action of the interossei and lumbricales. At a later time the wrist becomes considerably adducted by the unopposed action of the flexor carpi ulnaris, and the metacarpal bone of the thumb becomes approximated to that of the index finger by the similar action of the adductor pollicis. The attitude of the hand therefore closely resembles that which may be produced by faradising the normal ulnar nerve in the middle of the arm. In the case given below, in which the median nerve was compressed in the forearm, the most marked feature of the hand, in addition to those just described, was the strong flexion of the little and ring fingers due to the action of the unopposed half of the flexor profundus digitorum supplied by the ulnar nerve, and therefore unparalysed. It is worthy of note that in this case contracture of this unparalysed half of the muscle was both more severe and more permanent than in the paralysed muscles supplied by the median. It is this liability of contracture (organic shortening) to affect both paralysed and unparalysed muscles which makes the determination of the exact cause of the attitude assumed by the limb in lesions of peripheral nerve trunks so difficult to determine.

If any doubt as to the existence of nerve lesion remain after the examination of the voluntary power, sensation, and attitude of the limb it can generally be set at rest by testing the electric excitability of the muscles and nerves to the faradic and galvanic currents. In doing this, one or two facts should be borne in mind. In the first place, when the limb has only recently been taken out of splints or plaster, the skin is usually so dry and non-conductive that the reactions cannot be accurately tested until the limb has been thoroughly washed and rubbed for several days in succession. Secondly, mere disuse of a limb from immobilisation produces such a diminution of faradic excitability of all the muscles that often for several days no reaction can be obtained from a strong current. Hence diminution or loss of faradic irritability cannot be relied on as a proof of nerve lesion unless it is confined to a group of muscles supplied by a particular nerve. If loss of faradic irritability is found, some alteration, quantitative or qualitative, or both, will be found in the reaction to galvanism. In all the cases which have come under my notice, except one, faradic irritability has been lost; galvanic irritability, on the other hand, was retained, and in some cases increased together with the usual qualitative alteration. In only one was there total loss of both kinds of electric excitability (Case rv).

The prognosis is a point of considerable importance. It is on the whole extremely favourable. Only one of the six cases now recorded failed to recover (Case rv). If galvanic irritability is not lost at the end of a month from the time of injury, a favourable result may always be expected under proper treatment. The time varied from three to twelve months. It was more rapid in children than in adults.

The treatment consists in passive movement, friction, and electrical stimulation of the affected muscles and nerves. As a rule, no attempt should be made to deal with the nerve injury until the fracture is soundly united—that is, from four to eight weeks after the injury. Adhesions and contracture should then be dealt with, an anæsthetic being used if necessary. The whole limb should then be treated with systematic rubbing and with electricity. Whether for this purpose the interrupted or the constant current should be used is a subject of dispute. At present it is generally recommended to use the constant current, to which the muscles still respond. It is certain, however, that the beneficial effects of electricity do not entirely depend on its producing a muscular contraction. The efficiency of faradism, even after the muscles have ceased to respond to it, is indisputable. Duchenne, to whom we owe the use of electricity in muscular paralysis, produced his remarkable cures entirely with the faradic current. One of my own cases was also successfully treated by faradism alone. The use of the latter has several advantages over the galvanic current; the battery is of simpler construction, less liable to get out of order, and requires much less care and skill in its application than the constant current. This allows the application to be entrusted to a nurse or patient's friend—a point of some practical importance when it has to be continued for some months. Finally, the nerve may often be faradised above the seat of lesion, and the muscles may in this way be got to respond long before volitional impulses

are able to pass. If desired, the faradic current may be applied daily by a nurse or friend, and the galvanic once or twice a week by the surgeon himself.

The question of operation will naturally arise in many of these cases. No operation should be advised, unless there is reason to believe there still exists some pressure or traction on the nerve which can be relieved. I believe that the number of cases in which such a condition exists is small. Of the three cases in which an exploratory operation was done no sign of still existing pressure was discovered in any: in one it is true the nerve lay in a groove of callus, but there was no evidence that it was compressed by it. If, however, there is any condition such as marked displacement of bony fragments, or any other cause which seems remediable by operation, no hesitation need be had in undertaking it and in at least ascertaining the actual state of the nerve.

For permission to publish these six cases I have to thank the honorary surgeons of the Wolverhampton General Hospital.

THE COMPOSITION OF THE FETAL URINE SECRETED IN UTERO.

By T. ARTHUR HELME, M.D., F.R.S.E.,

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My purpose in this communication is to publish a new observation which is, I think, important, in that it provides the missing link of the chain of evidence that the foetal urine is one possible and probable source of the amniotic fluid. Numerous analyses have demonstrated the presence of urea in the liquor amnii, the amount varying in different cases, but always increasing as gestation advances, and proportional to the size and weight of the foetus.¹

The presence of urea has been explained in three ways: (1) By transudation from the maternal structures; (2) by excretion from the foetal kidneys and skin; and (3) from both these sources. Very few observations on the urine from the foetal bladder have been made. Urine obtained from two sources, however, has been examined: (1) That obtained from the bladder of the dead-born child; (2) that obtained by catheterisation from the bladder of the live-born child. Analyses of these urines have demonstrated the presence of urea in each series, but in one case only has a quantitative estimate been made, by Gusserow-Wislicenus,² the amount found being stated as 6.06 per cent.; probably, however, this is a printer's error, as has been suggested by Krukenberg;³ for von Ziemssen, in his *Cyclopædia*, says: "On the other hand, when fluids have been abstained from up to a point when really unendurable thirst has been sustained, I have never found that more than 6.1 per cent. of urea was contained in the urine. The twenty-four hours' urine of a strong, well-nourished adult, himself no great consumer of fluid, contains an average quantity of 2 or 3 per cent. of urea."⁴

The results of analysis of the urine obtained from both these sources are, however, vitiated: (1) In the first series the urine was obtained *post mortem*, and had presumably, therefore, undergone *post-mortem* changes; these analyses must therefore be excluded in discussing phenomena of life. (2) In the second series the analyses are useless for our present purpose because the children had breathed before the urine was obtained.

We have, therefore, no single analysis of healthy foetal urine—that is, of urine from the bladder of a healthy child before it has breathed; in other words, the chain of evidence that the foetal kidneys are one source of the liquor amnii breaks down at this one point; there is no evidence that the urinary secretion of the healthy living foetus contains urea.

¹ Prochownick, Beiträge zur Lehre vom Fruchtwasser, Arch. f. Gyn., Band xi, 1877.

² Zum Lehre vom Stoffwechsel des Fötus, Arch. f. Gyn., Band iii, 1872.

³ Kritische und experimentelle Untersuchungen über die Herkunft des Fruchtwasser, Arch. f. Gyn., Band xxii, 1884.

⁴ Cyclop. of the Practice of Medicine, translated by A. H. Buck, 1877, vol. xv, p. 27.

The fact has been assumed—and quite correctly, as the analysis I now submit shows.

I happened to be called to a lingering labour, in which I delivered the child with forceps (low operation). The child was a healthy full-time male and was born head first. Immediately the breech was expelled, and before any attempt to breathe was made, the child began to micturate. I seized the penis with one hand and a clean cup with the other, and then caught the remainder of the urine—just over half an ounce. This urine was carefully preserved and analysed. For comparison I obtained another specimen of urine from the same child—6 drachms, passed eight and a half hours later.

TABLE I.—Qualitative Analysis.

Specimen I.— $\frac{1}{2}$ ounce. Passed immediately after birth, and before inspiration.	Specimen II.—6 drachms. Eight and a-half hours after birth.
Examination showed— No sugar. No albumen. No phosphates. No sulphates. Chlorides present. Urea " Kreatinin "	Examination showed— No sugar. No albumen. No phosphates. Sulphates present. Chlorides " Urea " Kreatinin " (in larger amount.)

TABLE II.—Quantitative Estimation of Urea (by the Hypobromite of Soda Method).

Specimen I.—Before child breathed.	Specimen II.—After child breathed.
Urea 0.15 per cent., that is, 0.66 grains per ounce.	Urea, 0.3 per cent., that is, 1.32 grains per ounce.

These analyses show that urea is present in considerable quantity in the urine of the normal living foetus. It follows, therefore, that this urine is a possible source of the urea found in the liquor amnii, and that the foetal renal secretion is a possible source of the liquor amnii itself. Kreatinin is known to be present in small amount in the amniotic fluid, but its presence in the foetal urine is especially interesting. Speaking of the presence of this substance in the normal urine of the adult Von Jaksch⁵ says:

The formation of kreatinin has been shown to be intimately associated with the decomposition of muscle substance, and the quantity produced is in direct relation to the amount of flesh meat consumed as food, and, under certain circumstances, to the muscle waste within the body. Under these circumstances muscle contains kreatin, which is changed into kreatinin as it passes through the body. This is to be borne in mind when a clinical inference is drawn from the observation of an increase or diminution of the quantity of kreatinin secreted. According to Neubauer, the quantity excreted by a healthy man is about 1 gramme. According to Pouchet, it is 1 gramme in the case of the male, 0.75 gramme in that of the female, while kreatinin is altogether absent from the urine of sucklings. This point, however, is contested by Grocco.

Its presence was quite evident in both the analyses here recorded—in the specimen before the child breathed the quantity was just enough to give the colour reaction, while in the second the colour obtained was very rich—showing that the quantity of kreatinin was larger: this increase, no doubt, being the result of increased tissue waste by the action of the muscles of respiration now superadded to those which acted while the foetus was still *in utero*. Further observations on the urine of the newborn child enable me to state, in contradiction of Pouchet's statement and in support of Grocco's, that kreatinin is normally present in the urine of sucklings. I have found it in the urine expressed from the child's bladder five minutes after birth, during the first, second, and third days, and at various stages during suckling. I make this as a preliminary statement. It depends on qualitative colour tests—Weyl's nitroprusside of soda with caustic potash, and Jaffe's picric acid and caustic potash. I have not yet isolated the substance.

To sum up:

1. The foetal kidneys secrete actively during intrauterine life.
2. The urine of the normal living foetus contains urea.

⁵ *Clinical Diagnosis*, translated by J. Cagney, M.D., 1890.

3. The urine of the normal living foetus contains kreatinin.
4. The foetal renal excretion is a possible source of origin of the liquor amnii; this is shown by the presence of urea and kreatinin in both these fluids.

5. The presence of sulphates and phosphates in the liquor amnii,⁶ and their absence from the foetal urine, would point to there being an additional source of origin of the liquor amnii.

6. The amount of urea and kreatinin excreted in the urine undergoes a marked increase during the first few hours after birth.

7. Kreatinin is normally present in the urine of the newborn child and suckling.

Lastly, I wish to express my best thanks to Professor Stirling for his courtesy in allowing me to make use of the Physiological Laboratory of Owens College for part of the above analyses.

THE PROBABLE DESTRUCTION OF BACTERIA IN POLLUTED RIVER WATER BY INFUSORIA.

By D. HARVEY ATTFIELD, M.A., M.B.CANTAB., D.P.H.

TOWARDS the end of June, 1892, Professor Dr. Emmerich, of the Hygienic Institute of the Royal University of Munich, suggested that possibly infusoria had something to do with that self-purification of impure water which is well known to occur in many circumstances. With the object of investigating this matter I carried on the following series of experiments in the laboratories of the Institute:

On July 1st two 1-litre flasks, which may be designated A and B, were sterilised. Into each of these was introduced about 500 c.c. of water drawn from a certain disused well. This water contained 10,000 bacteria per c.c., as estimated by counting the colonies on gelatine plates in the usual manner, but was almost free from infusoria. To A was added 10 c.c. of River Isar water. The latter was collected about 150 metres below the influx of one of the main Munich sewers. The Isar water contained much *beggiatox alba* and swarmed with infusoria. The latter were for the most part *paramoecia aurelia* and *P. caudatum*. After diluting a portion of A with sterilised water, three gelatine plates were made containing respectively:

- (1) 0.008 c.c. of A
- (2) 0.016 " "
- (3) 0.024 " "

Three plates were also made from the contents of B—that is, the well water without Isar water:

- (1) 0.01 c.c. of B
- (2) 0.02 " "
- (3) 0.05 " "

The plates and the two flasks were then placed in the cellar of the Institute, because the temperature there resembled that of the well from which the water was drawn.

July 2nd. No colonies were as yet found on the plates. The infusoria in A were observed to be in active movement. More plates were prepared from A and B.

July 4th. From the counting of the colonies on the plates prepared on July 1st,

A had 259,000 bacteria per c.c.
B had 10,000

Additional plates were made from A and B. The plates prepared from A on July 22nd showed that A had on that day 364,000 per c.c.

July 5th. The plates made on July 4th had not developed sufficiently for counting the colonies. Further plates were prepared from A and B.

July 6th. The plates made from A on July 4th showed 48,587 bacteria per c.c.

July 8th. The plates made on July 5th from A showed a bacterial contents of 47,500 per c.c.; from B, 5,450.

These results may be thus tabulated:—

A.	B.
July 1st, per c.c., 259,000 bacteria	July 1st, per c.c., 10,000 bacteria
" 2nd " 364,000 "	" 2nd " "
" 4th " 48,587 "	" 4th " "
" 5th " 47,500 "	" 5th " 5,450

It thus appears that in the water (A) swarming with infusoria the bacteria had decreased to less than one-fifth of their original number in six days, whereas in the water (B) containing very few, if any, infusoria, the decrease of the bacteria in six days was only one-half of their original number.

The suggestion was here made that in the foregoing experiments perhaps light had something to do with the destruction of the bacteria, for a certain amount of direct sunlight had access to the cellar. In the next series of experiments, therefore, similarly-prepared vessels were protected from light by means of covers of black cardboard.

The contents of the two bottles were kept in constant movement by blowing a slow current of air through each bottle by means of a forcing air pump, the air being filtered through plugs of sterilised cotton wool.

⁶ *Landois and Stirling's Physiology*.