of extensive fibrinous pneumonia, certainly of infective nature.

In the former case, besides the diagnostic indication supplied by the biological analysis of urine, which in the rabbit reproduced the toxic symptoms in a more serious form than in the human patient, the knowledge thus gained was extremely useful to me from the point of view of prognosis and treatment. With regard to prognosis, I was able to foretell that improvement would take place at a time when the urotoxic coefficient of the urine was diminished in spite of the fact that, owing to an intercurrent attack of diarrhoea, the patient's general condition seemed to be worse, and when the clinical prognosis therefore was extremely doubtful. From the therapeutic point of view while the treatment (consisting of very large doses of hydrochlorate of quinine) employed before I was called in had been of little service, as soon as I had satisfied myself as to the close resemblance, as revealed by biological analysis, between the experimental results and the clinical picture presented by the patient, I ordered copious draughts of sulphuric lemonade with the object of destroying the circulating ptomaines and favouring their elimination.

In the second case—one of fibrinous pneumonia following influenza—whilst the specific inflammatory process successively invaded new zones of pulmonary tissue, there supervened suddenly eclamptic and tetanoid symptoms which made the medical attendant suspect the onset of infective cerebro-spinal meningitis. The condition of the patient was most serious; a temperature of 40.07° C, pulse 125, respirations 55; fluid yellowish-green expectoration, profuse sweating, etc., in short the classic picture of a most violent acute infection. The doubt as to the diagnosis was fully warranted, and it will readily be understood of what importance that was relatively to prognosis and treatment. I did not, however, feel justified in accepting without much reserve the diagnosis of cerebro-spinal meningitis, and before giving my opinion I caused experiments to be made in the laboratory of my Clinical Institute on the biological analysis of the urine which the patient had passed in the last twelve hours. The experiments were conducted by my able assistant Dr. Traversa and my demonstrator, Dr. Falcone.

The result of that biological analysis as well as of the chemical analysis was as follows: Urine deep yellow in colour, clear, acid, specific gravity 1020, containing no chemically appreciable pathological elements, no uroerythrin, biliary pigments, or ethyl-diacetic acid. The injection of 5 cubic centimetres of this into a guinea-pig weighing 250 grammes, and of 10 cubic centimetres into a rabbit weighing 1.050 grammes, was immediately followed by general tremors, alternating with tonic convulsions and extreme dyspnoea, exactly resembling the clinical symptoms and convulsions seen in the patient, and clearly showing that part of the dyspnoea, the severity of which was certainly disproportionate to the extent of the pneumonic process, was of bulbo-paralytic origin. I no longer hesitated as to the diagnosis, but declared that the convulsions did not indicate an attack of cerebro-spinal meningitis, but simply a most acute intoxication, due to the formation of new special ptomaines in that great and mysterious biochemical laboratory of which the human organism when attacked by influenza is the seat.

Such being the diagnosis, the prognosis naturally became less serious, and, instead of antiphlogistic treatment directed to the new seat of the supposed morbid process, I pursued my ordinary method of treatment in such cases, especially giving large doses of iodide of sodium, which I look upon as a powerful means of elimination in the treatment of infective diseases. The further progress of the disease completely confirmed this diagnosis; the convulsive attacks completely ceased twenty-four hours after their onset, and the urine passed after their subsidence, although the pneumonic process continued with hardly any appreciable improvement, and the temperature persisted at 40° C., showed itself on biological analysis to be inoffensive. In fact, the injection of 90 cubic centimetres into a rabbit did not induce the slightest toxic manifestation referable to the classic clinical type already described.

I do not think I need add other details to show the great assistance which may be derived in clinical work from the biological analysis of the urine, and I believe that these

analyses should be made *pari passu* with other semeiotic researches, since the results thereby obtained will enable one to foretell the impending development of serious clinical symptoms in the course of certain acute febrile infections, which begin suddenly with very high fever, and of which it is impossible in the earlier days of the illness to determine the nature.

I think it necessary before concluding to remind all those who propose to adopt this new diagnostic method of one thing, and it is this. There are two indispensable conditions for attaining all the clinical advantages which may be looked for from biological analysis: 1, the researches should be carried out by assistants thoroughly versed in pharmacological experiments, especially those concerning the nervous and muscular systems; 2, the experiments should be begun with small doses of urine (always having regard to the body weight of the animal), because in acute infective diseases the organism sometimes gives rise to the formation of most powerful poisons, with regard to which it would be impossible to obtain a precise knowledge of the clinical symptoms of intoxication produced by them unless one proceeded step by step. Lastly, the reader need hardly be reminded that the total quantity of urine passed in the twenty-four hours should be taken into account wherever it is possible to do so.

ON WOOLDRIDGE'S METHOD OF PRODUCING IMMUNITY AGAINST ANTHRAX BY THE INJECTION OF SOLUTIONS OF TISSUE FIBRINOGEN.

Being a Paper Communicated to the Bacteriological Section of the International Congress of Hygiene.

By A. E. WRIGHT, B.A.CAMB., M.D.DUBL.

ACCORDING to Wooldridge¹ rabbits which have received an intravascular injection of a solution of tissue fibrinogen, which has been sterilised by boiling, are thereby rendered immune against anthrax when it is subcutaneously inoculated. Wooldridge, however, described certain difficulties to exist in the application of this method. He found that when "strong alkaline solutions," that is, solutions in which no coagulation took place on boiling, were employed, the solutions did not possess any immunity-conferring power (this power having presumably been lost by the action of heat in the presence of free alkali). On the other hand, when the alkali of the solutions was reduced to a point which permitted of coagulation taking place on boiling, that coagulation tended to be too complete, and the filtrate in consequence to become inert, because entirely free from tissue fibrinogen.

The purport of this communication is to make a contribution towards defining the expression "tissue fibrinogen" from a chemical² point of view, and to report a few experiments upon the immunity-conferring power of a solution of tissue fibrinogen sterilised by passing it through a Chamberland's filter.

Method of Preparing the Fibrinogen Solution.—The perfectly fresh cellular organs (testicles, thymus, etc., in the experiments to be reported the fluid was always prepared from bulls' testicles) are reduced to a pulp in a mincing machine, and are extracted for twelve hours with water to which, for antiseptic purposes, an addition of 1 in 200 of chloroform has been made. The watery extract is then either centrifugalised or filtered, or simply decanted. It is then precipitated by the addition of 0.5 cc. of 33 per cent. acetic acid to every 100 cc. of the extract employed. The precipitate is collected and washed with NaCl solutions of moderate strengths, and lastly with distilled water. (All these washing fluids are rendered slightly acid by the addition of a trace of acetic acid.) The precipitate is now dissolved in a 1 per cent. solu-

¹ Arch. J. Anat. u. Phys., 1888.

² I have devoted a considerable portion of this paper to the discussion of the chemistry of the tissue fibrinogen, because the obscurity which surrounds it appeared to me to be responsible in great part for the fact that Wooldridge's experiments upon immunity do not appear to have been verified by subsequent observers.

tion of Na_2CO_3 , and is filtered first through an ordinary paper filter, and afterwards through a Chamberland's filter.

Characters of this Solution.—The solution obtained by the method just described is a perfectly clear straw-yellow fluid, which gives a heavy precipitate when enough acetic acid is added to it to make the reaction of the fluid slightly acid. The precipitate thus obtained is not very readily dissolved in excess. When boiled the solution does not undergo any coagulation. The fluid is also characterised by the following reactions: (a) It gives a marked biuret reaction at ordinary temperatures when treated with NaOH and CuSO_4 , the colour becoming more marked upon standing. (b) When it is poured on to the surface of strong nitric acid it gives a heavy precipitate, which is to some extent dissolved, when it is shaken up with the acid, a very marked xanthoproteic reaction being then obtained even at ordinary temperatures. On heating, the remaining precipitate is almost entirely dissolved, and on cooling the precipitate falls out again to a considerable extent. At first sight these reactions would naturally be interpreted as indicating the presence of albumose in the solution. I think, however, that the following considerations will show that this conclusion is not in any way a necessary one: (a) It is well known that both the biuret and the xanthoproteic reactions, when obtained in the ordinary way after the application of heat, indicate nothing more than the presence of certain substances which may have been derived from the disintegration of any proteid substance whatever. Where however, as here, these reactions are obtained at ordinary temperatures, the correct inference to draw is only an inference that we are dealing with some relatively unstable proteid, and the evidence of the reactions plainly does not entitle us to draw the conclusion that the unstable proteid we are dealing with is actually an albumose or a peptone; in point of fact, the albumoses and the peptones have by no means, as seems often to be inferred, a monopoly of these colour reactions as obtained in the cold. It has, for instance, long been known that the nucleo-albumen which can be prepared from yeast gives a brilliant biuret reaction in the cold. That it would do so might indeed have been expected *a priori* from the fact that this nucleo-albumen is notoriously unstable in the presence of alkalies. (b) With regard to the so-called nitric acid albumose test, it seems plain that any solution which contained a substance from which nitric acid could split off an albumose must of necessity give this reaction for albumoses in the same way as a solution would in which albumose was present preformed. I believe this to be the condition of things which obtains in the case of our tissue-fibrinogen solution. I hope to show that it is not a solution of preformed albumose, but a solution of a nucleo-albumen which can be easily broken down, and which yields an albumose among its disintegration products. The evidence I propose to submit to you upon this subject may be treated of under three headings: (a) the evidence afforded by the solubilities of tissue fibrinogen, (b) the evidence obtained by submitting a tissue-fibrinogen solution to an artificial peptic digestion, (c) the evidence afforded by boiling the solution and by the examination of the ash.

(a) *Evidence Derived from a Study of the Solubilities of Tissue Fibrinogen.*—1. Extraction of Tissue Fibrinogen from the Organs: Tissue fibrinogen can be extracted from the cellular organs employed either by water or by weak (for example, 5 per cent. NaCl) solutions. With stronger neutral salt (for example, with 10 per cent. NaCl .) solutions we obtain only an unworkable viscid gelatinous mass. The watery or saline extracts have a slightly alkaline reaction, but they are naturally very impure solutions, and as such they are not calculated to throw much light on the solubilities of tissue fibrinogen. From these impure solutions, as has been seen, we can precipitate our tissue fibrinogen by acidifying with dilute acetic acid. 2. Precipitation of Tissue Fibrinogen from the Extracts by Acetic Acid: With reference to the precipitation of tissue fibrinogen by weak acetic acid, and its bearing upon the chemical characterisation of this substance, the matter may perhaps be summed up shortly as follows: (a) The precipitate is not a neutralisation precipitate of an alkali-albuminate because the fluid must be made distinctly acid before any precipitation occurs. (b) The precipitate is not a neutralisation precipitate of a globulin or of

a globulin-like albumose, both for the reason assigned under (a), and also because it is plain, in view of the alternate solubility of the globulins in either neutral salt solutions or in dilute acid or alkaline solutions, that such a neutralisation precipitate can be formed only when there is not enough neutral salt present³ to undertake the solution. In the case, however, of our tissue-fibrinogen solution we obtain the same precipitation both in the salt-free extract and in the extract to which 5 per cent. of NaCl has been added before undertaking the precipitation. Further (c), the precipitation obtained on the addition of acetic acid cannot be considered as a case of the formation of such an albumose precipitate as is obtained on acidifying strongly an ordinary albumose solution after previous addition of a large quantity of NaCl , for we have just already seen that the precipitation of the tissue fibrinogen is best obtained when the strength of the acid does not exceed $\frac{1}{4}$ per cent. of acetic acid. We have also seen that it is obtained just as well in the salt-free as in the saline solutions. (3) Solubilities of the Acetic Acid Precipitate: The precipitate, as it is obtained from the extract, has of necessity an acid reaction from included acetic acid, and if we now, without previously correcting this acidity, proceed to treat the precipitate with either distilled water or with neutral salt solutions of moderate strengths we find that very little appreciable solution occurs, even after prolonged agitation in the shaking machine. The filtrates obtained give only the merest trace of a biuret or HNO_3 albumose reaction. If, on the other hand, we add to our distilled water or neutral salt solution enough alkali to effect a correction⁴ of the acidity of the precipitate and to leave over a small excess of alkali we immediately notice that solution is occurring, if from nothing else at least from the colour of the fluid changing from a grey-white to a yellow. The filtrate also now acquires the characters described above as those of a solution of tissue fibrinogen. It will be seen that the practical insolubility of tissue fibrinogen in either water or in salt solutions when there is even a trace of acid present; and, on the other hand, its solubility in the presence of any alkali are consistent with the view that we have to deal with a nucleo-albumen, but hardly with the assumption that we are dealing with a precipitate consisting of either globulins or albumoses.

(b) *The Evidence obtained from Artificial Peptic Digestions as to the Chemical Constitution of Tissue Fibrinogen.*—We may sum up this very shortly. When the washed acetic acid has been dissolved in 1 per cent. Na_2CO_3 and has been filtered, we obtain, as has as been already stated, a perfectly clear solution. On acidifying this solution slightly with hydrochloric acid we of course reprecipitate the fibrinogen, but this precipitate redissolves again completely before we have brought up the strength of the solution to 0.4 of HCl . On adding a few centimetres of Benger's liquor pepticus to this clear solution and digesting for a few hours at 37°C . we obtain a copious precipitate of nuclein.

(c) *Evidence obtained from Boiling the Solution of Tissue Fibrinogen.*—The 1 per cent. Na_2CO_3 solutions employed generally undergo no visible alteration on boiling (coagulation occurring almost exclusively in solutions containing a smaller proportion of alkali). After boiling and subsequent cooling down, weak acetic acid is still found to produce a precipitation in these solutions, presumably a precipitation of still unaltered fibrinogen. The filtrate obtained by filtering of this precipitate will, however, now be found to give a macroscopically visible precipitate of phosphates when treated with magnesia mixture. Under the microscope this crystalline precipitate can be observed to dissolve in a trace of acetic acid. Barium chloride and ammonia and ferric chloride will also be found to give a similar precipitate of phosphates in filtrate, the precipitate in the latter case being of course soluble in excess of the reagent. In this connection it may also be stated that when the washed acetic acid precipitate has been collected on a filter and dried it yields on ignition with soda and saltpetre an ash which when tested with nitric acid and molybdate of ammonium gives the

³ It will be remembered that in order to obtain a neutralisation precipitate of, for instance, serum globulin in serum it is necessary to make its solution almost salt free by dilution with many volumes of water.

⁴ Owing to the acetic acid being held fast by the precipitate it is almost impossible to arrive at an exact neutralisation.

yellow characteristic precipitate of phosphates. Besides the chemical evidence here adduced, which evidently points very distinctly in the direction of regarding tissue fibrinogen as a member of the class of proteids described by Hammarsten under the name of nucleo-albumens we have a good deal of light thrown upon the constitution of tissue fibrinogen by a study of its behaviour when injected intravascularly. I am at present engaged in preparing for publication an account of the experiments I have made in this direction. I may, however, perhaps state here the general results of those investigations as far as they have reference to the present question. At the same time I give these results as merely probable—I think very probable—conclusions from evidence which I hope shortly to publish in full. 1. The tissue-fibrinogen solution prepared as above will not under ordinary circumstances produce intravascular coagulation. That it none the less is a solution of the active principle Wooldridge's coagulating fluid is, however, evident from the fact that it produces coagulation in peptone plasma, and that the precipitate obtained from this solution by acidifying with acetic acid will produce coagulation when injected into the vessels in a more concentrated form. 2. The loss of the power of causing intravascular coagulation, which was described by Wooldridge to result from boiling a tissue-fibrinogen solution is not an entire loss of coagulative power, but rather a weakening of this power, depending on a partial disintegration of the tissue fibrinogen, a disintegration of which, from the chemical side, we have obtained evidence in the fact of the presence of free phosphoric acid in the boiled solution. That the disintegration of the tissue fibrinogen and concomitant loss of coagulative power is not complete is established by the fact noticed by Wooldridge, and repeatedly verified by myself, that the boiled tissue-fibrinogen solution will still produce coagulation in peptone plasma. From the chemical side we have evidence of the incompleteness of the disintegration in the fact, among others, that acetic acid still produces a precipitate in the boiled solution. 3. After prolonged boiling the filtered tissue-fibrinogen solution no longer clots peptone plasma, and, when injected intravascularly, instead of producing coagulation, it now renders the blood permanently liquid, the plasma obtained reacting in every respect like peptone (or, more correctly, albumose) plasma. In the light of this fact it appears to me that the loss of coagulability in the extraportal blood, which is a regular occurrence in the dog after the intravascular injection of a tissue-fibrinogen solution which coagulates the portal blood, would be perhaps most easily explained by assuming that it depends on the presence of albumose, which has been split off from its mother substance—tissue fibrinogen. I have already shown⁵ that the blood from the extraportal tracts under such circumstances possesses the physiological properties of albumose plasma. We have thus from our physiological as well as from our chemical study of tissue fibrinogen arrived at the conclusion that tissue fibrinogen is not an albumose, but that it is a substance which can readily be broken down into an albumose. We have further had chemical evidence that it belongs to the class of the nucleo-albumens. Confirmation of this chemical datum from the physiological side being, however, still lacking, I endeavoured to obtain it in the following manner: I took about 500 grammes of German yeast, and prepared the nucleo-albumen from it by treating the washed yeast with 0.5 per cent. of NaOH, and filtering directly into hydrochloric acid of a strength of 1 in 1,000. When I now proceeded to inject a concentrated solution of the nucleo-albumen I had thus prepared into the jugular vein of a rabbit, I found that I obtained the most widespread intravascular coagulation. I think that this fact is at least suggestive, and I am engaged in pursuing it further, although on repeating the experiment a second time I did not succeed in obtaining the same striking result. Having thus prepared the way by a preliminary study of the constitution of tissue fibrinogens, I may proceed to give a very short account of some preliminary experiments which I have made in connection with the production of immunity against anthrax with solutions of this substance. (I have also made some preliminary experiments to determine whether tissue-fibrinogen would produce immunity in guinea-pigs against *charbon symptomatique*, but am

not reporting them here because the results I obtained were all negative). These experiments were conducted with a solution prepared as described above, which was sterilised by passing it through a Chamberland's filter. In the first experiment made, 1 cc. of this protective fluid was injected directly into a vein of the right ear of a rabbit, and the other ear was immediately inoculated with virulent anthrax. A control was inoculated at the same time. Both these rabbits died between the thirtieth and the fortieth hour after inoculation. The inoculated (left) ear of the rabbit was intensely oedematous, but there were no other differences detected between the two rabbits. Assuming that the cause of failure was the smallness of the dose administered, I began a new series, this time making daily injections of 1.5 cc. hypodermically for several days previous to inoculating with anthrax. The results obtained in this series were as follows: Control rabbit dies between the thirty-sixth and the fortieth hour. Rabbit 2. Previous to inoculation with anthrax, five successive daily injections of 1.5 cc. of protective fluid, an interval of twenty-four hours being allowed to elapse between the last of these injections and the anthrax inoculations. Death from anthrax in forty-six hours. Rabbit 3 treated in the same manner as Rabbit 2, with the exception that only three successive daily injections were made. Death from anthrax in sixty-four hours. Rabbit 4. Exactly the same treatment as Rabbit 2, previously to anthrax inoculation, but the injections were repeated daily also after the inoculation. Death from anthrax in *circ.* ninety-six hours. Rabbit 5. Same treatment as Rabbit 3 before anthrax inoculation, subsequently to inoculation the injections were continued daily. Death from anthrax between the 120th and the 135th hour. In the above series we have in all cases a lengthening out of life in rabbits which had been treated with the protective fluid, life being most lengthened out in the rabbits which had received injections of the protective fluid both previous and subsequent to the anthrax inoculation. With the knowledge thus gained I undertook a new series, this time a series of twelve rabbits. Two of these rabbits were set aside as controls, and in the case of four others I proposed to determine the effect that would be exercised upon the course of the anthrax by injections of protective fluid when made only subsequently to the anthrax inoculation. The result of this third series was as follows:—Rabbit 1. Four antecedent daily injections of 2 cc. protective fluid; also subsequently to inoculation daily injections of 2 cc. Death from anthrax in sixty eight hours. Rabbit 2. Three previous injections of 2 cc. of protective fluid; also subsequently to inoculation daily injections of 2 cc., decreased afterwards to 1 cc. This rabbit (ten weeks after) still survives. Rabbit 3. Four previous daily injections of 1 cc., also subsequently to inoculation daily injections of 1 cc. Death from anthrax in sixty hours. Rabbit 4. Three previous injections of 1 cc. daily, also subsequently injections of 1 cc. daily continued for fourteen days. This rabbit also survived for eight weeks, after which it was killed. Rabbit 5. Two previous injections of 1 cc. daily, also subsequently 1 cc. daily. This rabbit also survived, but was killed by a rabbit epidemic seven weeks after. Rabbit 6. Two previous injections of 2 cc. daily, also subsequently to inoculation injections of 1 cc. daily. The rabbit survived, and was killed at the end of the eighth week. Rabbit 7. Control. Death from anthrax in forty-eight hours. Rabbit 8. Control. This rabbit still survives. There was some hæmorrhage from the seat of inoculation, as noted at the time, and the virus was probably washed away. Rabbits 9, 10, 11, 12. All these rabbits received daily injections of protective fluid, but the injections were only begun subsequently to their inoculation with anthrax. They died from anthrax at *circ.* 36, 72, 72, 72 hours respectively. In this series, as has been seen, four out of six rabbits treated with daily injections of 1 to 2 cc. of protective fluid; both, before and after inoculation with a very virulent anthrax, survived that inoculation. This is the last series of experiments which I can regard as completed, and therefore the last I can record before sending this communication to press. I hope, however, soon to be in a position to report on some further series. I do not propose at this stage to discuss these experiments further than to say that they appear to me to be, as far as they go, entirely confirmatory of Wooldridge's results. With respect to the questions raised by them, I must, however, state that I have succeeded in grow-

⁵ *Journ. of Phys.*, 1891, No. 2.

ing anthrax, not luxuriantly, it is true, but quite sufficiently to be macroscopically visible in sterile tubes of this protective fluid. This, as is well known, had been previously done by Wooldridge, who, in his first communication on the matter to the Royal Society, ascribed the immunity-producing results he obtained to this growth of anthrax. The fact that anthrax will grow at all here in a solution which is probably very poor in the requisite salts seems hardly reconcilable with the suggestion that Wooldridge's results were due to the action of proteids directly poisonous in their action upon the micro-organisms of disease. Lastly, I am bound to say in self-criticism of the experiments reported above, that it is undoubtedly possible, though it appears to me improbable, that the results I obtained may have been due either to (1) a "mixed infection," or (2) to the injection of bacterial products. I have sought to guard against the latter contingency as far as possible by very careful and repeated washing of the precipitate before it was used for the preparation of the protective fluid. To guard against a "mixed infection," the solution was, as has been stated, sterilised by filtration through a Chamberland filter, and the flask, plugged with sterile wool, was kept continuously in a mixture of ice and salt. The chances of a mixed infection were thus minimised. I cannot assert that they were absolutely excluded, even though perfectly pure cultures were successfully made from the animals which died in the second series, and though the cover-glass preparations made from the animals which died in the third series uniformly showed the presence of pure anthrax. In conclusion, I have to thank Dr. Cartwright Wood and Dr. Woodhead, the Director of the Laboratories of the Conjoint Colleges, where the experiments reported above performed, for much kindly and self-sacrificing assistance afforded to me by them in the bacteriological part of my work.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

RETROPHARYNGEAL ABSCESS.

PROFESSOR CHIENE has done good service in drawing attention to his method of opening retropharyngeal abscess, in his Address in Surgery, which was remarkable for its suggestiveness and practical value to the surgeon. I do not find that method described in the textbooks to which I have access, but I practised it with gratifying success in the early part of March last. It was suggested to me by a case of Mr. H. Littlewood's, where in dissecting out some deep-lying glands he obtained a ready access to the front of the cervical vertebræ. Mr. Littlewood assisted me in my own operation here described.

H. T., a weakly, ailing man, weaver by trade, came to me in March with a large swelling in the posterior wall of the pharynx, which had been growing for some weeks, and had now become tense and painful, altering the voice, interfering with deglutition, and resulting in loss of flesh and impairment of general health. Thirteen years ago he was the subject of diabetes, but at this time his urine contained neither sugar nor albumen. There were also vomiting, dyspnoea, and pain in the head and right side of neck, with sleeplessness. The swelling was rather to the left of the middle line. Mr. Ward kindly saw him at the Leeds Infirmary, and thought the case was one of lymphadenoma or retropharyngeal abscess.

The symptoms increasing, I operated on March 12th, making an incision along the posterior border of the sterno-mastoid, and dissecting carefully down till the fascia covering the transverse processes of the cervical vertebræ was reached; coasting round to the back of the pharynx it was an easy matter to push through the wall of the abscess, evacuate about an ounce and a half of thick, sweet pus, and flush the cavity with iodine water and insert a drainage tube. There was no bleeding point requiring ligature, and only one small nerve was seen and avoided. I syringed the cavity daily with iodine water, and removed the drainage tube about a week later, the wound soon closing.

A few weeks later the swelling returned, and my successor,

Dr. Shaw, of Kirkburton, aspirated the contents; but in a few days the old operation wound recommenced to discharge, and the patient is now in good health.

The operation is quite safe, and is, I believe, a very useful one for this condition. We are enabled safely to evacuate and drain the abscess, and ascertain the condition of surrounding parts, without endangering the important structures in the immediate vicinity. The bodies of the vertebræ can be most readily examined, and any disease noted. Now that Professor Chiene has drawn attention to the method it will probably receive the attention it deserves.

Clifton.

G. A. HAWKINS-AMBLER.

POISONING BY GELSEMIUM SEMPERVIRENS.

ABOUT two months ago Miss W., aged about 40, an inmate of my house, was seized with very severe neuralgia about both temples. I gave her tincture of gelsemium 10 minims, with a bismuth mixture to be taken every two or three hours. After taking this for about a day and obtaining no relief—but rather she grew worse, being, as is described, "nearly mad with pain,"—I gave her the full dose of the tincture of gelsemium, according to Squire's *Companion of the Pharmacopœia*, 1882, and Whitt's *Materia Medica*, 3rd edition, namely, 20 minims in a quinine mixture. This was taken every three hours, but with only moderate relief, three or four doses having been taken during the night. At about eight o'clock the following morning Miss W. was able to speak pretty well, and said she thought she was better. At about 9 o'clock she was speechless and in the greatest distress of mind and body; there was total loss of power in the tongue; it could not be protruded, she could not articulate, and with very great difficulty could she swallow the brandy and water we forced upon her. There was alteration in vision; she could not distinguish us clearly, and the pupils were widely dilated. She had uncertain power over the muscles of the hand and arm, so that she could not write her name. All this time she was perfectly conscious, and nodded her head in answer to questions. She was greatly alarmed as to herself, and, as she informed us afterwards, she thought she was about to have a fit. Not knowing of any special antidote for gelsemium, and seeing that there was no time to lose if we wanted to avert any increase of the paralysis, it fortunately came into my mind to give her a subcutaneous injection of strychnine, using 1 minim of the liquor strychninæ, or 1-120th part of a grain. Ten minutes after this the change for the better was most marked; there was return of power in the tongue and in the hands, and an improvement in the vision.

At this juncture I was glad to have the help of my neighbour, Dr. Barron, and consulted with him the various authors of books on the use of this drug and its antidotes, but with no satisfaction, and with his approval I again injected a minim of the strychnine, and with further improvement in the condition of the patient. After this she took food and stimulants, and all paralysis disappeared. The vision was not perfectly restored for some hours, the pupils being less dilated. She had some return of the neuralgia, and was very weak for a few days, but eventually she quite recovered, and has had altogether better health since this event than she had prior to it.

Durham.

EDWARD JEPSON, M.D.

DOUBLE URETHRA.

THE occurrence of a double orifice of the male urethra is, I think, of sufficient rarity to make it worth while to record an instance of it which has come under my notice recently. My patient, a middle-aged gentleman, married and with a large family, came to me suffering from urethritis, and rather puzzled me by discriminating between an upper and a lower passage. On examination, however, I found that such existed; the two passages being separated by a narrow but considerable fleshy septum, and both completely formed and in the usual position. He had also an undescended testicle on the one side. He was unaware that the existence of two passages was unnatural, and believed the upper to be the urinary and the lower to be the genital. Whether this view was founded on imagination or not I was unable to determine. During the inflammatory attack, for the first time, according to him, water was passed by both channels, the stream of