must in some way secure its elimination from the body. In some cases this may be accomplished through the ordinary channel of the kidneys; but usually we must call in the skin and gastro-intestinal tract as auxiliaries, and often we must depend entirely on this vicarious elimination, and leave the kidneys entirely out of our calculations. Could we estimate accurately the amount of poison to be eliminated and the exact condition of the secretory function of the kidneys, I believe we should divide our cases of eclampsia into classes, and

treat the different classes quite differently. A very mild case of post-partum eclampsia presents a different problem from that of a severe case of ante-partum eclampsia. In the former case, the original cause of the trouble (the child in the womb) has disappeared. The kidneys are freed from obstruction and will shortly resume their function fully. Let us suppose that the kidneys are very slightly affected, there is an accumulation of only a small amount of poison in the blood, the patient is easily susceptible and her nervous system has lost a good deal of its resistant power through a tedious labor. She has a mild convulsion, as a result of slight poisoning. Such a case may be successfully treated by using a sedative to render the nervous system less susceptible to the effect of the poison and waiting for nature to eliminate the poison by the rapidly improving action of the kidneys. The difficulty lies in the recognition of just such a case; and the first case reported in this paper shows that we must always be ready to change our tactics completely and push vigorous treatment if necessary. As a general rule, it is not wise to place much reliance on the kidneys in the immediate treatment of eclampsia. The safer plan is to supplement the action of the kidneys with more or less diaphoresis and catharsis, according to the severity of the case, and always to be ready to act temporarily as if the woman had no kidneys. I believe that the successful treatment of eclampsia depends more upon an intelligent understanding of the severity of the case than upon the adoption of any routine treatment, however good it may be.

Other questions of treatment arise, and important ones, too, but it is not necessary to discuss them at this point. In seeking to emphasize one or two aspects of the treatment, I do not wish to be understood as underestimating the importance of other measures. My object is not to give an exhaustive discussion of the treatment, but rather to explain the line of treatment which is common to the following cases.

CASE I. Primipara, married, eighteen years of age. Post-partum eclampsia after a labor at full term.

When first seen by me she was in labor, with the os about the size of a silver dollar. This was at 3 P. M., September 1, 1892. She had had irregular pains all day. The labor was uneventful, except that the first stage was tedious and the patient showed a very nervous temperament. Labor ended at 2.15 A. M., September 2d, by the birth of a female child weighing seven pounds.

Eleven hours later I called again. The patient was apparently all right. She had not slept, however, although she had had little sleep for forty-eight hours. She complained of headache and was rather restless, but this was not unexpected on account of her temperament. About two hours previous to my visit she passed nearly a pint of urine. It was high-colored, but otherwise apparently normal. During the labor she had passed a satisfactory amount of urine. She was given a dose of bromide of sodium before I left, and the nurse had orders to repeat it if she did not sleep.

An hour later I was summoned on account of a convulsion. This occurred twelve hours after delivery. I was prepared for a nervous explosion of some kind. With a nervous temperament there were family troubles and reasons why the child was very unwelcome. She had taken a violent dislike to it, was opposed to nursing it, and was greatly disturbed by the urging of the

family and nurse that she should nurse it. Shortly before the convulsion she had complained of pain in the abdomen, but it had been over the uterus and not in the epigastrium, and she was quickly relieved by hot applications. There had been no other symptoms except the headache and nervousness before mentioned.

At the time of my arrival she was fully conscious and was quiet. Her mind was perfectly clear. The convulsion was of very short duration, and the description which I obtained was not characteristic of any particular form. There was no œdema of legs or feet. She said she had worn during the latter part of pregnancy the same shoes as usual without discomfort. She was the picture of health, and could remember no sickness.

I decided that the convulsion was probably hysterical in character, on account of the secretion of urine and the absence of confirmatory evidence of eclampsia, and felt so sure of it that I did not catheterize her for a specimen of urine, fearing that she might get too much worked up by so doing. Subsequent events proved that I was wrong. However, it may be remarked in passing, to show that my view was not unreasonable, that about a week later (when eclampsia was out of the question) she had an hysterical attack not unlike this but more violent, and that she continued to be excited and verged on mania for two days. The patient was given another dose of bromide, and was soon asleep.

The second convulsion occurred at 6.30 P. M., four hours after the first. She awoke from a quiet sleep, and passed almost immediately into this convulsion. She was catheterized, and more than half a pint of urine was obtained — the secretion of seven hours. It was darker in color than that passed at noon, and contained an abundance of albumin. Recognizing the seriousness of the case, I advised a consultation and Dr. Clement responded to the call.

It was thought that this would be a favorable case to treat by the subcutaneous injection of morphine to check the convulsions. The convulsions had been rather mild. The patient quickly regained consciousness, and was perfectly clear mentally between the attacks. She had secreted one and one half pints of urine in sixteen hours, and it seemed probable that the kidneys would quickly resume their normal excretion. The skin was moist, and it seemed sufficient to favor perspiration by blankets and heaters. In addition, she was to have elaterium to favor the action of the bowels. The prognosis seemed, on the whole, favorable.

8 P. M., September 2d. Morphine, gr. $\frac{1}{4}$, subcutaneous injection. She soon fell into a deep sleep.

9.30 P. M. Third convulsion. I was not present, but arrived shortly afterwards. She passed into this convulsion from a deep sleep due to morphine, without waking. This convulsion was much more severe than the previous ones and lasted longer. I found her at 9.45 extremely drowsy from the morphine, not easily roused, and with pupils well contracted.

9.50 P. M. Elaterium, gr. $\frac{1}{2}$.

10.30 P. M. Elaterium, gr. $\frac{1}{2}$. The effect of the morphine is not quite so profound and she was given a subcutaneous injection of morphine, one-eighth of a grain. This produced complete narcosis. Pulse 84.

12 midnight. Fourth convulsion. She passed into this from deep narcosis. This was the first of the convulsions which I witnessed. It lasted eight minutes. The movements were not excessively violent. The

nurse said it was longer than the last one, but not so violent. It was probably modified by the morphine. The recovery from this convulsion was decidedly slower than before. The pulse during the convulsion was 96. The temperature in axilla immediately afterwards was 99.4°.

12.30 A. M., September 3d. Elaterium, gr. $\frac{3}{4}$. She could be aroused to swallow with the greatest difficulty, and probably could take nothing more for some while, as the stupor was increasing. She was catheterized, and four ounces of dark urine were obtained — secreted in five hours. The situation was now grave in the extreme. Convulsions had continued in spite of the morphine — in fact, they came oftener. The interval between the first two convulsions was four hours, the next interval was three hours and the last interval two and one-half hours. The succeeding stupor was becoming more marked, and with the aid of morphine threatened to be continuous. The amount of urine secreted was diminishing instead of increasing, and was of a worse character. The skin had become dry and harsh. The patient's countenance had changed decidedly for the worse. I left to get a reliable croton oil and some fresh pilocarpine for subcutaneous use.

1.30 A. M. Pilocarpine, gr. $\frac{1}{8}$, by subcutaneous injection. Five minutes later she vomited a small amount of green, watery vomitus. At 1.40 she was sweating profusely. Fresh heaters and blankets were applied.

1.40 A. M. Croton oil, two drops in some olive oil, on the tongue. In five minutes there was more green, watery vomitus.

2.10 A. M. Croton oil repeated. Vomited immediately.

2.30 A. M. This was the time when another convulsion was due, if it came after the same interval as the last one. I could not help feeling anxious, although the patient was coming out from her stupor somewhat, and her countenance looked decidedly better.

2.35 A. M. Pilocarpine, gr. $\frac{1}{8}$, by subcutaneous injection. She was still perspiring, but not freely. In five minutes there was profuse perspiration; and between this and 3 A. M. she vomited several times profusely.

3.40 A. M. Owing to the elimination by perspiration and vomiting, patient had improved so much that she could be roused to swallow. She was given elaterium, gr. $\frac{3}{4}$.

5.20 A. M. First dejection. This was thin, fecal, and moderate in amount. This was seven and one-half hours since a cathartic was first given. During this time she took six-fifths of a grain of elaterium, which was apparently retained, and two doses of croton oil, which were vomited.

At 5.45 she vomited copiously. Three dejections followed before 8 o'clock, and two more before 10.30 A. M. This made six dejections.

The patient was now entirely conscious and her mind clear and bright. She had no memory of the night. There was no headache. Her pulse was good. Four ounces of urine were drawn by catheter. This was the secretion of ten hours, and it showed that the kidneys were of little use as yet.

Patient was ordered thirty grains of compound jalap powder at 12, and at 1 P. M. cream-of-tartar water, ad lib. Her diet was plain milk. At 3 P. M. there was another dejection. By this time threatening symptoms had set in again. She was getting decidedly dull and

had a severe headache. Evidently the elimination of the poison was not keeping abreast of its formation.

3.30 P. M. Elaterium, gr. $\frac{1}{2}$. She continued the cream-of-tartar water in large amounts. She was now given ten grains of chloral hydrate to guard against convulsions. This was repeated at 4.30 and 6.15 P. M. Still her headache grew worse.

At 4 and 5 P. M. she had dejections. At 5.45 P. M. she was given one-fifth of a grain of elaterium. Four dejections followed before 8 P. M. She also passed a few ounces of urine, with one dejection. About five ounces of urine were drawn by catheter at 8 P. M., which was of a lighter color and showed evident improvement in the action of the kidneys.

Notwithstanding this her headache grew worse and worse. She was restless in spite of the chloral, and she could not sleep. Her skin had been moist and she was perspiring at 5.30 P. M., but her skin was now getting dry and harsh again. Pulse 84. Symptoms grew worse until 9 P. M. She was then given one-tenth of a grain of pilocarpine subcutaneously. This was followed by vomiting and profuse perspiration. A dejection followed at 10 P. M. and 1 A. M. Elaterium was given, in the dose of one-fifth of a grain, at 2, 5 and 7.15 A. M. There were three dejections between 6 and 9 A. M.

It was now the morning of the second day after the convulsions. She was again clear in her mind. There was no headache. Pulse 96. Temperature normal. The kidneys were acting much better. The urine was lighter colored and was passed in fair amounts. The amount was not accurately determined, for part was passed with her dejections.

From this time on there was steady improvement through convalescence. The bowels were kept freely open for two or three days, by compound jalap powder. Acetate of potash was substituted for cream-of-tartar water as a diuretic. The urine improved steadily. After a few days there was only a trace of albumin, and on the twentieth day after confinement, the albumin had entirely disappeared.

The feature of this case which is to me of most interest is the threatened recurrence of the eclampsia on the second night after complete relief for some hours during the day. This was apparently due to the slowness of the kidneys in resuming their functions and the failure of elimination to keep pace with the continued formation of the poison in the system. I felt obliged to use pilocarpine again and to resort to brisk purging. The pilocarpine at no time in the case caused salivation to any degree nor any accumulation of secretion in the bronchi.

CASE II. Primipara, married, twenty-two years of age. Ante-partum eclampsia between the sixth and seventh month of pregnancy. This patient entered the Boston Lying-in Hospital February 12, 1890.

For two weeks previous to this she had had headaches. Two days before her entrance to the hospital she complained of a severe, shooting pain in the epigastrium and the right iliac region. There was considerable nausea, but no vomiting. She complained of a "sick headache." On the day before entrance to the hospital there was a good deal of vomiting and retching. During these two days she was under a physician's care, and was treated symptomatically for vomiting, pain, etc.

Between midnight and seven o'clock in the morning of the day she came to the hospital she had four con-

vulsions. The doctor saw her first at 7.45 A. M. She was partially unconscious, but could be roused if spoken to sharply. Four ounces of urine drawn by catheter showed a large amount of albumin. Convulsions occurred at 8.20 and 9 o'clock. Ether was administered. Later she was brought to the hospital in a hack, lightly etherized. She was admitted about 11.30 A. M., February 12th.

At that time she was unconscious, and could not be thoroughly aroused. She was restless, resisted any interference, and needed restraint. There was some oedema of the feet, slight oedema of the face, but none of the hands. Patient could not swallow. Nine ounces of urine were obtained by catheter. Albumin was present in abundance — more than one per cent. The sediment showed numerous casts of all kinds, blood-globules and renal epithelium.

Dr. C. M. Green was sent for. It was decided not to attempt to bring on labor. Labor had not begun. For the mother, a forced labor would be detrimental, and delivery did not promise immediate relief. As for the child, its chances were almost too small to consider. There was just a possibility that labor might not come on if the patient were let alone. If labor came on itself the child might be born alive, but its chances of surviving would be exceedingly poor. In a forced labor it would probably be still-born. No good and a probable harm would attend an attempt to bring on labor. It was decided to rely entirely on vicarious elimination through the skin and bowels.

1.10 P. M., February 12th. Croton oil: three drops were given in olive oil by rectum, as the patient could not swallow.

1.15 P. M. Pilocarpine: subcutaneous injection of one-quarter of a grain. Profuse perspiration in five minutes. There was marked temporary salivation. Blankets and heaters were applied, and a profuse perspiration lasted more than an hour. Perspiration lasted to a less degree for a longer time, and the pilocarpine was not repeated until the end of two and one-half hours.

1.30 P. M. A severe, typical convulsion. Ether was applied, but did not shorten the attack.

2.10 P. M. Croton oil: two drops were placed on the tongue, diluted with olive oil. One hour had now elapsed since the croton oil was given by rectum.

2.25 P. M. Turpentine enema, without result.

3.10 P. M. Chloral: thirty grains by rectum, as patient is very restless. The pulse is 120, of good strength, but not full or bounding.

3.30 P. M. Profuse vomiting of greenish, watery material.

3.45 P. M. Pilocarpine: subcutaneous injection of one-sixth of a grain. It is two hours and a half since the first injection, and the skin is not very moist.

5 P. M. Croton oil: three drops by rectum.

5.30 P. M. Catheterized. Only one ounce of urine was obtained, of the same character as before.

5.45 P. M. Her condition had improved enough for her to be able to swallow. Elaterium, gr. $\frac{1}{2}$, by mouth.

6.30 P. M. Turpentine enema.

7 P. M. A large, loose, watery dejection. This was six hours from the beginning of treatment.

7.15 P. M. Pilocarpine: one-sixth of a grain subcutaneously. It was now three and one-half hours since the last injection, and it was desired to continue the elimination by the skin for some time yet. Re-

tween this time and eight the next morning the patient took forty ounces of cream-of-tartar water and about half a pint of milk. To the milk was added two drachms of brandy every two hours after 8 P. M., as the pulse was weak.

8 P. M. Turpentine enema, without result.

10 P. M. Elaterium, gr. $\frac{1}{2}$. This was repeated at 11 P. M., at midnight and at 1 A. M.

12.30 A. M., February 13th. A turpentine enema gave a small defecation. One ounce of urine, drawn by catheter, had a better color, and contained only one-half per cent. of albumin. The kidneys have secreted only about an ounce of urine in seven hours.

At midnight the patient was found to be in labor, with the os dilated to about half-an-inch in diameter.

4.30 A. M. Profuse vomiting began, and lasted at intervals for one and one-half hours.

4.30 A. M. Large watery defecation.

5 A. M. Large watery defecation.

Patient had now (5 A. M.) been under treatment for sixteen hours. Her condition was much improved. She was still unconscious, but was quiet, and breathed quietly and easily. Her countenance looked much better. Pulse was 100 and of good strength. Drugs were given for twelve hours.

Labor progressed easily during the day, and the child was born at 4.40 P. M. It was alive, but was premature, and lived only a short time.

In the meantime (at 8.30 A. M.) nine ounces of urine (secreted in eight hours) were drawn, showing one-fourth to one-half per cent. of albumin and a sediment with fewer casts and blood-globules. A number of loose defecations occurred during the day.

In the evening she began to pass urine in bed involuntarily. At 10 P. M. she could answer questions in a semi-intelligent manner when aroused.

The next morning, February 14th, she was entirely conscious. Steady improvement followed. She was discharged on the fifteenth day after entrance to the hospital. She was well, except for a slight trace of albumin in the urine. The urine contained no casts.

Summary.—The child and the uterus were left entirely to nature. A quiet, easy labor gave a living child, which died because it was so premature (about six and a half months). Entire dependence was placed upon vicarious elimination. The patient was too restless to take a hot-air bath if it had been desired.

Elimination was obtained first through the skin by the aid of pilocarpine. The first dose was one-quarter of a grain, followed in two and one-half hours by one-sixth of a grain, and again in three and one-half hours by one-sixth of a grain. In all, the action of pilocarpine was continued about nine hours. Vomiting aided in elimination at the end of two and one-half hours.

At the end of four and one-half hours she could swallow, and was given one-fifth of a grain of elaterium. She had previously had croton oil twice by rectum and once by mouth, and had had two enemata. The first defecation occurred at the end of six hours. The second, a small one, at the end of eleven and one-half hours, after four-fifths of a grain of elaterium and an enema. The third and fourth defecations were copious ones. They came fifteen and one-half and sixteen hours after treatment began, but were undoubtedly due to the elaterium given between 10 P. M. and 1 A. M.—that is, from six to four hours pre-

viously. In all, one grain of elaterium was used, besides croton oil and enemata.

There were six convulsions before entrance to the hospital and one afterwards, seven in all.

CASE III. Primipara, single, nineteen years of age. She was at full term, but whether the convulsions preceded the onset of labor or not is not known. She was admitted to the Boston Lying-in Hospital March 29, 1890.

The first convulsion occurred about 5.30 on that morning. She recovered consciousness, and had a second convulsion about 6 A. M. She was never fully conscious after this. Before reaching the hospital at 2.45 P. M. she had had sixteen convulsions.

At the time of her arrival she was unconscious; her breathing was deep, but not stertorous; her pulse was 144 and weak; the abdomen was in size that of full term; and the os was half dilated.

The seventeenth convulsion followed while preparations were being made to catheterize her. The pulse ran up to 210 during the convulsion. Ether was applied immediately as soon as the convulsion began. No urine was obtained; the bladder was empty, and it was not known when she passed urine last. The ether was continued, and I immediately set about the manual dilatation of the os. Dr. Green had been notified by the telephone in the meantime.

3.02 P. M. Eighteenth convulsion. Pulse 200.

3.08 P. M. Nineteenth convulsion. Pulse 200.

3.12 P. M. The os is fully dilated. Pulse 168.

3.14 P. M. Twentieth convulsion, during which the membranes ruptured.

3.18 P. M. Forceps were applied by Dr. Green, who had arrived a few minutes before.

3.25 P. M. The child was delivered. This was forty minutes from the time the carriage reached the hospital door. The child gasped, and was finally coaxed into living, after twenty convulsions.

The woman's pulse was 180 after the birth of the child, and 210 after the delivery of the placenta.

3.40 P. M. Hot-air bath. As the pulse was 210 and weak, she was given subcutaneously six syringefuls of brandy. The pulse improved.

3.55 P. M. Croton oil: two drops in oil on the tongue.

4.10 P. M. Pilocarpine, gr. $\frac{1}{10}$, subcutaneously. The reason for this was that the hot-air bath alone had not caused any perspiration after being in working order for twenty minutes. The dangers of using pilocarpine with the patient in such poor condition were fully recognized. At the same time her condition demanded speedy and effectual relief even by desperate measures. Under the dose of one-tenth of a grain of pilocarpine and the heat, the perspiration was profuse in less than five minutes. There had been some œdema of the lungs at the time of her entrance to hospital. This was somewhat increased by the pilocarpine and there was some salivation, but both disappeared in a short time. The pulse was not materially affected in rate or strength, running from 180 to 200.

The twenty-first convulsion occurred about the same time that the pilocarpine was given, 4.10 P. M. It was a short and slight one. The twenty-second convulsion at 4.40 P. M., however, was a severe one. The pulse became weaker and the subcutaneous stimulation, which had been given from time of brandy and digitalis, was increased. Patient rallied; and at 5 P. M.

was breathing easier, had a better pulse and was still perspiring freely.

5.25 P. M. Twenty-third convulsion.

6 P. M. Twenty-fourth convulsion.

The pulse again became very weak, rate 180. It rallied, however, under stimulation; and the condition improved enough so that she could swallow at 7.25 P. M. She was given one-fifth of a grain of elaterium. She also took a little brandy by mouth. The improvement was short, however, and she began to fail. By 8.30 P. M. she failed to respond to stimulants, and died at 9.35 P. M., March 29th. The baby lived and did well.

This case was almost hopeless from the beginning of our care at the hospital. She had had sixteen convulsions previously, and four followed in the next half-hour before delivery could be accomplished. The pulse ranged from 144 to 160 at the start, running up to 210 during a convulsion; and it was weak. There was some pulmonary œdema. And yet the patient rallied enough to be able to swallow. This is rather more surprising than that she died; and it is also interesting that the child was still alive and survived after twenty convulsions.

The cases here reported are too few in number to be in themselves the basis of any general conclusions regarding treatment. A number of interesting questions might be raised which would involve a general discussion of treatment, but that is not the object of this paper. Vicarious elimination is the feature common to all three cases; and I wish to speak of one or two points in this connection. I was surprised at the difficulty experienced in producing catharsis, and at the length of time which elapsed before the bowels moved. Elimination by the bowels is very valuable, but is not immediately available as a rule.

Elimination by the skin is, as a rule, more quickly obtained. For this purpose I prefer the hot pack to the hot-air bath. This is often enough. If, however, the perspiration does not come quickly enough or in large enough quantities, What shall we do? Shall we use pilocarpine? It was used in all three of these cases — with marked benefit in the first two cases, and it seemed to me to be of some aid in the third. In the last case I am quite confident it did no harm; and I feel that if the same line of treatment could have been begun two hours earlier, we could have saved the mother's life, but she came to the hospital too late.

Professor Hirst, in "Hare's System of Practical Therapeutics," Vol III, p. 870, says: "Pilocarpine seemed at one time an ideal remedy to secure diaphoresis in eclamptic cases, but it has lately fallen into well-deserved disrepute. It much increases the danger of pulmonary œdema and is too profound a depressant. It is no longer employed by experienced and educated obstetricians." And three pages later he adds: "Pilocarpine is simply mentioned to be condemned."

It seems to me that this condemnation is rather too severe. I do not advocate pilocarpine in all cases, and I should try to get along without it if suitable action of the skin could be obtained by a hot pack. But I think it still has a place in the treatment of eclampsia. If, as in the first case reported in this paper, I felt the case urgently demanded a profuse perspiration as soon as possible, then I think I should be justified in using pilocarpine in a suitable dose. It is, of course, a powerful depressant, but so are other drugs which are advocated for eclampsia, such as vera-

trum viride, chloral in large doses, and chloroform (for convulsions). If used intelligently and in small enough dose, I believe there need be no serious pulmonary œdema or depression of the heart. Combined with moist heat externally a small dose works admirably. The function of pilocarpine is only a temporary one, to bridge over the period until elimination by some other channel than the skin has been established.

Medical Progress.

RECENT PROGRESS IN GENITO-URINARY SURGERY.

BY FRANCIS S. WATSON, M.D.

CASTRATION FOR THE TREATMENT OF HYPERTROPHY OF THE PROSTATE.

At the third triennial meeting of the Congress of American Physicians and Surgeons, before the Section of the American Association of Genito-Urinary Surgeons, May 30, 1894, Prof. J. Wm. White, of Philadelphia, read an interesting paper on this subject. Professor White had already published¹ the results of his observations upon the effect of castration on the prostates of dogs, in which it was shown that atrophy of the prostate followed the operation invariably and within very short periods of time, and recalled to notice the suggestive facts that the prostates of eunuchs are known to be small; and that Gruber and Caviale, when operating by lateral lithotomy for stone, had noticed that the prostate of a patient who had been previously castrated, was very small. He also referred to the supposed analogy between the prostate and the uterus, and to the effects of oöphorectomy upon the latter. In this first communication he made the suggestion that castration might be in the future successfully applied to the treatment of prostatic hypertrophy, to produce a diminution in the size of the organ.

In his paper, read as stated on May 30th, White urged definitely that castration be tried in appropriate cases with the view of bringing about atrophy of the enlarged prostate, and adduced the four successful results already obtained, in support of his proposition.

Of these four cases two were operated on by Frederick Ramm, of Christiania;² the first operation being in 1893 (April), and the second case on January 17, 1894. The third case was operated upon by a surgeon in Los Angeles, Cal. The fourth was a case of Dr. Fremont Smith's, in St. Augustine, Fla., in 1894.³

In all of these cases marked atrophy of the prostate, attended with entire relief of all distressing urinary symptoms, followed castration within a short time; and the patients all recovered a satisfactory degree of health.

Ramm's conclusions with regard to the matter are as follows:

- (1) The prostate is a sexual organ.
- (2) It develops with or after puberty.
- (3) It retains its infantile size in cases of malformation of the sexual organs.
- (4) It shrinks in adults after castration.
- (5) This diminution in size begins at once after castration, and continues as time passes.

¹ Annals of Surgery, 1893, vol. xviii.

² Reported in the *Centralblatt für Chirurgie*, April 28, 1894, no. 17.

³ Reported in the *Annals of Surgery* for July, vol. xx, no. 1.