THE OPHTHALMO-REACTION OF CALMETTE IN THE EARLY DIAGNOSIS OF PHTHISIS.

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WHILE resident medical officer at the Royal Hospital for Diseases of the Chest I had the opportunity of studying the effects of Calmette's reaction on patients exhibiting various stages of pulmonary tuberculosis. I think it may be of some interest to bring these cases before the profession.

The solution of tuberculin which was employed consisted of glycerine-free desiccated tuberculin of strength 0.1 per cent. prepared from Koch's old tuberculin manufactured by Farbwerke vorm Meister Lucius and Brüning. One to two drops of this solution are dropped into one eye of the patient near the inner canthus. In the meanwhile the lower lid is drawn down by means of the finger, which is kept there for a minute or so to ensure absorption; otherwise it is inclined to be squeezed out, as the patient naturally closes his eye. In all cases before the drop is inserted the eye should be carefully examined for any sign of disease, old-standing or recent, in order to avoid any risk of serious inflammation. Unless the eye is to all appearances free from disease no tuberculin should be placed in it.

The resulting inflammation differs in different individuals, both in time of onset and in the intensity, irrespective of the amount of the involvement of the lungs. In my experience some cases have shown definite reaction as early as three hours after the application of the test; on the other hand, some patients have not shown any reaction until as late as 36 to 40 hours after the test was applied, but in the majority of cases the maximum reaction is obtained in about 12 hours.

The reaction produced may consist of slight conjunctival injection, of marked injection of the whole conjunctiva, both ocular and palpebral, or of marked injection with a mucopurulent discharge, and the patient experiences the usual symptoms of simple conjunctivitis, which entirely clears up in the course of three or four days. In one case, in which a stronger solution was used (1 per cent.), there was not only a marked conjunctival reaction but also an inflammatory condition of the surrounding tissues closely resembling that produced by erysipelas. As a rule, no treatment is required. If the patient complained much of the irritability of his eye the usual treatment for conjunctivitis was employed, but in my experience this was rarely required.

Some observers have recorded bilateral results; none of our cases have shown this. If Calmette's test has been applied to the eye of a patient with pulmonary tuberculosis and several weeks afterwards Koch's old tuberculin has been injected in the usual manner with a positive result, the same eye in some cases has been relit up by a secondary inflammatory reaction.

In some cases of early involvement of the lung no *physical signs whatsoever were discovered*, and the disease was only suspected on a history of hæmoptysis. These are the cases which I should like to emphasise, and in which Calmette's reaction came to our aid and proved to be of the greatest value, the diagnosis being confirmed later by the appearance of crepitations at one or other apex. Thus a positive reaction may be said to carry great weight, and the patient would be wisely treated from the first as if physical signs had existed. One often sees a patient who complains of "bringing up blood," and in whom no physical signs can be found; hence it is difficult to say where the blood is coming from, but, as I have just stated, if Calmette's test is positive, in such a case it throws a grave suspicion on one or other lung.

A negative reaction by no means excludes phthisis, as the test may fail in some early cases, but more especially in old-standing ones, and those who are moribund. If Calmette's test proves negative, and pulmonary tuberculosis is suspected, then a second application has been made in from three to four weeks' time; on this occasion, however, to the other eye. If a reaction now takes place, in my opinion the patient is undoubtedly tubercular, but this was found only to be required in a few cases. I have seen two cases in which a

third application was necessary before a positive reaction was obtained. Patients who have had the test applied have been seen some considerable time after, even as late as a year, and I have *never* had a case which showed any bad results whatever.

Summary.—Out of 225 cases in which Calmette's reaction has been tried, 100 were cases of pulmonary tuberculosis, which all reacted with the exception of seven. These seven were undoubted phthisis, the sputum containing tubercle bacilli. Five of these seven were in an advanced condition; two of these five gave a reaction to a subcutaneous injection of Koch's old tuberculin. The remaining two cases were early, and, as I have stated above, their sputum contained tubercle bacilli.

Of the remaining 125 cases which were not phthisis, and so did not give the test, 25 were cardiac cases of all varieties; 31 were simple bronchitis which cleared up; 20 were chronic bronchitis and emphysema; 23 were of various kinds—e.g., gastric ulcer, dyspepsia, enteric fever, empyemata, neuroses; 8 were bronchiectases; 6 were simple pleuritic effusion of pneumococcal origin; and 12 were cases of lobar pneumonia all of which resolved. In all these 125 cases tubercle bacilli, although repeatedly looked for, were found to be absent from the sputum, and also from the six cases of simple pleuritic effusion.

Conclusions.—1. A negative result by no means excluded pulmonary tuberculosis. Some very advanced cases failed to give a reaction. 2. I have *never* obtained a positive result except in true cases. 3. I never saw any bad results whatever from the use of the test as regards the eye. 4. If a patient in a suspicious case fails to give a reaction, then the test should be applied on three different occasions, with an interval between each application of from three to four weeks.

I am greatly indebted to the physicians of the hospital for their permission to publish these results. My thanks are also due to my colleague, Dr. A. J. B. Leckie, the house physician, for his kind assistance in compiling the cases.

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RESEARCHES IN EPILEPSY.

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THE following investigations into the pathology of epilepsy were undertaken in connexion with the Maghull Epileptic Colony. They are varied in character and do not pretend to have followed any special scheme of line or study, though the majority of them have originated from an idea that the conditions in the intestinal tract have an influence upon the disease. Several are in the nature of repeat experiments upon the work of previous workers. A few have not been attempted before, as far as can be discovered, in connexion with epilepsy.

The work done falls into several categories, and a classification at the outset may tend to simplify subsequent description.

I. *Brain.* 1. Attempts to produce an epileptic animal. 2. The histology of an epileptic brain.

II. *Blood.* 1. The agglutination reaction of epileptic blood with bacillus coli. 2. The opsonic index of epileptic blood for bacillus coli.

III. *Urine.* 1. The urea-ammonia ratio. 2. The evidences of intestinal putrefaction that are afforded by chemical examination of the urine. 3. Correlation of these evidences with the actual putrefactive organisms present in epileptic faeces.

IV. *Faeces.* 1. A general bacteriological survey of the faeces of eight epileptics. 2. A special bacteriological survey of the faeces of an early case.

I. *Researches on the Brain.*

1. *Attempts to produce an epileptic animal.*—It is a noticeable feature of epileptic brains, always when they are seen during life upon the operating table and generally at a post-mortem examination immediately after death, that they present a markedly oedematous appearance to the eye. Closer examination reveals that the oedema lies under the dura mater, in the arachnoid and pia mater, the immediate coverings of the brain. The fluid lies along the vessels and in the interstices of the arachnoid tissue. When extensive, it forms a single sheet of fluid, more or less divided into little drops and ponds by the

divisions of fine fibrous tissue springing from the arachnoid and pia mater. Neither chemical nor bacteriological examination of this oedema fluid by any known method has afforded the discovery of the reason for its presence in epilepsy. It consists of normal cerebro-spinal fluid, and has all the properties and characters of that fluid. Some observers state that it contains more albuminous matter than is normal, and contend that this points to the fluid being the result of an inflammatory process.

It is possible that the mere presence of this fluid might conduce to a condition of the cortical nerve cells of the brain, which would make them peculiarly sensitive and prone to nerve discharges, or from their hypersensitiveness more exuberant in their reaction to stimuli. It was with a view to decide upon the importance of the mere presence of this fluid that the following experiments were undertaken. First, to produce this oedema experimentally in animals. Secondly, to determine whether this oedema, thus produced, was capable of producing epileptic convulsions.

It was found impossible to produce *permanent* oedema of the membranes by any method of fluid injection, and hence the answer to the second question still remains undecided. Altogether 37 rabbits were trephined, various fluids containing albumin were injected under the membranes in varying amount, and the bone was replaced. Natural albuminous fluids were used in the hope that they would be slowly absorbed, and that the artificial oedema produced would last long enough to produce effects. Blood sera and epileptic cerebro-spinal fluid were employed. Pig's serum saturated with common salt was used in the hope of attracting fluid from the vessels and increasing the oedema. Indol, which has always been looked upon as a convulsant, and which is normally manufactured in the intestinal canal by the bacillus coli, was also injected in a very dilute solution, which also contained albumin. Some observers have remarked that the stools of epileptics contain a toxic body of nature unknown, which has convulsant effects when injected into animals. An extract of epileptic faeces was therefore sterilised and injected into the brain membranes of three rabbits which all died, probably poisoned, but even they showed no convulsive seizures.

Table I. gives the results in tabular form:—

TABLE I.

—	Fluid used for injection.	Died operation.	Died afterwards.	Fits.	Recovered.
18	Pig's serum, 0.5, 1.0, 2.0 c.c.	3	2	0	13
5	Pig's serum saturated with NaCl.	0	2	0	3
3	Epileptic cerebro-spinal fluid.	0	0	0	3
8	Solution of indol in peptone water.	1	1	0	6
3	A sterilised extract of epileptic faeces.	2	1	0	0
37	—	6	6	0	25

It was found that 1 c.c. was the largest amount that could with safety be injected at one time, and with this amount great care had to be taken to carry out the injection very slowly, otherwise the animal died almost at once from cessation of respiration. Of the animals, six in number, which died after the operation, two succumbed to septic meningitis, one from a hæmorrhage into the brain through accidental puncture of a vessel, and three from symptoms of pressure from too large a dose. One rabbit injected with normal pig's serum had a spasm of some sort early next morning, but recovered entirely and showed no more symptoms.

Since these experiments were undertaken, six rabbits have been injected with different fluids to discover if it were possible to project the fluid along the vessels into the spaces in such a way that it might be retained. Ten other rabbits which had died from other causes were trephined post mortem, and attempts made to produce an oedema of the arachnoid and pia mater spaces. All these attempts ended in failure. It was found impossible to inject the fluid in any way that it might be retained.

2. *Histology of the epileptic brain.* (a) *Histology of the choroid plexuses.*—With this oedematous condition of the membranes the epileptic brain frequently presents an excess of fluid in the ventricles, which are sometimes distended and distorted out of all shape and size by the liquid they contain. Since this liquid originates in the secretory cells of the choroid plexuses, which lie in the lateral ventricles, it was considered worth while to examine these histologically to discover if any abnormality might be present accounting for this hypersecretion or transudation. In all, the choroid plexuses of seven epileptic brains were removed post mortem and placed in fixing fluid. Sections were made of various portions, which were compared with similar sections made from the choroid plexuses of normal brains. No abnormalities of structures were found.

(b) *Histology of the epileptic brain cortex.*—The histology of the cortical cells has been investigated by innumerable observers, and, except in very chronic cases, very little has been remarked. Turner examined the cortex for the purpose of investigating the conditions of the blood-vessels. He was able to discover minute clots in the arterioles, which he considered to be the cause of fits by the irritation of their presence. Nine epileptic brains have been sectioned and various parts of the cortex suitably stained. No clots such as Turner described have been found.

II. Researches on the Blood.

1. *The agglutination of B. coli communis.*—The toxic theory of epilepsy is one that has obtained great support in past times, and the favourite origin of the manufacture of the toxin has been placed in the intestinal canal. Very many and very various have been the attempts to discover a specific toxic body in epilepsy, and the minds of the greatest number of observers have been directed towards an investigation of the intestinal contents of epileptic patients. The bacillus coli has fallen under suspicion as the producer of nerve toxins, and it was for this reason that indol, which is formed by the B. coli from albuminous materials, was employed in some of the experiments on rabbits.

In all, the sera of 24 epileptics and six normal individuals were tested against four strains of B. coli communis. One strain came from a healthy stool, two were isolated from epileptics at the Colony, and the last was a laboratory specimen. Dilutions were carried up to 1 in 100, and were made 1:5, 1:10, 1:20, 1:25, 1:50, and 1:100. The first three dilutions were made first, and if any agglutination occurred in the 1:20 dilution within two hours the further dilutions were then proceeded with.

In three of the controls some slight agglutination occurred in two hours in the 1:20 dilution; in four of the epileptic sera a little more than a tendency to agglutinate was observable in the same period. In none of the 30 sera tested did agglutination take place in higher dilution than 1:20 in two hours.

2. *The opsonic index against B. coli.*—Fifteen epileptic sera were examined with the laboratory strain and against one strain of B. coli recovered from an epileptic stool. The control sera, single on one occasion only, plural in all others, were usually formed of a pool of three normal healthy sera. The results of this investigation were quite negative. The indices varied within normal limits, and on the whole the variations amongst the 15 epileptic sera were not so wide as between the individual sera of six healthy persons which were examined against one another.

III. Researches on the Urine.

1. *The urea-ammonia ratio* is of little value in itself as evidence of liver activity. It was used in this case, probably mistakenly, to gain some idea of the urea production. Urea is formed in greater part by the re-combination effected by the hepatic cells of ammonia and carbonic oxide, in the form in which it occurs in venous blood (ammonia carbamate). If the liver loses this power to any extent less urea will be manufactured, and ammonia in some form will escape into the blood. Too much ammonia or too little carbonic oxide, either of which conditions might occur through an error in metabolism, would lead to the same result. The alkalinity of the blood would be affected in consequence, though its restoration to normal would take place rapidly by the automatic readjustment of the phosphates. But such an alteration in reaction, though slight and transient, might be capable of affecting sensitive nerve-cells. The gravity of such an altered condition is heightened if the powerful

convulsant effects that ammonia can produce when injected experimentally into the circulation be remembered.

So far only six cases have been examined, but these have been investigated for some months. The 24 hours urine was collected in a covered receptacle containing some thymol to prevent decomposition. The urea was estimated in a Gordon-Little's ureometer. The ammonia determination was performed by three methods, and finally that of Schlössing was adopted as it was the simplest, and if attention were paid to uniformity of the surface-area of urine exposed the error as estimated by the control methods was very small.

It was found that the urea-ammonia ratio varied very much; at times it reached 100 or even more; at its lowest it became as low as ten, in one case 9.3. These numbers were obtained as per following example:—

$$\frac{\text{Amount of urea passed in 24 hours grammes}}{\text{Amount NH}_3 \text{ passed in 24 hours grammes}} = \frac{41.77}{1.081} = 38$$

The fits appear to have some correspondence with a low ratio, although occasionally there is a fit when a high ratio occurs. For example, the ratio was obtained daily in Case 1; it runs:—

CASE 1.—9.33 (1 fit), 12, 36, 82, 28 (1 fit), 38, 55 (1 fit).
 CASE 2.—97 (1 fit), 10 (1 fit), 18, 154, 37 (1 fit), 36, 33, 35.
 CASE 3.—134, 45.2, 24, 10 (2 fits), 15 (2 fits), 36, 45 (1 fit), 29 (1 fit).
 CASE 4.—17 (1 fit), 48, 30, 28 (1 fit), 83.
 CASE 5.—40, 40, 40, 40, 28 (1 fit).

These are weeks taken at random from the charts of daily investigations and speak for themselves. A larger series is required before anything definite may be said.

An attempt was made to obtain the first urine passed after a fit. This was difficult, for the patient so frequently micturated unconsciously. But eight specimens were eventually obtained from different epileptics and passed not more than four hours after the attack. The urea-ammonia ratio of these eight specimens runs: 7.6, 12, 5, 5.7, 3.0, 12, 38, and 15; low in every case.

2. *Evidences of intestinal putrefaction.*—This was investigated by means of Ehrlich's test. A solution of para-amidobenzaldehyde in hydrochloric acid is added to urine, using 10 c.c. of urine and 1 c.c. of the test solution for uniformity. A deep red cherry colour indicates putrefaction, and its degree is said to be measured by the depth of colour produced. Herter has found a correspondence between the amount of skatol in the faeces and the intensity of this reaction. The test was performed on each urine in the following way. Ten cubic centimetres of urine were measured into each of three test tubes, and 1 c.c. of the Ehrlich solution added. One tube was placed aside, the second was gently warmed, and the third rapidly raised to boiling-point.

The results were tabulated in four classes, indicating the production of colour in the cold, on gentle heat, on boiling, or only after boiling and standing some time. Twelve control urines of healthy individuals were first investigated. Of these, two only gave a reaction in the cold at once, seven a reaction on gentle heat being applied, and three a slight reaction on or after boiling. Twenty-three epileptic urines were examined. No urine gave an immediate reaction in the cold. Eight gave a reaction in the cold after standing from varying periods from half an hour to two hours; five gave a colour only after gentle heat. Four required to be boiled to obtain the test; the remaining six required to be boiled and allowed to stand for some hours before a definite cherry tinge appeared.

3. *Correlation of urinary and intestinal evidences.*—Many theories of epilepsy accuse the alimentary tract as the source of the disease. There is no doubt that intestinal irritation frequently precipitates an attack, and, on the other hand, that attention to bowel functions and diet has always some effect, and often a pronounced ameliorating influence, upon the number of attacks. The common cause of intestinal irritation proceeds from constipation and its consequences, and is due to the putrefactive products of retained excreta. The products chemically fall into a class of substances of which indol, phenol, and skatol are good examples. The body in protecting itself combines these bodies with sulphur to form sulphates, in which form they are comparatively harmless. Thus indol is converted into indoxyl-sulphate, and can be recognised as indican in the urine with its well-known blue colour. When thus converted the bodies are excreted by way of the urine and can there be estimated. Other sulphates appear normally in the urine, such simple salts as sodium and potassium sulphate. In the normal body

the complex sulphates comprise a tenth of the total sulphates excreted.

In Table II. the total number of putrefactive organisms has been estimated in a gramme of faeces, and in another column the ratio of complex to total sulphates in the urine of the 24 hours previous to the motion. The putrefactive organisms are here estimated as *B. aerogenes capsulatus*, which is the anaerobic inhabitant of the lower intestine capable of attacking proteid material directly.

TABLE II.

Total putrefactive organisms.	Sulphate ratio.	Total putrefactive organisms.	Sulphate ratio.
1. 1000, not 10,000 ...	1 : 5.7	5. 10,000 ...	1 : 15.6
2. 10,000 ...	1 : 10.3	6. 1000 ...	1 : 14.8
3. 100,000 ...	1 : 5.3	7. 10,000 ...	1 : 11.8
4. 10,000 ...	1 : 12.6	8. 1000 ...	1 : 10.3

Herter and Smith have tried to show that fits run parallel with the ethereal sulphates of the urine. According to their investigations, the fit is preceded by a rise in the ratio, and this rise they suggest to be due to intestinal putrefaction. In the table above there seems to be no parallelism between the intestinal contents and the ethereal sulphates.

IV. *Researches on the Faeces.*

1. A complete bacteriological examination has been made in eight cases of epilepsy from Maghull. In each case as many various organisms as possible were isolated and tested upon various media—(1) to identify them; and (2) to examine their cultural and morphological characters. Special attention was paid—(1) to the strains of *B. coli* isolated; (2) to the numbers of putrefactive organisms present; (3) to the number of staphylococci; (4) to the number of streptococci; and (5) to the number of aerobic spore-bearing organisms.

All results were made comparable by the use of a routine method of dilution of the faeces. In each case a gramme of the stool was carefully weighed out and diluted with 99 c.c. of water, making a dilution of 1 in 100; 10 c.c. of this were transferred to another flask containing a further 90 c.c. of water, making a second dilution of 1 in 1000. This process was carried on until the final dilution was 1 in 100 million. 1 c.c. of this final dilution contained 100 millionth part of a gramme of faeces. 1 c.c. of each dilution was used for inoculating plates of neutral-red-lactose-agar, lactose-litmus-agar, gelatin, often ordinary agar. These were incubated aerobically and anaerobically, and the colonies were counted from day to day. Dilutions were also heated to 80° C. for 20 minutes to kill off all but spore-bearing bacilli, and milk tubes were inoculated from them.

To act as controls some 20 normal faeces have been examined in a similar fashion, so that comparisons might be made. It has been found impossible to adopt any standard of normality for healthy faeces. The variations that take place from day to day in the same individual and from one individual to another swing within wide limits. The only standard that appears to remain fairly fixed is relationship of the classes of organisms one to another.

The epileptic faeces examined have shown no striking points of divergence from the normal. The results of this work have been extremely disappointing, especially after the amount of time and labour that has been put into it.

2. *A special case.*—In May, 1909, a boy, aged 7, was seen, who suffered from *petit mal*. His history was as follows: He had always been a delicate child, and at the age of 3 years contracted a severe attack of gastro-enteritis, profuse diarrhoea with very offensive, slimy, and bloody stools. At this time he had a fit and remained unconscious for 24 hours. The fits recurred after the intestinal condition was relieved up to the age of 5, but they were few and far between. From May, 1907, to June, 1908, he had no fits, but they began again in this latter month. His mother noticed that his motions had become very offensive, and also that he cried out that he had pain in the abdomen before a fit came on.

Treatment with bromide had little effect. Grey powder gave no relief. His motions were examined bacteriologically and the condition was that of great diminution in the colon group of bacilli and a great rise in the putrefactive group. The stools were soft, gaseous, and extremely disagreeable to the nose. They were examined twice, and he was then taken into the Royal Southern Hospital, where he received lactic

acid bacilli in milk. The last two weeks he was in hospital he had no fits, and for a week after returning home he had none. The intestinal condition was entirely changed under the influence of suitable food, attention to the bowels, and the lactic acid bacilli. But the influence upon the fits was practically nil.

Conclusions.—It will be noticed that the results of these investigations are uniformly negative with one exception. There appears to be a relationship between the fit and a low urea-ammonia ratio.

Literature—1. Herter: Journal of Biological Chemistry, January, 1906. 2. Herter and Smith: New York Medical Journal, August and September, 1892. 3. Laplace: Semaine Médicale. 4. Turner, John: Brit. Med. Jour., March 3rd, 1906.

Liverpool.

Clinical Notes:

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

NOTE ON A CASE OF ACUTE ANTRUM OF HIGHMORE WITH EYE COMPLICATIONS.

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A WORKMAN was sent to me on June 30th of this year with the following history. Fourteen days prior to his visit to me he had a severe attack of influenza. On the day following the beginning of this illness he explained to me that he had very acute pain of a neuralgic character, confined to the left side of his face and head. Two days following the neuralgia he began to notice that the sight of the left eye was becoming dim, also that his sight was double; this latter feature he especially noticed on descending the stairs from his room. I found his condition as follows:—

The patient was a young man 22 years of age. From the position in which he sat, and the distortion of his face, it was evident that he suffered great pain. His left eye had a marked vertical squint, only the lower half of the cornea being visible. The pupil was dilated and sluggish to strong light, and it was under the circumstances difficult to estimate his vision accurately, but it appeared to be about 6/60. When I palpated between the eyeball and the inferior orbital wall there was a distinct swelling, irregular and firm, only slightly painful on pressure; there was also some oedema of the lower lid, which the patient explained was not so bad as it had been on the previous day. The fundus of the eye showed the edges of the optic disc blurred and the vessels distended and tortuous; I could not make out any difference in the level of the disc and the surrounding retina. With red and green glasses well-marked diplopia was shown, both horizontal and vertical in character, the vertical being the more marked.

The diagnosis rested, in my opinion, between tumour of the antrum and acute antrum. I punctured the antrum below the inferior turbinate and syringed out some pus with a very offensive smell. It appeared to me that the best thing under the circumstances was to make a good exploratory opening through the canine fossa. I did this and found nothing in the antrum except a small quantity of pus and some polypoidal mucous membrane. A gauze drain was inserted, and in a few days all symptoms disappeared.

West Hartlepool.

RUPTURE OF AN EMPYEMA INTO THE OESOPHAGUS.

BY WILLIAM A. BULLOUGH, M.Sc., M.B., CH.B. VICT.

A GIRL, aged 16 months, was admitted to the Royal Albert Edward Infirmary and Dispensary, Wigan, on Feb. 8th, 1911. Her medical attendant had explored the chest and found pus. The child was in an extremely emaciated condition, and cried in a peculiar way that suggested extreme hunger. On exploring the left side of the chest some turbid

fluid was withdrawn. A portion of rib was resected, but very little pus was found. The lung was contracted and although a number of adhesions were broken down, did not "come up" well. Next day she continually vomited the milk and other fluids which were got down her throat. At the first dressing it was noted that the discharge resembled curds of milk. Consequently the child was given a little milk to swallow, and thereupon a portion of it came through the wound. The quantity swallowed and the quantity coming through the wound were roughly measured and it was found that about half the milk escaped through the wound. On interrogating the parents it was elicited that the child had been continually vomiting her milk for a week prior to admission. We were of the opinion that it was a case of ruptured diverticulum of the oesophagus. However, the child died next day and we made a post-mortem examination.

Necropsy.—The left lung was contracted and firmly adherent to the posterior pleura. On opening the mediastinum a mass of adhesions was found between the oesophagus and the left pleura. An oval-shaped hole was found on the left side of the oesophagus about 3 inches above the diaphragmatic opening. The hole was three-quarters of an inch long and a quarter of an inch wide, the long axis being in the axis of the oesophagus. The edges of the hole looked as though it had existed for some little time, being nicely rounded off. No diverticulum was found.

The presumption is that an empyema had burst into the oesophagus, that the pus had been expectorated by the oesophagus and mouth, and that the child had died from starvation.

I am indebted to Dr. J. Blair for permission to record this case.

Wigan.

Medical Societies.

ROYAL SOCIETY OF MEDICINE.

OBSTETRICAL AND GYNÆCOLOGICAL SECTION.

Large Fibro-cystic Tumour of the Perineum.—Specimens.

A MEETING of this section was held on Oct. 5th, Dr. AMAND J. M. ROUTH, the President, being in the chair.

Mr. J. D. MALCOLM showed specimens and photographs of a remarkable case in which a tumour measuring 37½ inches in horizontal circumference hung from a woman's buttock to the level of her knees. Her age was 30; she had one child, aged 11 years, and six years before admission to the Samaritan Free Hospital she was under the care of an experienced surgeon who found a lax cyst to the left of the vagina and another behind the uterus. It was thought that there was a double vagina, and an incision was made into the lower swelling, but no pus and very little fluid escaped. A year later another surgeon of large experience found three tumours, one in the abdomen, one protruding from the vagina, and one bulging the buttock. After that time the patient did not consult any medical man from July, 1906, until April, 1911, when the buttock tumour had attained the enormous dimensions above mentioned. The anus was dragged out of place, and the finger, when inserted into the rectum, did not pass upwards into the body but downwards into the external mass, which had an area of resonance on its upper surface corresponding to the position of the lower bowel. There were also a tumour projecting from the vagina and an ill-defined growth in the abdomen. The removal of the new growths was obviously a very formidable procedure, but as there was no hope of relief except by operation the patient was told that the treatment would be very dangerous, and she still was anxious to have it carried out. There was a tumour in front of the peritoneum below the umbilicus, another behind the uterus, a third 7½ in. long projecting from the vagina, and the enormous mass of the buttock tumour. These all united in the anterior part of the pelvis on the left side, and they were removed by combined abdominal and perineal incisions, with the exception of a small part in the pelvis. After much fluid had drained from them the parts removed weighed 39½ pounds. Three of the tumours were soft very oedematous solids; the large buttock growth was partly soft solid and partly cystic. The