urine secreted over some period must be measured; by employing ratios valuable indications are given from examination of a single sample, especially if the specimen is selected by the method we have indicated.²

Considerable variation in concentration of urine may even affect these ratios, and for this reason results obtained from very dilute urines are not considered. This variation is a matter with which we hope to deal in a further communication.

The question might very reasonably be asked, Are these ratios merely empirical, or is there any physiological reason for their use? That is to say, have they a theoretical as well as a practical basis? For R_A there is such a basis of theory, as it indicates the effort that is being made to maintain the constant reaction of the blood, giving information of value in assessing its state and more particularly the amount of buffer salts present. The ammonia secreted may be compensatory, or in certain cases may arise from incomplete metabolism; with its source we are not immediately concerned.

With the ratio R_U there appears to be no blood mechanism which relates ammonia to urea; the urea in blood may normally vary within wide limits, and R_U may or may not be a purely fortuitous ratio depending upon the source of the excess of ammonia. The value of the ratio may really be in the numerator which happens arithmetically to be little disturbed by usual variations in the denominator.

It is interesting to note that Palmer and Henderson make use of an inverted form of R_A , but attach no special significance to it, though they find it a convenience in their pathological studies.³

Opinions differ as to the normal value for R_U ; Wells⁴ gives the proportion of nitrogen in urine in the form of ammonia as from 2 to 5 per cent., which in diabetic acidosis may rise to 10 to 25 per cent. Todd⁵ states that normally about thirty times more nitrogen is excreted as urea than ammonia, and when the ratio of urea to the ammonia nitrogen is as 10 to 1 or more, we are justified in saying that acidosis is present, but figures between 10 to 1 and 20 to 1 point to defect of the liver. Burgess⁶ gives an average value in a series of normal children of 7.04. The figures we have obtained are more in agreement with those of Burgess. There would seem to be something unsatisfactory when a ratio which is given normally in the neighbourhood of 4 (Wells and Todd) has to increase to as much as 10 before it has indicative value.

The normal value of R_A ranges up to 1.5; a value exceeding this for an early morning specimen, having a first or acid titration greater than 5 c.cm., indicates some derangement. This is in agreement with the values given by Palmer and Henderson,³ where the quantities are comparable.

To summarize briefly: Of the two ratios R_A is more accurately and readily determined, and it is definitely connected with a well established mechanism. R_U , including as it does the same numerator as R_A , must in a measure give similar indications, but is affected by a denominator which does not appear to have any definite connexion with the numerator such as is found in R_A .

Experimental.

Some fifty samples of early morning urine, presumably normal, gave the following values:

	Average.	Minimum.	Maximum.
R_A ...	1.02	0.6	1.5
R_U ...	6.6	3.4	9.0

so that in normal urines the two ratios run along fairly parallel lines. More interesting are the results of a similar comparison in cases not normal.

Case 1.—A case of cyclic vomiting in a little girl which we were fortunately able to observe also during normal health.

In normal health:

R_A ...	1.08	1.0	1.05	1.50	1.07	1.23	1.13	0.87	1.11
R_U ...	8.2	7.1	7.8	6.1	5.9	6.7	6.6	6.5	6.8

During attack and recovery:

R_A ...	4.47	4.09	1.56	0.84	1.35	1.34
R_U ...	8.9	10.4	8.0	6.4	8.7	9.3

It will be seen that whereas R_A indicates clearly a considerable derangement, R_U gives no very definite information.

Case 2.—A case of simple acetonuric vomiting. We were able to observe this patient also during normal health.

In normal health:

R_A ...	1.23	0.87	1.01	1.22	1.20	0.96
R_U ...	7.3	6.7	7.0	4.9	3.8	7.3

During attack and recovery:

R_A ...	2.10	1.75	1.88	2.0	1.21	1.40
R_U ...	9.9	9.8	4.6	5.9	7.6	8.1

Case 3.—A case similar to 2; the ratios were recorded during attack and recovery:

R_A ...	2.49	4.25	2.57	1.55
R_U ...	10.7	11.3	5.35	5.35

Case 4.—This was a case of diabetes, the patient being an adult male.

R_A ...	2.07	1.82	2.60	3.60
R_U ...	14.8	5.9	15.4	13.0

Here the ratio R_A is uniformly consistent with a marked derangement of function, but R_U fails to indicate this in one case.

Case 5.—A case of catarrhal jaundice in small girl.

R_A ...	2.13	3.36	3.25
R_U ...	15.2	17.3	14.6

Case 6.—A case of catarrhal jaundice in a girl aged 10 years.

R_A ...	1.62	2.0	2.0
R_U ...	4.4	7.1	7.5

A few cases in health and when unwell where a series of specimens of urine were not available:

	Ill.	Well.	Ill.	Well.	Ill.
R_A ...	3.54	1.29	2.22	1.36	3.62
R_U ...	14.5	6.6	8.2	4.7	15.8

It will be seen from these tables that on some occasions both ratios give similar indications, but in certain instances when R_U fails to denote any derangement R_A shows distinct abnormality—that is, R_A appears to give more generally reliable information than does R_U . As the value of these ratios is affected by taking alkalis or acids, the above results were drawn from cases where the administration of drugs containing these substances was not immediately essential in the treatment.

There still remains a considerable volume of work to be carried out upon this and similar ratios, and it would seem highly probable that by careful comparison of values obtained from various ratios it might be possible, not only to discover a derangement in physiological mechanism, but also in a measure to locate it, and we are carrying out a further investigation on the subject.

Conclusion.

Two ratios have been compared, and of the two the first—namely, the ratio of ammonia to acid present in a specimen of urine—seems to give generally better information, and has the advantage of being much more readily and quickly determined.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, July 7th, 1923, p. 21. ² Davis and Rixon: *Clinical Journal*, February 11th and 18th, 1925. ³ Palmer and Henderson: *Journ. Biol. Chem.*, 1914, vol. 17, p. 305. ⁴ Wells: *Chemical Pathology*, fourth edition, 1920, p. 550. ⁵ *Clinical Journal*, April, 1925. ⁶ Burgess: *Guy's Hospital Gazette*, July 5th, 1924, p. 285.

THE PERINEAL AND PELVIC FLOOR MUSCLES IN THE SECOND STAGE OF LABOUR.

BY

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THE care or management of the perineum consists in the use of means for the prevention and limitation of perineal rupture in labour. It applies to the later period of the second stage, the period of perineal distension. The teaching, express and implied, that during the pains of this period the perineal muscles are in a state of contraction seems opposed to some obvious phenomena of labour. This prompted a reference to a number of books on midwifery in order to discover if the teaching were common, and in hopes of learning the evidence on which it is based.

Sixteen textbooks and manuals of midwifery, published from 1899 to 1923, were consulted. Twelve do not mention

the matter, four do. In two of the four the same author is concerned. The terms used are such as these:

Between the pains the pelvic floor is relaxed, not rigidly contracted; try to deliver between pains, the perineal muscles being then relaxed instead of tightly contracted; spasmodic rigidity of the muscles of the pelvic floor obstructs delivery; during a pain there is contraction of muscles of the pelvic floor and outlet; this makes the perineum more rigid and therefore more liable to tear.

All this may be summarized to mean that whilst the perineum is becoming more and more stretched (that is, during the pains) its muscles and those of the pelvic floor are in a state of spasmodic, tight, or rigid contraction, and that this obstructs delivery and favours laceration. This seems to signify that the mechanism of a physiological function normally so acts as to obstruct its completion and favour its injurious accomplishment—a view hard to accept without very satisfying evidence in its favour.

A little consideration of the conditions of the second stage compared with the effects of contraction of perineal and pelvic floor muscles will serve to indicate the difficulty of accepting this teaching. The coccygei and levatores ani in contracting flex the coccyx forwards and upwards upon the sacrum and raise the ano-coccygeal raphe; during the pains the advancing head forces the coccyx downwards and backwards in extension upon the sacrum and depresses the ano-coccygeal area. The pubo-coccygeal fibres of the levatores ani in contraction oppose bilateral distension of the anal canal; during the pains the advancing head spreads the anal canal bilaterally and forces the adjacent edges of the levatores ani far apart. The sphincter ani in contraction closes the anus; it is precisely during pains that the anus is stretched more and more widely open. The compressor vaginae in contraction constricts the vaginal orifice; this orifice is stretched more and more widely open by the head advancing during the pains.

Thus it appears that during the pains of the perineal "management" stage the muscles of the pelvic floor and perineum are stretched, as indeed are all the perineal tissues. They are not contracted, and therefore it is not true that their contraction opposes delivery and favours laceration. Considering the condition of the perineum during a pain just before delivery—bulging, stretched in all directions, tense as a drumhead—one wonders what is the evidence and how precisely it was obtained for the teaching in question. But on these points the books are silent.

A CASE OF HYDATID PNEUMOTHORAX.

BY

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THE rupture of a hydatid cyst of the lung into the pleural cavity is a rare cause of spontaneous pneumothorax, and it is therefore considered that the case detailed below is worthy of record.

This condition was first described by Fouquier in 1828, two years after the death of Laennec. He states that at the autopsy on a case of spontaneous pneumothorax "the right chest was half filled with a yellow turbid fluid in which floated a sac the size of a large nut, rather soft, of milk-white colour, and semi-translucent, which one could not fail to recognize as a ruptured hydatid cyst." In 1925 Professor F. Dévé of Rouen¹ collected thirty-one authentic cases. Since then one other case has been reported by Bacaloglu and Tanasesco.² The condition occurs most frequently in young adults, and in males twice as often as in females. The rupture is usually precipitated by a sudden effort.

The perforation in the pleura is commonly situated on the inferior lobe of the lung and more often on the right side than the left, thus differing in this latter respect from pneumothorax secondary to tuberculous disease. It may be round or linear, admitting the tip of the finger or measuring several centimetres in length. The perforation leads into a cavity in the lung which is not usually larger than an orange. The cyst itself is commonly found free in the pleural cavity and in most cases is ruptured, but

in the case described by Mercier in 1838 it was intact and filled with a clear yellow fluid. It is sometimes accompanied by daughter cysts, and, rarely, there is a secondary growth of echinococcus on the pleura, caused by the dissemination of parasites at the moment of rupture of a fertile cyst.

By studying sections of the lung of a rabbit in which the condition had been artificially produced Dévé came to the following conclusions as to the course of events which take place. There is not a sudden rupture of the cyst into a bronchus, but a gradual invasion of the bronchus by the cyst wall, followed by necrosis of the wall of the bronchus at one point. A pneumocyst is thus produced. The necrotic plug acts as a valve, the tension in the cyst rises and its wall becomes stretched. Owing to the fact that the part of the cyst wall which is adjacent to the pleura lacks support and that its blood supply is diminished, a slight increase of intrabronchial pressure is sufficient to cause it to rupture.

The hydropneumothorax thus produced may remain sterile for several months. Sooner or later, however, the entrance of organisms from the bronchus, combined with the irritation produced by the presence of the hydatid fluid and the cyst wall in the pleural cavity, leads to suppuration and the formation of a pyopneumothorax.

Case Report.

J. P., a groom, aged 36, had been in South Africa in 1913, and had served in France during the late war. He stated that he had always been fond of dogs and that he kept one in the house. He had suffered from cough since being gassed. There had been no haemoptysis or loss of weight.

On December 17th, 1926, at 10.30 a.m., his cough became much worse, and during the next hour and a half he expectorated about two pints of frothy sputum. He felt better during the afternoon, but at 5 p.m., while walking upstairs, he developed rapidly increasing dyspnoea and severe pain all over the chest, not localized to either side. That evening his temperature rose to 102°. On the following day Dr. MacCormack of Steeple Claydon found signs of pneumothorax on the left side. The temperature remained high until December 21st, and then began to settle down. On December 23rd the sputum was purulent. It was examined, and no tubercle bacilli were found. There was never any haemoptysis. On December 28th and January 2nd air was aspirated from the left pleural cavity, giving relief to the dyspnoea. There was no urticaria. There was no family history of tuberculosis.

He was admitted to the Radcliffe Infirmary on January 16th. He was a thin man with flushed face, orthopnoeic, and obviously ill; temperature 100°, pulse 100, respirations 28. The tongue was dry and furred. Cardiac pulsation was visible below the right nipple in the fifth intercostal space. The area of cardiac dullness was entirely to the right of the sternum. The heart sounds were normal. The left side of the chest was larger than the right; movement of the chest was impaired and vocal fremitus absent on the left side. The percussion note was hyper-resonant on the left side down to the seventh rib, and dull below this level. Weak vesicular breathing was present above the seventh rib and absent air entry below. The bell sound was clearly audible on the left side between the fourth and seventh ribs. The liver and spleen were not palpable. The sputum contained pneumococci.

On January 18th the left pleural cavity was aspirated in the ninth intercostal space. About two pints of creamy pus with air were removed. At operation, the same evening, under gas, oxygen, and ether anaesthesia, one inch of the ninth rib in the left scapular line was resected. When the pleural cavity was opened air at first bubbled out, and was followed by about two pints of pus. There was a large cavity the walls of which could not be reached with the finger. No adhesions were felt. A few flakes of fibrin were removed and a tube was inserted.

The pus was found to contain pneumococci, but was sterile. The temperature continued to swing, rising to 100° every evening until January 24th, when a ruptured cyst was discharged through the wound. The temperature immediately fell and remained normal.

The pathological report on the cyst was to the effect that it measured in its collapsed state four inches in diameter, and consisted of a yellowish substance. On the inner aspect were a few small excrescences, but neither in these nor in the scrapings from other parts were any hooklets found. Microscopically the cyst consisted of a laminated, translucent, non-cellular structure. It was undoubtedly a hydatid cyst.

The chest was x-rayed on February 9th, and no evidence of other cysts in the lungs was found. There was no eosinophilia. The patient made an uninterrupted recovery.

According to Dévé the initial cough in these cases is usually dry. The large quantity of sputum expectorated by the patient in the above case might be due to pulmonary oedema caused by hydatid fluid leaking out when the cyst opened into the bronchus.

The noteworthy points in this case are (1) the opening of the cyst into a bronchus, (2) the production of a pneumothorax six and a half hours later, (3) the valvular nature