place one electrode over the sacrum and the other in the groin, and apply the secondary faradic current very weak to begin with, and gradually increase its strength as much as the patient can bear. In other cases one electrode may be placed in the vagina and the other in the groin. In another set both poles may be placed in the vagina, using the bipolar vaginal electrode. It is only by testing each case that it is possible to say which mode of administration will give the most relief.

The effects derived from the secondary faradic current in these cases, and used as we have described, are as follows:—

1. A marked diminution in the sensitiveness of the parts in the course of the current, and to which the electrodes have been applied. If it is a case of a painful and prolapsed ovary that at the beginning of the sitting cannot be touched without inducing great pain, it can be firmly pressed on at the end of the sitting without causing pain, and this condition will last some hours after the application. To obtain permanent relief, the application must be made night and morning for about ten days, then daily for about twenty more days; at the end of this time, in the majority of cases the nerves in the painful organs or tissues cease to have the power of conveying pain, yet the organs or tissues still remain as they were before the electricity was used.

2. We believe the current does assist absorption of effused products, but of this we cannot be sure. Our principal object in using this form of electricity is to relieve pain, and in the majority of cases where a woman complains of pelvic pain, one can relieve her to a very great extent, and remove entirely in a large number of cases the pain she complains of.

This electrical treatment is of very great value in a class of cases where any aid is welcome; treatment can be carried out so easily—any nurse can do it, or the patient can do it herself—and the expense is slight, as these batteries can be hired for a few shillings a week.

THE PUERPERIUM AS A FACTOR IN THE ETIOLOGY OF MULTIPLE NEURITIS AND DEGENERATIONS OF NERVE TISSUE.

By H. HANDFORD, M.D., M.R.C.P.,
Physician to the General Hospital, Nottingham.

GOWERS has mentioned the instance of a lady who, a few days after childbirth, on two occasions, suffered from a neuritis of the ulnar nerve, followed by paralysis and muscular wasting, but ultimately by recovery.

I have to relate two instances of multiple neuritis, both probably of alcoholic origin, where the onset seemed most clearly to be determined by childbirth. In a third case the exact diagnosis is more difficult, but its onset immediately after a confinement was equally clear. Gowers suggests that the "tissue health is lowered," and, consequently, slighter causes are efficient to excite neuritis. The rapid progress of *tabes dorsalis*, and the frequency of optic atrophy in it when sexual relations are actively maintained, has been frequently noted. The relation of childbearing is probably only of a similar nature, and it is not suggested that it is an efficient cause *per se*. On the other hand, childbearing is a common occurrence in women, and the neuritis may have been simply a coincidence; but multiple neuritis is a comparatively rare disease, and for 3 out of 6 or 7 cases, in both sexes, to commence with childbirth seems an undue proportion. Is the influence of childbirth one of the factors which renders alcoholic neuritis so much more common in women than in men, while drinking habits are more prevalent in the latter?

CASE I.—M. K., aged 43, married seven years, three children, has always been a stout, florid, healthy woman. Her husband keeps a publichouse and is a habitual drunkard, but evidence of drinking habits in the patient could not be obtained. Three days after her confinement she lost power in the legs completely, and, at the same time, felt strange sensations like pins and needles in the arms. This was soon followed by paralysis. She had been able to walk about up to the date of her confinement. The general character and course of the paralysis was typical of an alcoholic multiple neuritis. There was loss of muscular sense, impaired cutaneous sensibility, but greatly increased deep sensibility, general muscular wasting, loss of knee-jerks, and the presence of the reaction of degeneration in the muscles of the arms and legs. The condition of the eyes was normal. She was able to walk a little in six months, and in twelve months she was comparatively well.

CASE II occurred in a stout well-nourished woman, aged 34, the wife of a publichouse-keeper. She suffered from complete paralysis of the extensors of the toes and the flexors of the ankle, with weakness of most of the other muscles of the leg, coming on immediately after her confinement. The ankle-joint was habitually extended and the toes pointed. There was much hyperæsthesia, both superficial and deep, and a little oedematous swelling. No contraction could be elicited in the muscles below the knee by as strong a faradic current (sledge at 7 centimetres distance) as the patient could bear. A voltaic current from twenty-six cells, registering 30 milliampères, caused much pain and produced some contraction of the rectus femoris, deficient in quantity but normal in quality. No response could be obtained from the muscles below the knee. Some weeks later there was considerable muscular wasting, extending as high as the right gluteal muscles. In six months' time she could walk without a stick, and eventually she completely recovered, except that the affected muscles remained rather small.

CASE III.—In this last case the exact diagnosis is much more difficult, but it lies between *tabes dorsalis*, sclerosis with patches in the crura and in the lumbar enlargement of the cord, and multiple neuritis. There certainly is ataxy; but against the affection being *tabes* are the facts that she has never suffered from lightning pains or gastric crises, the pupils are dilated, there is no atrophy of the optic discs, the arms were affected as early and as severely as the legs, there was distinct loss of power, and there is now distinct localised muscular wasting. There was no evidence of syphilis, and her children are very healthy looking. E. S., aged 31, married six years; three children. She suckled the last a year and a half. Difficulty in walking and weakness in the arms came on immediately after her confinement two years ago. In the house she used to have to assist herself by the furniture, and in the street walk along by the wall. On admission, in July, 1888, she could not stand, and could only feed herself with one hand with a spoon. The legs were very feeble, and occasionally jumped and became drawn up without the patient's knowledge. The knee-jerks are totally absent, as are also all the superficial reflexes. Cutaneous sensibility is much diminished in the arms and legs. There is double ptosis with divergent strabismus and widely-dilated pupils, moving neither to light nor to accommodation—a complete paralysis of both third nerves. Sight is good in each eye, and the discs are normal. There is inco-ordination and loss of muscular sense. She soon went home and I have been able to keep her under observation up to March, 1891. The duration of the illness is, therefore, five years. There is now no appreciable alteration, except that voluntary movements of the arms are more irregular and the left is much weaker than the right. There is no rigidity of the legs. The thighs are well nourished, but there is considerable wasting of the muscles below the knee in the left leg. She cannot stand but spends her time entirely in bed, eats and sleeps well, and is free from pain.

In further illustration of the influence of fatigue on the progress of nerve degeneration in general and *tabes* in particular the following case is noteworthy:—

M. A. L., aged 48, married twenty-six years, no children, was always a poor walker, and usually did not walk more than a mile a day. She had suffered occasionally for the past two or three years from shooting pains in the legs, but considered herself quite well. In May, 1889, she had occasion to walk more than four miles, was very much fatigued, and next day was obliged to keep her bed, where she remained until she was admitted into the hospital in October of the same year, and was found to be suffering from well-marked *tabes dorsalis* with Charcot's affection of the left hip.

CASE OF ECHINOCOCCUS OF SPINAL CANAL, WITH OPERATION.

By WILLIAM B. RANSOM, M.A., M.D., M.R.C.P.,
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AND

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CASES of operation for the removal of tumours compressing the spinal cord are as yet so few, and the production of symptoms by an echinococcus cyst within the spinal canal is so rare an occurrence, that the following case, although not successful in the result, seems to us worth publishing. The failure of the treatment does not make the case less instructive, and the case as a whole offers distinct encouragement to surgical procedure.

HISTORY OF CASE (MEDICAL, BY DR. RANSOM).

J. U., a married man, aged 42, a mechanic, was admitted into the General Hospital, Nottingham, under my care on December 31st, 1890, with paraplegia of a fortnight's duration. Twenty years before he had had gonorrhœa and a chancre, probably soft, but there was no history of secondary syphilis. He had been somewhat addicted to alcohol. For six years he had had a hydrocele of the right tunica vaginalis, which had been tapped on several occasions. He was admitted into a surgical ward in 1889; pure carbolic acid was injected, but without satisfactory result, and after leaving the hospital the fluid reaccumulated. When a surgical in-patient, no symptoms of nerve disease beyond occasional pain in the back were observed. The patient said that he first began to suffer from pain in the back eighteen months before his admission in December, 1890. The pain began gradually, and increased to such severity that he would spend hours of the night walking about, sleep being impossible. The pain was worse at night; it was of two kinds: a constant gnawing and burning pain, and occasional shooting pains which ran down the legs to the soles of the feet. At this time he had no difficulty in walking, in

retaining or passing water, and no loss of sensation. After four months the pain rather suddenly left him, only recurring occasionally and with moderate intensity. During the eighteen months before admission he attended occasionally as a surgical out-patient, both on account of the hydrocele and the pain, and took during that period considerable quantities of iodides. During most of this time he was able to do his work. In October, 1890, the pain returned in its original severity, and in another month there supervened stiffness, weakness and numbness in the legs. He now lost sexual power. On November 30th he was seen by the house-surgeon, who noticed an ataxic gait and loss of knee-jerks. By the middle of December he was unable to walk, and had difficulty in passing his water. On December 31st, these troubles continuing and the pain becoming more intense, he came into hospital under my care.

State on January 4th, 1891.—Patient is an intelligent, powerfully built, but fat man. He is unable to stand, but sits propped up by pillows, as lying down increases the pain. He presents no signs of past or present syphilis. The circulatory, respiratory, and digestive systems are normal. Urine normal. There is a hydrocele of the right tunica vaginalis. There is no sign of cerebral disease or of affection of cranial nerves. He complains of severe, constant, dull, aching pain across the lumbar spines, which sometimes radiates into both the iliac regions, and sometimes shoots down the legs and causes twitching sensations. There is slight and variable spinal tenderness over and at the sides of the lumbar and four lower dorsal spines. There is no visible deformity of spines. On the right buttock is a small tumour which feels like a lipoma. (This was found to be so at the necropsy.) The pain has prevented much sleep for the last two nights, being but slightly relieved by antipyrin. Sensation was normal in the arms and on the trunk as low down as an inch above the pubes. In the area of the ilio-hypogastric and ilio-inguinal nerves there is incomplete anaesthesia, and some confusion between a touch and a prick. On the thighs there is incomplete anaesthesia in front and behind, and some dulling of the temperature sense. On the shins and upper surfaces of the feet sensation is rather better, but on the calves it is again deficient. On the right sole sensation is nearly absent, but is present on the left. There is no zone of hyperæsthesia. Motor power is normal in the arms. The patient cannot lift either leg from the bed, but can move the toes, better on the right side. He has difficulty in passing water, but succeeds sometimes. There is occasionally involuntary escape from the distended bladder. No paralysis of sphincter ani; the stools passed fairly well. Knee-jerks both absent. No ankle-clonus. Slight plantar reflex, more marked on left. Cremasteric reflex absent. Abdominal reflex present on both sides above umbilicus; below it absent on right, present but slight on left. There is no muscular rigidity, and no obvious wasting. All the muscles of the legs and thighs react to a moderately strong faradic current.

Treatment.—In view of the possibility of antecedent syphilis, the patient was from December 31st given the biniodide of mercury.

Progress of Case.—During the next week the severity of the pain increased, until it could only be controlled by morphine, but it did not become more localised, and no distinct girdle sensation was developed. Spinal tenderness continued diffused over the lumbar and lower six dorsal spines. The retention of urine became complete, and occasional involuntary evacuation of feces occurred. By January 9th there was complete loss of motor power in the legs, and complete anaesthesia of the legs and as high as the area of distribution of the hypogastric branch of the ilio-hypogastric nerve. Sensation was not quite lost in the upper part of the gluteal region. No spasms of the legs were observed. On January 9th a moist bed sore commenced over each tuber ischii, and the temperature rose to 101.2°. On the two following days the temperature fell to normal, but in spite of antiseptic dressings the sore increased, and on January 12th the temperature reached 101.8°. On January 12th a narrow zone of slight hyperæsthesia was noticed above the anæsthetic region. The muscles still reacted to a strong faradic current.

The patient was now (January 12th) clearly getting rapidly worse, the nutrition of the muscles was suffering, and although the area of disease was not extending, yet it was evident that, even if the bed sore did not cause an early death (as was probable), irreparable injury would soon be done to the cord if the destructive process could not be quickly arrested. From the history of long-continued pain and the subsequent paraplegia I had inferred that the cause of the symptoms was probably a tumour compressing the spinal cord. It appeared also possible, if not probable, that the tumour was a gumma. At any rate, it was thought right first to see what could be done with antisyphilitic treatment. The fact of the patient having taken iodides during the year previously militated against this view, but it was rather doubtful whether he had taken them in sufficient and regular doses.

Medical treatment was now however completely failing to arrest the disease, and the question of operation became urgent. The exact position of the tumour became therefore important. On the motor side there was evidence, from the diminishing irritability of the recti, of damage to the anterior roots of the second, third, and fourth lumbar nerves, or to the cells of the grey matter from which they arise. The loss of sensation showed the lesion to involve the sensory tract either in the cord or roots as high as the first lumbar, while the twelfth dorsal nerve appeared only partially affected. The acute bed sore over each tuber ischii indicated that the degenerative changes had involved the upper sacral nerves. The evidence, on the whole, then showed damage to the cord from its lower extremity to the origin of the twelfth dorsal nerve, or if the zone of indistinct hyperæsthesia, which

developed later, be considered, to the level of the eleventh dorsal nerve.

It is now well known that in compression of the cord the compressing agent is not infrequently somewhat higher than the symptoms would suggest. The case of Dr. Gowers, on which Mr. Horsley operated successfully, was an illustration of this, and some of the cases of injury to the spine reported by Mr. Thorburn indicate the same fact; but the evidence of implication of the centres for the lumbar and sacral nerves pointed to downward extension of the assumed growth. It was therefore considered best to cut down on the twelfth dorsal spine as a centre, and thence advancing two vertebrae higher and one lower. The variable length of the spinal cord made localisation in this region more difficult.

After a consultation on January 13th, operation was agreed upon, and performed on January 15th. On the morning of January 15th it was found that neither the anaesthesia nor the paralysis had altered in extent, but the extensors of the legs were more flabby and did not react to faradism. The bed sore on the buttocks was sloughing, and small ones had appeared on the great toes. The temperature before operation was 102° F. The urine remained normal, as it did till the end.

(SURGICAL, BY MR. ANDERSON).

On the morning of January 15th the patient's temperature was 103°, pulse 120, tongue furred and dry. This constitutional disturbance was to be attributed to septic absorption from the sloughing sores over the sacrum, but as delay could only make matters worse I operated at 2 P.M. On the previous evening his back had been well washed with soap and water and a wet mercuric dressing applied and left on during the night.

An incision was made through the skin and fat, of which there was a vast amount, over the spinous processes of the vertebrae from the ninth dorsal to the second lumbar. It afterwards turned out to be one vertebra lower than was thought, so that the incision was really made from the tenth dorsal to the third lumbar. The bony outlines were much obscured by the adipose tissue, and the exact position was not discovered until after careful examination at the *post-mortem*. I then rapidly cleared the muscles from the left side of the spine, and exposed the laminae. There was free oozing of blood for a minute or two, and a few vessels which spouted were seized with pressure forceps. The hollow which existed between the spinous processes and the retracted muscles was plugged with sponges, and the muscles of the right side of the spine dissected off in the same way. There was less bleeding here than on the opposite side, and what little there was was easily arrested. The spinous process of the second lumbar vertebra was cut off with bone forceps, and a trephine applied to the laminae, which were cut through and removed after some trouble.

The remaining spinous processes and laminae of the first lumbar and eleventh and twelfth dorsal were cut through and removed quite readily by means of a pair of powerful, but fine-bladed, angular bone forceps. There was plenty of room for the blade under the laminae, and this method of removal I consider to be much quicker, easier, and indeed safer than the slow and doubtful process of trephining.

After the removal of the laminae the fat over the cord was taken away, and the dura mater exposed throughout the length of the wound. I could discover nothing in the shape of any localised swelling. The membrane was then incised, and between 4 and 5 inches of spinal cord and cauda equina exposed, of a greyish-blue colour and distinctly softened. An aneurysm needle was passed round it, but nothing felt on the anterior surface. Cerebro-spinal fluid flowed freely on opening the dura mater. As nothing could be discovered, it was decided to close the wound. The dura mater was replaced over the cord but not sutured, the wound douched with 1 in 4,000 solution of perchloride of mercury, and closed by four silk and superficial horsehair sutures. A large dressing of cyanide gauze was applied.

Two days afterwards (January 17th) the wound was dressed, as there was some appearance of discharge (cerebro-spinal fluid) through the dressing. The wound looked well, and there was no sign of pus. The discharge of fluid was very moderate in amount. January 18th. The wound was again dressed. There was no change in its appearance. The patient was sinking, and died at 5 P.M.

POST-MORTEM EXAMINATION BY DR. RANSOM.

At the necropsy, made twenty-two hours after death, the whole body was found distended with gas, and *post-mortem* changes were advanced. On enlarging the incision in the back and exposing the spine, there were found in the substance of the erector spinae to the right of the lumbar spines two cavities, one of the size of a walnut and the other of a Tangerine orange, with grey opaque walls and grey pultaceous contents, among which were in the larger cavity two or three typical hydatid daughter cysts. Shreds of hydatid membrane were found in the walls of the cavities, which did not seem to be in communication with each other or with the spine. Careful examination of the spine showed that the arches removed at the operation were those of the eleventh and twelfth dorsal and first and second lumbar vertebrae. The subsequent removal of the next, or tenth dorsal vertebra, at once showed the tumour of which we had been in search. It appeared as a whitish, soft, cheesy-looking mass, about the size and shape of a horse-chestnut, attached by a pedicle to the arch of the tenth dorsal vertebra, and pressing on the dura mater for an inch along its dorsal surface. To the naked eye its nature was not obvious, and it broke up immediately when placed in alcohol.

Microscopical examination, however, proved it to be a caseating hydatid cyst, among the debris of which numerous fragments of typical hydatid membrane were found. The cut in the dura mater made at the operation had reached to within half an inch of the mass, and we felt some-

what surprised that we had not seen its lower end when looking upwards under the tenth arch. The spinal cord itself was much flattened, and almost fluid for half an inch at the point of pressure, and it was also extremely soft below down to its end. Above the tumour it was obviously softened somewhat for an inch, but in the rest of its extent the cord showed no naked-eye changes except general slight softening due to *post-mortem* changes.

Microscopical examination of the cord, after hardening in ammoniac bichromate, showed complete disintegration of its structure at the point of pressure, very extensive diffuse disorganisation of both grey and white matter an inch below that point, and similar but less intense changes for about an inch above. Sections in the mid-dorsal region showed no definite degeneration.

With regard to the hydatid cyst, it had a narrow attachment close to the summit of the tenth vertebral arch, and had no visible continuity with the cysts in the muscles. No abnormal hole was found in the bone. No other hydatids were found, and the brain presented no changes beyond a slight chronic arachnitis. There was an enormous quantity of subcutaneous, pericardial, and peritoneal fat, and *post-mortem* changes were everywhere advanced, the heart muscles being honeycombed with small cavities containing gas. It was obvious that rapid infection had proceeded from the bed-sore. The right testicle was atrophied, and the right tunica vaginalis much thickened.

It is much to be regretted that the patient did not come under observation until such a late period of his illness, as, therefore, several interesting phenomena of the development of the malady escaped an accurate record. The history of intense spinal pain radiating to the legs, diminishing or even disappearing for weeks or months at a time, but at the end of a year and a half recurring in greater severity, together with loss of sensation and motor power in the legs, pointed clearly to pressure on the spinal cord; but the diffuseness of the pain and its variable character made it impossible to use this symptom pain for the purpose of accurate localisation, though these characters made it slightly probable that the tumour was extra- and not intradural. Growths of the arachnoid or pia mater tend more to produce unilateral and root symptoms. It is possible that in this case the hydatid cysts in the substance of the muscles had also some share in causing the pain.

So far as could be ascertained, the motor and sensory loss seem to have come on together; and although the patient said that his legs had been rather "stiff" for two months before admission, there seems no doubt that muscular rigidity was never marked. No increase of reflexes was at any time noted, and a month before his admission as an in-patient, it was found by the house-surgeon that the knee-jerks were absent; which fact, combined with some ataxy and loss of sexual power, suggested then the diagnosis of locomotor ataxy. It seems probable that pressure on the posterior roots of the lower dorsal region led to a certain simulation of tabes dorsalis, for the absence of knee-jerks was then not due to wasting of the muscles. With regard to the march of paralysis and anaesthesia, it should be noted that motor power was retained longest in the toes, while sensation disappeared first from the soles, and from the calves before the shins. The foot which first lost sensation retained its motor power longer than the other. Hyperaesthesia at the upper limit of the anaesthetic area was only observed, and then indistinctly, on one day.

A remarkable point is the rapidity of the final pressure effects. The patient was able to walk until the middle of December. Diminished electrical irritability of the muscles showed extensive damage to cells of the anterior cornu on January 12th, and on January 18th he was dead.

The case is of interest in regard to the diagnosis of the level of a tumour pressing on the cord, and shows how it may be higher than the anaesthesia or paralysis would suggest. Extradural tumours probably in this respect resemble dislocations of the vertebrae, in which, as Thorburn's statistics show, the damage to the cord is frequently two vertebrae higher than the level of the anaesthesia. The level of intradural growths can probably be estimated with greater accuracy. The diagnosis of the nature of the tumour was in this case impossible. There were no grounds from either history or physical signs for suspecting hydatids, and the extremely rare occurrence of echinococcus in the spinal canal made it *a priori* improbable. Neisser (quoted by Leuckart in his *Parasites of Man*) collected 986 cases of echinococcus, in only 13 of which were cysts found in the spinal canal; and Dr. Maguire,¹ after searching through the literature of the century, could find only 20 cases in all. In several of these cases the hydatids had eaten into the vertebral column from outside (pleura, peritoneum, muscles), and in one case (Reydellet) a cystic tumour pointed from the lumbar spine, was opened,

and daughter cysts escaped. This patient died a year later from suppuration in the wound. In the great majority of Dr. Maguire's cases several cysts were present, so that our case is exceptional in there being but one. In several cases the cysts were found along nearly the whole length of the spinal cord. In only 4 of Maguire's cases were the hydatids within the dura mater, but in 2 cases of cysticerci the parasite was in the substance of the cord. The duration of symptoms in the 20 cases varied from three months to four years, but most lasted between six and eighteen months. Pain seems to have been a marked and early symptom in most, but in some was not specially mentioned. Anaesthesia became usually but not always complete, and the paraplegia seems in two or three instances to have come on suddenly. No details are given as to the state of the reflexes except in Dr. Maguire's own case. In this case the cysts extended along the upper half of the spinal canal on the dorsal side of the dura mater. The paralysis of the legs was complete and extended to the arms, but sensation was never quite lost. There was retraction of the head, increased knee-jerks, and ankle-clonus.

To sum up, we may say that hydatids of the spinal canal are usually extradural and multiple, but their presence cannot be diagnosed from benign growths, such as fibromyoma and lipoma, unless other cysts are found elsewhere in the body. The fact that they are usually multiple renders them less amenable to surgical interference, but the present case shows clearly that such treatment might in some cases be of very great use.

MEMORANDA:

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

BELLADONNA AND MERCURY POISONING BY EXTERNAL APPLICATIONS.¹

CASE I. *Dangerous Poisoning from Absorption from a Belladonna Plaster.*—Mrs. E., aged 65, seen by me with Dr. Robert Browne of Bourne on August 24th, was lying unconscious with occasional muttering delirium, answering incoherently when roused. The limbs were rigid, with convulsive twitchings, arms flexed, the clenched hands pressed against the throat, pupils widely dilated, urine retained. The symptoms had commenced 24 hours before with giddiness and staggering gait; this was followed after an interval by delirium, complete loss of power and unconsciousness.

On the patient's back I found a perforated belladonna plaster, about eight inches square, the skin below it having the appearance of a healing blister. The plaster was removed and the skin well cleansed. Later, the patient, after being catheterised, became violently excited, and morphine was injected hypodermically; there was immediate improvement, the patient passing into a quiet sleep from which she woke rational and quiet, and in twenty-four hours she was convalescent.

The explanation of the raw condition of the skin which led to the absorption of the poison was, that three weeks before, Mrs. E. had applied a so-called strengthening plaster manufactured by the same firm. Ten hours after its application it had inflamed the skin, producing vesication, and Mrs. E. removed the plaster in consequence. At the end of the three weeks the back having, as she considered recovered, Mrs. E. applied the belladonna plaster; seven hours afterwards the first symptoms of poisoning set in. Dr. Browne has since had another case in his practice in which a belladonna plaster by the same maker had in six days caused inflammation of the skin with serous discharge, to cure which Dr. Browne was called in.

The manufacturers have been informed of the dangerously irritating nature of the basis of their plasters, and will doubtless change their composition. In the meantime, it is desirable that the members of our profession should know of this possible source of danger to their patients.

¹ Notes of these cases were read at the meeting of the South Midland Branch of the British Medical Association, held at Northampton October 6th, 1891.