Clinicopathological Conference

A CASE OF BRONCHIECTASIS WITH OSTEOPOROSIS

DEMONSTRATED AT THE

POSTGRADUATE MEDICAL SCHOOL OF LONDON

This is the case of a man with bronchiectasis whose death was accelerated by a rapidly progressive kyphosis (Case No. 161,617; P.M. No. 8,429).

Clinical History

Dr. C. M. FLETCHER: This man, an electric-truck driver, was aged 59 when he was first seen in the out-patient department of Hammersmith Hospital in April, 1954. He had always been healthy apart from gonorrhoea in the first world war and some form of dermatitis, three years previously, which had been treated with an arsenical medicine. He denied any cough (although he smoked 25 cigarettes daily), until January, 1954, when he developed a cough with profuse greenish-yellow sputum. He also became wheezy and slightly short of breath. He stopped smoking, but his symptoms continued until he first attended an out-patient clinic in April, 1954. The chest radiograph showed extensive chronic inflammatory changes (Fig. 1). A diagnosis of chronic bronchitis and emphysema was made. He was given a course of breathing exercises and discharged.

His symptoms persisted until January, 1955, when I first saw him. He was now more short of breath and had lost a stone (6.4 kg.) in weight. On examination he had a marked "snowflake" pigmentation of the skin and hyperkeratosis of the palms and soles, due to his arsenical medicine which he had recently discontinued. (This arsenical dermatitis subsequently cleared up.) He had slight kyphosis (Fig. 2) and widespread basal rales. The chest radiograph was

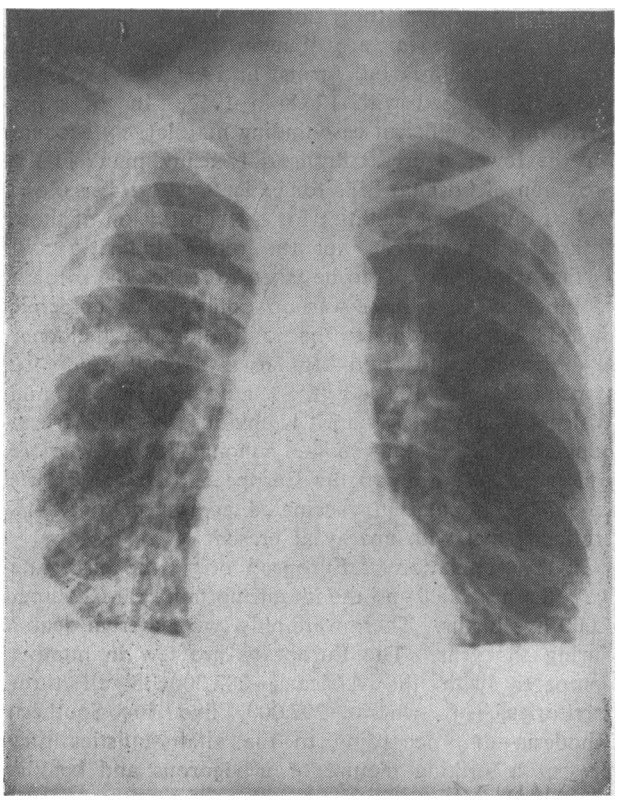


FIG. 1.—Postero-anterior radiograph of chest. April, 1954.

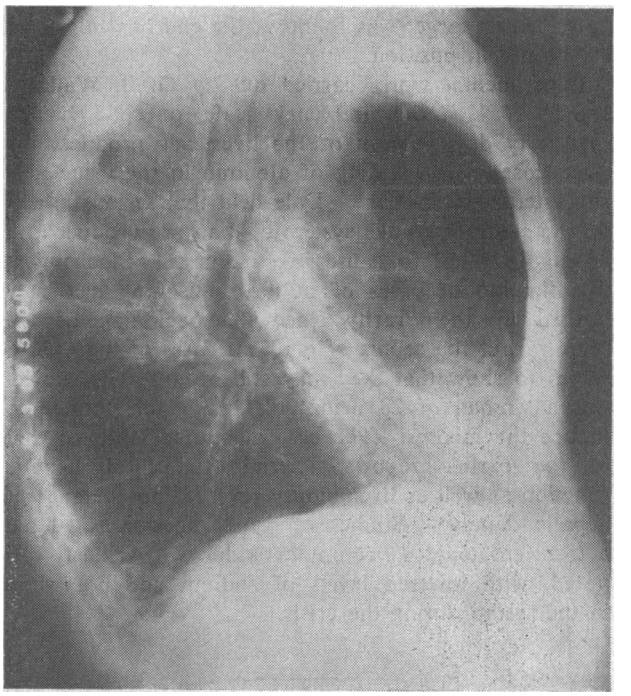


FIG. 2.—Lateral radiograph of chest. March, 1955.

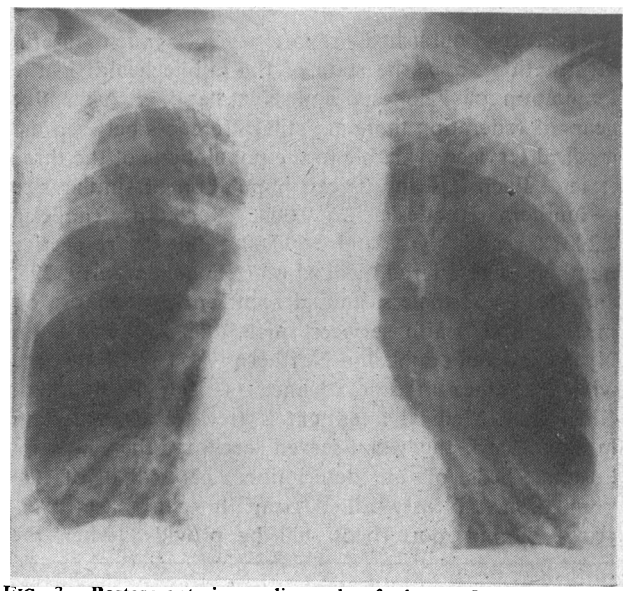


FIG. 3.—Postero-anterior radiograph of chest. January, 1957. Note shadows at both apices and bases. Compared with Fig. 1 there is crowding of the upper ribs indicating increased kyphosis.

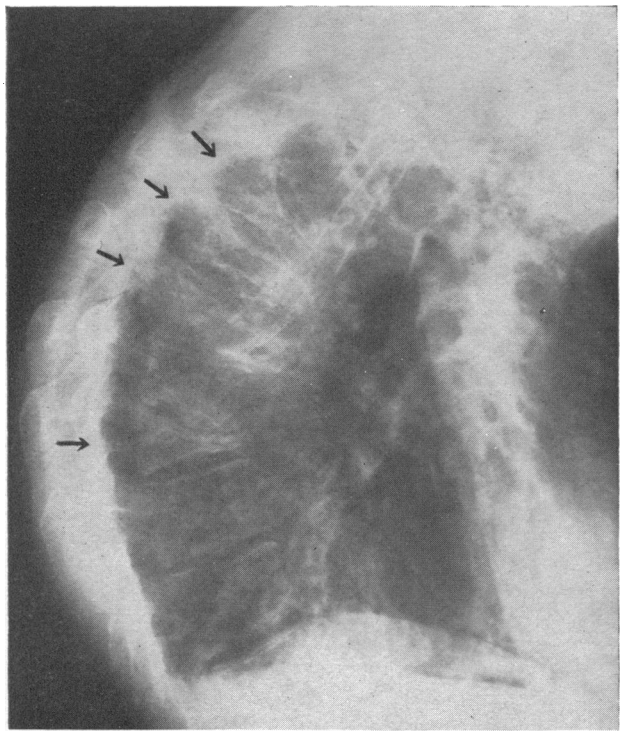


FIG. 4.—Lateral radiograph of chest. August, 1957. Arrows indicate the collapsed vertebrae.

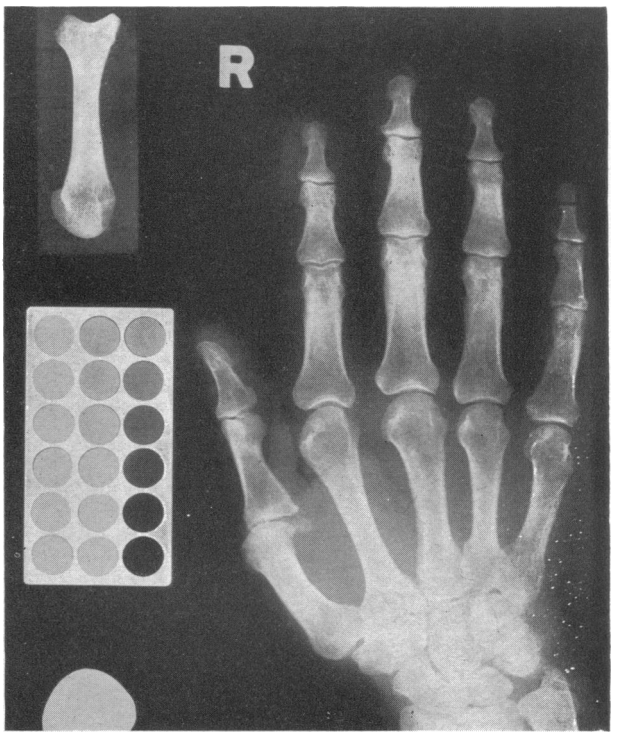


FIG. 5.—Radiograph of right hand. August, 1957. A normal metacarpal is shown for comparison.

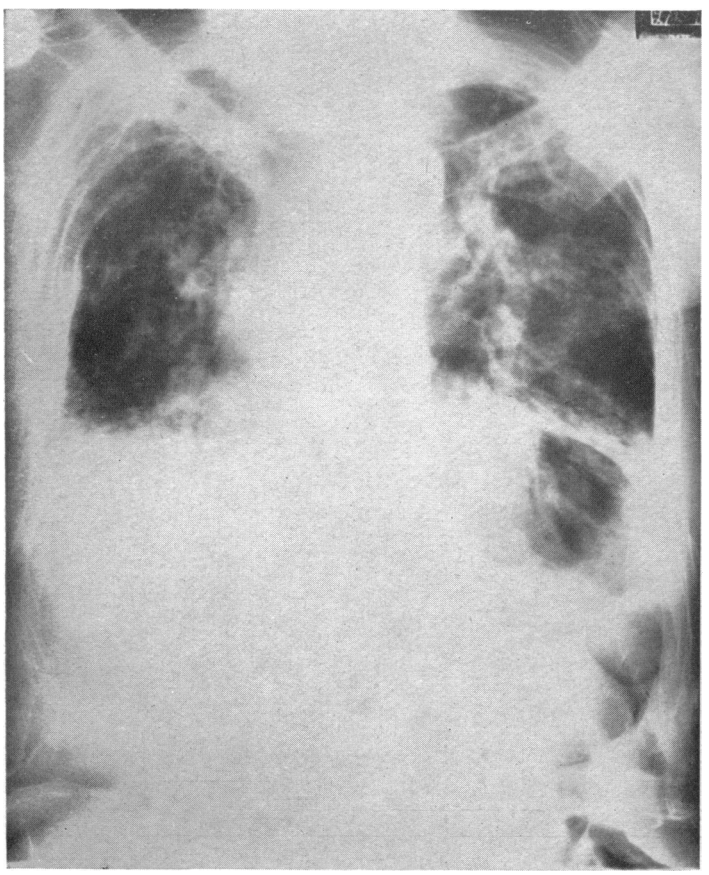


FIG. 6.—Postero-anterior radiograph of chest. May, 1958. Note overlap of ribs and iliac crest.

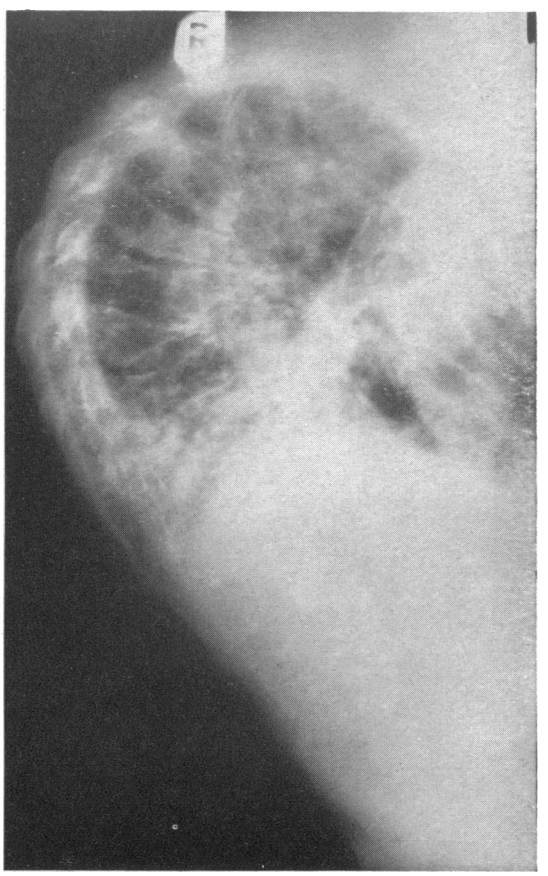
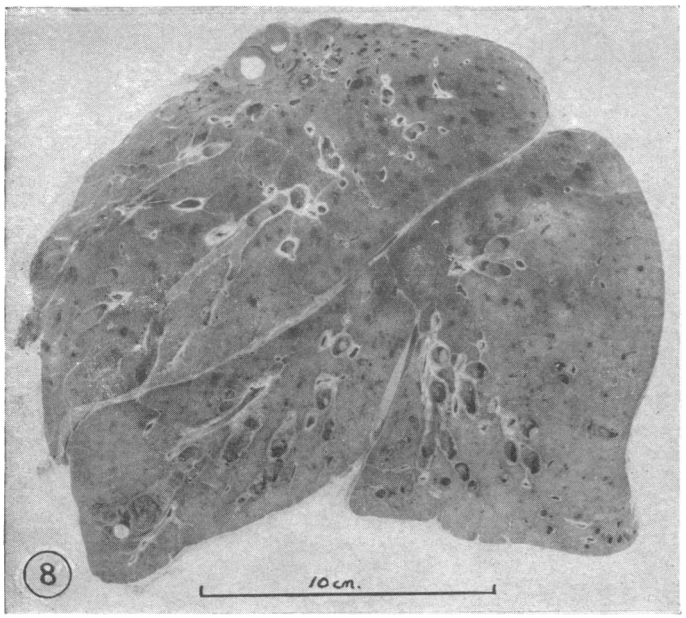


FIG. 7.—Lateral radiograph of chest. May, 1958. Note the ribs overlapping the iliac crest.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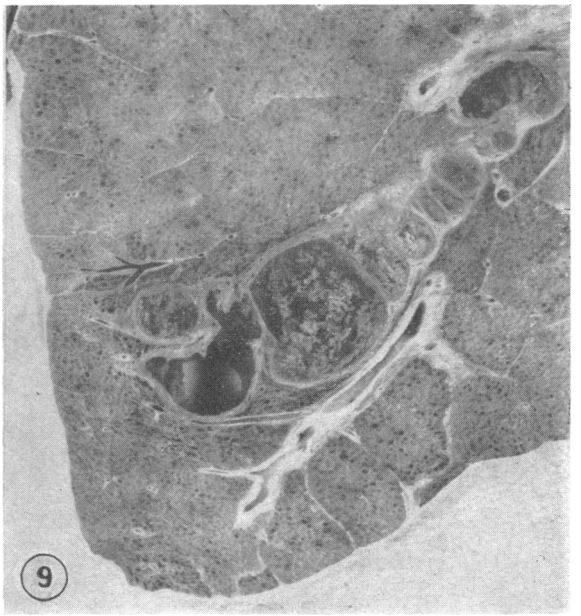
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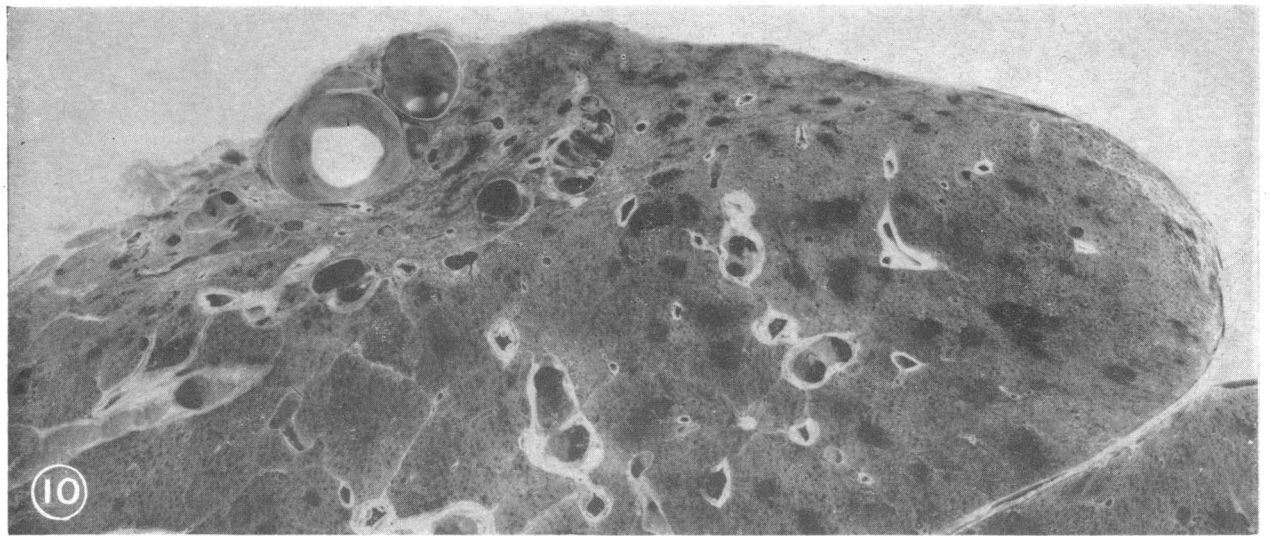
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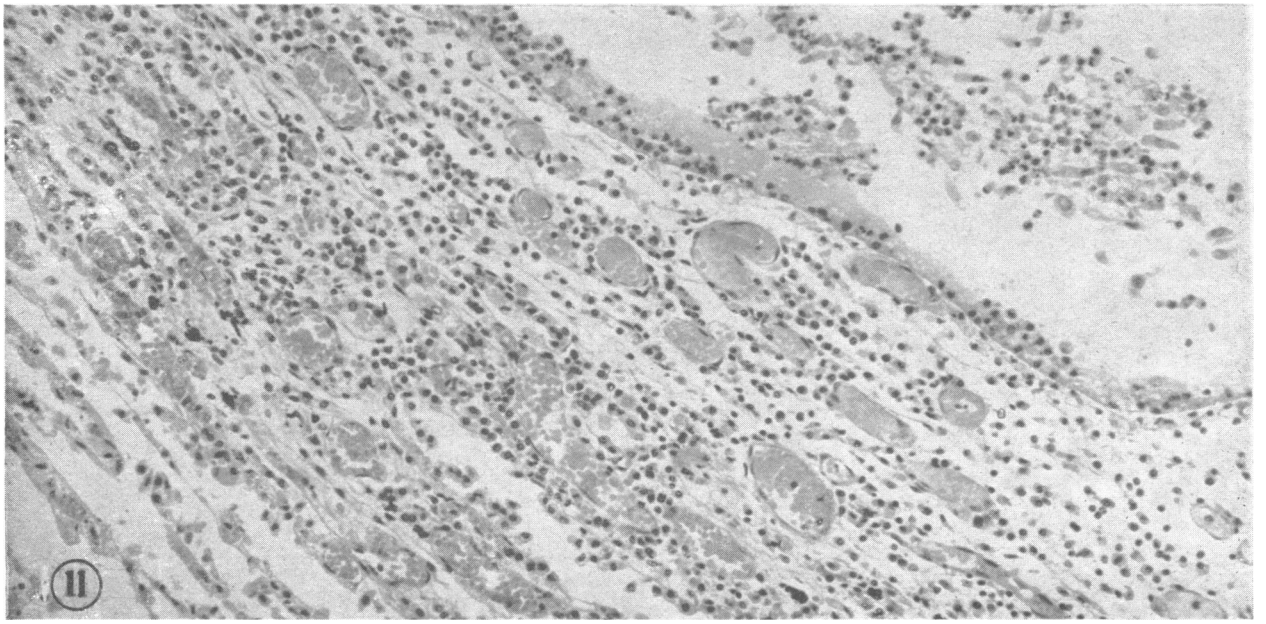
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unaltered and a diagnosis of bronchiectasis was made. He was instructed in postural drainage, and given a fortnight's course of penicillin injections. He improved slightly, but *Haemophilus influenzae* was cultured from the sputum and tetracycline (1 g. daily) was given for a week. His sputum became mucoid and reduced in volume and his shortness of breath decreased. The *x*-ray shadows cleared considerably and he returned to work. He remained fairly well but his sputum repeatedly became purulent, but became mucoid after further short courses of tetracycline in March and June, 1955. From September, 1955, to April, 1956, he was given 0.25 g. tetracycline twice daily. It was then stopped because a resistant *Klebsiella pneumoniae* predominated in his sputum. He remained at work even in foggy weather, but his sputum, though smaller in amount, remained purulent. An estimation of his indirect maximum breathing capacity (M.B.C.) in September, 1955, was 60 litres/min. (normal 64–122). He was able to walk as far as he liked on the level, but was breathless on hurrying.

In January, 1957, he had an episode of acute breathlessness with increased sputum. His own doctor gave him tetracycline and he improved. After this, apical shadows were noted on his chest radiograph (Fig. 3) in both posterior segments. These shadows increased in intensity over the next six months, but seven sputa were negative for acid-fast bacilli. The shadows were probably due to bronchiectasis developing in the upper lobes. Continuous tetracycline therapy was given again, the dose being restricted to 0.25 g. twice daily as before because a larger dose caused diarrhoea.

In May, 1957, the patient was more breathless and he noticed he was getting more round-shouldered. This was confirmed clinically. He developed pain in his shoulders and hypochondrium which was worse on breathing or coughing. He had lost 4 lb. (1.8 kg.) in one month. He was much more breathless and unable to work. A radiograph (Fig. 4) showed very thin vertebrae with collapse of D5, 6, 7, and 9. He was admitted to the Metabolic Unit under Professor T. Russell Fraser.

INVESTIGATIONS

Skeletal.—Further skeletal radiograph (Fig. 5) showed widespread osteoporosis.

Cardiovascular.—Electrocardiograph tracing normal.

Serum.—Calcium, 5.3; phosphorus, 2.1 mEq/litre; alkaline phosphatase, 8.0 K.-A. units; acid formol stable, 1.4 units.

Calcium Balance.—Diet, 15.2; faeces, 12.2; urine 10.8 mEq/24 hours.

Iliac Crest Biopsy.—Osteoporosis. Seams up to 5μ thick.

Strontium Test of Bone Uptake.—Ca_E, 12.7; Ca_B, 2.15 (high normal).

*Ventilatory Function**.—F.E.V., 1.04 litres (normal 8–3.5). V.C., 1.61 litres (normal 2.9–3.9). Indirect M.B.C. 36 litres/min. (normal 64–122). F.E.V.%V.C. 64% (normal). No response to isoprenaline. These tests show severe restriction of ventilatory capacity of the restrictive rather than obstructive type, since 64% of the ventilatory capacity was exhaled in 1 second.

SUBSEQUENT COURSE

On discharge, a 600-mg. testosterone implant was given together with 95 mE calcium, 1 mg. stilboestrol, and 0.5 g. tetracycline daily. A spinal brace was fitted but the patient found it too heavy to wear. During the winter the tetracycline was changed to chloramphenicol, 0.25 mg. twice daily, and this kept his sputum mucoid. His breathlessness remained severe and was present even at rest. Oedema of the ankles developed but was thought to be due to varicose veins. On discontinuing chloramphenicol in March, 1958, the sputum soon became purulent and he had great difficulty in coughing because of increasing kyphosis.

In May, 1958, he was readmitted. He was very breathless but not definitely cyanosed. The kyphosis was more extreme, his lower ribs resting on his iliac crest (Figs. 6 and 7). The jugular venous pulse was difficult to see because of his kyphosis, but did not appear to be raised. There was no clubbing. The day after admission he suddenly became cyanosed and comatose. The head of the bed was lowered, a large quantity of purulent sputum was aspirated, but the patient died in 10 minutes.

Clinical Diagnosis

(1) The persistent and profuse purulent sputum was attributed to bronchiectasis affecting both lower lobes at first, and later the upper lobes on both sides.

(2) The kyphosis was due to an unexplained osteoporosis.

A diagnosis of emphysema was made in two radiological reports and it appears in several letters to doctors and on the case notes after his first admission. I do not accept the radiological diagnosis, and the ventilatory function tests were not characteristic of emphysema. I do not believe there was significant anatomical emphysema. The breathlessness was due partly to bronchial obstruction by excessive secretion, but chiefly to the restrictive effect of the kyphosis, and his death was due to suffocation from bronchial secretion which he could not expectorate.

The only evidence of right heart failure was ankle oedema, but he had varicose veins. He was never grossly cyanosed and his E.C.G. was normal. I do not diagnose significant abnormality of the heart.

LEGENDS TO SPECIAL PLATE

FIG. 8.—An antero-posterior slice of the right lung. The upper lobe is flattened by the chest-wall deformity. There is bronchiectasis in many parts, most obvious in the middle lobe and the basal segments of the lower lobe. (Prepared by pressure-fixation and barium sulphate impregnation.)

FIG. 9.—A higher magnification of the middle lobe to show bronchiectasis to advantage. (×15.)

FIG. 10.—The apical segment of the right upper lobe is shrunken and bronchiectatic. The black foci in the posterior segment of the upper lobe are centrilobular emphysema. In the shrunken segment, they also have shrunk. (Natural size.)

FIG. 11.—The wall of a small bronchus from the posterior basal segment. It showed bronchiectasis macroscopically. Microscopically the wall is heavily infiltrated by lymphocytes, plasma cells, and some neutrophils, and the vessels are engorged with blood. (×212.)

*$F.E.V._{1.0}$ = Forced expiratory volume. V.C. = Vital capacity. F.E.V.%V.C. = Forced expiratory volume as percentage of vital capacity. M.B.C. = Maximum breathing capacity. (See Thomson, W. B., and Hugh-Jones. P., *Brit. med. J.*, 1958, **1**, 1093.)

Post-mortem Findings

Dr. B. E. HEARD: The body was thin and there was a marked dorsal kyphosis. The sternum was depressed and the lower costal cartilages projected forwards prominently. The skin of the upper abdomen was creased. There was no clubbing of the fingers.

The *trachea* contained a little yellow mucus. Both pleural sacs were obliterated by adhesions. The *left lung* showed shrinkage and cystic change of the apical and posterior segments of the upper lobe and dilatation of the supplying bronchus. There was bronchiectasis in the lower lingular and posterior basal segments, where bronchi up to 0.8 cm. in diameter reached to within 0.5 cm. of the pleura. The lung was oedematous and congested and showed patches of bronchopneumonia. The *right lung* was prepared by pressure-fixation and barium sulphate impregnation. Fig. 8 shows that the shape of the lung has been altered by the severe kyphoscoliosis. The apical segment of the upper lobe is flattened, the middle part of the lung is ballooned out, and the lower lobe is shrunken. Bronchopneumonia was present and there was bronchiectasis of the middle lobe and most of the lower lobe bronchi. In Fig. 9 the bronchiectasis is shown in detail, sacculations increasing in size as the bronchus is traced distally. In Fig. 10 the flattened apical segment of the upper lobe is shown in more detail. The shrunken zone shows bronchiectasis and solid lung between. The black foci in the posterior segment of the upper lobe are centrilobular emphysema, and it is interesting to note that in the shrunken parts the black emphysematous foci are also shrunken. A section of the main upper-lobe bronchus showed a trace of goblet-cell hyperplasia but no inflammatory changes in the submucosa. Sections of the dilated bronchi of the lower lobe showed heavy infiltration of the whole wall of each bronchus by lymphocytes and plasma cells and destruction of some mucous glands (Fig. 11). Section of the area of apical collapse showed evidence of old carnification but no sign of active tuberculous inflammation.

The *heart* was displaced to the right beyond the costal cartilages and presented through the incision. No abnormalities were noted in the valves or muscle. The right ventricle was of normal thickness. *Coronary arteries* showed an occasional small plaque of atherosclerosis. The *aorta* showed very mild atherosclerosis. About 10 cm. below the arch there was a kink in the aortic wall, probably produced by the vertebral deformity.

The *liver* (1070 g.) was slightly reduced in size and distorted by pressure from the costal cartilages. There were prominent cough-furrows and the cut surface showed centrilobular congestion. Both *kidneys* were normal, but the *prostate* contained a nodule of hyperplastic smooth-muscle 1 cm. in diameter. The *spleen* showed thickening of the capsule and trabeculae. The bone-marrow was red in all the usual sites. The *lymph nodes* at the pulmonary hila were enlarged and oedematous and histologically showed moderate infiltration by neutrophils. The left *testis* was normal; the right showed some parenchymal pallor. Histologically there was atrophy of many of the seminiferous tubules with hyalinization. Leydig cells were very scarce, presumably owing to testosterone therapy.

The *brain* was not examined (permission refused).

The *mid-thoracic vertebrae* were collapsed and some of the *ribs* were fractured. The 6th rib showed a healing fracture which was seen histologically to be united by an adequate amount of woven bone containing small islands of cartilage. The sternum was of a rubbery consistence and easily indented with the finger-tips. Histologically all bones showed osteoporosis with moderate osteoclastic activity unaccompanied by the formation of fibrous tissue. The only fibrous tissue found in the marrow was at small fracture sites in collapsed thoracic vertebrae. The *thyroid* appeared normal, as did the *suprarenals*. The *parathyroids* were dissected out. Histologically there were large islands of pale oxyphils, a not uncommon finding at this age. The fat content was reduced, suggesting slight hyperplasia.

Pathologist's Diagnosis

(1) Idiopathic osteoporosis with severe kyphoscoliosis.
(2) Bronchiectasis and terminal bronchopneumonia.

Discussion

Dr. FLETCHER: There are two aspects of this case: the kyphosis, which I shall leave Professor Fraser to discuss, was the main cause of his death; and the bronchiectasis, which played an important contributory role. I do not quite know what would happen to a man with normal lungs with this degree of rapidly advancing kyphosis, but I think he probably would survive. We know that people with very severe kyphoscoliosis ultimately tend to hypoventilate and go into respiratory failure. In our patient the kyphosis was very acute, causing severe impairment of ventilatory capacity. Death was due to his kyphosis preventing him from expectorating the profuse sputum coming from his bronchiectasis.

BRONCHIECTASIS AND MAINTENANCE CHEMOTHERAPY

The history is a little unusual. He was fairly reliable and he denied cough or sputum before his illness in 1954. He very likely did have a smoker's cough, for he had been a heavy smoker. He sensibly gave up smoking when he first got ill. I think that he already had bronchiectasis when he was first seen; this was four months after the onset of his illness, which may have been a pneumonia which left him with infected basal bronchiectasis. This subsequently spread to involve the upper lobes. We know that bronchiectasis can extend to previously unaffected lung in adults. This has been clearly shown at lobectomy for local bronchiectasis, especially in subjects with pre-operative evidence of generalized bronchitis.

I should like to say something about his management by chemotherapy. He was given no chemotherapy until 18 months after the onset of his illness. In the light of our present experience, it is conceivable that, if he had been vigorously treated from the onset, he would have remained with relatively undamaged lungs. When I first saw him we were not accustomed to giving long-term maintenance chemotherapy. He was given only a week's course of tetracycline, which was then our custom. This was temporarily effective, but he continually relapsed. When we did start maintenance chemotherapy with tetracycline, we were prevented from giving full dosage by the fact that he got

diarrhoea. This, of course, is an annoying side-effect of tetracycline, which we encounter in something like 10–15% of our cases. It usually can be controlled by a simple opium mixture and very often does not persist. Later on we were forced to take the risk of maintenance on chloramphenicol, which was much more effective, but the bronchial infection relapsed as soon as it was discontinued.

Professor J. MCMICHAEL: Two questions. When he came in last no clinical cyanosis was noted. Did we have an arterial sample?

Dr. FLETCHER: No. He died within a day.

Professor MCMICHAEL: Was he never grossly cyanotic?

Dr. FLETCHER: There are occasional out-patient notes of slight cyanosis, but he was never grossly cyanotic.

Professor MCMICHAEL: What was the cardiac state?

Dr. FLETCHER: It seems to have been normal. There was no evidence of right hypertrophy. I do not think his ankle oedema was cardiac.

PROGRESSIVE OSTEOPOROSIS

Professor T. RUSSELL FRASER: Well, this is an instance of osteoporosis, unfortunately a condition which is not very well understood nor very easy to treat. But I suspect that his disorder must have been more severe than is usual in men, and perhaps more difficult to treat because of his associated cough. It is at least not at all common to see a progressive kyphosis advancing as rapidly as that. How much the cough contributed to it is hard to say. From the fact that he had osteoporotic bones in the radiological meaning of the term we can infer that the man must already have lost over 50% of his bone mass. If there is, in addition, the mechanical problem of coughing, I suppose progression may almost be inevitable.

The normal treatment would include a spinal brace to place the support of his shoulders on to his iliac crest; but doubtless he could not manage with his brace, since it must have increased his dyspnoea very considerably. On the other hand, progressive collapse was probably inevitable without it; for even with the best response to the conventional methods of treatment osteoporotics can only be made to recalcify slowly.

The diagnosis of osteoporosis, like that of emphysema, is made by exclusion of other causes. Here the data have shown that there was no evidence of osteomalacia or hyperparathyroidism; especially from the normal serum electrolytes—particularly the serum calcium, phosphorus, alkaline phosphatase.

Professor MCMICHAEL: What were the figures?

Professor RUSSELL FRASER: The calcium balance can be interpreted only in the light of the dietary intake. These balance figures are all given in mEq per 24 hours when he was on a diet of 15.2 mEq (or 304 mg.) of calcium per day. His faecal loss was 12.2 mEq calcium/24 hours and his urine loss was 10.8. So he was obviously in abnormally strong, negative calcium-balance (−7.2 mEq/24 hours); though that does not tell you anything about the nature of the decalcifying process.

Professor MCMICHAEL: Is this characteristic of osteoporosis?

Professor RUSSELL FRASER: In osteoporosis it is a matter of whether the disease is advancing and active at the time when you do the test, or has become quiescent and stationary. Very often the patient is seen at this last stage when you would not see a negative calcium-balance. But of course it should be said that a negative calcium-balance is a normal finding on that dietary intake. This particular patient was balanced on a low normal calcium-intake, chosen as the diet on which the faecal calcium and the dietary intake are approximately equivalent. He was also balanced later on a high calcium-intake when he went into positive calcium-balance It is a very frivolous custom, therefore, to talk about somebody being in negative calcium-balance on a test, or in positive calcium-balance, for this really means nothing unless you relate it very carefully to his dietary intake and whether that is what his usual diet is. The only abnormality in this patient's balance data is that he had a high urinary calcium. The rest of the data, the faecal loss, was probably normal. His serum levels were also normal (serum levels of calcium, phosphorus, and phosphatase), and that is the reason for concluding that he had shown no primary defect of calcium metabolism.

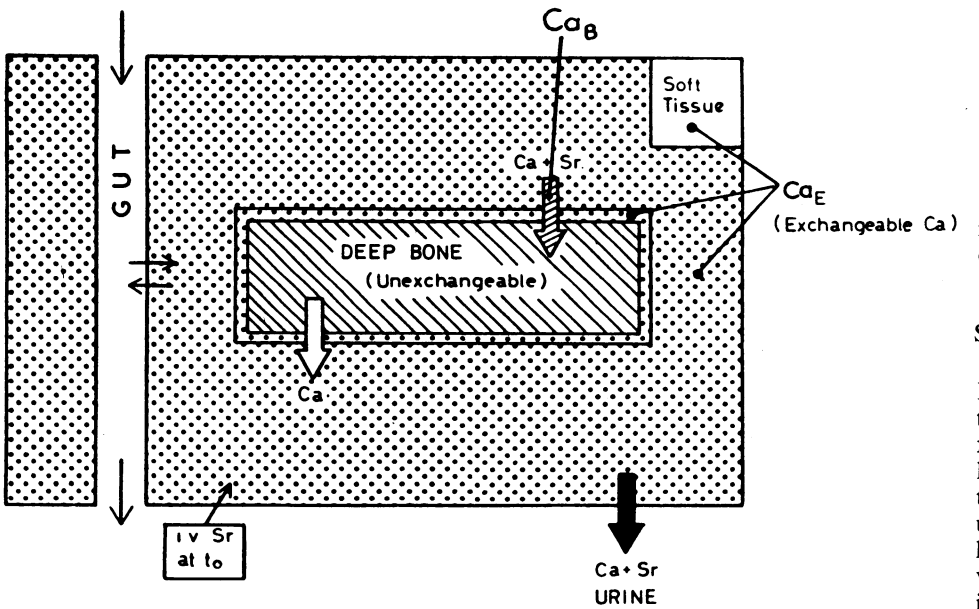

FIG. 12.—Diagram illustrating the distribution of the body's calcium and the strontium tracer test.

STRONTIUM UPTAKE TEST

Professor MCMICHAEL: I am sure about half of the people here, Professor Fraser, do not know about the strontium test for bone uptake. The figures are here in the record and we should be very glad if you could explain them.

Professor RUSSELL FRASER: Well, it is possible to use strontium, somewhat in the same way as radio-iodine is used, to measure the rate of calcium uptake in the bones. From a pool in the body fluids, calcium goes to two main external sites, the urine and the faeces, and it goes also to the bones (see Fig. 12). The pool is replenished from the absorbed dietary calcium and also from the steady resorption of bones. Now, with an injection of strontium—or for that matter of any tracer of calcium—into the body fluid's pool of calcium, you can measure the rate at which calcium disappears from the pool, and also the rate at which it is excreted into the urine and the faeces. The loss not so accounted is therefore the uptake of calcium by the bones. The test is primarily concerned with measuring the size of the bone uptake. The statement that this patient had a normal uptake means that he did not show any signs of an excessively rapid turnover of calcium through the bones. Such a rapid bone turnover is found when you have primary overaction of the parathyroid, and it is also a finding in osteomalacia, in which, for perhaps rather more complex reasons, the bones similarly have a very rapid turnover of such calcium as the body can offer, the defect perhaps being primarily in the absorption of calcium.

Professor MCMICHAEL: Now your answer is expressed here as Ca_E, 12.7; Ca_B, 2.15. What does that mean?

Professor RUSSELL FRASER: Well, calcium E is the exchangeable calcium mass, that is what is illustrated in the diagram as the pool of calcium into which an injection is rapidly mixed. That is compounded of the plasma, the extracellular fluids, soft tissues, and the surface of the bone. The units are "total plasma" units. These rather unusual units are needed because you have to incorporate a correction for body size. A Ca_E of 12.7 means that he had 12.7 times as much calcium in the exchangeable pool as he had in his plasma. The normal figures for this are 10–15. Now Ca_B, shown by an arrow on the diagram, is the calcium deposited in bone each day. And that again is expressed in plasma units. The test showed that he deposited 2.15 plasma contents per day, and the normal range for this being 0.8–2 plasma units he was therefore at the upper end of normal. You might expect simple osteoporosis to show defective laying down of bone by this test, but it is interesting that this has not been our experience. In fact, the results in osteoporosis have been remarkably similar to those in normals. We must, however, think carefully what is to be concluded from that. Let us compare the situation in, shall we say, obesity. Nobody really doubts that the obese patient eats a little more than he needs for the energy he expends, but if you attempted to demonstrate this on a metabolic test it would be very difficult. It probably takes many years to develop osteoporosis, just as it takes many years to develop obesity, and our tests are not sufficiently sensitive to show that this little arrow has perhaps been reduced over a very long period of time. The important diagnostic feature is that the uptake is not increased, as it is in those two other conditions which may readily be confused clinically—hyperparathyroidism and osteomalacia.

Dr. M. D. MILNE: Isn't it very odd, though, that in a very active case it was right at the upper limit of normal?

Professor RUSSELL FRASER: I think it's very probable that, when we discover what osteoporosis is due to, we will find it isn't the conventional explanation that the bones do not lay down matrix normally and so lay down insufficient bone; there is a chronic excessive loss of calcium from the body and so it may well be that excessive resorption is the explanation. Certainly that is, for example, the case in thyrotoxicosis, where you may ultimately get an identical clinical picture of thin bones as a part of the syndrome in a proportion of the patients. But when you examine these or other thyrotoxics bone deposition is found to be rapid and excessive; however, it is not so great as bone resorption, and it is the balance between these two which will determine whether the bones end up as being thin.

Professor MCMICHAEL: Has Nordin's theory of osteoporosis resulting from defective absorption been accepted?

Professor RUSSELL FRASER: No, not generally accepted yet. You can produce the disorder of osteoporosis by a low-calcium diet in rats. Osteoporosis results if you give them a low dietary intake of calcium, along with otherwise adequate diets. So this is one way of producing atrophy of the bones, which is what we are talking about. Osteoporosis is an atrophy of the bones without any indication of the cause of the atrophy, and there may be several causes of the bone atrophy. When we come to the question of the osteoporosis seen clinically, we have no adequate clues yet. Calcium deficiency could cause it, but this has not been shown to be the cause. Calcium therapy is being tried. Defective bone formation is still a possible cause, for with thin bones there would be an excessive stimulus to calcium deposition, yet all these patients achieve is a normal rate of deposition. You may say that is abnormal.

HOW BRONCHIECTASIS SPREADS

Dr. J. G. SCADDING: I agree with Dr. Heard's analysis of the situation rather more than with Dr. Fletcher's. As I understood it, what Dr. Heard suggested was that the primary event here was the development of a bronchitis of the truly inflammatory type. I agree with him that the first event here was a succession of purulent infections of the bronchi and that the bronchiectasis developed as a consequence. As I saw it, what Fletcher was suggesting was that a local bronchiectasis developed somewhere as a result of an acute pneumonic episode and the bronchiectasis "spread" from that focus. Admittedly, bronchiectasis may appear to spread in the lungs, but I think that the word "spread" is rather an unfortunate way of expressing it. It rather suggests that bronchiectasis is a little demon that goes about dilating people's bronchi. My thought is that, when bronchiectasis appears to spread, it is certainly a repetition of the process that caused the original bronchiectasis: another focus of a similar inflammatory change occurs in another part of the lung and bronchiectasis develops there. I think the bronchiectatic changes observed finally at the post-mortem examination in this case were of a distribution which suggests that Dr. Heard's explanation—that mainly they were a consequence of chronic and recurrent widespread inflammatory changes in the bronchi—is probably correct.

EMPHYSEMA NOT A CLINICAL SYNDROME

The really unusual feature of this case, to my mind, from a pathological point of view, is the extremely

small amount of emphysema that was in evidence at the end of the process. I would have expected, in a case where all these changes had been going on for so many years, a good deal more emphysema. I was very glad to hear pin-pointed the difficulties over the diagnosis of emphysema. We are trying to establish the idea that the only tenable definition of emphysema is a morbid anatomical one, so that the only person who can say for certain whether emphysema is present is the morbid anatomist. When, as clinicians, we are bold enough to say, "I think this patient has emphysema," what we should mean is that we think that the morbid anatomist will find certain morbid anatomical changes. We may be wrong or we may be right, but that is what we should mean. Unfortunately the word "emphysema" has become one of those witchwords which misleads everybody, because it has become equated with a clinical syndrome without any real justification. Indeed, I think this happens all over the world; certainly it happens in the United States. Recently I received, from a centre in the United States for the journal which I edit, a paper in which a case report recorded that the patient had had "recurrent attacks of emphysema." This makes clear that the word emphysema is used by some people there in the same sense in which most of us in this country refer to recurrent or chronic bronchitis. I don't think we'll overcome this confusion until we can get it established that ultimately emphysema is a morbid anatomical concept.

What we urgently need is a convenient word to describe the syndrome of chronic respiratory disease leading up to eventual respiratory insufficiency, which is so common in this country, without necessarily implying any untenable hypothesis. The position in respiratory disease at present, with the current use of the word emphysema, is rather similar to that which would exist in cardiology if you could not refer to "cardiac failure" without implying that there was valvular disease of the heart. We want functional terms in respiratory disease that would be as commonly used and understood by everybody as "congestive cardiac failure" is in cardiology. Having introduced that term into our respiratory work, we could then go on to add, to complete a diagnosis, the morbid anatomical changes among which might be emphysema.

A VOICE: What about "respiratory failure"?

Dr. SCADDING: Well, that would be a nice term to use, but unfortunately it's not quite precise enough. "Respiratory failure" might imply failure of the respiratory centre as well as of the lungs. "Pulmonary insufficiency" is the nearest I can get to it, implying a local insufficiency in the lungs.

Dr. FLETCHER: I should just like to say that I do not look on bronchiectasis as a demon. I entirely agree with Dr. Scadding that its spread is due to extension of infection with destructive results, but I do not think this case was one of bronchitis in the ordinary sense, because we do not see this process in most of our bronchitics. There was something different about this bronchitis. Did Dr. Heard say that he did not find hyperplasia of mucus-secreting glands in the bronchi? In my mind bronchitis is characterized primarily by goblet-cell and mucous gland hypersecretion. In this case there was no real history of chronic bronchitis preceding the first illness. There was a rapidly progressive bronchiectasis starting, I believe, with an acute infection. Later on bronchiectasis developed elsewhere, so that some processs was spreading in his lungs which had as a secondary consequence bronchiectasis. This may have been a peculiar kind of bronchitis, but it was not the ordinary kind in which there is persistent mucoid sputum with recurrent episodes of infection. This man had a persistently purulent sputum and radiological evidence of bronchiectasis at first in the lower lobes and subsequently in the upper lobes.

EFFECT OF THE KYPHOSIS

Dr. R. E. STEINER: The appearance of lesions in the upper lobes coincided with the kyphosis.

Dr. FLETCHER: Yes, they appeared in January, 1957, when the P.-A. radiograph shows increased kyphosis, but this wasn't noticed clinically till about four months later. Do you suggest that he had a collapse of his upper lobe and got a secondary infection in it, which may have led to the bronchiectasis?

Dr. STEINER: Yes, the mechanical compression was the first thing, at least in the case of the upper lobes. You are not dealing with a primary infection of the bronchi, on which you base your explanation of the bronchiectasis.

Dr. P. C. ELMES: I wonder which was causing the lung disease, the vertebral collapse or the bronchial infection? The disease process may have been primarily a bronchial infection, perhaps associated with some other pulmonary disease. He was coughing up about 200 ml. of purulent sputum per day, containing 10% of protein, for many years, and his disease was associated with anorexia. Thus protein deficiency could have occurred and given rise to bone absorption with vertebral collapse and progressive symptoms. This in turn made it impossible for us to clear his lungs of infection. Two processes were going on at the same time and you never could say which came on first, or which was the more important; they added to each other all the time. I wonder whether the final picture of minimal emphysema was due to the fact that here were lungs which were getting progressively scarred but were not being asked to occupy the normal space. Had they been asked to occupy the normal 5 or 6 litres of thoracic volume, then there would have been very much more likelihood of emphysema because the lungs would have been stretched to fill that large volume, whereas because of the collapse they were not being stretched at all.

Professor MCMICHAEL: Isn't it rather unusual, Dr. Heard, to see completely collapsed segments of the lung in these deformed hunchbacks? My observation of the ordinary kyphoscoliotic is that the lungs are small but otherwise normal.

Dr. HEARD: Complete collapse is unusual, but some shrinkage is not uncommon with deformities of the chest wall or with pleural thickening. The defect is not always one of compression. We have had a few cases of severe kyphoscoliosis recently. One of them was a man of 53 who developed kyphoscoliosis early in childhood. He had very small lower lobes to both lungs. The defect was not so much due to compression by the deformity, but to the fact that his lungs appeared never to have developed to the normal size. Incidentally, he had no bronchiectasis and no emphysema was demonstrable though the lungs were prepared by pressure-fixation and barium sulphate impregnation.* As regards the bronchial histology in the present case,

* Heard, B. E., *Thorax*, 1958, **13**, 136; ibid., 1959, **14**, 58.

the small bronchus showing marked goblet-cell hyperplasia was of the sort described by Reid.*

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EXTRACTS AND HOWLERS TAKEN FROM EXAMINATION SCRIPTS

BY

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The following are authentic extracts from examination papers collected through the years by the senior sister tutor of a well-known hospital.

Anatomy

Wax glands in the ear protect the organ from foreigners.

The pupil is nothing more than a whole in the eye. In some people it is big, in others small, but unless you have been peculiarly born it is there in some size or another.

The stomach is covered with a serious membrane.

The stomach is supplied by an involuntary nerve called the pneumatic.

The aorta is a long tub whose contents are very precious to the body.

The eye is the organ of site, the sight of which is in a holler in the temple.

At the back of the eye one may see a retinue.

The heart is the only organ in the body made of heart muscle.

The heart is a muscular organ acting like a human pump, which pumps the heart around the body at regular intervals.

The heart is an organ the shape of a heart. It is not quite heart shaped because there are tubes protruding from it, 4 from top left, an umbrella handle from bottom left, a "Y" shaped connection from bottom right and 2 tubes from top right.

The brain will soon become a weekend one.

At the back of the throat are 2 long pillars of faeces.

The mouth is situated in the lower part of the face and is known as a buckled cavity.

Personal and Communal Health

The best room for eye trouble is one which is well alight.

The shopkeeper should thoroughly rap all cakes before selling them to make sure all disease is eliminated.

Food should not be picked up by hand but with tongues.

Clean overalls or trousers should be worn to polish the fruit on.

These are some of the types that help in hygiene of the country: (1) Road sweepers. (2) Dustmen. (3) Nurses in the district and so on. All these add up to the answer prevention of spread.

If blue bottles come through the larder window trouble is around.

Cooks should be trained to wash their hands after a visit to the toilet it is through wiping them on food that diseases are spread.

Overcrowding is not necessary. People when they get married their parents say they can live with them until they get a house built. Some people think, well as long as we can live with them it is all right. BUT IT IS NOT. After they have been married a few months they start having children, these grow up and this goes on. Overcrowding then occurs air is not allowed to enter, with too many in the bed diseases are caught. Conditions like this can be likened to the many hundreds of people who were put in a black hole in Calcutta and burried alive.

Rest is essential if habits are to be good ones, if you do not rest then habits will be bad ones, so proper rest and good cleaning of the body will produce a nurse who can be relied upon to perform any old job the sister may have on her list.

Our Tutor has stated that in her opinion all infection in hospitals is caused by dirty nurses, therefore personal hygiene and cleanliness is next to godliness, and is the first thing to remember if you want to prevent wounds from becoming pussy.

The important thing in the maintenance of health is personal cleanliness. This commences when the person is getting up in the morning, and it continues until the person is ready for getting up again.

You always find a person who does not practice personal cleanliness is not nice to be near.

Everything about personal cleanliness should be kept very clean, making sure that the habits are not forgotten.

The body needs a daily inspection to make sure every part is in proper working order.

The teeth should be brushed with a brush and toothpaste. The best method is to have 2 brushes and keep one of them in a septic solution.

Clothes should be well washed and ironed for hygienic conditions and also for decorating purposes.

In bad light people may screw up their eyes and the damage they do may never be undone.

Housewives when baking should wear the appropriate head gear.

Flies may land on composed heaps then pass through a crack in a house, settle on persons within and cause trouble amongst them. Few escape this vital pest.

Flies spread disease "you can say that again."

Food should be kept covered in shops because of the diseased types who buy it.

If overcrowding continues water vapour increases and people will shout for air, this when heard denotes air hunger.

We seek them here, we seek them there, we seek those horrid flies everywhere.

Flies are abundant wherever rubbish, manure, filth and hospital food are found.

Flies may be shot down whilst in flight by the aid of D.D.T. bombs.

If open shoes are worn on duty the toe nails should be kept clean and short as they are on view to all the patients.

Curt shoes and swede shoes are generally baned by the Matron.

A nurse has no choice in the footwear she wears, the Matron chooses her shoes for her.

The ear is the organ of earing.

Nursing

Treatment of Shock

The best treatment is to rape the patient in old blankets making sure she is not overheated.

The woman should have blankets under and over her but onlookers must be kept outside.

Coronary Thrombosis

The severity of the attack depends upon the sight of the clot.

The patient must adhere closely to the bed rest.

Common Cold

The common cold is caused by an organism called droplet bacillus.

The patient will suffer from a steaming nose.

*Reid, L. M., *Lancet*, 1954, **1**, 275.