Correspondence

Erysipeloid

SIR,—I was interested in the medical memorandum by Dr. R. A. Bush (October 29, p. 964) and I agree that most cases of erysipeloid are penicillin-sensitive, though during the past few months I have seen two cases which have dragged on for a month or so in spite of adequate doses. I do not agree with the statement that the disease is by no means common. I believe it is more correct to say that it is by no means commonly recognized. At a recent examination for a higher surgical diploma I found that only about one candidate in ten was familiar with the condition.

Erysipeloid is more common in certain localities, and is seasonal—June to October being the optimum months. The reddish-purple area on the hand, which follows a prick by a fish-scale or poultry bone, is very typical. Untreated, this intradermal, *not* subcutaneous, inflammation slowly extends over the adjacent palm or fingers and subsides in from four to six weeks, during which period disability results from itching and stiffness. Regional lymph nodes are occasionally enlarged, probably as a result of the initial skin puncture. I have never seen lymphangitis as described by Dr. Bush.

It is important that this disease should be recognized, both from the point of treatment and maltreatment. The majority of cases respond to penicillin but are resistant to sulphonamides, and surgery is contraindicated—in spite of which I have seen two cases which were incised as indolent whitlows. It is interesting to recall that erysipeloid was described as long ago as 1873 by Morrant Baker, who termed it erythema serpens, and it is now commonly referred to as "fish-handler's disease."—I am, etc.,

London, W.1. R. J. MCNEILL LOVE.

SIR,—Mr. Thomas G. Lowden has made several observations on erysipeloid (November 26, p. 1231) and I am prompted to add my comments on this interesting and often undiagnosed condition, since I treated a fairly large number of cases during a period of 18 months at the Royal Northern Hospital. I would agree that the incidence is in the region of 1% of septic hands seen, that the cases vary in severity, and that the diagnosis is of necessity made clinically, as incision is not required and thus no material is available for pathological study.

"Untreated" cases—i.e., those patients who have only attended for treatment after self-medication with poultices, etc., for several weeks—do not resolve readily in spite of penicillin, taking 2–3 weeks further to clear (i.e., as long as 5–6 weeks from the original date of onset). The condition sometimes spreads slowly on to the hand but is often surprisingly stationary. Although the majority of cases respond to penicillin (250,000 units b.d. for seven days), the response is often slow, and there are a number of cases that do not respond at all but tend to linger on for weeks like the "untreated" cases. These may have not come for treatment until the second week, but early cases too may react in this way. Sulphonamides appear to be entirely ineffective, as shown by cases referred by general practitioners who have tried sulphonamides without success, and by personal clinical trial.

The commonest cause appears to be a minor prick with a fish, meat, or rabbit bone, but pricks with a variety of sharp objects have been seen to cause this condition. I have also seen one case which followed a small burn of the finger, there being no history of any obvious source of contamination. The burn had healed before the patient attended hospital, so there was no opportunity of isolating the organism, but clinically the appearance was quite typical. For some reason the disease appears to have a seasonal incidence, and I have noticed a very definite increase in spring and autumn. I have been told that the disease has a definite topographical distribution, and I would be very interested to hear of its incidence in other parts of the country, in order that this contention may be proved or disproved.—I am, etc.,

London, N.12. CYRIL BORODA.

Lymphoid Hyperplasia and Rheumatoid Arthritis

SIR,—Professor Hans Selye (November 19, p. 1129) discusses possible reasons for the favourable modification of "formalin arthritis" by ACTH, "cortisone," the "alarm reaction," and the worsening effect of LAP and DCA. He suggests that the inhibitory action of ACTH and cortisone may be due to their antihistaminic or anti-hyaluronidase properties.

For some time past I have felt that the lymph-gland enlargement found in rheumatoid arthritis (and some cases of "fibrositis") might be an important factor in the production of the disease, rather than secondary to chronic inflammation in the joints as was formerly believed. Selye's paper on the general-adaptation syndrome[1] encouraged me to pursue this hypothesis, particularly as it lent some support for a basic psychogenic aetiology which I believe to be present in many cases. It is known that the synovial cavities of joints are at least in part drained by lymphatics,[2] and Indian ink injected into these spaces may be found very soon in the appropriate lymph nodes. Professor E. W. Walls and I[3] have recently worked on lymphograms obtained by putting "thorotrast" into the synovial cavities of cadavers and moving the joint concerned. This fills the lymph channels and outlines the nodes in a few minutes. My object has been to gain some idea of the normal, to compare with cases which have previously suffered from rheumatoid arthritis. The snags will be obvious, and the scope of the work has been limited to the cadaver owing to our unwillingness to put thorotrast into the joints of living subjects, though this has been done abroad.

Now the photographs (Fig. 7, p. 1133) and description of what happened to the lymph nodes in Professor Selye's experiments have prompted me to put forward tentatively an alternative mechanical theory—that the changes in the joints are due to lymphatic oedema caused by a partial obstruction by lymphoid hyperplasia. In the experiment referred to the rat's glands are small under the influence of ACTH, and the deep staining shows that absorption of ink was rapid. When the glands were hyperplastic from LAP little ink had reached them. Applying the hypothesis to the aetiology of "formalin arthritis," formalin solution is irritant and is probably dealt with subcutaneously by the lymphatic system. It may well be that the system becomes overloaded or damaged, thereby rendering the mild and uncertain "arthritis-producing" property of DCA and LAP more clear-cut. The intensity of local skin reaction could also depend on the speed of removal of formalin.

Finally, Selye's unexpected finding in his rat experiments of main involvement of the ankle-joint, though it was some way from the site of injection, is also capable of explanation by mechanical block of lymphatics. Clinical observation, too, shows that it is the peripheral joints that are mainly affected in rheumatoid arthritis, and perhaps those in which the periarticular tissue is lax are affected most—e.g., proximal interphalangeal points, as opposed to distal interphalangeal. It should not be difficult, given facilities for animal work, to show whether lymphoid hyperplasia can in itself impede lymph drainage.—I am, etc.,

Ipswich, Suffolk. J. W. PAULLEY.

REFERENCES

1 *J. clin. Endocrinol.*, 1946, **6**, 117.
2 Davies, D. V., *J. Anat., Lond.*, 1946, **80**, 21.
3 Ibid., in the press.

Autotransplantation of Joint Capsule

SIR,—With reference to your annotation (November 26, p. 1221) on this subject, it may be of interest to note that in 1938 we trephined a small controlled series of cases of rheumatoid disease and osteoarthritis at the Royal National Hospital for Rheumatic Diseases, Bath.[1] In the rheumatoid cases no significant improvement was noted, but in those suffering from osteoarthritis there was some relief from the bone pain certain patients experience at night. We felt that some reduction of tension might be the explanation of this improvement in symptoms.—I am, etc.,

Bath. G. D. KERSLEY.

REFERENCE

1 *Reports on Rheumatic Diseases*, 1938, **4**, 68.