thrombosis. It is remarkable, too, that a glycosuria so severe as to require 40 units of insulin per diem for its control should have cleared up in spite of progressive deterioration in the state of the cardio-vascular system. This is all the more surprising when it is remembered that during the patient's first residence in hospital the blood sugar had been well above the limits of normal, and that at post-mortem examination the pancreas was found to be shrunken and fibrosed. It seems possible that these variations in the carbohydrate metabolism may have been related to the chronic inflammation which was present in the biliary tract and to the pathological state of the kidneys.

SUMMARY

1. A case is described in which syphilitic aortitis, coronary thrombosis, and extensive myocardial infarctions were associated with glycosuria and hyperglycaemia.

2. Widespread atheroma with degenerative and fibrotic changes in the kidneys and pancreas and chronic inflammation in the biliary tract were found at necropsy in addition to the cardiac lesions.

3. The relationship between glycosuria and coronary thrombosis is discussed. It is considered more probable that these two conditions result from a common cause (vascular degeneration) than that glycosuria is an etiological factor in the production of coronary thrombosis.

I wish to thank Professor W. K. Hunter for permission to use the clinical records of this case, Dr. J. H. Wright who made the electrocardiogram, and Dr. Alice J. Marshall who performed the necropsy.

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PLEURAL EFFUSION*

BY

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Pleural effusion occurs under several conditions, and its treatment is largely dependent on the cause and character of the fluid. For practical purposes effusions may be considered as either clear or thick, and I suggest the following classification.

A. CLEAR EFFUSION

Causation	Appearance	Coagulable Protein	Specific Gravity	Cytology
1. Transudates:		Per cent.		
Renal	Very pale	2	1008	Endothelial cells
Cardiac	Pale yellow	3	1015	
Neoplasm	Pale yellow or haemorrhagic	3	1016	
2. Exudates:				
Tuberculous	Straw colour	Over 4	1020	Small lymph.
Pyogenic	Turbid yellow	Over 5	1024	Polymorph.

B. THICK EFFUSION

1. Non-purulent opaque fluids vary from the cloudiness due to a slight excess of cells to the dense opacity of true chylous effusions. The density may be increased by the presence of blood. A true haemothorax is usually the result of trauma, but bleeding with an effusion is not uncommon, especially in malignant disease; it occurs in about 50 per cent. of cases of carcinoma of lung. Haemorrhage does occur in tuberculosis and other effusions, and should not therefore be taken as diagnostic of neoplasm. The fluid may be rendered opaque by the presence of cholesterol in an effusion of long standing,

or by cells which have undergone fatty degeneration. True chylous effusions are not common, but may occur as a result of occlusion of the left subclavian vein, injury to the thoracic duct or pressure on it by tumours, or by filariasis.

2. Purulent effusion or empyema may be due to one of many causes, such as malignant disease, ruptured abscess of lung, septic emboli, actinomycosis, etc., but for practical purposes it may usually be considered as (a) pneumococcal, (b) streptococcal, or (c) tuberculous.

CLEAR EFFUSION

Before considering treatment I should like to say a little more about the laboratory findings of these fluids. On microscopical examination a few red corpuscles are usually seen. Their presence may be due to bleeding as a result of the pleural puncture, but they are not of clinical significance unless they are sufficiently numerous to be observed by the naked eye. Polymorphonuclear cells may also be present in proportion to the red corpuscles, but if these white cells are in excess it suggests that the fluid is due to a pyogenic organism, and that it will probably become purulent. Eosinophil cells may be regarded as intermediary between an acute infection where polymorphonuclear cells are in excess and a chronic state in which small lymphocytes predominate. A large number of eosinophil cells is said to be evidence against tuberculosis; it also suggests that the fluid will not become purulent. Eosinophilia is present in only 35 per cent. of cases of hydatid disease. As a rule, hydatid cysts of the lung do not leak; unless they do, eosinophilia does not occur. Hydatid fluid may be confused with pleural effusion; it is characterized by the presence of hooklets, not of eosinophil leucocytes. Usually it is like water, but during suppuration it resembles water into which one or two drops of milk have been put. Small lymphocytes occur in chronic inflammatory conditions, the commonest example of which is tuberculosis; therefore an excess of small lymphocytes strongly suggests this disease. Syphilis of the pleura is extremely uncommon, but when it does occur there is an excess of lymphocytes. Endothelial cells come from the lining of the pleura, and are in excess in transudates. They are rarely present in excess in cases of tuberculosis or of pyogenic infections, so that if renal or heart disease can be excluded, a preponderance of these cells in a pleural effusion makes us think of neoplasm. They have sometimes been mistaken for carcinoma cells, especially when they have undergone some degree of degeneration. Lymphocytes have been confused with sarcoma cells, but it is doubtful if neoplastic cells are ever present in an effusion; if they occur, it is very seldom, though they have often been described in error. The absence of tubercle bacilli should not be taken as evidence that the condition is non-tuberculous, for these organisms are hardly ever found in the early stages of an effusion unless a very large quantity of the fluid is used. The bacilli can usually be found in long-standing effusions, and they are present in almost every case of tuberculous empyema.

Treatment of Clear Effusions

When pleural effusion forms in a case of renal disease it is usually bilateral. Repeated aspiration is necessary, as the fluid tends to reaccumulate quickly. In cardiac cases it is best to remove the fluid early, as even a small collection in the pleural cavity embarrasses the heart. In the case of neoplasm, aspiration is necessary only when dyspnoea or pressure symptoms make the patient uncomfortable. I have seen a case of mediastinal tumour and a large effusion which, although the patient lived for six months, did not re-form after aspiration. As a rule, the fluid rapidly reaccumulates, especially in a case of endothelioma of the pleura. Indeed, the presence of an effusion which re-forms after each aspiration is very

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suggestive of endothelioma. In one case I withdrew a quantity of fluid three times in one day.

Most of the cases are tuberculous, and I think every case of pleural effusion should be regarded as of tuberculous origin unless some other definite cause for it can be found. It is necessary to take a few cubic centimetres of the fluid for analysis as soon as a diagnosis is made, but, apart from this, I think aspiration should not be performed except for one of three reasons:

1. To relieve urgent pressure symptoms, or to treat a case in which the fluid is not absorbing naturally.
2. To convert an effusion into a pneumothorax if the underlying lung has active disease.
3. To allow the lung to re-expand if there is active disease spreading in the other lung.

With regard to the first indication, it is not often necessary to aspirate, as almost always the fluid becomes absorbed in time. There is usually a rise of temperature at the beginning; this may continue for several weeks, suggesting an empyema, but in time the temperature will fall without aspiration, unless there is active disease in the lung. If the temperature persists and the patient is ill after six weeks, some of the fluid should be aspirated, but if more than 10 ounces are removed it is best to replace the fluid with air or oxygen. Air should be used if it is intended to make a permanent pneumothorax, but oxygen, which is more quickly absorbed than air, is indicated if one wishes the lung to re-expand. If, in spite of removing some of the fluid, the temperature does not settle, the remaining fluid should be completely aspirated and replaced with air or oxygen. In this case, however, it is almost certain that there is active disease in the underlying lung, and as the lung re-expands it should be carefully examined for any sign of disease, and repeated x-ray examinations should be made. One must remember that tuberculosis starts in the lung and spreads into the pleura; it does not start in the pleura and spread back into the lung; consequently when one sees a case of pleural effusion one should assume that there is disease in the lung, although in most cases it is not active, and does not develop into clinical disease. Some 40 per cent. of cases, however, do go on to clinical tuberculosis, and for this reason it is necessary to keep a patient under careful supervision and give him thorough after-treatment following pleural effusion.

In treatment by pneumothorax pleural effusion frequently forms, and in my opinion this is a true tuberculous exudate, and is not due to irritation of the pleura, or to any mechanical cause. It has been stated that if the gas is heated, pleural effusion is not so likely to occur, but I have tried heating the gas, and have found an effusion developing to the same degree as in my other cases. I have also noticed no difference between the influence of summer and winter on the accumulation of fluid.

When the fluid is examined the only organisms found are tubercle bacilli, and in the great majority of cases these can be demonstrated after the first two months. Moreover, the cytology of the fluid suggests an exudate, and I have not known an effusion form in any case I have treated with pneumothorax for diseases other than tuberculosis, except in one case of an acute abscess, when there was a small amount of fluid for a short time. Pleurisy is such a common complication of pulmonary tuberculosis that one would expect an effusion to form if the parietal and visceral pleura were separated so that they could not become stuck together. An effusion forms in practically every case of spontaneous pneumothorax due to tuberculosis, but in those cases where air is quickly absorbed and there is no effusion the patient makes a complete recovery, and subsequently no evidence of tuberculosis can be found.

When an effusion occurs during treatment by artificial pneumothorax it keeps the lung collapsed so that the intervals between the refills can be increased by weeks, or even months. The fluid may remain clear and unabsorbed, but there are two dangers; one is that it becomes tuberculous pus, and the other is that, as it is absorbed, organization takes place, and the pneumothorax cavity becomes gradually obliterated. On thoracoscopic examination, white strands of fibrin may be seen in the fluid. Many cases of pneumothorax which terminate by obliteration in this way do very well, a satisfactory fibrosis occurs in the diseased part of the lung, and the healthy part begins to function again. If, however, it is desired to maintain the pneumothorax, the fluid should be aspirated from time to time, and if any signs of obliteration are found, the fluid should be completely removed and replaced by a 5 per cent. solution of gomenol in olive oil. Usually, after one or two aspirations, the fluid does not reappear.

TUBERCULOUS PURULENT EFFUSION

If an effusion changes into tuberculous pus the condition is much more serious. I have had a few cases in which the pneumothorax has been maintained and the patient has done well, in spite of having had a tuberculous empyema; but these are very exceptional, and the majority die. For this reason, as soon as a tuberculous empyema forms, I encourage the lung to re-expand by frequent wash-outs with Dakin's solution, leaving a negative intrapleural pressure. In most cases this is successful, the lung can be made to re-expand, and in favourable cases becomes a chronic fibroid lung. If, however, it does not re-expand, I advise thoracoplasty, provided there are no serious contraindications to the operation. The principles of treatment in a case of tuberculous empyema are therefore:

1. Remove the pus by aspiration, and not by open drainage. Wash out the pleural cavity with Dakin's solution in order to remove the fibrinous lining round the pleura and to encourage the lung to re-expand. If there is a bronchial fistula or perforation into the lung, as in a case of spontaneous pneumothorax, Dakin's solution is too irritating to use, and the pleura should be washed out gently with saline or methylene blue. When there is a large perforation, however, it is unusual for the lung to re-expand, and thoracoplasty becomes necessary.
2. If the lung fails to re-expand and to obliterate the pneumothorax or empyema cavity in spite of this treatment, some thoracoplastic operation should be performed to obliterate the cavity.
3. Attend to the general condition of the patient.

PYOGENIC PURULENT EFFUSION

The treatment of non-tuberculous empyema depends almost entirely on whether the pus is free in the pleural cavity or whether it is pocketed. If it is free, the condition is, in effect, pyothorax, and any operation for drainage will convert it into an open pyopneumothorax, a condition in which the mortality is very high indeed. If there are no adhesions and the mediastinum is free, a wound which opens the pleural cavity on one side affects the pressure not only on that side, but to practically the same degree on the other. The result of this is that, during inspiration, some of the air that should enter the lungs through the trachea enters the pleural cavity through the open wound. If, therefore, the opening into the pleura is larger than that into the lungs through the larynx, it follows that the lungs will not expand, and the patient will die of asphyxia. This was found to be the case in gunshot wounds during the war, and also by experiments on dogs. In the case of pneumonia the lungs are already getting insufficient air, so that the effect of an open pneumothorax is still more disastrous.

The first principle of treatment, therefore, should be to avoid an open pneumothorax. This may be done by using a drainage tube with a valve, which allows the pus to leave but prevents air from entering the pleural cavity. It is usually better, however, to aspirate the fluid at first until adhesions have formed, when incision and open drainage can be performed if necessary, although a certain proportion of cases get well with aspiration alone. In practice it is found that pneumococcal empyema in lobar pneumonia occurs after the illness. The pus is thick, and there are adhesions, so that it is safe to resect a rib and drain the cavity. Moreover, by the time the empyema forms, the patient has, as a rule, recovered from the toxæmia of the pneumonia. For these reasons treatment by incision and drainage is usually successful in pneumococcal cases, and when once the pus is found to be thick, it is better to drain the empyema.

In streptococcal cases, however, the effusion forms early, whilst the patient is still suffering from the toxæmia of the disease. The fluid is at first cloudy and thin, and there are no adhesions, consequently incision and drainage is likely to be fraught with danger. Dr. W. J. Stone published the results of three series of cases.¹ In the first series 85 cases were treated by early operation and drainage, and the mortality was 61.2 per cent. In the second series 96 cases were treated by early aspiration and late operation, and the mortality was 15.6 per cent. In the third series 94 were treated by early and late operation, and had a mortality of 9.5 per cent. In some cases aspiration alone is sufficient, but in the majority subsequent drainage becomes necessary, and when once the fluid has turned into frank pus it is usually safe to operate. After operation, in every case of empyema, it is essential to wash out the pleural cavity frequently with Dakin's or some similar solution in order to encourage re-expansion of the lung, and to disinfect the pleural cavity. The principles, therefore, in the treatment of pyogenic empyema are:

1. Remove the pus, but always avoid an open pneumothorax.
2. Encourage the lung to re-expand and obliterate the empyema cavity. This should be done by washing out the cavity, and any drainage should be arranged so that air cannot enter from outside.
3. Sterilize the cavity by frequent irrigations.
4. Attend to the general condition and nourishment of the patient.

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BATH TREATMENT FOR DEAFNESS

BY

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Any measure which will help, even in a minority of cases, what is commonly called chronic progressive deafness is worth consideration, especially if it sometimes succeeds where the usually approved methods fail, and if it is a simple procedure capable of supervision by the general practitioner. The following case illustrates the point.

CASE I

A man, aged 42, consulted me in December, 1927, about deafness in the left ear, which had increased during the previous two years. There was no obvious retraction or thickening of the tympanic membranes, but he had difficulty in self-inflation. With the right ear he could hear a whisper across the room (say 15 feet away); with the left ear, 5 feet away. After treatment by catheter, bougie, and inflation for a year, the condition was worse. A whisper was then heard by the right ear at a distance of 5 feet; by the left ear, 2 feet. He was ordered hot baths, with a pound of commercial Epsom

salts added to each bath; immersion for ten to fifteen minutes was advised at bedtime every second night for a week, once during the following week, and then once a fortnight. Within a week, the hearing distances for whisper were 15 and 9 feet (instead of 5 and 2 feet) for right and left ears respectively. By May, 1930, the distance for each ear had increased to 30 feet. Improvement is still maintained, and is valuable not only to himself but to his colleagues. The baths are still necessary, but they are taken at longer intervals.

As often happens with new treatment, this first case has perhaps been the most striking. The success induced me to try the method in other cases, and sometimes gratifying results were obtained.

CASE II

For many years the manager of a large business, aged 64, had increasing deafness for which I tried in vain the usual measures. Some years elapsed, during which he did not consult me, but after the experience mentioned I asked him to see me. On examination, the right membrane was dull and indrawn, the left was ruptured and atrophied, and the ossicles were bound in cicatrix. Conversation voice was heard 4 feet away on the right side, 4 inches away on the left side. The latter figure is naturally open to question, because of possible hearing by the right ear even when closed. After treatment by baths for two months, the distances were 10 feet and 2 feet respectively, to his great delight.

CASE III

A hospital matron, aged 33, had tinnitus in the left ear, and slight deafness. The Eustachian tubes were narrow; systolic blood pressure 100 mm. Hg. Resection of nasal septum, removal of tonsils, and passage of Eustachian catheter and bougie were all tried, with no obvious benefit. When the patient reached the age of 38, the tinnitus was worse and was in both ears. When, two years later, in May, 1930, a whisper was heard by both ears at 8 feet, I asked her to try baths. A fortnight later the figures were 21 and 25 feet respectively, tinnitus not having improved.

CASE IV

A lady, aged 60, complaining of deafness beginning in the left ear ten years earlier, came to see me in October, 1930. Both membranes were dull, and inflation by catheter was difficult and imperfect. She heard the conversational voice 15 inches away on the right side, and 5 inches away on the left; Weber referred to left. As there was no improvement on inflation, she was asked to try baths. At the middle of December, conversation was heard on the right side at 16 feet distance (instead of 15 inches), on the left at 10 inches.

CASE V

A man, aged 58, had noticed deafness and tinnitus in the right ear for five months. Both membranes were indrawn. A whisper on the right side was heard 2 inches away, but after three weeks of bath treatment the distance had increased to 7 feet; tinnitus was still present, but less marked.

The less chronic cases respond more readily. Those described above were probably "catarrhal" cases, with narrowing of the Eustachian tube—the type in which sweating might be expected to help. Encouraged, however, by the improvement in the cicatricial ear of the patient (Case II) mentioned above, I resolved to try baths in the following case:

CASE VI

A man of 39, who complained of deafness in the right ear after an explosion in the trenches in 1916, came to me in June, 1930; there was also tinnitus in the right ear. The left ear was normal; the right drum was "gone," ossicles adherent, no discharge. He was asked to try baths. The hearing for whisper before treatment was at a distance of 3 feet; after two months' treatment, at 60 feet.

One might expect benefit from this treatment in patients suffering from Ménière's complex, if it is tried early. I have had the opportunity in only two such cases, both late ones. In the first there was complete failure; in the second there seems to have been some success, as shown by the history.