My best thanks are due to Professor Dr. Einhorn, of Munich, who has on several occasions assisted me in the most courteous manner; also to Drs. Hodgkinson, Hutton, Moritz, Haring, and Fenn, on the staff of the Manchester Consumption Hospital; to Dr. Wild, Physician to the Manchester Skin Hospital; to Dr. Mercer and Dr. Hodge, all of whom have very kindly given me clinical material or other generous help.

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A NEW VOLUMETRIC METHOD OF ESTIMATING URIC ACID IN URINE.

By F. W. TUNNICLIFFE, and OTTO ROSENHEIM,
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In a preliminary communication in the *BRITISH MEDICAL JOURNAL*,¹ one of us (Tunncliffe) drew attention to the solubility in water of urate of piperidine, the salt formed by the action of piperidine on uric acid. This property of piperidine to combine in molecular proportion with uric acid and the solubility of the resulting salt form the fundamental principles of our method.²

If the uric acid is separated from the urine by any of the known methods, and the resulting acid be suspended in water to which a few drops of an alcoholic solution of phenolphthalein have been added, there will be no colour reaction upon the addition of piperidine solution until all the acid is dissolved. If after this point has been reached any further piperidine be added it will remain as such, and by virtue of its alkaline reaction will cause the development of a red colour. Until all the uric acid is dissolved, any piperidine falling into the liquid will be at once converted into piperidine urate, which latter produces no colour reaction with phenolphthalein. By using a solution of piperidine of known strength, the amount which must be added to produce the clearing of the liquid and the development of the purple colour, gives by a simple calculation the amount of uric acid present.³

The method resolves itself into three distinct procedures:

1. The preparation of the standard piperidine solution.
 2. The obtaining of the uric acid in a pure state from the urine.
 3. The titration of the same with the standard solution.
1. We found a $\frac{1}{10}$ normal piperidine solution most suitable. This may be standardised by estimating the amount of it which is required to neutralise a certain amount of a $\frac{1}{10}$ normal acid solution. We do not purpose here giving any fuller directions with regard to the preparation of the $\frac{1}{10}$ normal piperidine solution. To those possessed of a chemical training this would be superfluous, those unpractised in the preparation of standard solutions are advised to purchase the solution from a firm of manufacturing chemists.⁴

2. The uric acid may be obtained from the urine in several ways. The one which we have used, and consider the best, is the method described by Hopkins,⁵ which consists in precipitating the uric acid from the urine with ammonium chloride, that is as ammonium salt, and subsequently decomposing with hydrochloric acid. The uric acid obtained from 100 c.cm. of urine by this method is carefully filtered and freed from hydrochloric acid by washing with water until the washing shows no reaction with Congo paper. For this purpose we have found 15 to 20 c.cm. of water suffice, if an unreasonable excess of hydrochloric acid has not been taken.

3. The pure acid is rinsed with 20 to 30 c.cm. of hot water off the filter paper into a small vessel. Although the urate of piperidine, as we have shown elsewhere, is soluble at 15° C. to the extent of 5.3 per cent., we regard it as advisable in order to obtain a sharp end reaction to bring the fluid which is to be titrated up to the boiling point. A few drops of an alco-

holic phenolphthalein solution are then added, and the piperidine solution allowed to run in from a burette, the liquid being the while kept hot. The complete saturation of the uric acid present is shown by the appearance of a red colour which remains on shaking, and also by the fact that no uric acid remains undissolved.

Since 1 c.cm. of a $\frac{1}{10}$ normal piperidine solution corresponds to 0.00425 g. piperidine, and this is equal to 0.0084 g. of uric acid, the number of c.cm. required to produce the end reaction described above multiplied by 0.0084 will give the amount of uric acid present. In those cases in which 100 c.cm. of urine was the amount originally taken, it is obvious that the number thus obtained will give the percentage of uric acid in the urine.

It may be of interest here to subjoin the following comparative table, which gives the amounts of uric acid present in several urines, as estimated by weighing and by titration with piperidine. The method was to take two portions, of 100 c.cm. each, of each urine, and to estimate the uric acid by titration in one case, and by weighing in the other.

	Uric Acid by Weighing (Hopkins).	Uric Acid by Titration.	Difference.
			Mg.
Urine with an abnormally large percentage of uric acid	0.1056	0.1042	- 1.4
Normal urine	0.0809	0.0809	± 0
"	0.0818	0.0827	+ 0.9
"	0.0849	0.0846	- 0.3
"	0.0640	0.0647	+ 0.7
"	0.0799	0.0786	- 1.3
"	0.0550	0.0559	+ 0.9
"	0.0424	0.0422	- 0.2
"	0.0716	0.0728	+ 1.2
"	0.0809	0.0809	± 0
"	0.0923	0.0909	- 1.4
"	0.0799	0.0777	- 2.2
"	0.0674	0.0674	± 0
"	0.0426	0.0423	- 0.3

From these experiments it will be seen that the average difference between these two methods amounts to $-\frac{1}{2}$ mg. The difference is so small as to be practically negligible. The probable explanation of it is that the urinary pigments thrown down with the uric acid are, in the case of the weighing estimations, weighed with it; in the case, however, of the titration, they remain unattacked by the piperidine solution, and thus do not vitiate the result.

NOTES AND REFERENCES.

¹ February 27th, 1897. ² For the proof that piperidine and uric acid combine in molecular proportions readers are referred to the authors' article in *Centralblatt für Physiol.*, xi, No. 14. ³ *BRITISH MEDICAL JOURNAL*, February 27th, 1897. ⁴ At the request of the authors Messrs. Baird and Tatlock, of Cross Street, Hatton Garden, have undertaken to supply the piperidine solution ready for use. ⁵ *Jour. of Path. and Bact.*, 1893, p. 458. See also G. v. Ritter, *Zeitsch. für phys. Chemie*, xxi, 288.

MITRAL STENOSIS: A STATISTICAL INQUIRY.

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In [the *BRITISH MEDICAL JOURNAL* of November 28th, 1896, I communicated a paper on the above subject, and endeavoured to show, from the *post-mortem* statistics of cases of mitral stenosis during four years at Guy's Hospital, that "the characteristic alteration in the left auricle in mitral stenosis is not dilatation but hypertrophy," and that the necropsy excessively exaggerates the importance of dilatation, which probably is introduced only with the auricular breakdown, and is thus an "old age phenomenon in the history of mitral stenosis."

I have lately analysed the *post-mortem* reports of all the cases of death at Guy's Hospital in the ten years 1886 to 1895 inclusive, in which stenosis of the mitral orifice was found present. The results appear to me sufficiently interesting to be worth recording.

In 4,791 necropsies during this period, there was a total of 196 cases (about 4 per cent. therefore of all examined) in which

the mitral orifice measured $3\frac{1}{2}$ inches or less. If the measurement exceeded $3\frac{1}{2}$ inches (2 fingers), the stenosis was considered doubtful, and neglected. The stenosed orifice exceeded $2\frac{1}{4}$ inches (1 finger) in circumference in 108 instances, and measured $2\frac{1}{4}$ inches (1 finger) or less in 85. It is not clear from the statistics in which of the two catalogues 3 cases should be included. Of the 196 cases, 107 were females and 89 males.

The average age of death for the males was 38½ years, that for the females was also 38½ years. The average age of death for the cases of severe stenosis (1 finger or less) was 33.6 years; for the less severe cases (1 to 2 fingers, or $2\frac{1}{4}$ to $3\frac{1}{2}$ inches), 43.6 years. The average was the same for males and for females.

In 32 cases the heart is stated to have weighed 20 ounces or upwards. Of these 24 were males and 8 females. The heaviest, that of a female, is entered, probably incorrectly, as 31 ounces.

In 32 instances tricuspid and mitral stenosis were present together. Of these 21 were females and 11 males. The tricuspid stenosis accompanied almost exclusively the severe form of mitral, which included 24 of the total 32 cases. In 28.2 per cent., therefore, of the more severe cases (1 finger or less) of mitral stenosis, tricuspid stenosis was also present, whereas in only 7.4 per cent. of the less severe was stenosis remarked on the right side. The presence of tricuspid stenosis at all with these less severe forms would seem to indicate that it was not consequent on the severity of the mitral stenosis; and that its frequent association with the more severe stenosis is due to a common cause, severe or general enough to affect both orifices, while producing considerable contraction of the mitral.

The tricuspid orifice measured less than 3 inches in circumference in 4 cases only, and between 3 and 4 inches in the remaining 28. The orifice was not considered stenosed if the circumference exceeded 4 inches.

In a large proportion of all the cases the aortic valves were thickened, distorted, or otherwise defective, but were seldom referred to as stenosed.

Neglecting the reports for the year 1886 (which were not made with sufficient care to be useful for most statistical purposes), there remain 77 cases of the severe, and 96 of the slighter: form of mitral stenosis for the nine other years. In the 77 severe cases the left auricle is entered as greatly hypertrophied in 22 instances, as hypertrophied (to what extent is not indicated) in 17 others, and as slightly hypertrophied in 5 more. In the 96 less severe cases it was considerably hypertrophied in 14, hypertrophied (amount not indicated) in 2 others, and slightly in 5 more. Auricular hypertrophy, more or less considerable in amount, was therefore recorded as present in 44 of the 77 cases of severe mitral stenosis, and in 21 of the 96 slighter cases, that is in more than half of the one, and in less than a quarter of the others; it is therefore associated especially with severe stenosis.

The left auricle is recorded in the 77 cases of severe stenosis as much dilated in 14, dilated (amount not stated) in 18 others, and slightly dilated in 7. Among the 96 less severe cases it was much dilated in 8, dilated (amount not indicated) in 15, and slightly dilated in 6. Dilatation would, therefore, seem on the whole to be more marked at the necropsies on the severe than on the slighter cases, but the statistics are not carefully enough recorded for conclusions of much value to be drawn on this point.

In the 77 cases of severe stenosis the right ventricle was given as considerably hypertrophied in 22 cases, hypertrophied (amount not defined) in 15 others, and slightly hypertrophied in 4. Hypertrophy, therefore, was present in 41 of the 77 cases; making thus about the same proportion as given for the hypertrophy of the left auricle. In the 96 cases of slighter stenosis hypertrophy of the right ventricle was considerable in 11, was present (amount not recorded) in 8, and slight in 6; which again differs but little from the proportion given for the auricle in these cases.

In the 77 severe cases the right ventricle was stated to be greatly dilated in 16, dilated (amount unrecorded) in 18, and slightly in 6. Among the 96 slighter cases dilatation of the right ventricle was considerable in 7, was present (amount unrecorded) in 17, and slight in 3. The right ventricle is therefore evidently more likely to be found dilated at the

necropsy in cases of severe than of slight stenosis of the mitral orifice.

The left ventricle was generally normal or small; rarely enlarged by either hypertrophy or dilatation.

Of the total number of cases 23 were from the surgical wards where death was not due to asystole from organic lesion. In these instances, though stenosis was present, dilatation of the left auricle was only found on 4 occasions. Dilatation of the auricle would therefore appear to be but rarely associated with mitral stenosis not directly fatal, hence it probably appears late in the disease, causing or accompanying cardiac failure. My statistics, published last year, led me then to the same conclusion.

Pericarditis had been present in nearly one-third of all the cases, and frequently where no definite pericarditis could be traced there was an unusual quantity of fluid in the pericardium. The pericardium in 35 instances was universally adherent. The influence of this pericarditis on cardiac hypertrophy or dilatation could not be determined from the statistics.

Sudden death occurred in at least 7 cases.

Advancing age—60 and upwards—seems to be a factor in the development of stenosis of the mitral orifice, as it was found on 19 occasions in persons of this age, in only 3 of whom was there a history of previous rheumatism. Dilatation of the mitral orifice appears more likely to be followed by dilatation of the left auricle than does stenosis.

My statistics further indicate that a presystolic murmur is heard some time in the course of the disease in about three-fifths of all cases, and a thrill in about one-third or rather less; while a history of rheumatism may be traced in upwards of 60 per cent. of all cases.

The above statistics are, unfortunately, limited in their value and accuracy by the *post-mortem* reports, from which, with some assistance from the ward reports, they are taken. The records of necropsies are sometimes very vague and unsystematic in their account of cardiac lesions. This applies especially to the manner in which hypertrophy and dilatation of the ventricles and auricles are recorded and distinguished.

REPORT OF A CASE OF HERNIA OF THE LUNG, WITH REMARKS.

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THE occurrence of hernial protrusions of the lung is exceedingly rare, and the literature on the subject is by no means abundant, and as one would naturally expect, is almost confined to papers published by military and naval surgeons. The standard textbooks, such as Erichsen's *Science and Art of Surgery*, and Treves's *System of Surgery* dismiss the subject with but a very brief allusion to this interesting condition, and we are left for the study of these herniæ to Morel-Lavallée's valuable essay.

As these cases then are so rare, every case that is met with ought to be put on record, and it is for this reason that I have been led to publish the following very rare form of pneumocele.

I must here acknowledge my indebtedness to Dr. George Patrick, under whose care the case came, for his ready assent to my publishing it.

D. F., aged 47, a striker, was seen on August 15th, 1896, and complained of a painful lump in his back.

History.—He had been playing his musical instrument (the trombone) on a certain Saturday about the middle of July; he felt nothing at the time, but on the Sunday night following, at about 6 P.M., whilst standing in the street he felt a sudden pain "like the stab of a knife" in his back. He went straight home and went to bed, and applied hot water and turpentine. He stayed in bed over Monday and Tuesday, and went to work on the Wednesday as the pain, although still present, was much better. He had no cough, no hæmoptysis, no vomiting. He noticed no lump till Thursday, August 6th, but had had pain, sometimes better and sometimes worse, since the first attack.

Present State.—On examination a swelling was found about $3\frac{1}{2}$ inches long and 2 inches wide, situated opposite the seventh and eighth dorsal spinous processes on the left side about $1\frac{1}{2}$ inch from the processes, freely movable in all directions, not tender, but painful; no dimpling of skin on pinching it up. The swelling was lobulated, and had a distinct edge, which slipped from under the finger. It felt very like a lipoma, and was evidently situated beneath the muscles.

Operation.—The patient was put under chloroform, an incision made,