mechanism of increasing cortico-adrenal function after damage is in some way deficient, or that damage may be so severe that the potentialities of the mechanism of increasing cortico-adrenal function of any animal or human being are exceeded.

It would seem therefore that the administration of adrenal cortical substances pre- and post-operatively and in conditions of damage such as infection, burns and other forms of trauma may be of clinical importance. This however, has not yet been definitely established.

SUMMARY

Adrenal cortical extract and desoxycorticosterone acetate given together both before and after trauma without other therapy reduce the mortality from experimental shock after intestinal manipulation in normal (non-adrenalectomized) rabbits. The difference in mortality between the treated and control animals is highly significant, statistically (P = less than 0.01).

Desoxycorticosterone acetate alone given before trauma is not effective for the difference in mortality between this group and the controls is not statistically significant (P = greater than 0.05).

Dr. Stephen Karady was associated with the early part of this work. The expenses of part of this investigation was defrayed by a grant from the National Research Council of Canada.

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BOECK'S DISEASE (SARCOID): ITS CLINICAL GROUPS AND DIAGNOSIS*

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THE extensive literature which is accumulating on Boeck's disease, particularly in Europe, is evidence of the growing importance with which it is coming to be regarded.[1 to 12] In 1899 Boeck[13] published his first description of what he considered a new skin lesion, calling it at the time "multiple benign sarkoid". Since then an integration of separately described symptom-complexes has been taking place to make up the disease picture as we now know it. Each of these syndromes had been heretofore regarded as a separate rare disease by the particular specialist in whose field it appeared to lie. The integration was accomplished through recognition of a common histological basis, and by the discovery that frequently the characteristic signs of two or more of these syndromes appeared in the same patient. Most observers now seem inclined to regard it as a specific disease, possibly caused by a virus. Schaumann[46] was apparently the first[26] to recognize it as a generalized disease with widespread incidence throughout the body; he emphasized its lymphatic distribution and renamed it "benign lymphogranuloma". Although uncommon, it is not rare, and we have during the past year observed several cases at the Royal Victoria Hospital. While essentially a disease of young adults, it affects all age-groups from infancy[15] to middle life; it is on the whole relatively benign.

PATHOLOGY

A very short description of the pathological appearances will suffice. Grossly, the lesions may be found in almost any tissue, as tumours in size varying between a millet seed and a walnut, with fairly sharply-defined borders; or else diffuse infiltration may cause general enlargement of

* Read at the seventieth annual meeting of the Canadian Medical Association, Section of Medicine, Montreal, June 21, 1939.

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such organs as the liver and spleen. Histologically,[6, 16] the lesions are seen to be granulomata, made up of nests of epithelioid cells and a few giant cells. They resemble tubercles in many respects, the chief point of difference being the absence of caseation.

Infiltrations have been found at necropsy in practically every organ and tissue of the body. Any one individual will, however, rarely show this state because of the tendency for the disease to heal in one region while remaining or becoming active in another. The sarcoid lesion may undergo complete replacement by fibrous tissue so that there remains histologically no indication of the character of the preceding granuloma. The fact that the chief incidence falls on different parts of the body at different times explains the frequent appearance of conventional groups of symptoms which may be replaced later by entirely different groups. Whenever the disease is widespread throughout the body, bizarre pictures may be encountered; on the other hand, the clinical picture may be extremely simple, as in one of our cases where the only finding was a single small nodule of the skin.

CLINICAL APPEARANCES

Mrs. R., aged 38, was admitted to the Royal Victoria Hospital on May 4, 1933. For three years she had had slight morning cough and expectoration, and for three months loss of energy. A month before admission she began to have conjunctival irritation and some discharge. Shortly after this she complained of anorexia, night sweats, a heavy headache, slight deafness, and such hypersomnia that she could with difficulty keep awake at all. The conjunctivitis persisted.

On admission her temperature was 102°. Three weeks later she developed left facial palsy and, a month after this, double iridocyclitis. The facial palsy was now passing off, but in a few days both parotid glands were found enlarged and slightly tender. During her four months in hospital she ran a low-grade fever which gradually settled down to 99°. Vague joint pains and neuralgias were almost constantly present, her cough persisted, and for three weeks in June moist râles were present at one base. Repeated sputum examinations were negative for tubercle bacilli, as were guinea-pig inoculations. X-ray examination of the chest showed an enlarged gland at the right hilum (see Fig. 5). The white blood count was normal. Macular and herpetiform rashes appeared from time to time. At the date of her discharge the iridocyclitis had cleared up, but the parotid swelling persisted for a year.

For the next four years she felt fairly well, but continued to have a cough, attributed to dryness of the mouth, which bothered her considerably. Several times the parotids swelled for short periods. Early in 1938 she again began to feel tired. Physical examination in the fall was negative. The leucocyte count and sedimentation rates were normal. There was slight anæmia, and 8 per cent eosinophilia. The sputum was again negative on guinea-pig inoculation. The Mantoux test was negative for 0.01 mg., slightly positive for 0.1 mg. O.T. X-ray films of the hands and of the chest showed pictures which will later be described as characteristic of Boeck's disease. The left breast, which was removed for suspected malignancy, showed an extraordinary amount of fibrosis.

Various generalized symptoms are encountered during the course of the disease, more frequently as prodromata, and especially in the symptom-group known as uveoparotitis. In the case described tiredness, loss of strength, anorexia, night sweats, headache, fever, and vague joint pains were noted. Nausea, vomiting, and diarrhœa are occasionally found. Œdema of the ankles has been reported by several authors and has been ascribed to infiltrations lying deep in the skin.[12]

Skin.—The cutaneous lesions were the first to be described. They are not found in every case, but when present are usually the most prominent feature. Schaumann[14] classifies them as, first, specific dermatoses, including tumours and erythrodermia; and second, lymphadenoides, or non-specific eruptions.

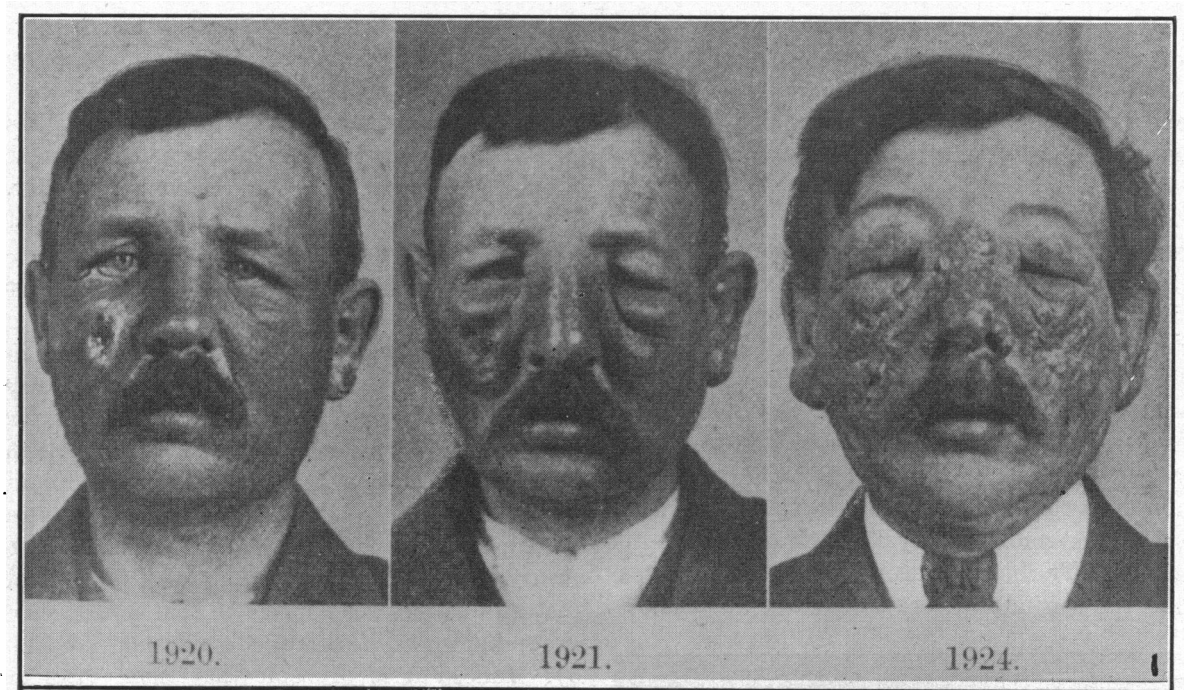
Of the tumours which we now recognize as sarcoid, the first to have been described was Besnier's[17] lupus pernio, so named because of its superficial resemblance to chilblain. It appears as extensive diffuse bluish-red infiltrations of the cheeks, nose, and ears, which are rather sharply defined and symmetrical (see Fig. 1). The backs of the hands may be affected, and also the fingers at the same time as are the underlying phalangeal bones.

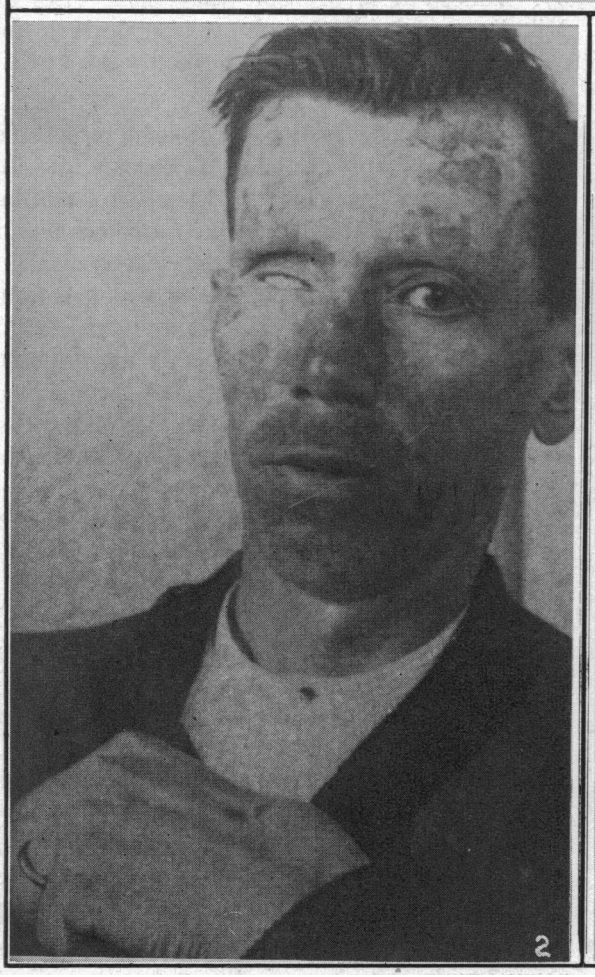
Boeck's cutaneous sarcoid is very similar to lupus pernio except for the asymmetrical distribution. It occurs chiefly on the face and limbs, and the nodules may reach the size of a small walnut. There is a tendency to central flattening during involution, but no ulceration (Fig. 1). This same tendency to flattening is seen in the so-called disseminated miliary lupoid. Here the lesions are small brownish-yellow smooth papules with a pink centre, which may remain isolated or spread out in patches or rings (see Fig. 2). They are usually asymmetrically distributed over the face, scalp, neck and shoulders, and the outer surfaces of the limbs.

Schaumann[14] has described a specific erythrodermia involving almost exclusively the legs and thighs in large irregularly-shaped scaly pinkish areas which are not raised above the skin surface and are not itchy (see Fig. 4).

All of these eruptions may be present at the same time, and one frequently undergoes gradual transformation into another. The specific skin lesions usually persist for years and then resolve completely.

Of the non-specific eruptions prurigo has been known to be present continuously for twenty





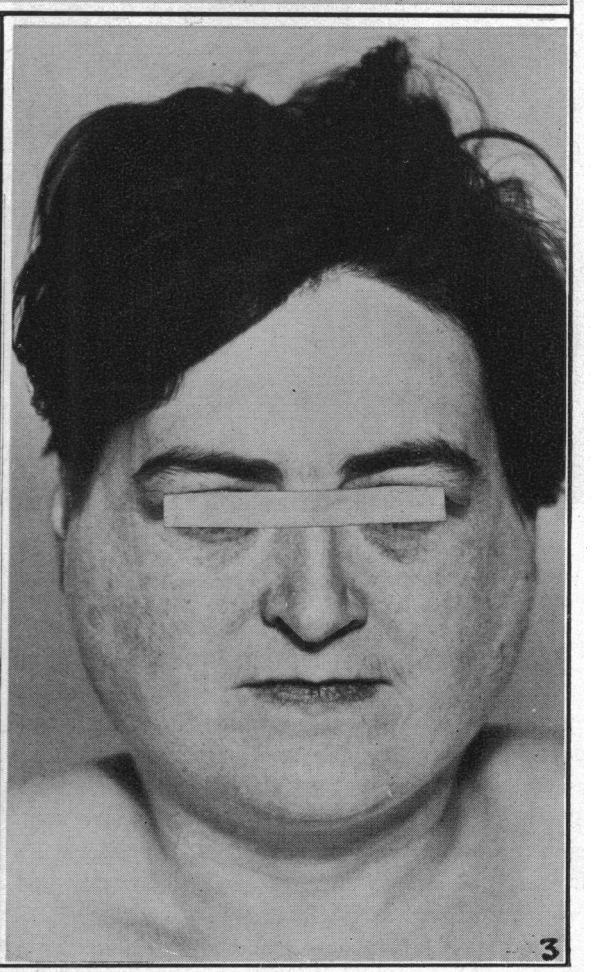


Fig. 1.—Serial photographs to show transformation of cutaneous sarcoid to lupus pernio (after Schaumann). (Courtesy of H. K. Lewis & Co., London). **Fig. 2.**—Disseminated miliary lupoid of the circinoid type. Note also destruction of the eyeball, and involvement of tendon on left hand (after Schaumann). (Courtesy of H. K. Lewis & Co., London). **Fig. 3.**—Involvement of lachrymal, parotid, and submaxillary glands (Mikulicz's syndrome). This patient also had facial palsy, and later developed iridocyclitis. (Kindness of Dr. Colin Russel).

years.[14] Urticaria is sometimes seen, and also various erythemata. Erythema nodosum sometimes appears in the early stages of the disease, and has been observed to persist for ten years before the development of the uveoparotid syndrome.[18]

Mucous membranes.—Invasion of the mucous membrane of the upper respiratory tract sometimes occurs early, bringing the patient first to the oto-laryngologist. Nasal obstruction may result,[19] while at the same time involvement of the nasal cartilage leads to its absorption, with consequent falling-in of the nose itself.[14, 20] Stridor may be caused by similar destruction of laryngeal cartilage, and infiltration of this region and of the neighbouring bronchial mucous membrane has ended fatally through œdema of the glottis.[21] The buccal mucous membrane, the gums, and the conjunctiva are frequently invaded.

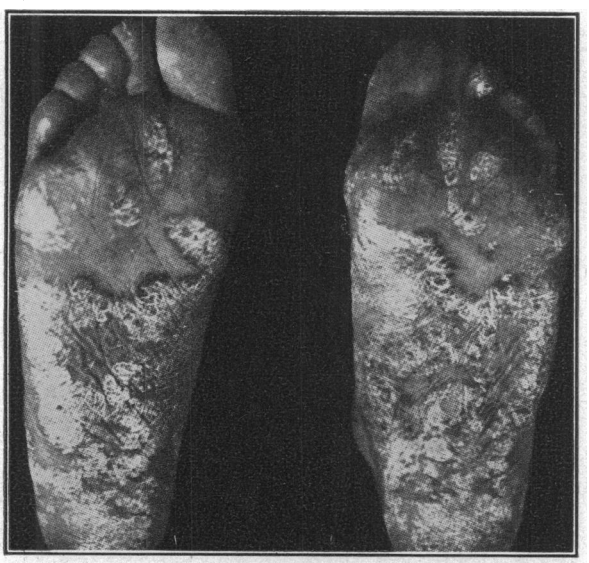


Fig. 4.—"Erythrodermie sarcoidique"—the lesions in this case were also present on the lower legs and thighs (after Pautrier). (Courtesy of Masson et Cie., Paris).

Uveoparotitis.—Perhaps the most interesting syndrome is that one originally described by the ophthalmologist Heerfordt[22] as subchronic uveoparotid fever. It may still be considered rare, as Pautrier[23] was in 1938 able to collect less than 120 published cases. It is probably more common however than a survey of the literature would lead one to suppose, and the writer last year saw three cases within a few weeks.

Prodromal symptoms are often present for a considerable period before the appearance of the characteristic iridocyclitis and parotid enlargement. These two conditions do not as a rule develop together, but each precedes the other in about an equal number of cases,[24] sometimes by several weeks.

Active inflammation of the uveal tract is present for several weeks; it is serious in that it nearly always leaves some residual disability. Practically all other parts of the eye may at different times be involved, this sometimes resulting in complete destruction of the eyeball (see Fig. 2).

The parotid swelling resembles that seen in mumps, is not necessarily bilateral, is usually painless, and persists for months, sometimes for many years. Dryness of the mouth, due to loss of salivary secretion, may give rise to cough and to considerable dysphagia. When parotitis is accompanied by coincident enlargement of the lachrymal and submaxillary glands, the clinical picture qualifies for a place in that non-specific group known as the Mikulicz syndrome (see Fig. 3). Indeed it is probable that those cases of Mikulicz syndrome heretofore ascribed to so-called pseudoleukæmia have in reality been uveoparotitis.[7]

Central nervous system.—For some reason the neurological manifestations of Boeck's disease are seen almost exclusively in the uveoparotitis group. Levin,[25] in his excellent review, found involvement of the central nervous system in about half the published cases of uveoparotitis, and by far the most common finding was facial palsy. Its high incidence is partly attributable to pressure on the nerve as it passes through the parotid swelling, but that a true neuritis also occurs is evident from the fact that the chorda tympani is sometimes involved, and that (as in the case here reported) facial paralysis often appears before either uveitis or parotid enlargement. Other cranial nerves may also be implicated; various grades of optic neuritis and papillœdema are seen, also strabismus, and ptosis; facial anæsthesia from unilateral involvement of the trigeminal, bilateral deafness, and impairment of the labyrinth have been described. The only cranial nerves that seem to escape are the spinal accessory and hypoglossal.[7] Widespread neuritis of spinal nerves may result in muscle tenderness, weakness and atrophy, paræsthesias and impairment of sensibility, foot and wrist drop. Boeck himself remarked that the nerve trunks may be so infiltrated with sarcoid lesions that the signs closely resemble those seen in leprosy.

Pyramidal tract involvement is rare but spastic paraplegia has been described. Infiltration of the base of the brain has resulted in the frequent appearance of hypersomnia as an early symptom. Indeed Waldenström[7] has suggested that the many cases of encephalitis lethargica in which parotid swelling was observed were actually cases of uveoparotitis. True diabetes insipidus has several times been reported.[7, 26]

Invasion of the lepto- and pachy-meninges has been found at necropsy, although clinical signs of meningeal irritation are rare. The cerebro-

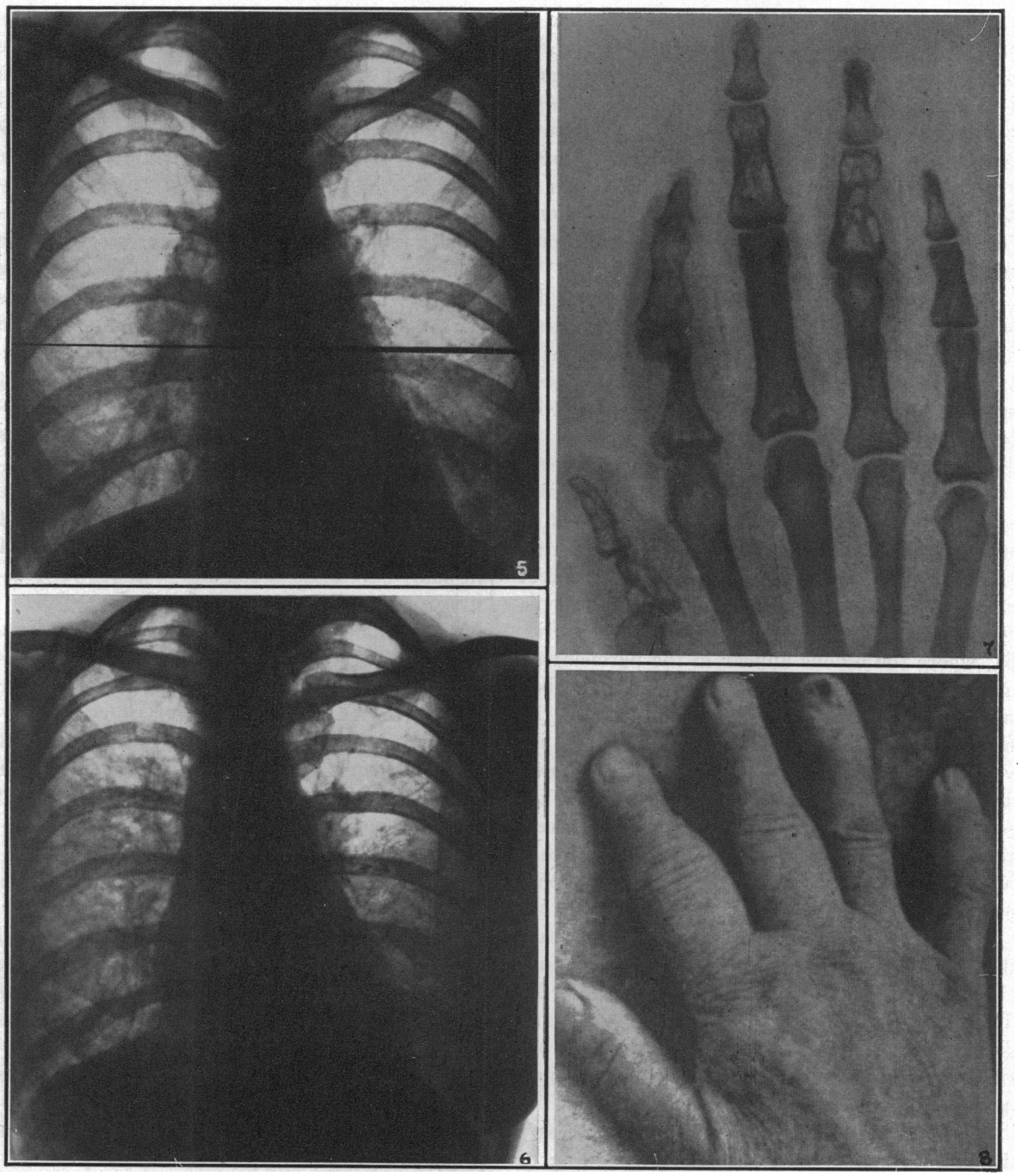


Fig. 5—Roentgenograms of chest to show right hilar lymphadenopathy. **Fig. 6.**—Same case as in Fig. 5, five years later. Note that hilar enlargement is not now prominent, and that "marbling" of the middle and lower zones has occurred, the apices remaining relatively free. **Fig. 7.**—Roentgenogram of hand, showing multiple lacunæ in phalanges. This is an advanced case, and pathological fracture of the index finger has occurred (after Nielsen). (Courtesy of Masson et Cie., Paris). **Fig. 8.**—Hand showing distortion of fingers, closely resembling appearance of rheumatoid arthritis. Note erosion of one nail-bed (after Whittle). (Courtesy of H. K. Lewis & Co., London).

spinal fluid may show a slight lymphocytosis and excess of protein.[25]

Lungs.—Involvement of the lungs is common and may occasionally be the only recognizable feature of the disease. Nevertheless, pulmonary symptoms are usually so insignificant that attention is not attracted to them. If the patient comes complaining of tiredness, cough, and a little fever, early tuberculosis is suggested. Loss of weight and night-sweats are not common, and it is doubtful whether hæmoptysis has ever been caused by sarcoidosis.[27] Infiltration of the pleura occurs, but hydrothorax has only rarely been reported. On the whole, clinical examination of the lungs is usually negative, and one may therefore be greatly surprised by the extensive pulmonary involvement revealed by x-rays.

These roentgenological changes have been described as of three main types.[28] The commonest, constituting perhaps two-thirds of those with lung involvement, is that group with pronounced enlargement of the hilar lymph glands (see Fig. 5). The picture is like that often seen in Hodgkin's disease, with which it is apt to be confused. Although this enlargement is usually asymptomatic, it may be so pronounced as to interfere with breathing through pressure on the bronchi, and in one case was the direct cause of death by suffocation.[29] The second type is the so-called "marbling" of the lung (see Fig. 6), where the lesions appear in the parenchyma as more or less nodular small circumscribed masses tending to radiate from the hila into the middle zones. The apices are almost never involved. The third form, about half as common as marbling, is that in which small nodules are seeded throughout both lungs. It is almost certain that in this group belong many cases that have been reported from time to time as chronic or healed miliary tuberculosis.[30]

The hilar type of involvement usually appears first, and may later be replaced by or merge into the other forms (as in the case here reported). The pulmonary lesions tend to remain stationary for months or years during which time the patient may be free from symptoms, or they may quickly resolve completely.

For diagnosing pulmonary lesions as Boeck's disease, the most important point, according to Dressler,[28] is the pronounced discrepancy between the extensive lung findings by x-ray on the one hand, and the well-being of the patient and the slight or absent acoustic findings on the other.

Bones.—Jüngling[31] at first regarded the bone lesions as tuberculous and called them "osteitis tuberculosa multiplex cystica"; this name is synonymous with the terms "pseudocystic polydactylitis", and "nodular polyosteitis of the extremities".[39] The most prominent features are the small, regular, cyst-like areas in the bones which by x-ray appear as rarefactions (see Fig. 7). They are found in the phalanges of the hands and feet, occasionally in the metacarpals and metatarsals, and very rarely in the distal end of the radius. Frequent x-ray search has failed to show them with certainty in other bones,[32] even though we know from necropsy evidence that the rest of the bony framework is usually involved. The cyst-like areas are in the cancellous tissue, the phalangeal epiphyses being the site of election. In more advanced stages there may be "complete disorganization of the whole structure of the phalanx with loss of distinction between the cancellous bone of the shaft and medulla, and replacement by uniformly distributed coarse trabeculation".[33] The result is an appearance of increased density and widening of the shaft with fraying of the edge of the shadow, suggesting a diffuse breaking down of the periosteal surface. In extreme cases most of the shaft may be absorbed and pathological fractures occur. Sequestra do not form.

In common with other sarcoid lesions the bone cysts may regress and undergo fibrous tissue replacement. When this occurs the x-ray picture may be normal or may perhaps show replacement of the cyst-like areas by small areas of increased density.[32] It is possible on the other hand for the bones of the extremities to be filled with granuloma without there being any roentgenological abnormality.[14]

The bony changes may never of themselves give rise to symptoms. Occasionally, however, the fingers are greatly distorted (see Fig. 8), and if lesions are not manifest in other parts of the body difficulties in diagnosis will arise. The spindle-shaped and nodular deformities may be like those seen in rheumatoid arthritis, osteoarthritis, or gout. The picture must also be differentiated from that of sarcoma and osteomyelitis, and there is some resemblance to the lesions of congenital syphilis and leprosy.

Lupus pernio involving the finger often accompanies the bony changes, and the digit then becomes painful, bluish-red, and swollen. There may be thinning and partial destruction of the nail, as well as erosion (see Fig. 8) and vegeta-

tion of the nail-bed.[14] The resemblance to osteomyelitis has mistakenly led to amputation.[32, 34]

Lymph nodes.—Enlargement of the peripheral lymph nodes may be the earliest, and the only, objective sign of the disease; it probably occurs at some stage in most cases. The glands are discrete, rubbery and painless, and the enlargement, which may reach the size of a walnut, is either generalized or localized in one or more groups. There is no regional relationship to any skin lesion that may be present.[9] These lymph node enlargements which are now recognized as Boeck's disease had previously been described as a hyperplastic sclerosing type of tuberculosis[35]; the so-called Sternberg tuberculosis probably also belongs here.

Heart.—Clinical evidence of cardiac involvement is not common, but at necropsy infiltrations of epithelioid cells in the pericardium and epicardium are often seen, not so frequently in the myocardium. Electrocardiographic changes include inversion of T waves[5, 6] and bundle-branch block.[5] Infrequently dyspnœa and cyanosis occur, some of which is apparently due to pulmonary disease.[6, 12] Death from heart failure has been observed, the myocardium at necropsy showing widespread granulomatous infiltration.[47]

Abdominal organs.—Of the abdominal signs splenomegaly is the most prominent. Slight enlargement of the spleen is common, and occasionally the increase in size may be great. The fact that splenectomy has been performed because of a mistaken diagnosis of Banti's disease[36] emphasizes the statement that Boeck's disease should always be considered in the differential diagnosis of chronic splenomegaly. The liver may be enlarged without giving rise to symptoms, although jaundice has been reported, and the Takata-Ara test has been positive.[5] Infiltration of the stomach and intestines is frequently found at necrospy, but symptoms directly referable to this are not common. It is interesting to mention here that in 1933 Homans and Hass[37] drew attention to the similarity of the histological pictures in Boeck's disease and in Crohn's regional ileitis, and that later Williams and Nickerson[38] obtained skin reactions in Boeck's disease from emulsions made from the lesions of regional ileitis. The two conditions seem closely related if not identical.

Glands.—Sarcoid tissue has been found at necropsy in most glandular structures, endocrine and otherwise. Invasion of the sex glands, including the prostate, is usually asymptomatic. Amenorrhœa has been observed, but was probably an expression of the general infection rather than of direct replacement of ovarian tissue. Thyroid infiltration has been seen at necropsy,[21] and has been suggested as a cause of myxœdema.[40] It has already been stated that diabetes insipidus has resulted from disease of the pituitary and the neighbouring parts of the brain. Mastitis is seen,[41] and in our own case the intense fibrosis found in the breast suggested that this had replaced a previous widespread sarcoid infiltration.

Blood.—Examination of the blood usually shows no pronounced abnormality. Slight hypochromic anæmia may be found, and the total white cell count is usually low, with a relative monocytosis. The eosinophile count is often slightly increased, and occasionally it may reach a very high figure, up to 80 per cent.[9] Hyperproteinæmia, due to increase in the serum globulin, has been reported.[5, 42*] The sedimentation rate may be normal or rapid.[7]

Kidneys.—Infiltration of the kidneys results in albuminuria as a frequent finding. Gross hæmaturia has been described, and there is sometimes impairment of the ability of the kidneys to concentrate the urine.[28] Tremendous renal enlargement has been seen at necropsy in a child, where the lung space was so seriously encroached upon by the elevated diaphragm as to cause death.[43]

Muscular system.—Although nodules have been found in skeletal muscles large enough to give rise to pseudo-hypertrophy or to cause surgical removal, involvement of the muscles seems to be rare.[44] The tendon sheaths may be infiltrated by large compact masses of epithelioid tissue, which have the appearance of subcutaneous tumours and cause tenosynovitis (see Fig. 3).

DIAGNOSIS

The diagnosis of Boeck's disease is occasionally obvious, as for instance when the complete picture of uveoparotitis is present. The significance of the isolated appearance of any of the signs is not so readily apparent, and the diagnosis is most often missed merely because the disease has not been considered as a possibility.

* A recent study of blood chemical changes in eleven cases confirms the finding of hyperglobulinæmia in active cases, along with elevation in the blood phosphatase level, and abnormal bilirubin excretion. These were taken to indicate impaired hepatic function (Harrell, G. T. and Fisher, S.: *J. Clin. Invest.*, 1939, **18**: 687).

The investigation of every suspected case should include radiological examination of the lungs and of the small bones of the hands and feet. In a good proportion of cases one or both of these will be positive.

The diagnosis is of course not difficult if surface lesions in the skin or lymph nodes, or even in the salivary glands, are available for biopsy. Splenic puncture, when this organ is enlarged, or sternal marrow puncture,[45] may reveal the characteristic histological picture. Another source of biopsy material has been indicated by Schaumann,[14] who pointed out that in his experience the tonsils were always involved. A small snipping under local anæsthesia is easily removed, and offers little inconvenience to the patient. It should be mentioned that the tonsils and peripheral lymph nodes may be thoroughly infiltrated by the disease without their being enlarged.[14] Also it should be remembered that replacement of epithelioid tissue by fibrous tissue may have occurred, so that inability to find the characteristic lesion in the section examined does not rule out the disease.

Williams and Nickerson[38] found that patients with Boeck's disease showed positive skin reactions to intradermal injections of emulsions of sarcoid tissue. This observation has not, to my knowledge, been repeated elsewhere, but seems worthy of further investigation.

Finally, reference must be made to the tuberculin test which in Boeck's disease shows a very high percentage of negative reactions. While it is known that in tuberculosis of the skin and in overwhelming pulmonary or miliary infections the Mantoux test may sometimes be negative, yet nearly all tuberculous disease gives a strongly positive reaction. As this is the one condition with which Boeck's disease is most apt to be confused, the Mantoux test assumes considerable importance. It may be stated that when a benign illness resembling tuberculosis is found, a weak or negative Mantoux reaction, in the absence of an extensive skin lesion, strongly suggests the diagnosis of Boeck's disease.

SUMMARY

The lesions of Boeck's disease may occur in every system of the body. It is therefore a disease of great importance to internal medicine. The unequal distribution in different organs and tissues results in a variety of syndromes, many of which were at first regarded as separate diseases. These are described, including uveoparotitis which is now generally accepted as a manifestation of Boeck's disease. The diagnosis is usually dependent on biopsy, but is frequently possible on clinical evidence alone.

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