statement as to acetone—that “its presence without symptoms has no effect on operative treatment or prognosis”—seems to me to require considerable qualification. I think I have shown conclusively that where acute acetonuria is present it requires very careful consideration, and that it renders the prognosis distinctly less favourable, especially when chloroform is the anaesthetic employed. If Hubbard refers merely to chronic acetonuria I agree with him.

CONCLUSIONS.

1. That two separate conditions should be recognized—acute and chronic acetonuria.
2. That ether and chloroform invariably induce a temporary acute acetonuria which may be very detrimental even to an apparently healthy organism.
3. That this acute anaesthetic acetonuria is accompanied by symptoms of acid intoxication, sometimes ending in death, when the kidneys are unable to cope with the increased formation of acetone by a corresponding capability of excretion.
4. That although ether may produce a greater acetonuria, this is less harmful than that produced by chloroform, because ether is less injurious to the cells of the liver and kidneys, and thus does not hinder their power of elimination.
5. That the more plentifully and rapidly excretion is carried on the less serious is the poisoning.
6. That the effects of the poisoning are mitigated by the administration of alkalies, which may also be given with advantage before operation if poisoning be anticipated.
7. That the usual risks of anaesthesia are not increased by pre-existent chronic acetonuria.
8. That anaesthesia is dangerous with pre-existent acute acetonuria, especially if the anaesthetic is chloroform.
9. That a guarded prognosis must always be given when acute acetonuria is present with symptoms of poisoning.
10. That death following the administration of chloroform with symptoms of poisoning may be due to the idiosyncrasy of the patient.

I am indebted to Dr. McAllum, Anaesthetist to the Hospital, for the anaesthetic notes, and my sincere thanks are due to the Sister of the Wards for her kindly co-operation, as without her assistance I could not have carried out a great part of the work.

Test for the Quantitative Estimation of Acetone in the Urine.

In examining urines, I have used the method described in Hoppe-Seyler's *Physiological Chemistry*; this takes on the average four hours to complete. The twenty-four hours' collection having been obtained 50 c.cm. is taken, and if it is not acid a drop or two of a 50 per cent. solution of acetic acid is added. This is distilled very slowly, and generally occupies two hours. When the distillate amounts to 45 c.cm. it should be mixed with a little calcium carbonate and about 25 or 30 c.cm. of cold water and then redistilled. This will occupy about half an hour. The distillate is then acidified with a little of a 1 to 7 solution of strong sulphuric acid and then redistilled. The final distillate is then ready for testing. A known amount of a decinormal iodine solution is run from a burette into the distillate, and a sufficient amount of nitric-free caustic potash is added to at once discharge the brown colour. The mixture is then shaken for half a minute and then allowed to stand for five minutes. Strong hydrochloric acid is now added until the mixture becomes acid, when it should at once become brown again if there is a superfluous amount of iodine present. Finally, a decinormal solution of sodium thiosulphate is run in from a burette until the colour of the iodine is neutralized. This is best estimated by adding a starch solution before all the iodine is neutralized. The amount of thiosulphate used will correspond to the amount of superfluous iodine which has not united with the acetone to form iodoform; from this it is easy to estimate how much iodine has united with the acetone. It has been chemically determined that 1 c.cm. of a decinormal iodine solution = 0.967 mg. of acetone.

Other tests for acetone employed:

To a specimen of the urine add a drop or two of a freshly-prepared solution of sodium nitro-prussiate, two or three drops of caustic soda, and a little acetic acid. Shake, and if acetone is present the foam will be of a purple colour, the depth of the colour depending on the amount of acetone present.

To a specimen of the urine add a few drops of a freshly-prepared solution of sodium nitro-prussiate and some 10 per cent. caustic potash solution. A red colour is produced, which

if left to stand becomes canary yellow, the red colour returns on adding a drop or two of acetic acid and shaking.

The object of this paper has been to show that certain symptoms after operation sometimes followed by death, which are either unaccounted for or attributed to wrong causes, are really due to an acid intoxication of sudden occurrence and variable intensity, originated by the anaesthetic and indicated by the degree of acetone excretion in the urine. Although other circumstances may sometimes have contributed to the fatal result, the intoxication is by itself capable of producing death.

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SUBPHRENIC ABSCESS COMPLICATING EMPYEMA: RESECTION OF RIBS: CURE.

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It was Maydl's well-known monograph in 1894, with its analysis of all the cases of subphrenic abscess published before that year, that stimulated scientific interest in this subject. The decade since 1894 has seen two further articles of a collective character, which have brought our knowledge of the question well up to date. Grueneisen¹ reports upon 60 cases from Koerte's clinic alone; while Perutz² gives a most excellent summary of 208 cases from the literature of the last ten years.

An examination of these sources reveals the clinical fact, possibly not thoroughly realized hitherto, that subphrenic abscess as a complication of an intrathoracic infection, is a decidedly rare event. Of the 447 cases reviewed in the three articles mentioned, only 21 are given as owing their origin to intrathoracic disease. Of these 21 cases we may probably exclude 4 as lacking proof, 2 of Maydl's cases (Nos. 4 and 8 in Paget's review³), and 2 of Grueneisen's (Cases LVI and LVII loc. cit.). Adding to the 17 remaining the case here reported, we get a total of 18 cases. The analysis of this material reveals certain facts of interest. Thus, as to the primary lesion, we find that it was in 6 cases an empyema following pneumonia, in 7 empyema following an undetermined source of infection (influenza? pleurisy?), in 1 an abscess of the lung, in 1 gangrenous bronchitis with pleurisy, in 2 gangrene of the lung, and finally, in 1 tuberculosis of the lung, the focus being in the right lower lobe. Although the cause of the empyema in our own case is set down as “probably grippe,” the finding later of pneumococci in the pus renders the diagnosis of pneumonia a more likely one.

In the reports collected by Maydl, antedating 1894, all but one of which represented *post-mortem* findings, the diaphragm was found perforated; while in later cases, almost all treated by operation, no perforation could be discovered—from which we may deduce that the abscess under the diaphragm originates as a metastatic lesion, by way of the lymphatics joining one cavity with the other, but that later, as the result of prolonged contact with pus, the diaphragm is gradually destroyed at one or more spots.

The mortality figures offer, as usual, a strong argument in favour of operation on modern lines. Of Maydl's 7 cases before 1894 only one came to operation. This patient recovered; the other six died. Of the 11 cases since 1894, 2 were accidental autopsy findings; while of the remaining 9 patients, who were all operated upon, 7 recovered, 1 dying as the result of a second undiscovered subphrenic abscess.

The difficulty of recognizing the subphrenic abscess before operation in cases in which it complicates a pleural effusion, and in which gas is not present, is well illustrated by the simple fact that, so far as I can ascertain, the condition has been uniformly missed. The abscess is discovered only during operation for a supposedly simple empyema. Frequently, as in the present case, the

diaphragm is pushed upwards (not the liver downwards), the two layers of the pleura over the lowermost area of the thorax become adherent, and thus the exploring needle, inserted in the seventh or eighth interspace, passes directly into the subphrenic region without invading the pleural cavity. If, now, the subphrenic abscess be small, the needle may easily miss it; and I believe this to be the reason why in so many of the reported cases one finds mention of one or more negative punctures. Finally, the needle happens to strike the pus; or, going higher in the chest, finds the empyema. In the case here reported the diaphragm was pushed so far upwards that resection of the fifth rib in the posterior axillary line was just sufficient to permit easy palpation of its upper surface for a considerable area.

In a few cases the subphrenic abscess has developed days or weeks after the draining of the empyema. Here the diagnosis is comparatively easy, from the recurrence of septic symptoms, combined with tenderness and bulging over the hypochondrium.

REPORT OF CASE.

The following notes are taken from the history of the case, written by Dr. English, Resident House-Surgeon:

History.

J. B., aged 17, was admitted to the Royal Victoria Hospital, Montreal, April 11th, 1904, complaining of cough. About two months previously he had been taken with what was called "the grippe," characterized by cough, expectoration, some dyspnoea, and slight fever. Pain in the right side soon became marked, and the other symptoms grew worse. While the pain ceased quickly, the other symptoms continued up to his entrance into hospital. About three weeks before admission he coughed up a slight amount of blood. He had frequent chilly feelings all through the illness, and had free perspiration, especially in the mornings. The dyspnoea grew decidedly worse; pallor increased; fever was constant; he lost weight, and upon admission he looked a very sick patient. About a week before the onset of the pulmonary symptoms he had acquired a gonorrhoea, and, at admission, he still had a slight urethral discharge.

His previous history was that of a strong and healthy boy. He could remember no serious illness, nor had he ever had any chronic cough or expectoration. His family history was good, save for the fact that at the time of his admission he had a brother in the surgical ward suffering from an empyema of the left side, tuberculous in origin—which, by the way, ended fatally some months later. There was also some tuberculosis on his mother's side.

Condition on Admission.

Upon examination in the ward the following condition was noted: Young man, apparently of the age stated; complexion muddy and sallow; mucous membranes pale.

Respiratory System.—He has cough with expectoration and some shortness of breath. Inspection reveals some lessening of expansion on the right side and slight bulging of the right lower thorax. On palpation one finds increased vocal fremitus over the right apex, diminished in the right axilla posteriorly. Percussion gives anteriorly a dull note which extends along a line between the second and third ribs inwards to the edge of the sternum, then downwards, merging into the heart dullness. The note below this line on the right side is everywhere dull, and towards the axilla becomes absolutely flat. In the axillary line it is flat as high as the apex. Posteriorly, the dull note begins about the level of the spine of the scapula, and extends, soon becoming flat, quite to the lower limit of the pleura. The only resonant area left occupies the apex anteriorly, the suprascapular region, and the space between the inner border of the scapula and the spinal column. This latter, however, narrows as it goes down, until, at the level of the inferior angle of the scapula, it practically ceases, the area of flat note taking its place down to the free rib border. Auscultation shows in a general way absence of breath sounds over the area of flat note, and diminished sounds over the dull areas. Anteriorly, over the apex one gets an exaggeration of the normal vesicular sounds and over the resonant area of the right side generally one can get a few moist râles. The left side appears clear.

Circulatory System.—Pulse regular, a little fast, of fair tension. The heart is pushed a little to the left side, the apex beat being in the fifth space $\frac{1}{2}$ in. outside the nipple line. The sounds are normal; no accentuation of the pulmonary second. His other organs, with the exception of the urethra, in the discharge from which gonococci can be demonstrated, are normal. In particular the liver is not displaced downward, or, at any rate, not sufficiently so to be observable by palpation. The urine is normal save for the presence of pus cells from the urethritis. During the eight days he was in the ward under observation before operation his temperature ranged from 97.4° F. to 100.3° F., being mainly of a remittent type. His pulse varied from 80 to 100, but was more frequently over 90 than below. Although the case seemed, on the face of it, to be a plain empyema, the two first

exploratory punctures failed to find pus. In the light of subsequent findings, it is probable that they went through the two layers of adherent pleura at the base, through the diaphragm and into the liver, missing the subphrenic abscess. At a third puncture, on March 15th, made immediately below the angle of the scapula, pus was struck and the diagnosis of empyema confirmed.

Operation.

On March 18th, under ether, a needle was inserted in the eighth space, just external to the angle of the scapula, without success. On going in, however, in the seventh space, pus was found, and thereupon an inch or more of the eighth rib was resected in the posterior axillary line. Upon incising the pleura the membrane was found to be much thicker than is usual in empyema. A small amount of bloody pus was evacuated, but on inserting the finger only a limited abscess cavity was discovered extending mainly backwards towards the spine, and but a very short way forwards. It seemed to be bounded anteriorly by vascular adhesions, which were partly broken down, and above and below by stiff tissue. As from the clinical signs the firm tissue above could hardly be the lung, it was assumed to be the diaphragm; and it was only at this point that the likelihood of subphrenic abscess was realized. It was thought possible, however, that we had come upon an encapsulated minor abscess in the thorax, cut off from the main empyemic collection.

The aspirating needle was now inserted above the fifth rib in the posterior axillary line, and clear serous fluid, followed by pus, was withdrawn. The fifth rib was resected at this spot and a large amount of pus evacuated. With the finger one could feel the lung posteriorly compressed against the spinal column, but the boundaries of the abscess in other directions could not be reached, save below, where one came upon a firm, dense membrane, the diaphragm. This was made especially clear by palpating with a finger in each of the openings; the flat membranous structure between the two could only be the diaphragm. A careful search was made, as far as one could reach with the finger, for a perforation, but none could be found. Rubber drainage tubes were placed in each cavity.

After-history.

This was uneventful. He remained in hospital twenty-four days, during which the fever gradually subsided to normal. Twelve days after the operation the tube draining the subphrenic abscess was removed, and a week later that from the empyema. Twenty-four days after operation he was allowed to go home, at his own demand, both sinuses still discharging, and drained by iodoform gauze. His physician, Dr. John West, of Magog, Que., has kindly written me to the effect that the sinuses, after discharging for six months, healed completely, and that the boy's general health was excellent.

The bacteriological examination of the pus (by Dr. Keenan) showed the presence of pneumococci alone, in both the empyema and the subphrenic abscess.

This case offers several interesting points. For instance, the difficulty of diagnosis. It is probable that it will always be impossible to diagnose before operation the presence of a subphrenic abscess complicating an empyema. A displacement of the liver downwards is of no certain value, for that may occur with an uncomplicated empyema. The signs of the one merge into, and are explainable by, those of the other. On the other hand, as here, the abscess may displace the diaphragm upwards. In either case, who can tell just where, in this area of continuous dullness, the diaphragm is situated? As a matter of fact, in the reported cases, the abscess has been always an accidental finding during an operation for empyema, or at an autopsy. The importance of not missing it can be imagined. In one of Beck's cases, quoted by Perutz, death occurred because, though an empyema and also one subphrenic abscess were drained, a second abscess under the diaphragm was overlooked. And we can easily believe that many a patient has been relieved of his empyema only to die of his subphrenic abscess. The main thing, as Perutz says, is to keep the possibility of the thing in mind; then it will not be missed.

It might be objected that the unsuccessful exploratory punctures, antedating the operation by a few days, might, in this and in other cases, be of themselves the cause of the subphrenic abscess by carrying infection from the pleura through the diaphragm into the subphrenic space. The natural answer lies in the fact that in such an event the puncture would not have been negative; to carry pus through would have been impossible without also aspirating it back.

An interesting problem concerns the transport of the infection from the pleural to the subphrenic space. In all probability we find the explanation in Kuettner's¹ work on the lymph stream of this region. This author, by a series of careful injection experiments, established

these facts: that the lymph paths of the parietal, peritoneal, and pleural serosae are in continuous communication with those of the diaphragmatic serosae; that there exist in the diaphragm numerous perforating lymph vessels, and that these pass through the diaphragm not only in the direction from peritoneum to pleura, but also, contrariwise, from pleura to peritoneum. The anatomical disposition is thus given for a spread of infection in the direction illustrated by the present case. On the other hand, the assumption of Burkhardt that the bacteria pass through the membranes and muscle by a process of active growth, and not by passive transportation, cannot be disproven. Probably the metastasis occurs in both ways.

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AN IMPROVED METHOD OF PERFORMING THE "PANCREATIC" REACTION IN THE URINE.

By P. J. CAMMIDGE, M.B.LOND., D.P.H.CAMB.

IN March, 1904, I described a new method of examining the urine in suspected cases of pancreatic disease, the results of which, when considered in conjunction with the clinical symptoms, had been found by Mr. Mayo Robson and myself to be of considerable assistance in diagnosis.¹ Since then numerous experiments have been made with a view to freeing the test of disturbing factors which, either from errors in manipulation or from inherent defects in the method, were liable to obscure the issue or render its interpretation at times a matter of difficulty, especially in inexperienced hands. Before describing the improved method, I should like to correct an erroneous impression which some appear to have received from my original paper. It was not my intention to suggest that the presence of glycerine in the urine had been proved in pancreatic disease, nor did I wish it to be thought that the methods of examination I described were tests for that substance; the glycerine theory was merely mentioned to make clear the genesis of the reaction finally described.

Examination of the phenylhydrazin precipitate derived from the urine in cases of pancreatic inflammation, after treatment with hydrochloric acid, showed that it consisted of a phenylhydrazin compound of glycuronic acid and the osazone of a sugar. Investigation of the precipitate obtained from the urine after treatment with mercuric chloride indicated that it consisted entirely, or almost entirely, of a glycuronic acid compound of phenylhydrazin, so that the difference noticed between the original *a* and *b* reactions in characteristic cases of pancreatic disease was probably dependent on some substance other than glycuronic acid. If, therefore, the glycuronic acid could be removed previous to employing the phenylhydrazin test, the result might be expected to be more definite and the necessity for two comparative tests be done away with. This was found to be possible by taking advantage of the fact that glycuronic acid is precipitated by basic lead acetate from acid solutions, while the sugars are only thrown out when the reaction of the fluid is alkaline. The details of the improved method based upon this fact are as follow:

A specimen of the twenty-four hours' urine, or of the mixed morning and evening secretions, is filtered several times through the same filter paper and examined for albumen, sugar, bile, urobilin, and indican. A quantitative estimation of the chlorides, phosphates, and urea is also made and the centrifugalized deposit from the urine examined microscopically for calcium oxalate crystals. If the urine is found to be free from sugar and albumen, and of an acid reaction, 1 c.cm. of strong hydrochloric acid (specific gravity 1.16) is mixed with 20 c.cm. of the clear filtrate, and the mixture gently boiled on the sand-bath in a small flask, having a long-stemmed funnel in the neck to act as a condenser. After ten minutes' boiling the flask is well cooled in a stream of water, and the contents made up to 20 c.cm. with cold distilled water. The excess of acid present is neutralized

by slowly adding 4 grams of lead carbonate. After standing for a few minutes to allow of the completion of the reaction, the flask is again cooled in running water and the contents filtered through a well-moistened, close-grained filter-paper until a perfectly clear filtrate is secured. The filtrate is then well shaken with four grams of powdered tribasic lead acetate and the resulting precipitate removed by filtration, an absolutely clear filtrate being obtained by repeating the filtration several times if necessary. Since the large amount of lead now in solution would interfere with the subsequent steps of the experiment, it is removed either by treatment with a stream of sulphuretted hydrogen or, what I have found to be equally satisfactory and less disagreeable, by precipitating the lead as a sulphate. For this purpose the clear filtrate is well shaken with 2 grams of finely-powdered sodium sulphate, the mixture heated to the boiling point, then cooled to as low a temperature as possible in a stream of cold water, and the white precipitate removed by careful filtration; 10 c.cm. of the perfectly clear transparent filtrate is made up to 18 c.cm. with distilled water and added to 0.8 gram of phenylhydrazin hydrochlorate, 2 grams of powdered sodium acetate, and 1 c.cm. of 50 per cent. acetic acid contained in a small flask fitted with a funnel condenser. The mixture is boiled on a sand-bath for ten minutes, and then filtered hot through a filter paper moistened with hot water into a test tube provided with a 15 c.cm. mark. Should the filtrate fail to reach the mark, it is made up to 15 c.cm. with hot distilled water. In well-marked cases of pancreatic inflammation a light-yellow, flocculent precipitate should form in a few hours, but it may be necessary to leave the preparation to stand overnight before a deposit occurs. Under the microscope the precipitate is seen to consist of long, light-yellow, flexible, hair-like crystals, arranged in sheaves which, when irrigated with

Crystals obtained from the urine by the improved method in a case of pancreatitis in which there was a gall stone in the pancreatic portion of the common duct.

33 per cent. sulphuric acid, melt away and disappear in ten to fifteen seconds after the acid first touches them. The precipitate should always be examined microscopically, as it may be difficult to determine the characters of a small deposit with the naked eye, and so cases giving only a slight reaction may be overlooked. To exclude traces of sugar, undetected by the preliminary reduction tests, a control experiment is carried out by treating 20 c.cm. of the urine in the same way as in the test described excepting for the addition of the hydrochloric acid.

The urine employed for the experiment should be fresh, and not have undergone fermentative changes. If alkaline in reaction it should be made acid with hydrochloric acid before the test is commenced; any glucose that may be present should be removed by fermentation after the urine has been boiled with the acid, and the excess neutralized. The administration of calcium chloride has been found to interfere with the success of the reaction.

I have arranged the cases examined by this improved method in seven groups, corresponding to the clinical