Whether the recurrence of vertigo in this case was due to hypersensitivity to adrenaline or adrenaline-like bodies following excision of the ganglion (for the patient was hard at work again and under considerable mental strain), or whether some sympathetic fibres to the ear may also originate on the opposite side to a greater or lesser extent in individual cases, is not known, but the fact that there has been a marked improvement on the administration of ergotamine would suggest that the former view is the more likely of the two, in which case division of the pre-ganglionic fibres together with stripping and ligation or division of the vertebral artery would be the operation of choice. This has now been done in more recent cases with the same beneficial result on the vertigo, added to which it has the advantage of avoiding a Horner's syndrome.

The result upon the tinnitus is apparently unpredictable, though on the whole it was reduced—in three cases it was completely relieved. The relief seems to be more pronounced with the pulsating type of tinnitus than with the continuous variety. Each patient suffered from concomitant nerve deafness, and in all except the last two most recent cases there has been improvement (in some ears very markedly) of hearing by both air and bone conduction. If the nerve deafness is very severe it is beyond relief.

It is felt that further work along these lines will greatly aid in elucidating the aetiology and pathology of this distressing complaint and its accompanying tinnitus and nerve deafness.

It is interesting to record that a further series of stellectomies has been performed for tinnitus alone (to be published later), but we forbear to make further comment on the role of the sympathetic on tinnitus at this stage.

In the meantime a new surgical method of treating Ménière's syndrome, having tremendous advantages over the old surgical destruction of the internal ear, is at our disposal.

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For the last two years 45 severely disabled men have been happily and gainfully employed at Haven Products, Ltd., a "sheltered workshop" on Clydeside. They work a regular 8-hour day five days a week and are paid a basic wage of 2s. 1½d. an hour plus bonuses. The Nuffield Provincial Hospitals Trust has recently published an illustrated pamphlet describing the development of the workshop and the work done there. The men manufacture electro-thermic quilts, and the machines are adapted to suit the needs of individual workers. Doors are made specially wide to allow invalid chairs to pass through, lavatories have railings fitted so that men who must use their hands to get about may be helped, and transport is organized from the workers' homes. Four travel to the workshop in motor-chairs and fifteen require ambulance transport. A medical advisory committee, consisting of consultants from the three large voluntary hospitals in Glasgow, a senior medical officer of the Ministry of Pensions, three almoners, and an observer from the Ministry of Labour, was set up to advise on the planning and equipment of the factory, to help in the selection of suitable disabled men, and to ensure that the men's disabilities were not aggravated by their work. The men are medically examined every year, but medical supervision is kept in the background so far as possible. The venture has been very successful both financially and as an experiment in the restoration of the men's self-confidence and contentment. Harmonious relationships prevail in the workshop, and the report emphasizes particularly the enthusiasm and the sense of corporate life enjoyed by the men. Copies may be obtained from the Nuffield Provincial Hospitals Trust, 12 and 13, Mecklenburgh Square, London, W.C.1, and 10, Duke Street, Edinburgh.

"RHEUMATOID DISEASE" WITH JOINT AND PULMONARY MANIFESTATIONS

BY

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AND

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It will be generally accepted that rheumatoid arthritis, like pulmonary tuberculosis, is a systemic disease with local manifestations, the former in the joints, the latter in the lungs. Moreover, its systemic nature may be manifested, as recent studies have shown, by widespread pathological changes in various tissues and organs.

Our own studies, confirming those of other observers (see Hench *et al.*, 1948), have shown that in certain cases the bones may exhibit atrophy and even widespread cystic changes, and the peripheral nerves may become involved, with resultant neuritic pains, paraesthesiae, and trophic changes. Biopsy studies of the muscles may reveal microscopic alterations in the form of perivascular lymphocytic infiltration and macrophages in the perimysium and endomysium akin to those sometimes seen in periarteritis nodosa and disseminated lupus. Cardiac lesions may also occur almost identical with those following rheumatic fever, while other accompanying lesions may be found in the spleen, liver, lymph glands, subcutaneous nodules, pleura, and even in the eye in the form of iritis and scleritis. In one recent case there was good reason for regarding a kidney lesion (Ellis type 2) as part of the rheumatoid process.

The purpose of this paper is to report three cases in which an accompanying pulmonary lesion appeared as an integral part of the "rheumatoid state." A careful search of the literature has not disclosed any earlier records.

Case 1

A fitter's mate aged 47 was admitted under the care of one of us (P. E.) to the Rheumatism Unit and later to the Leatherhead Emergency Hospital, and when first seen on Dec. 14, 1945, gave the following history. Two and a half years previously, while he was in a fair state of general health, pain and stiffness started in the right knee-joint. During the next few months the fingers, wrists, elbows, and left knee became involved. Swelling of the affected joints was present while active and subsided with rest. Until the time of admission he had had several courses of physiotherapy at different hospitals. He had lost 2 st. (12.7 kg.) in weight since the onset of the illness. There was a previous history of pleurisy progressing to empyema at 11 years of age, and malaria at 21 and 24 years. There was no significant family history.

On examination he was pale and toxic, and wasting involved subcutaneous tissues and muscles. Examination of the locomotor system revealed evidence of swelling of the proximal interphalangeal joints, wrists, elbows, and knees, and fibrous nodules were present over both olecranon processes, sacrum, and scalp. Examination of the lungs showed poor air entry, and breath sounds diminished at both bases, where numerous crepitant rales could be heard. The heart, abdomen, and central nervous system were normal. Blood pressure 130/90.

Pathological investigations showed: E.S.R., 56 mm. at first hour (Westergren); red cells, 3,670,000. Hb, 66%; C.I., 0.83; white cells, 8,000 (polymorphs 67%, lymphocytes 29%, monocytes 3%, eosinophils 1%); G.C.F.T., negative; serum uric acid, 1.2 mg. per 100 ml.; Mantoux, weak positive. Repeated sputum examinations, including concentration techniques, were all negative for tubercle bacilli. General bacteriological examination of the sputum was not significant. Fluid aspirated from the left knee-joint was purulent and contained many polymorphs, but was sterile on culture.

Radiological examination of the wrists, knees, and finger-joints showed osteoporosis, loss of joint space, and erosions characteristic of the rheumatoid type of arthritis. There was evidence of a fine reticulation throughout both lung fields with a chronic bronchopneumonic lesion.

On March 26, 1946, he began to complain of cough and produced small amounts of mucopurulent sputum. In April he developed a generalized purpura, but the bleeding-time was normal and platelets numbered 310,000 per c.mm. His general condition gradually deteriorated, and he became increasingly wasted until June 17, when he developed a pyrexia ranging about 99–100° F. (37.2–37.8° C.), became dyspnoeic and cyanosed, and coughed up much frothy sputum. These symptoms gradually increased in severity, being unaffected by penicillin therapy, until his death on June 23.

Post-mortem Examination (performed by Dr. D. N. Nabarro 18 hours after death).—The body was that of a middle-aged poorly nourished man with polyarthritis. The dura mater was somewhat adherent over the surface of the brain, which was

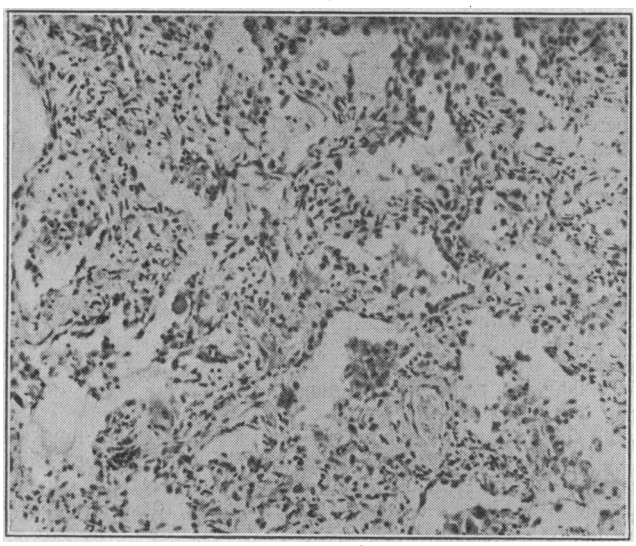

FIG. 1.—Case 1. Photomicrograph showing interstitial pneumonitis with well-marked fibrosis between the lung alveoli. (× 100.)

slightly congested but was otherwise normal. *Respiratory system:* There were fairly dense pleural adhesions, more especially marked over the lower lobes; no free fluid was seen. The trachea and bronchi contained muco-pus. The lungs were firm throughout, with nodular areas suggestive of a bronchopneumonia. Both lungs were congested and oedematous, and on section showed many areas of recent bronchopneumonia. However, the lungs as a whole were firmer than normal and suggested a chronic fibrosing pneumonitis. There was no evidence of tubercle or sarcoidosis. The mediastinal glands were enlarged and showed evidence of chronic inflammation. *Cardiovascular system:*—The pericardium contained some 30 ml. of clear straw-coloured fluid. The heart was dilated and slightly enlarged, and the myocardium was pale, soft, and flabby. The valves were all normal and showed no vegetations or any evidence of endocarditis. There was very little atheroma in the blood vessels. *Gastro-intestinal tract,* normal. The *liver* was somewhat enlarged, appearing firm with a rather coarse mottled surface. There were no gall-stones. The *spleen* was considerably enlarged (weight 630 g.), with a fair amount of perisplenitis. The *kidneys* were not abnormal. The other organs were all normal, and none showed any evidence of tubercle or sarcoidosis.

Histological Report.—Sections were cut of the lungs, lymphatic glands, pancreas, suprarenals, heart, kidney, spleen, liver, and knee-joint and stained haematoxylin and eosin and haematoxylin and Van Gieson. *Lung* (Fig. 1):—The changes chiefly seen were those of an interstitial pneumonitis with a terminal bronchopneumonia, together with the formation of some small abscesses. The alveoli contained considerable exudate; in some the exudate was chiefly albuminous fluid, but in most there was infiltration by polymorphs and some lymphocytes. There was well-marked fibrosis between the lung alveoli, and infiltration with mononuclears and some polymorphs was prominent. There were many alveolar phagocytes, and some giant cells were also to be seen. Blood vessels were largely normal, but a few could be seen, more especially near the small abscesses, in which the muscle coat showed some fibrinoid degeneration with endothelial proliferation. In these there was infiltration of the wall of the vessel by mononuclear inflammatory cells. There was no evidence of tubercle or sarcoidosis. The *lymphatic glands* showed chronic inflammatory changes only. The *pancreas* and *suprarenals* were normal. The *heart* muscle showed no abnormality, but there were one or two small vessels in the fatty tissue immediately next to the muscle which showed changes from the normal. These consisted of mononuclear infiltration in the wall, of endothelial proliferation, and very slight necrosis of the muscle coat. No abnormality in the vessels in the heart muscle itself was found. The *kidney* showed slight tubular epithelial desquamation and a little lymphocytic infiltration in the interstitial tissue. A few blood vessels showed a change similar to that seen in the sections of the lung and heart. (The appearances were very similar to those depicted in Fig. 15 in the paper of E. F. McKeown, 1947.) The *spleen* showed a reactive hyperplasia. The appearances in the *liver* were those of venous congestion. The *knee-joint* showed a typical rheumatoid change with cell proliferation, even to the formation of giant cells in a few places.

Case 2*

A female cotton-mill worker aged 48 was admitted to hospital on Dec. 15, 1946, complaining of cough, loss of weight, and arthritis. The onset of an acute polyarthritis characterized by pain, swelling, and stiffness of the knee-joints had occurred for the first time in April, 1946, previous to which she had been quite well. During the four weeks following the onset the wrists, elbows, and shoulders had been involved. In November, 1946, she started to complain of lassitude, palpitations, and dyspnoea, and since the onset of the illness she had lost 2 st. (12.7 kg.) in weight. She gave a previous medical history of recurrent attacks of bronchitis. There was no relevant family history.

On examination there was a morbilliform rash of the arms and trunk; the temperature was 100° F. (37.8° C.), and dullness and bronchial breathing were present at both lung bases. There was no evidence of abnormality in the heart, abdomen, or central nervous system. The blood pressure was 105/60. *Pathological investigations* at this time showed Hb 70% and white cells 8,900. *Radiological examination* of the chest revealed bilateral basal opacities indicative of consolidation and, above this, marked reticular shadows extending into the mid-zones. Pneumonia was diagnosed and a seven-day course of penicillin was given, in spite of which a pyrexia ranging between 99° and 100° F. (37.2° and 37.8° C.) persisted. Except for occasional rises of temperature to 103° F. (39.4° C.) the patient remained in this condition until Jan. 16, 1947. During this time repeated sputum cultures for tubercle bacilli were negative, the general bacteriological examination of the sputum revealed nothing abnormal, and the radiological appearances of the chest remained unchanged on repeated examinations. At the beginning of February, 1947, she developed a spiking temperature ranging between normal and 103° F. From this time her condition gradually deteriorated, and she became more anaemic. Blood examination showed: red cells, 2,570,000; Hb, 50%; serum albumin, 2.2 mg. per 100 ml.; serum globulin, 4.5 mg. per 100 ml.; serum sodium, 320 mg. per 100 ml.; chlorides, 533 mg. per 100 ml.; blood urea, 26 mg. per 100 ml.; W.R. negative. Agglutination tests: *S. paratyphi A* "H," *S. paratyphi B* "H," *S. typhi* "H," negative 1 in 20, Comp. *S. paratyphi B* "O," *S. typhi* "O," *Br. abortus* negative 1 in 20.

On March 24 oedema of the legs developed, and on the 27th the patient died.

*This case was kindly brought to our notice by Professor Robert Platt, of Manchester, who, having seen the observations of one of us (Ellman, 1947)—namely, the interpretation of the joint and pulmonary lesions as part of a "rheumatoid disease"—felt that the case, which was in the wards of the Manchester Royal Infirmary under the care of Dr. Fergus R. Ferguson, was of an analogous nature. We are much indebted to him and to Dr. Ferguson for kindly suggesting that we might include the case.

Post-mortem Examination (performed by Dr. J. T. A. Lloyd 36–48 hours after death).—The body was that of a slightly pale middle-aged woman. *Respiratory system*:—There were a few fine pleural adhesions on both sides, with some 50 ml. of straw-coloured fluid on each side. The lower lobes of each lung and the lower part of the upper lobe of the left lung were firm and hard to the touch. They were congested, and also showed small areas of consolidation together with minute abscesses containing a little pus. The appearances suggested a terminal bronchopneumonia with a fibrosing pneumonitis. The remainder of the lung tissue appeared normal. No evidence of tubercle or of sarcoid was seen in the lungs. The trachea was normal. The mediastinal glands draining the lungs were slightly enlarged, but showed no evidence of tubercle. *Cardiovascular system*:—The pericardium contained about 50 ml. of greenish-yellow clear fluid. The heart was slightly dilated but not enlarged. The myocardium was soft and somewhat pale. The mitral valve cusps were very slightly thickened along the free margins, but no evidence of any endocarditis was present in any valve. The blood vessels showed slight atheroma. The *intestinal tract*

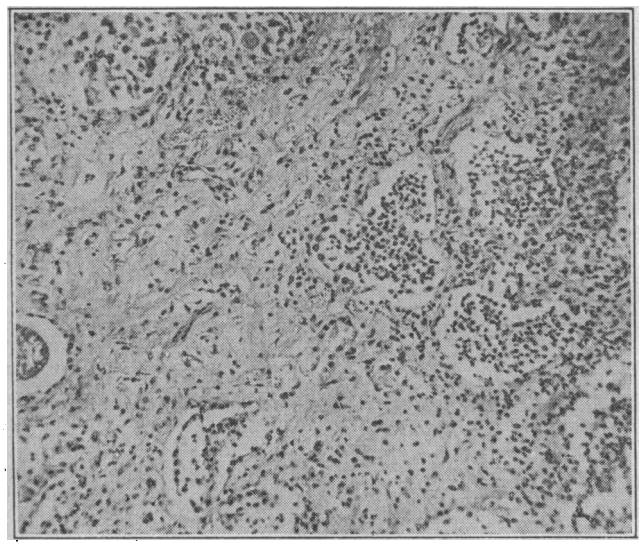

FIG. 2.—Case 2. Photomicrograph showing interstitial pneumonitis and secondary bronchopneumonia. (× 90.)

was normal throughout. The *liver* was not enlarged, but showed some degree of fatty change. The *spleen* was soft and slightly toxic as a result of the pulmonary infection. The *kidneys* showed no abnormality apart from some degree of pallor. There was a small cervical polypus, but the uterus and ovaries were normal. None of the organs revealed any evidence of tubercle or of sarcoidosis. The brain was not examined.

Histological Report.—Sections were cut from the kidney, spleen, lung, and lymphatic glands from blocks kindly supplied by Professor S. L. Baker. *Kidney*:—There were relatively few changes. Those present consisted of collapsed empty glomerular tufts, some tubular epithelial desquamation, and a few tubular casts. The blood vessels showed some atheroma only. The *spleen* revealed some reticular-cell proliferation with relatively empty sinuses. The *lung* (Fig. 2) showed considerable general disorganization. The main appearances were those of an interstitial pneumonitis with a terminal bronchopneumonia. The *alveoli* were of varying sizes, some being emphysematous, but many showed infiltration with polymorphs together with some mononuclear cells and also some desquamated alveolar phagocytes. A few of the alveoli were lined with cuboidal epithelium. However, the most striking change was seen in marked interalveolar fibrosis, with fairly well developed fibrous tissue which was infiltrated with mononuclear cells, plasma cells, and a few polymorphs. The blood vessels showed no change from the normal except that in one large vessel there was a little atheroma. There was no evidence of tubercle or sarcoidosis. The mediastinal lymphatic glands showed some chronic inflammation only.

Case 3

A housewife aged 55 was admitted into the rheumatism unit under the care of P. E. on Sept. 26, 1946, when she complained of pain, swelling and stiffness of the hands, shoulders, knees, and feet of three months' duration. There was no history of a precipitating factor in the form of an acute infection. Frequent attacks of tonsillitis had occurred during the past few years.

On examination she was thin and wasted; weight, 6 st. 9 lb. (42.2 kg.). No clinical abnormality was present in the heart, lungs, abdomen, or central nervous system. Examination of the locomotor system showed fusiform interphalangeal joint swellings and a swollen flexed left knee-joint. There was no lymphadenopathy.

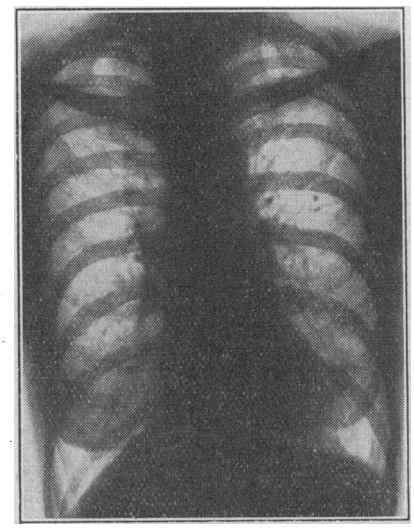

FIG. 3.—Case 3; Oct. 1, 1946. Radiograph of chest showing no gross abnormality.

Radiological examination of the chest showed no gross abnormality (Fig. 3). There were some calcified hilar foci; those of the hands and wrists showed osteoporosis and narrowing of the joint spaces.

Pathological investigations showed: E.S.R., 60 mm. in the first hour (Westergren); red cells, 5,920,000 per c.mm.; Hb, 102%; C.I., 0.85; white cells, 22,200 per c.mm. (polymorphs, 68%; lymphocytes, 26%; monocytes, 6%; eosinophils, 2%). Gastric residue before histamine: free HCl, nil; total acidity, 18 ml. N/10. Gastric residue after histamine: free HCl, 22 ml. N/10; total acidity, 44 ml. N/10. Urinalysis, N.A.D. Throat swab: no Klebs-Loeffler bacilli or haemolytic streptococci grown on Oct. 4, 1946; copious growth of haemolytic streptococci on Nov. 27; scanty growth of haemolytic streptococci on Dec. 5; W.R. and Kahn, negative; serum uric acid, 2.6 mg. per 100 ml.; alkaline phosphatase, 7.5 units.

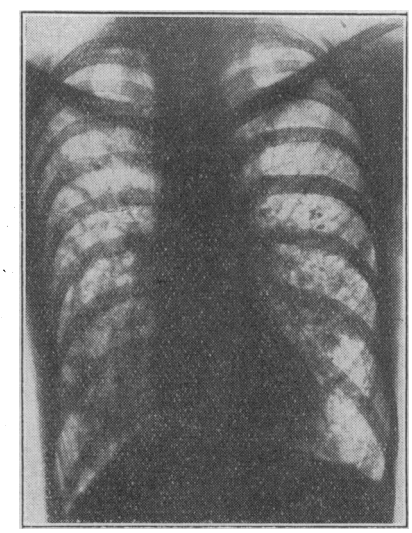

FIG. 4.—Case 3; June 16, 1947. Radiograph showing widespread heavy reticulation and miliary mottling throughout both lung fields.

The patient's general condition remained the same until June 12, 1947, when she complained of cough with sputum, sweating, and loss of weight of 7 lb. (44.5 kg.). There were now scattered rales throughout both lung fields, and the breath sounds were bronchovesicular in type. Serial skiagrams were unchanged until the one on June 16, which showed widespread heavy reticulation and apparent miliary mottling throughout the whole of both lung fields (Fig. 4). A presumptive diagnosis of pulmonary tuberculosis, Boeck's sarcoidosis or polyarteritis nodosa was made. Repeated sputum examinations and Loewenstein's culture, together with gastric lavage and concentration techniques, failed to

reveal the presence of tubercle bacilli. General bacteriological examination of the sputum revealed no abnormality. On Aug. 30 there was no significant change, and the radiological appearance of the lungs was regarded by several experts as being compatible with that of polyarteritis nodosa. A further radiograph on Jan. 16, 1948, showed the lung fields to be cleared a little, but heavy reticulation was still present. The patient now weighs 6 st. 2 lb. (40 kg.), but her general condition is fundamentally unchanged.

Discussion

In a recent discussion on the aetiology of chronic rheumatism one of us (Ellman, 1947) suggested that the theory of bacterial allergy or that due to foreign protein or hormones provided an attractive and rational aetiological basis for this rheumatoid state and at the same time strengthened the unitary theory. Fundamentally the acute and chronic rheumatisms may be regarded as anaphylactic diseases with multiple lesions in the mesodermal system produced by continual antigen–antibody reactions in or on tissue cells. Visceral and other lesions may be explained as hypersensitivity manifestations in tissues elsewhere in the body. Some co-ordination is thereby provided for the rheumatoid group of diseases and suggests a relationship between the acute rheumatoid type of arthritis and rheumatic fever.

It is possible, too, as has been suggested by Parkes Weber (1946), that the syndromes of a recurrent and transitory nature included under the term "palindromic rheumatism" of Hench and Rosenberg (1944) can be extended to include the so-called intermittent hydrarthrosis, serum joint disease, allergic arthritis, and allied syndromes. Again, the work of Rich and Gregory (1943, 1946) and their co-workers has indicated an even wider association on the basis of hypersensitivity between the rheumatoid type of arthritis and such hitherto obscure conditions as sclerodermia, dermatomyositis, polyarteritis nodosa, and acute disseminated lupus erythematosus. Bohrod (1947) has described their essential lesions as "rheumatoid granulomata," and Cecil (1946) refers to "these rheumatoid diseases of the collagen matrix." In clinical practice we have been impressed on more than one occasion by the association of sclerodermia with a rheumatoid type of arthritis (Ellman and Parkes Weber, 1948). While the experimental production in rheumatic fever of anaphylactic pulmonary lesions with a peculiar type of "pneumonitis" has received some attention (Rich and Gregory, 1943, 1946), pulmonary and other lesions are now known to occur also in such widespread systemic diseases as polyarteritis nodosa (Daley and Miller, 1946) and disseminated lupus (Rakov and Taylor, 1942; Klemperer, 1948).

So far as the lung lesions are concerned, Gouley (1938) and Neubuerger *et al.* (1944) found histologically in "rheumatic pneumonia" three successive stages of change in the interalveolar septa: (1) fibrinoid necrosis in collagen, (2) infiltration with round cells, plasma, and giant cells; (3) fibroblastic proliferation and fibrosis. All these are essentially changes in the collagen matrix.

In support of the anaphylactic or hypersensitivity theory in the mechanism of production of these lesions Rich and Gregory were able to carry out necropsy studies on patients with serum sickness. They found changes in the endocardium, myocardium, and pericardium, all of which were histologically indistinguishable from those in rheumatic fever. Further experiments have been carried out on animals rendered anaphylactic with horse serum or egg albumen. Similar changes were again produced in the heart, and more recently pulmonary lesions characterized by interstitial fibrinoid necrosis.

From those observations, and in view of the widespread systemic nature of some of the cases, it will be understood how the term "rheumatoid arthritis" is really misleading. "Rheumatoid disease" may be open to criticism, but it is preferable as the parent term, with joint lesions seen as the principal clinical manifestation, in the same way that the term "gout" describes the parent lesions of a metabolic dysfunction whose principal clinical manifestation is the joint involvement.

The three cases we have described exhibit common features in what appears to have been the development of lung lesions during the early active phase of the joint process. In Case 3 the onset of the lung disease is known from the serial radiographs to have occurred nine months after the onset of the polyarthritis, and attention was drawn to it by the symptomatology of cough, dyspnoea, and weight loss initially ascribed to possible pulmonary tuberculosis. All investigations disproved this diagnosis, likewise the possibility of Boeck's sarcoidosis. A consensus of expert opinion on the radiological appearances of the lung reticulation has favoured the diagnosis of polyarteritis nodosa, and the relationship of this condition with the "rheumatoid granulomata" is of considerable interest. It would be fair to postulate a hypersensitivity phenomenon involving equally the joint and lung tissues. In Case 2 symptoms referable to the pulmonary disease occurred six months after the onset of the polyarthritis. The radiological lung changes were those of a basal reticulation. Unfortunately no radiograph of the chest before this time is available.

In all three cases the clinical course is similar, the joint lesions preceding the pulmonary lesions, and one might not unreasonably assume, without wishing to appear to be in any way dogmatic, that the joint and lung lesions are manifestations of one and the same pathological process.

Studies of the two cases that came to necropsy present certain features in common, also suggestive on histological investigation and predominantly evident in the lungs, where fibroblastic thickening of the alveolar walls (chronic fibrosing pneumonitis), plus infiltration by mononuclear, plasma, and occasional polymorph cells, was present; the kidney in Case 1 revealed fibrinoid necrosis in a few blood vessels. This case also showed similar changes in blood vessels at another site—namely, in the fatty tissue immediately next to the heart muscle. In both cases the section of the lung has shown a chronic fibrosing type of "pneumonitis." It was certainly not sarcoid, and at the same time was rather different from the "rheumatic pneumonia" of acute rheumatic fever. Our own opinion was that the lung changes could not be dissociated from Gouley's Stage 3, already described, where interalveolar fibroblastic proliferation predominated on histological examination. However, it cannot be held that this histological change is specific by itself, but its being coupled with the necrosis found in the muscular wall of the artery in other parts of the body suggests that an acute arteritis had been present in the past as part of a fibrinoid necrosis. These arterial changes were found only after careful and thorough search, and, taken together with the lung lesions, would appear to suggest a widespread disseminated lesion. Further than that we are not prepared to go from the pathological studies alone. However, on clinical grounds the similarity of the lung lesions is undoubted, and many features of one are mirrored in the other.

It is of interest to note that Klemperer has recently reported a case of disseminated lupus erythematosus with lung lesions where the histology was of a fibrinoid necrosis in the interalveolar septa. A similar pathological process has been observed in the allied group of so-called granulomata already noted.

Summary

The widespread systemic nature of certain cases of rheumatoid arthritis has been noted.

It is suggested that "rheumatoid disease" is a preferable term whose principal clinical manifestation is "arthritis."

Three cases of joint and lung lesions are recorded, and it is suggested that they are among the clinical manifestations of "rheumatoid disease."

We are greatly indebted to Dr. J. N. Cumings for help on the pathological side, for the photomicrographs, and for his close study of the histology of Cases 1 and 2; to Dr. A. Signy for much helpful criticism; to Professor Robert Platt for kindly allowing us to include Case 2; and to Dr. G. Batten for help on the radiological side.

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CORD COMPLICATIONS DURING PREGNANCY AND LABOUR

BY

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Considering the length and slenderness of the umbilical cord, it is surprising that cord complications do not occur more frequently. Prolapsed cord, which is the commonest of these complications, occurred in only 50 cases out of a total of 10,000 deliveries—i.e., 0.5%. Munro Kerr (1937) gives an incidence of 0.25% and DeLee (1943) an incidence of 0.8%.

The 42 cases treated in this hospital are listed in Table I. Eight patients were admitted with dead babies and therefore received no special treatment A summary of the 50 cases is given in Table II.

Three of these cases are given in further detail because of their special interest.

Mrs. A., aged 35; 2-gravida; married 12 years. Admitted with severe pre-eclamptic toxaemia, not improved by rest. Surgical induction performed at 28 weeks with a Drew-Smythe catheter. At half dilatation, hand and cord prolapsed beside head. Attempt at reposition unsuccessful. Forceps then applied to foetal head, but, though small, the head was too large to be delivered through the thick half-dilated cervix. Two incisions made in cervix, and infant, weighing 2 lb. 8 oz. (1.13 kg.) delivered alive. Cervix sutured. Mother made uneventful recovery. Infant lusty at birth and remained healthy. It is now 2 years old.

This delivery may appear to be rather heroic treatment but is justified by results.

Mrs. B., aged 28; 2-gravida; admitted at term as a transverse lie. External version to vertex performed, and patient sent to *x*-ray department for pelvimetry. Membranes ruptured and cord prolapsed while patient was on the *x*-ray table. Patient was returned to the ward in the knee–chest position and the cord replaced through a quarter-dilated cervix. Pulsation in the cord returned after reposition, and caesarean section was performed. At operation the cord was found to be stretched tightly across the head, and the child was stillborn.

Mrs. C., aged 26; primigravida; term. Breech presentation at term. Fairly easy external version to vertex, but foetal heart became irregular and slow. A few minutes afterwards the heart returned to normal and the foetus was left as a vertex, with the head above the brim. Membranes ruptured early in labour, and the foetal heart became irregular. The head was still high, but when it was pushed down into the pelvis with a hand above the symphysis the foetal heart stopped altogether, returning as soon as the pressure was released. It was decided that this was the so-called "occult type" of prolapsed cord—i.e., the cord was probably nipped between the foetal head and the pelvic brim. Caesarean section was therefore performed and a healthy infant obtained. The cord was twisted loosely round the child's body, but the actual prolapsed loop was not seen, as it would be dislodged as the head was delivered.

Apart from prolapse of the cord, the following cord complications can occur:

Cord Round Neck or Body

With an active foetus, or with repeated antenatal versions, the umbilical cord may be wound round the neck or body once, twice, or three times. Only once have I seen the cord four times round the neck of the foetus. Strangulation *in utero* or during birth may result. This condition of the cord is usually recognized only during labour or at delivery, but in a few cases it can be diagnosed during pregnancy. The following three cases illustrate the point.

Mrs. D., aged 35; primigravida; breech presentation at term. Easy antenatal version, but directly after version the foetal heart dropped to 80. The heart rate gradually returned after a few minutes and the foetus was left in the vertex position. Labour started spontaneously a few days later. Delivery was normal, after a rapid labour, but the infant was stillborn, with the cord tightly round the neck twice.

Mrs. E., aged 35; primigravida. Breech presentation at 36 weeks. Easy antenatal version, but foetal heart stopped. Foetus was turned back to a breech, and the heart restarted as soon as the foetus reached the transverse position. This manœuvre was repeated the following week with exactly the same result. It was therefore concluded that the foetus had a short cord or the cord was round the neck, and that this would cause delay in delivery and probably stillbirth. It was decided to deliver by caesarean section at term. At operation it was found that the cord was twice round the infant's neck and that there was an area of haemorrhage at the root of the cord where it had been pulled upon during the antenatal version. The infant was healthy, weighing 6 lb. 15 oz. (3.15 kg.), and the mother made an uninterrupted recovery.

Mrs. F., aged 34; primigravida; term. Breech presentation. Easy version, but foetal heart dropped to 100 and became muffled and irregular. The foetus was turned back to a breech. A second attempt a few minutes later produced the same result, so that the foetus was left as a breech. In this case also caesarean section was performed at term, and the cord was found to be wrapped round the neck and body. The infant was healthy, and the mother made a satisfactory recovery.

Over-activity of the foetus with self-strangulation is illustrated by the next case.

Mrs. G., aged 29; 2-gravida. At 38 weeks the head was high and the foetal heart was heard. The patient was healthy, with a blood pressure of 120/68, no albuminuria, and a negative Wassermann. Three days before term the foetus became increasingly active and then all movements suddenly stopped. Labour began spontaneously, and when the membranes ruptured the liquor was deeply stained with meconium. The infant was stillborn and macerated, and the cord was wound tightly round the neck three times, with tension at the umbilicus.

Cord round the neck, apart from causing foetal distress, may prevent engagement of the head and so increase the duration of labour.