Radiological Reports.—March 6:—Both middle zones showed impairment of a congestive type, more pronounced on the right side. Both lung fields were infiltrated with generalized miliary shadowing, more intense in the basal zones (Plate, Fig. 1). The nodules were discrete and averaged 0.5 to 2 mm. in diameter. No pleural effusions were seen. The heart was enlarged in its transverse diameter and had a contour suggestive of mitral stenosis. *March 26:*—An effusion had developed in the right costo-phrenic angle. The miliary opacities remained unchanged, showing no tendency to resolution or confluence. The tentative diagnosis of haemosiderosis was made.

Necropsy

Macroscopic Appearance.—The subject was well nourished and showed slight oedema of the lower extremities. About 300 ml. of clear straw-coloured fluid was found in each side of the thorax. The heart weighed 570 g. and exhibited gross right ventricular hypertrophy. Both auricles were hypertrophied and dilated, a large ball thrombus being found in the left auricle. The tricuspid valve admitted four fingers. Thickening of the cusps of the aortic valve was present, their edges being adherent and giving rise to some stenosis. The mitral cusps were thickened and shortened, with adherent edges leading to stenosis of the funnel type. Thickening of the chordae tendineae was pronounced. The lungs showed cyanotic induration. Numerous large and small infarcts were evident, especially at the bases. Some were suppurative. The pleurae were studded with miliary calcified plaques 1 to 2 mm. in diameter. Nodules of bone in the lung parenchyma, described below, were neither seen nor felt. The spleen was enlarged, hard, and dark red, and there was moderately severe chronic congestion of the liver. There was also congestion of the mucosa of the stomach and of the renal parenchyma. The brain was not examined.

Microscopical Appearance.—Well-marked chronic congestion was seen in the lung substance. The interstitial tissue carrying the vessels showed some fibrous increase, with large numbers of pigment-carrying cells. Throughout the lung substance were seen numerous rounded nodules of bone with irregularly crenated outlines, many showing a concentric lamellar appearance (Fig. 2). The bone was mostly well calcified, but many nodules had a calcified centre with peripheral additions of osteoid tissue. This was well seen in the subpleural plaques, which showed an inner calcified and an outer osteoid zone. Vessels were present in the bone, but no medullary cavities were seen. The size of the nodules was several times that of an alveolus. A striking feature was that most of these nodules had no fibrous surround and under the microscope gave the appearance of lying free in the lung substance. In some areas, however, the bone was embedded in fibrous tissue containing pigment-laden phagocytes. The vessels in the interstitial tissue of the lungs showed no abnormality.

Discussion

The aetiology of the condition is obscure, and is likely to remain so until more cases are discovered. Past rheumatic infection with advanced mitral stenosis is invariable. Cases which have come to necropsy have all shown some degree of chronic passive congestion of the lungs. The question arises whether this alone can give rise to disseminated ossification or whether another factor—e.g., rheumatic pneumonia—is necessary to produce foci of necrosed or poorly vascularized tissue in which calcification and ossification can take place.

In the above case there is not sufficient histological evidence to warrant the presence of any other aetiological condition than chronic passive congestion. It is suggested that the bone may arise from organization of congestive haemorrhages or of intra-alveolar collections of pigment-bearing phagocytes. It should be appreciated that rheumatic pneumonia as an entity is not universally accepted.

My thanks are due to Dr. M. M. Deane for permission to publish the case, to Dr. A. C. Counsell for the pathological report and for advice and criticism, to Dr. D. G. Arthur for the radiograph, and to Mr. D. A. Vinten for the photograph.

PULMONARY LESIONS IN RHEUMATOID ARTHRITIS

BY

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AND

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[WITH PHOTOGRAVURE PLATE]

The paper by Ellman and Ball (1948) on "rheumatoid disease" with lung lesions will no doubt provoke many observers to report similar experiences. We have so far regarded as unique a case of juvenile rheumatoid arthritis in which a transient diffuse pulmonary lesion formed an intimate part of the disease, which was severe and progressive. We have not made an exhaustive study of the formidable literature, but the inquiries we have made have not produced any serious study directed to this aspect of the disease.

The case mentioned was that of a boy aged 6, weighing 34 lb. 12 oz. (15.76 kg.), in whom the pulmonary episode was heralded by a mild tonsillitis but was symptomless except for a slight increase in malaise. Intermittent pyrexia over eight days increased to 101.8° F. (38.8° C.) and subsided by lysis over a similar period. There was no leucocytosis or eosinophilia. The blood sedimentation rate, which had been 80 to 100 (Westergren) over many months, fell to 15 on one occasion at the height of the fever, rising again almost immediately afterwards. This boy was in hospital throughout in a separate cubicle, and his Mantoux reaction was negative before and after the incident. The radiographs show the condition of his lung in November, 1947, four months before the onset of the lung condition: on March 3, 1948, at the height of it; and on April 6, when it was resolving (Plate, Figs. 1, 2, and 3). Pulmonary congestion from left heart failure, possibly precipitated by a blood transfusion given three weeks before, was considered as a possible explanation of the attack, but this idea was rejected because of the absence of any evidence of heart disease and the rapid recovery without treatment. During this transfusion, the latest of several, an extensive urticarial rash appeared. In an earlier transfusion there had been rather more severe allergic symptoms. The further history of the case is as follows

Case History

A boy aged 6 was admitted to hospital on Dec. 12, 1946. He was the youngest of six children, and his parents and sibs were all healthy. In November, 1945, he had scarlet fever, followed by otitis media. He then remained well until August, 1946, when swelling of various joints occurred, each lasting for only a few days. There was pain on movement, but no tenderness. From time to time all the joints of his body, except those of his hands, feet, and spine, were affected. He was febrile during the attacks of pain, he lost weight, and his energy and appetite decreased.

At the time of admission there was swelling of the right ankle and right knee, but little interference of movement and no tenderness. He was afebrile. There was slight generalized adenopathy, but no apparent enlargement of the spleen then or subsequently. The liver, however, was always palpable. A diagnosis of juvenile rheumatoid arthritis was made, and he was given two courses of gold treatment with a maximal single dose of 25 mg. (total, "myocrisin" 212 and 210 mg.). During the next year he remained in hospital, and the disease was steadily progressive. Profound muscle-wasting occurred, and there was some limitation of movement at the wrists and severe osteoporosis with a tendency to periostitis in the small bones. Anaemia was a persistent feature and was only slightly improved by treatment,

which included repeated small blood transfusions. After about six months of observation the small joints of the hands became involved. He tended to get urticaria after transfusions, and on one occasion had symptoms suggestive of a mild anaphylaxis during a transfusion, there being no evidence of group incompatibility. Improvement in general condition followed a high-protein diet (2 g. per lb. body weight). The sedimentation rate during the earlier period of observation fluctuated between 20 and 30 mm., but later was consistently in the neighbourhood of 100, and after August, 1947, the only observation made of a sedimentation rate which was below this level was on March 3, 1948 (15 mm. at one hour), during the episode of pulmonary involvement. This was unfortunately not repeated at the time. His Mantoux reaction up to 1 in 100 was tested from time to time and was always negative. The white cell count was persistently low. The serum proteins were found to be low (albumin 1.45, globulin 5) in August, 1947, and following high-protein diet increased to albumin 5.4, globulin 2.5. This effect was produced some months before the pulmonary episode. Estimates of serum calcium, phosphorus, and serum iron, and urinary analyses, were all within normal limits.

When last seen (August, 1948) the boy's condition was unchanged. He was at this time transferred to an orthopaedic centre.

Discussion

We agree with Drs. Ellman and Ball in considering the lung condition to be a part of the allergic state, of which the entire symptoms of rheumatoid arthritis are also manifestations. The linkages suggested between acute rheumatism, rheumatoid disease, Henoch's purpura, arthritis, and possibly glomerulonephritis may well turn out to be too nebulous as a theory, but, as in the reticuloses, a conception which postulates a synthesis of phenomena which are similar in some part of their characters may well be profitable, and should lend itself to critical examination and experiment in a number of ways.

Hypothetically, the course of events might be as follows: A parasite, presumably a streptococcus, is introduced for the first time to an individual. In infancy he reacts to it according to his genetic constitution and according to the degree to which this has been influenced by humoral factors derived from his mother's circulation via the placenta. In his tissues this first contact is "sensitizing," and by analogy with tuberculosis the tissue reaction will be "productive," confining, and healing, and will establish a tissue and humoral pattern. For most individuals this process will proceed to a satisfactory immunity; for some it will be incomplete, either generally or locally. These latter are the potential victims of rheumatic disease. The groups will merge into each other; individual reaction will proceed at varying speeds and in varying degree, and will be manifested in different ways according to the intensity of subsequent contacts. There is a higher incidence of rheumatic fever in the children of families in which other manifestations of rheumatoid disease are present, which suggests that the failure of the immunity reaction to proceed to completion is in part decided by genetic constitution.

The universal experience of streptococcal infection makes it unlikely that the chances of contact provide the main cause of differences in incidence. Environmental differences, of which nutrition is probably the most important, cannot be ignored. The decreased incidence of acute rheumatism has coincided with the provision of school milk and meals and the introduction of food subsidies and rationing. Chemotherapy may also have had a modifying action. The response of the sensitized individual to secondary contact would correspond to the exudative phase of tuberculosis and, as in tuberculosis, might be initiated by autogenous activation under the stress of different circumstances, such as nutritional defect. The silent period of the first three to five years of life is the period of developing sensitivity, with school life introducing increased opportunity for streptococcal infection. Reinfection, autogenous or exogenous, in the sensitized but non-immune child may then show itself in various ways: (1) polyarthritis and the specific cellular reaction of the Aschoff nodule in heart, skin, blood vessels, etc., constituting the acute rheumatic attack; (2) in the basal ganglia as chorea; (3) as localized tissue reaction in the capillaries, as in the Henoch–Schönlein complex, and possibly in the glomerular tufts as nephritis; (4) in the arterial wall as periarteritis nodosa; and (5) in the lung as rheumatic or rheumatoid pneumonia.

Such a thesis can probably be tested in a number of ways—by a critical examination of heredity in rheumatic disease, by a study of the influence of nutrition and chemotherapy in the course of streptococcal infection both in animals and in man, by further efforts to establish a satisfactory test of sensitivity, and by observation of its application in groups of children.

Acknowledgments are due to Dr. J. Bracken, radiologist, and Mr. R. G. Pearman, photographer, for their co-operation.

REFERENCE

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ENDOMETRIOSIS OF THE GROIN

REPORT OF THREE CASES

BY

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[WITH PHOTOGRAVURE PLATE]

Swellings of the groin are an everyday problem in diagnosis to many doctors. Endometriosis at this site is uncommon, and when such a case is encountered its true nature may be overlooked if one is unmindful of the possibility. Lyall (1945) describes a case of inguinal endometriosis in a housewife, who, on her doctor's instructions, wore a truss over the lump for six years. The following three cases, two with inguinal lumps and the third with a femoral lump, are recorded as interesting examples of this unusual condition.

Case 1

A married woman aged 41, with two children, was admitted to the Radcliffe Infirmary on Dec. 2, 1941. About 22 months previously, on the day before a period was due, she first noticed a lump in the right groin. It rapidly swelled up until it was "nearly as big as an egg" and was very painful. On the last day of the period it began to shrink and became "not much bigger than a pea." After that she had a clear warning of an approaching period by the onset of pain, tenderness, and swelling of the lump on the preceding day, but the swelling did not again reach its initial size. During the period walking increased the symptoms; at the end of each period the swelling diminished and the symptoms subsided.

On examination a firm, mobile, finely nodular lump about 1½ by ¾ in. (3.75 by 1.88 cm.) was found in the right groin at a site just lateral and superior to the pubic tubercle. It was not adherent to skin or deeper structures and there was no impulse on coughing. Examination of the pelvis revealed no abnormality, and all other systems were normal.

On Dec. 4 Mr. Stallworthy made an incision over the mass, and, finding it intimately fused with the surrounding tissues, he excised it completely. Subsequent wound healing was uneventful.

Histological Report.—"The section shows ectopic endometrium both in lymph-nodal and connective tissue. Most of the tubules are irregular and non-secretory in type, but a

D. G. LEYS AND P. N. SWIFT: PULMONARY LESIONS IN RHEUMATOID ARTHRITIS

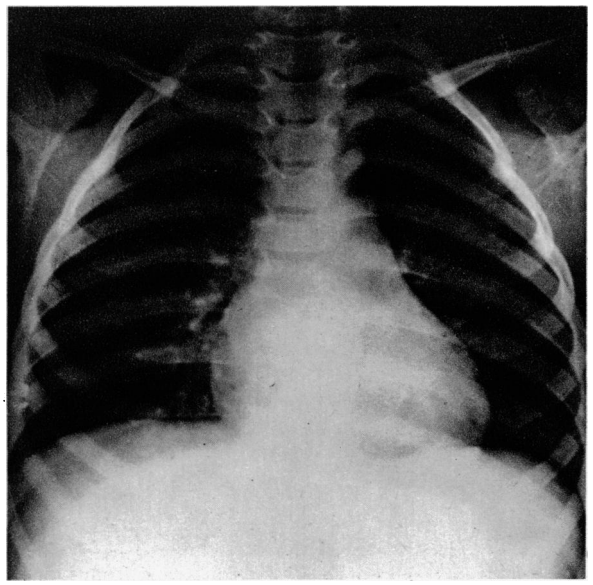


FIG. 1.—November, 1947. Four months before onset.

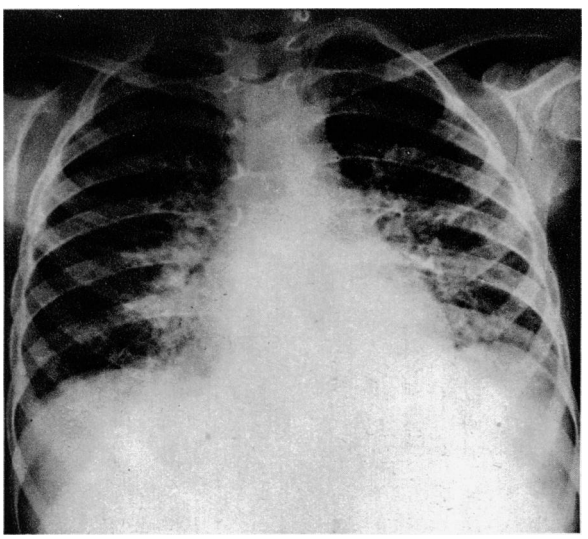


FIG. 2.—March, 1948. Pulmonary episode.

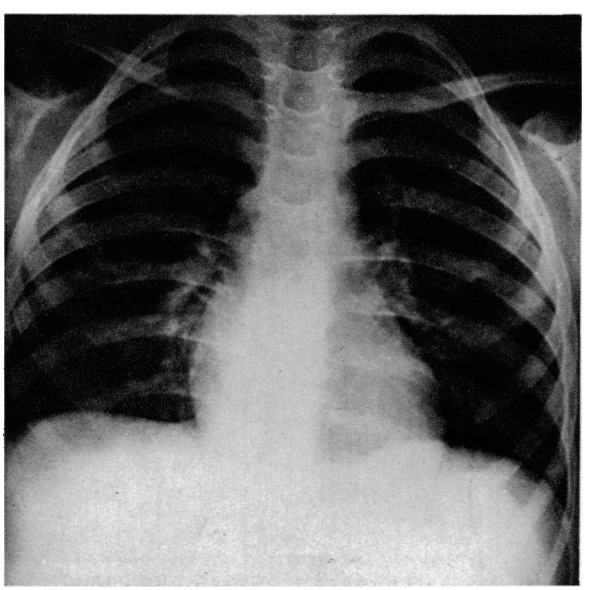


FIG. 3.—April, 1948. Resolving.

A. IAN L. MAITLAND: SEVERE HYPERTENSION WITH RECOVERY AFTER NEPHRECTOMY

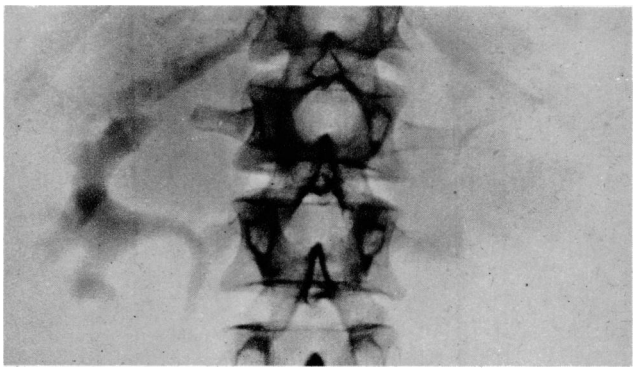


FIG. 1.—Normal outline right renal pelvis. The left renal pelvis and ureter are only visible as a thin line.

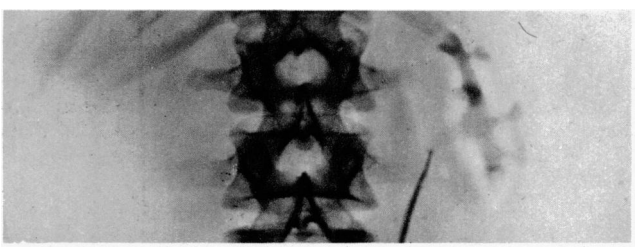


FIG. 2.—Retrograde pyelogram showing on left side normal outline of the small apelvic type.

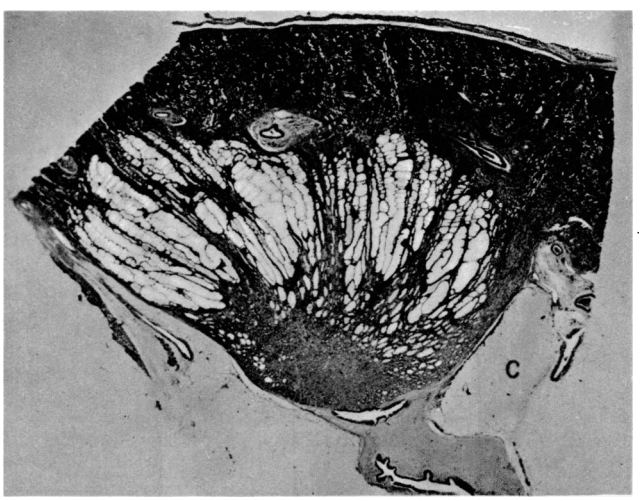
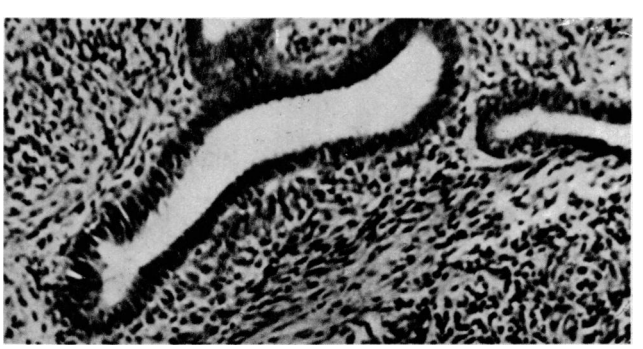


FIG. 3.—Section of renal parenchyma (× 5). Note narrowing of cortex and enormously dilated tubules filling the central area of the pyramid. C marks the cavity of the calix.

G. E. MOLONEY: ENDOMETRIOSIS OF THE GROIN



Case 2. Endometrium in lymphatic tissue.