2. *Water*.—All the patients were drinking Sheffield water, which has a low calcium content. To test this point the amount of calcium of a few Sheffield persons was obtained and compared with the amount found in a few London residents. The average quantity of calcium in the salivas of the ten Sheffield persons was: adults 8.07 and children 7.19 mg. in 100 c.cm. of saliva. The average quantity of calcium in the saliva of the ten London cases was: adults 10.61 and children 9.34 mg. in 100 c.cm. of saliva. Röse³ carried out investigations with the idea of relating the extent of caries to the calcium content of the drinking water in different districts. He found that, on the whole, the children living in districts where the water was hard suffered less from caries than those living where the water was soft. The amount of calcium in the saliva of these subjects, however, was often less when the water was hard than when it was soft. Röse states that the full influence of a calcium-rich diet is only seen after several generations. It was found in the present investigation that the addition of calcium phosphate, calcium lactate, and calcium carbonate over a short period to the dietary of patients had no effect on the calcium content of the saliva.

3. *Age of the Patients*.—No definite relation was noticed between the age of the patient and the amount of calcium in the saliva.

4. *Hour of Collection*.—The amount of calcium in the saliva varied slightly at different times of the day in individual persons, but this variation was not constant.

5. *Rapidity of the Secretion of the Saliva*.—It was not found that the percentage amount of calcium varied appreciably with the rapidity of secretion.

6. *Hydrogen-ion Concentration*.—This varied between a pH of 6.5 and 7.5, and bore no relation to the amount of salivary calcium, nor to the degree of dental caries.

SUMMARY.

1. The amount of calcium in the saliva is influenced by diet. (a) A high calcium content of the saliva can be produced by a diet having a large fat-soluble vitamin content. (b) A low calcium content can be produced by a diet containing much cereal (especially oatmeal) and comparatively little fat-soluble vitamin, although the amount of calcium ingested in this diet may be the same as, or even greater than, in (a).

2. The calcium of the saliva is not increased by increasing the calcium of the diet over a short period.

3. Tuberculous children appear to have a lower calcium content of the saliva than normal children, and, on the whole, low resistance to the infection is accompanied by low calcium content. This relationship between resistance to infection and salivary calcium requires further investigation before it can be regarded as established.

4. It will be seen that the calcium content of the saliva is controlled by the same factors of special nutrition as those on which the calcification of teeth and bones also depend. If, therefore, caries of the teeth be influenced by the chemical composition of the saliva, then it is evident that the problem of dental caries is at least partially one of general metabolism. This point of view is corroborated by other results showing the relation of diet and caries, published elsewhere.

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PLEURAL EFFUSION COMPLICATING PNEUMOTHORAX.

BY

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PLEURAL EFFUSION is the most common complication of pneumothorax. If every patient with pneumothorax were examined daily with the x-ray screen there would be seen at some time or other in practically every case a small pool of fluid lying in the costo-diaphragmatic angle. Such small collections of fluid, however, are quickly absorbed; they come and go, and are of no importance. In speaking of pleural effusion I mean cases where the fluid at least covers the diaphragm and lasts more than a week, and I do not include those small transient effusions discovered accidentally by x rays.

Out of 309 consecutive cases in which I have successfully induced artificial pneumothorax for pulmonary tuberculosis an effusion formed in 128 (41.4 per cent.), and of these 47 (36.7 per cent.) are dead. Of the 181 cases in which no fluid formed, 57 (31.5 per cent.) are dead. Of the 128 cases the fluid remained clear in 92, and 27 (29.4 per cent.) of these are dead. In the other 36 cases the fluid was tuberculous pus, and 20 (55.6 per cent.) of them are dead. In time, no doubt, more of these cases will die of tuberculosis, but this applies equally to cases with and without fluid, so that the figures can be taken to show that, roughly speaking, cases which have clear pleural effusion do neither better nor worse than dry cases, but that if a tuberculous empyema forms the outlook is much more serious.

It should be clearly understood that these 309 cases do not include non-tuberculous cases, nor those where artificial pneumothorax was attempted but failed or was only partially successful. I have divided them into three groups: C1, where the disease was unilateral; C2, where there was some disease in the other lung but only slight; C3, where more than one-third of the better lung was affected. The last group consists largely of cases where artificial pneumothorax was induced as a last resort, and so the mortality is very high. Had the patients in this group lived longer many more would undoubtedly have developed effusion.

The following table shows the number of cases in the various groups which developed fluid.

	C1.		C2.		C3.	
	Cases.	No. Dead.	Cases.	No. Dead.	Cases.	No. Dead.
Total ...	126	24 (19%)	125	47 (34.8%)	48	33 (68.7%)
No effusion ...	78	13 (16.6%)	72	22 (30.6%)	31	22 (71%)
Clear fluid ...	35	5 (13.9%)	45	14 (31.2%)	11	8 (72.7%)
Pus ...	12	6 (50%)	18	11 (61.8%)	6	3 (50%)

In my series, effusion in the great majority of cases developed in the first six months and rarely occurred after the first year. The number of months after the induction of artificial pneumothorax when the effusion formed was:

1 to 3 months ...	45 cases
4 to 6 " ...	43 "
7 to 12 " ...	29 "
13 to 18 " ...	5 "
Over 18 months ...	6 "

Various explanations have been given to account for the frequency of pleural effusion in cases of pneumothorax. Irritation of the gas, its temperature, the repeated punctures of the parietal pleura, or the intrapleural pressure have all been described as playing a part in the production of fluid. In my opinion, however, these effusions are true tuberculous exudates, and in my experience they occur with equal frequency whether the gas is heated or not and whether air, nitrogen, carbon dioxide, or oxygen is used. We know that pleurisy is a common complication of pulmonary tuberculosis, and that it often leads to

adherent pleura. If the parietal and visceral pleurae are separated, as they are in cases of pneumothorax, one would expect effusion to form if pleurisy developed. In almost every case of spontaneous pneumothorax (where, of course, the disease has spread to the pleura) effusion develops. The cytology of the fluid is that of a tuberculous exudate, and although at first tubercle bacilli are not usually found, in long-standing effusions they can almost always be demonstrated. In none of my cases has any other organism been found. If my opinion is correct we would expect to find that in cases of non-tuberculous disease artificial pneumothorax would not be complicated by pleural effusion, and this is actually the case. In one of my cases of abscess of the lung a small pool of fluid appeared for a short time, but in none of my other 54 non-tuberculous cases of artificial pneumothorax did any fluid form—a percentage of less than 2, as against 41.4 for the tuberculous cases.

In spontaneous pneumothorax effusion is usually present, being absent in only 4 of my 22 cases, and these 4 all did well. As a rule, the fluid in spontaneous pneumothorax becomes thick tuberculous pus, the lung never re-expands, and the outlook is very serious; 13 of my 18 cases in which fluid formed are dead, and only 2 can be said to have done really well. I think thoracoplasty should be considered early in many cases of spontaneous pneumothorax, for unless the pneumothorax cavity can be obliterated by re-expansion of the lung it will sooner or later become infected, and the outlook is almost hopeless.

The treatment of pleural effusion complicating artificial pneumothorax depends on many factors, and each case must be taken on its merits. If the fluid is left it will usually cause obliteration of the pneumothorax cavity by pleural adherence and fibrosis starting at the bottom of the pleural cavity and working upwards. If this result is desired, therefore, the fluid should be left, but the patient must be kept under careful observation lest too much fluid develops and causes displacement of the mediastinum. Before this happens a sufficient quantity of the fluid must be aspirated. Many cases in which artificial pneumothorax treatment is allowed to terminate in this way do extremely well. As the lung is drawn out a satisfactory fibrosis develops over the diseased areas and the healthy parts of the lung begin to function once more. If, however, it is thought better to prevent obliteration of the pneumothorax cavity and to continue the refills it is very important to keep the pleural cavity dry by aspirating the fluid at each refill. Usually after one or two aspirations the fluid does not re-form. When fluid forms, a lung which was previously partially collapsed will become flattened out, and as the fluid accumulates the pressure rises, but it should never be allowed to rise sufficiently to displace the mediastinum. Sometimes the first indication of the presence of fluid is an unusually high intrapleural pressure. In one of my cases a pressure which was usually negative before a refill was found to be +5+10, and after giving 100 c.cm. of air it rose to +10+15. The pressure may, however, be negative in spite of the fluid, but a small refill will render it positive. For example, in one of my cases with fluid the pressure before refill was -10-6, but it became +17+19 after only 100 c.cm. of air had been given.

In replacing fluid the quantity of gas given must be much smaller than that of the fluid removed; roughly speaking, about one-quarter the quantity is required. In one case 225 c.cm. of fluid was removed and 200 c.cm. of air given, and the pressure was altered from -8-0 to 0+6. In another case where the initial pressure was -17-9, after removing 600 c.cm. of fluid it became -22-13, and after another 200 c.cm. had been removed it was -28-15. On adding 200 c.cm. of air the pressure at once rose to -16-9, another 200 c.cm. made it -10-6, and a third 200 c.cm. sent the pressure to -4-0. In one more case a pressure of +7+14 became -7+4 after removing 1,700 c.cm. of pus. It is not wise to allow the intrapleural pressure to alter too suddenly, and in replacing fluid with gas I think it is best to use two needles, the gas being introduced through one and the fluid flowing out through the other; by this method the pressure can be raised or lowered or kept constant at will. If it is wished to encourage the lung to re-expand the fluid should be removed without adding any gas, or if this is not thought wise

oxygen should be used, as this is absorbed more quickly than air.

If the fluid is purulent the case is very much more serious. The pus should be aspirated and the intrathoracic pressure kept as low as possible in order to help the lung to re-expand. If the lung does re-expand the outlook is hopeful, but if x rays show that there is no sign of re-expansion in the lung then we must face the fact that the patient will probably have a permanent pyopneumothorax and be liable to any of the many complications of that condition. In these cases I have washed out the cavity with saline, normal or hypotonic. I have injected 2 per cent. formalin in glycerin and other disinfectants into the pleural cavity, and in cases where the pus is thick I have injected some modifying fluid and rendered the pus more liquid and easier to aspirate, but the end is the same. If, therefore, the fluid is purulent and the lung fails to re-expand I think the best chance for the patient is to perform thoracoplasty as soon as possible. Before the operation, however, it is necessary to get the patient as fit as possible by washing out the pleural cavity and allowing him a period of rest in bed and good feeding. In most cases a preliminary operation for evulsion of the phrenic nerve is desirable. It is hardly necessary to add that active disease of the other lung is a contraindication to thoracoplasty, but in this case the outlook is almost hopeless. If, however, the better lung shows no sign of active disease I feel strongly that in these cases thoracoplasty offers the best chance to the patient.

A CASE OF CHRONIC OBSTRUCTIVE JAUNDICE:

FOCAL NECROSES IN THE LIVER: MYELOID TRANSFORMATION OF THE SPLEEN.

BY

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I HAVE recently encountered a case of chronic obstructive jaundice which presents some points worthy of comment. I will relate only the main points of the patient's last illness.

A man, aged 62, had chronic obstructive jaundice (complete obstruction) owing to primary adenocarcinoma of the hepatic duct at its junction with the cystic duct. He died on March 11th, 1926, after having had jaundice for only about one month. He had had occasional haematemesis, but at the *post-mortem* examination Dr. E. Bock found no lesion in the oesophagus, stomach, or upper bowel to account for it. The adenocarcinomatous growth was small and macroscopically scar-like. No metastases were seen. The liver weighed 2,300 grams (70½ oz.), and the intrahepatic bile ducts were distended with colourless bile. On microscopic examination sections of the liver were found to contain numerous foci, recalling by their small size and their irregular distribution the focal necroses of infectious diseases (enteric fever, etc.). They were distributed in various parts of the hepatic acini and contained necrotic liver cells and bile pigment; in many of them there were also quantities of large phagocytic cells (macrophages), and in some of them there were also collections of polymorphonuclear leucocytes, as if suppuration were threatening.

There had been no fever during life, and an infective origin for the focal necroses can hardly be maintained. It seems to me that these necroses are most probably a minor variety of what Horst Oertel¹ called "multiple non-inflammatory necrosis of the liver with jaundice," an example of which I described with a coloured illustration in 1909.² One possibility is that they are merely capillary "cholérhagies" due to overdistension of bile capillaries (apoplexia biliaris hepatis), but biliary focal necroses of a peculiar kind in the liver have been found in the absence of obstructive jaundice.³

Another point of interest was the enlargement of the spleen, which I have not yet mentioned; it had been a puzzling feature of the case during life. This splenomegaly had much diminished (as splenomegaly generally does) after haematemesis, but at the necropsy the spleen still weighed 770 grams (27 oz.); it was evenly enlarged, of