If there is no thrill, a left parasternal murmur unaccompanied by any of the clinical features of V.S.D. mentioned above should be regarded as innocent.

Simple pulmonary valvular stenosis can usually be distinguished by the higher position of the thrill and murmur, by the softness of the pulmonary element of the second heart sound (if it can be heard at all), by the tapping cardiac impulse, and by dilatation of the pulmonary artery in the absence of pulmonary plethora. Subvalvular stenosis is more difficult, because turbulence may then be lower and the pulmonary artery may not be dilated; but the second heart sound and the quality of the cardiac impulse should prevent mistakes. Severe pulmonary stenosis is easily distinguished by the electrocardiogram. Mild subaortic stenosis may be clinically indistinguishable from the "maladie de Roger" if the murmur and thrill are low; if the left ventricle is enlarged, however, absence of other evidence of V.S.D. strongly favours subaortic stenosis.

Organic mitral incompetence with left ventricular enlargement, a mitral diastolic murmur, and a systolic thrill and murmur rather centrally placed may deceive the most experienced; but skiagrams should reveal the tell-tale systolic expansion of the left auricle if there is mitral incompetence, and pulmonary plethora if there is V.S.D.

Patent ductus without a Gibson murmur should be recognized by the peripheral pulse. Confusion is scarcely possible unless V.S.D. is complicated by aortic incompetence; and if systolic turbulence then happens to be high, real difficulty may be encountered. Clinical or electrocardiographic evidence of right ventricular dilatation (as well as left) favours V.S.D.

Atrial septal defect with a relatively low systolic thrill and murmur, and with a hyperdynamic right ventricle forming the apex beat, may closely simulate V.S.D. But the second heart sound is much more widely split in A.S.D., and the pulmonary element is not accentuated. Moreover, the Roesler type of thrusting cardiac impulse is much less common in A.S.D. than was at one time thought: in clinical diagnosis few mistakes will be made if a left ventricular type of cardiac impulse is accepted at its face value.

Terminology

It is suggested that the term "maladie de Roger," if retained at all, should be applied only to mild cases showing nothing more than the Roger murmur and thrill. The majority of cases of V.S.D. present a very different picture from that described by Roger himself and from all textbook descriptions of the "maladie de Roger." Nor is Taussig's classification of high and low defects acceptable. She regards cases with pulmonary plethora and dilated pulmonary arteries as examples of high V.S.D., the left ventricle pumping much blood almost direct into the pulmonary artery. She thinks that trivial shunts mean low V.S.D. This is incorrect. Selzer (1949) has already reviewed the literature from the pathological point of view and shown that only 10% of all cases of uncomplicated V.S.D. have low defects in the muscular septum and that these present no distinctive clinical features. In the present series cardiac catheterization has shown that high and low right ventricular samples are similar to one another and to samples from the pulmonary artery in nearly all instances (Table III); only in the mildest case was there any significant difference, and then the lowest sample was the least saturated.

(The conclusion of the paper will appear next week, with a full list of references.)

A circular from the Ministry of Health to local health authorities points out that many soldiers discharged from the Army on psychiatric grounds need social rather than medical aftercare. A special procedure has therefore been devised for these people as an experiment for one year. Army Form D.487, with which medical officers of health are familiar, will be marked "psychiatric aftercare" and sent to the M.O.H. of the area where the soldier will live. The local health authority should arrange for an initial visit to be paid to the ex-soldier by a social worker soon after the form is received. The authority may then arrange for aftercare services to be provided.

DIFFUSE EOSINOPHILIC ARTERITIS

BY

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[WITH SPECIAL PLATE]

During the past years considerable interest has centred on the problems of tissue changes in conditions of allergic origin, and an increasing quantity of evidence has been collected demonstrating that certain abnormalities, such as vascular proliferative lesions, focal necrosis, and tissue oedema, are to be found in a variety of apparently different diseases linked only by the probability of their having an allergic background.

Essentially similar changes have been found in cases of rheumatic polyarthritis and polyarteritis nodosa (Bergstrand, 1946), whilst evidence of associated allergic skin phenomena have been demonstrated in these conditions; and the association of polyarthritis with clear-cut cases of polyarteritis nodosa is not unknown (Herlitz, 1930-1; Friedberg and Gross, 1934). Further, Rich and Gregory (1943) have shown that the lung lesions in rheumatic pneumonia are essentially similar to those found in cases in which hypersensitivity to sulphonamide drugs exists. There is also certain evidence (Salvesen, 1938; Ehrström, 1941) that glomerulo-nephritis—also considered to be of allergic origin—may, more often than was first supposed, be an associated factor in cases of rheumatism.

More recently, examples of vascular and tissue changes of a similar type, occurring particularly in the lungs, have been described in cases of asthma, and Wilson and Alexander (1945) have shown that 18% of their cases of polyarteritis nodosa had an associated bronchial asthma.

A group of cases has therefore emerged which, although presenting widely differing clinical pictures, has in common the possibility of a similar type of aetiology—namely, an allergic manifestation. Experimental proof of such a theory is so far lacking, and the threads of evidence which do exist are disappointingly inconclusive. Evidence of similar or related morbid anatomical and histological changes in the tissues of patients suffering from this group of diseases provides another method of approach to this problem which has been followed principally by Bergstrand. Opportunities for post-mortem examination have been scanty, for diseases of the asthma group are of relatively benign type, and it is considered justifiable to place on record a further case in which necropsy was performed and full clinical details are available, in order to add to the few examples of this type already in the literature.

Case Report

A married woman aged 33, with one healthy child aged 3, first consulted her doctor in the autumn of 1948 complaining of a cough which had been present for nine months. Previously she had been healthy, with no personal or family history of any complaint of an allergic nature. She gave a history of frequent streaking of blood in the sputum, and for two months she had suffered from dysphagia and hoarseness associated with a swelling of the thyroid gland, together with some increase of nervousness and irritability. She was admitted to hospital on October 10, 1948.

A blood count showed: Hb, 82%; red cells, 4,570,300; white cells, 7,400 (23% eosinophils). Sputum examined for tubercle bacilli was negative on three occasions, but 9% of the total number of nucleated cells were eosinophils. A radiograph of

the chest revealed no abnormalities. It was considered that a non-toxic goitre was causing some non-specific laryngitis which by this time had disappeared. Whilst in hospital the patient had a low-level pyrexia, which settled, and she was discharged improved on November 11.

Within a week of her leaving hospital laryngitis returned, and she was readmitted on December 29. The thyroid enlargement had increased and was giving rise to definite respiratory obstruction and dysphagia. Additional symptoms of swelling of the eyelids and oedema of the dorsum of the hands, together with chemosis of the conjunctivae, had manifested themselves. She was again running a low-grade pyrexia. The white cells numbered 13,200 (19% eosinophils). Subtotal thyroidectomy was performed on February 1, 1949, and histological examination showed no signs of toxicity. The patient was discharged improved after operation, but in April and again in June required hospital treatment on account of dyspnoea, dysphagia, and swelling of the eyelids. Each time a blood count was performed eosinophilia was revealed. On April 5 a radiograph of the chest showed for the first time abnormal irregular shadows in both lower zones, which had increased in size and distribution by June 20 (Plate, Fig. 1).

In view of her increasing dyspnoea at this time she was recommended for early admission to Westminster Hospital, where she arrived in the early evening of July 1 dyspnoeic but

shotty feel on palpation. Examination of the cut surface proved this to be due to very widespread irregular patches of consolidation of greyish yellow colour, involving the vast majority of the lung substance, with small intervening areas of compensatory emphysema (Fig. A).

Heart.—No abnormalities of valves or coronary vessels were found, and the musculature appeared normal on naked-eye examination, with the exception of one small area at the base of the left ventricle anteriorly, where it was very soft and yellowish in colour.

Spleen.—This was normal in size. The cut surface was rather pale and fatty, showing numerous scattered areas of greyish yellow infiltration varying in size from a pinhead to 0.5 cm. in diameter.

Kidneys.—These were normal in size, but scattered through the cortex were numerous yellowish areas of infiltration similar to those seen in the spleen and liver.

Thyroid.—A small remnant remained on the right side which was normal in appearance.

Lymphatic System.—Numerous enlarged lymph nodes were found in the neck, mediastinum, and para-aortic regions. The nodes were rubbery and discrete, and on the cut surface some of them had a yellowish appearance similar to the nodules in the liver, spleen, and kidneys.

The cause of death was considered to be acute respiratory obstruction associated with extensive bilateral pulmonary consolidation. The possibility of some bacterial infection of the lungs leading to a septicaemia was considered, but cultures taken from the blood, lungs, and the nodules in the spleen and kidneys were all sterile, and material injected into guinea-pigs and hamsters caused no abnormalities in these animals.

Microscopical Features

Lungs.—The normal architecture was replaced by a diffuse fibrous process showing irregular collagen bands and inflammatory cells, including numerous eosinophils, a few plasma cells, and some multinucleate giant cells (Fig. 2). Frequently the collagen strands were seen to be split up by oedema, and areas of hyaline degeneration were seen which failed to take up the aniline blue by Mallory's method or even to retain the first dye—in this case azocarmine. This collagen change has been described particularly in the so-called "Frühinfiltrat" of rheumatic pneumonia. Foci of necrosis were frequent and were invariably surrounded by a dense infiltration of eosinophils. Where an alveolar network remained signs of compensatory emphysema were seen. The bronchi were everywhere grossly thickened by oedema and infiltrated by eosinophils. The arterioles showed considerable intimal proliferation with thrombosis; necrosis of the wall with eosinophilic infiltration of the type found in polyarteritis nodosa was often seen (Fig. 3). Hyaline degenerative masses in the walls of the arterioles were a frequent finding.

Heart.—The myocardium showed a generalized degeneration of muscle fibres and these were widely separated by oedema and an infiltration of eosinophils. In the yellow area noted macroscopically focal necrosis with intense eosinophilic infiltration had occurred (Fig. 4).

Spleen.—There was a diffuse infiltration of eosinophils throughout the organ, and in the macroscopic yellowish areas focal necroses packed with eosinophils were seen. The majority of arterioles showed intimal proliferation.

Liver.—Accumulations of eosinophils were found around many portal tracts, and isolated cells of this type were found lying free in the sinusoids. No areas of necrosis were seen.

Kidneys.—Both had focal necrotic areas scattered in the cortex, where a few convoluted tubules had been destroyed and accumulations of eosinophils existed. Glomeruli were for the most part normal, but occasional capillary thrombi were seen (Fig. 5). The arterioles showed intimal proliferation and occasional thromboses, but no vascular necrosis was found.

Lymph Nodes.—The three lymph nodes examined revealed sinus catarrh with numerous eosinophils.

Lacrimal Gland.—This showed oedema and considerable infiltration by eosinophils.

FIG. A.—Cut surface of lung, showing diffuse patchy consolidation.

not unduly distressed. She ate a reasonable dinner soon after admission, but three hours later became extremely distressed with inspiratory and expiratory stridor. Direct laryngoscopy revealed no obstruction and she finally lapsed into a coma and died 12 hours after admission.

Post-mortem Examination

This was performed three and a half hours after death. The body was that of a young woman in a good state of general nutrition. A well-healed scar at the site of thyroidectomy was seen in the neck. There was some oedema of the face, more marked on the right side than on the left, and considerable swelling of the right lacrimal gland caused prominence of the right eyelid.

Respiratory System.—The pharynx and larynx were filled with thick sticky mucus, and the walls of the pharynx, larynx, trachea, and main bronchi were thickened and intensely oedematous. The mucous surface was flattened and very pale. The vocal cords were normal, and there was no abnormality in relation to either recurrent laryngeal nerve. Both pleurae showed easily broken-down adhesions, more marked on the right side, and at the right base a small collection of serous fluid was found. Both lungs were heavy and had an irregular

Discussion

To summarize, one may say that here was a woman with no relevant family or past history, who had complained of cough for 18 months, and who gave a nine-months history of associated dyspnoea and dysphagia. Partial thyroidectomy had been performed five months before death, with apparent temporary relief. The first sign which might have led to the suspicion of an allergic background was the eosinophilia discovered nine months before death. The first time any specific lesion was found in the lungs was two and a half months before death, whilst the oedema of the face and hands began six months before death. The only attack which might have been called asthmatic was the terminal one during which the patient died.

The morbid anatomical and histological changes, however, seem to be identical with cases described by a number of authors under various titles, notably, Meyer-Dörken (1934), Harkavy (1943), Bayley *et al.* (1945), Bergstrand (1946), Smith (1948), and others. The generalized lesions found in the above case may be said to fall under three headings—namely, intimal proliferation, necrosis, and thrombosis of small vessels; focal necroses; and tissue oedema and eosinophilia.

The most striking lesions were the vascular changes in small arteries and the eosinophilia, which was constant in the peripheral blood throughout the course of the disease and was such a pronounced feature of the histological picture. For this reason in discussing this case I have decided to use the non-specific term diffuse eosinophilic arteritis rather than attempt to make any particular diagnosis.

In studying this case it is at once apparent that there are certain similarities to a number of other conditions. Thus the vascular changes are almost identical with those seen in polyarteritis nodosa. The focal necroses which have not been described in this latter condition are common, however, in other conditions in which hypersensitivity plays a part, as, for instance, after the use of sulphonamide drugs (Lederer and Rosenblatt, 1942). In this connexion it is interesting to note that, despite the patient's pyrexia, no drug of this group was at any time administered. The lung fibrosis is reminiscent of rheumatic pneumonia, and the eosinophilia and oedema are common findings of allergic phenomena.

It is difficult to escape the conclusion that the changes described both in this case and in the literature represent not a particular disease syndrome but a non-specific tissue reaction of allergic type which may vary from the benign lesions of the so-called Loeffler's syndrome (von Meyenberg, 1942) to lethal conditions such as the one described here. Examples of intermediate pathology may be quoted from the numerous varieties and gradations of polyarteritis nodosa.

In the cases of rheumatism, hypersensitivity to sulphonamide drugs, and nephritis many data exist concerning the exact precipitating factors, whereas in conditions such as polyarteritis nodosa no such evidence is yet forthcoming.

The above record is therefore presented as one of violent allergic reaction to some unknown precipitating stimulus, with widespread lesions of a type seen in a number of other conditions, and which it is perhaps justifiable to regard as representing part of the general pathology of the allergic state.

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OBSERVATIONS ON SYMPTOMATOLOGY AND TREATMENT OF CERVICAL EROSION

BY

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The large number of cases of cervical erosion seen in this out-patient department has focused attention on that condition, which was present in 36% of all the cases seen there from April, 1947, to June, 1949—some 870 cases of erosion in all. This prevalence may be due in part to the lack of post-natal care in the surrounding districts, though Fulkerson (1926), in his study of all cases attending the gynaecological department of the Cornell University Medical School, found that the incidence of cervicitis with erosion was 33%.

All patients were subjected to speculum examination of the cervix, and diagnosis was by direct observation either of an erosion or, in a few cases, of Nabothian follicles associated with a mucopurulent discharge from the external os.

The diversity of symptoms presented when cervical erosion was the only lesion found became evident. These symptoms have been analysed in 128 cases out of the 870 treated. The remaining cases of erosion have been set aside because they were associated with other conditions which clouded the issue, or because follow-up was impossible. In all other respects these 128 cases are unselected.

The symptoms of which the patients complained, and the numbers cured by healing of the erosion, are presented. The patients were examined six weeks after treatment, and were followed up for a period of from six months to two years, in order to observe the permanency of cure and to eliminate any purely psychological effect of treatment.

The treatment employed was thermo-cauterization of the cervix, using the electric cautery. The results of treatment in the 128 cases are analysed, and, in addition, the complications which occurred after cauterization in the 870 cases treated are reviewed.

Symptomatology

The symptoms observed in the 128 patients (summarized in Table I) were, in order of frequency, as follows.

Leucorrhoea.—As is generally recognized (Young, 1930; and others), the cardinal symptom was leucorrhoea. The discharge was typically not more than moderately profuse, mucoid, and white or yellowish in colour. No fewer than 102 (80%) complained of leucorrhoea, and 92 (90%) of these were cured as a result of treatment. Of the remainder four were found to have persistent or recurrent erosion,

GEORGE LUMB: DIFFUSE EOSINOPHILIC ARTERITIS

FIG. 1.—Radiograph of chest taken on June 20, 1949.

FIG. 2.—Lung. Fibrosis, giant-cell formation, and eosinophilic infiltration.

FIG. 3.—Lung. Necrosis and eosinophilic infiltration of the wall of a vessel. ($\times 150$.)

FIG. 5.—Kidney. Necrosis of convoluted tubules, capillary thrombosis in a glomerulus, and eosinophilic infiltration of the interstitial tissue. ($\times 150$.)

FIG. 4.—Heart. High-power view of eosinophils. ($\times 240$.)

L. J. MAHONEY AND H. J. BARRIE: ADRENAL INSUFFICIENCY AFTER SURAMIN

FIG. 1.—Adrenal. Vacuolation of zona fasciculata and haemorrhagic necrosis of zona reticularis. H. and E. ($\times 45$.)

FIG. 2.—Adrenal. Vacuolar degeneration of zona fasciculata with isolated islands of autolysing cells. H. and E. ($\times 115$.)