Sir Harold Gillies

SIR,—May I add a postscript to the delightful and fully deserved eulogy of the late Sir Harold Gillies by your correspondent Mr. Patrick Clarkson (10 September, p. 641).

I was present in Boulogne one day in the autumn of 1915 with a Captain Harry Grey, a Canadian E.N.T. surgeon, when the meeting I am about to describe took place. He and I, as officers of the same motor ambulance unit near Poperinghe, had often discussed the horrors of facial wounds; and he, Harry Grey, was insistent that these and other injuries should be the aim of a special brand of surgery, and that it should be developed by the E.N.T. surgeons. Gillies, it will be remembered, was up to the outbreak of the first world war specializing in the E.N.T. field.

Walking that day along the front at Boulogne I saw a curious-looking stooping figure creeping rather than walking towards us, dressed in a curious uniform that was neither Army nor Navy. "Giles" it was. He was even more depressed than he looked; full to overflowing with complaints. Here he was at the beautifully equipped and staffed Red Cross hospital donated by the Duchess of Westminster at nearby Wimereux—they were rotting away, no worthwhile cases ever being sent to them—victims of the R.A.M.C. jealousy, no real officer status, and so on and on. Here then was Harry Grey's hungry soil, ready to lap up and respond to his eloquent and enthusiastic advocacy of specialization in facial surgery.

Some months later I visited my old friend "Giles," now happily and actively installed at the Cambridge Hospital in Aldershot. I think this chance meeting between the New Zealander and the Canadian must have been the spark that set alight the genius of this unique character.—I am, etc.,

Chipping Campden, Glos. C. B. HEALD.

Clofibrate in Lipoid Dermato-arthritis

SIR,—I have recently seen a case of lipoid dermato-arthritis (reticulo-histiocytosis of skin and joints) in which there was a favourable result following treatment with the cholesterol-lowering agent Atromid-S (clofibrate).

A 40-year-old man had complained of almost constant pain in the forearms and at the backs of the wrists, and recently had observed pain and swelling of the medial metacarpo-phalangeal joints of the hands. His mother had diabetes mellitus, but there was no family history of xanthomatosis, arthritis of other metabolic origin, or rheumatic disorder.

Examination revealed arthritis of the right third metacarpo-phalangeal joint and tenosynovitis of the abductor pollicis longus/extensor pollicis brevis tendon sheaths at both wrists. On the extensor aspects of the elbows there were lobulated, soft, rubbery masses, having the appearance of xanthoma tuberosum. No other abnormality was found.

A blood count was normal, the haemoglobin being 98% and E.S.R. 10 mm. in one hour (Westergren). The serum was cloudy, owing to lipaemia, and the cholesterol raised at 390 mg./100 ml. Serum uric acid was 6.6 mg./100 ml., serum proteins and electrophoresis normal, blood glucose 2 hours p.m. 75 mg./100 ml., latex and sheep cell agglutination tests for rheumatoid arthritis negative. *X*-ray of the hands and feet showed patchy osteoporosis of the phalanges. A biopsy of one of the skin lesions on the elbows showed circumscribed fibroblastic proliferation interspersed with lipoid-laden histiocytes, and fine crystalline clefts, which proved to be composed of lipid and cholesterol.

A diagnosis of lipoid dermato-arthritis was made and he was given Atromid-S (clofibrate) 1.5 g. daily. He was seen two months later and reported that he had become symptom-free a few weeks after starting the drug, and that the wrist pains had recommenced three weeks after he had finished his supply of tablets. The serum cholesterol had fallen to 220 mg./100 ml., but the skin lesions had not appreciably diminished in size.

Clofibrate is known to be effective in lowering serum lipid levels in most patients where these are raised within two weeks of starting the drug. Several reports of the effectiveness of clofibrate in hyperlipidaemia with xanthomatosis have appeared[1-3] but a search of the literature has not revealed a report describing the effect of clofibrate in lipoid dermato-arthritis,[4] which is characterized by the occurrence of multiple nodules composed of lipoid-containing histiocytes in the tendons and bone-ends, together with similar lesions in the dermis. While the diagnosis has not as yet been proved by joint or tendon biopsy in this case, the clinical evidence of tenosynovitis and arthritis with hyperlipidaemia and hypercholesterolaemia, and the absence of other causes for arthritis, are very suggestive that early bone involvement and tendon lesions were indeed present.

It has been considered important to report this case in view of the fact that subsequent invasion and destruction of involved bone by the lipoid-containing lesions may result in severe destructive arthropathy, a form of arthritis mutilans.[4-6] In the experience of these workers therapy with low fat diets has not been successful, though corticosteroids may cause temporary regression of skin nodules. It seems probable that early recognition of this condition and prompt treatment with lipid-lowering agents might avert destructive bone changes.—I am, etc.,

D. N. GOLDING.
Princess Alexandra Hospital, Harlow, Essex.

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Aberdeen Typhoid Epidemic

SIR,—May I comment on two minor points in your admirable summary of the Aberdeen typhoid epidemic (10 September, p. 601)? The statement "The classical picture with rose spots and splenomegaly was seldom seen" is true only of the earlier admissions with presenting symptoms of gastro-enteritis. Rose spots were observed in 234 (46%) and splenomegaly in 196 (39%) of the patients.

There was no particular reluctance to use corticosteroids where the indication seemed sound. Five critically ill patients received intravenous hydrocortisone with good effect. With one doubtful exception this treatment was withheld from no patient who could conceivably have benefited from it. Of the three deaths, one was from massive and unheralded pulmonary embolus and two were in old people with severe cerebrovascular and cardiac disease.—I am, etc.,

City Hospital, Aberdeen. W. WALKER.

Treatment Before Diagnosis

SIR,—Dr. Leon Walkden (Points from Letters, 3 September, p. 591) writes that treating suspected *Trichomonas vaginalis* infestation before diagnosis is reasonable. Perhaps; but not inevitable. Put a slide and an ampoule of normal saline in the hand-washing water before examining the patient. After the examination mix a little of the discharge from the speculum with the warm saline on the warm slide, and examine without coverglass under the 1/6-in. objective. The organism can be seen wriggling before the patient has dressed, if she is a little slow. However, as Dr. Walkden says, confirming one diagnosis does not exclude others.—I am, etc.,

Edinburgh 10. J. M. FORRESTER.

Placental Localization by Thermography

SIR,—In his paper "Placental Localization by Thermography" (25 June, p. 1571) Dr. K. G. Millar concludes that thermography cannot be relied upon to predict the placental site accurately. This prompts me to answer in defence of a research technique that being in its infancy is vulnerable to theoretical mistakes. Dr. Millar was unable to detect placentae on the posterior wall of the uterus, and obtained no better results with anteriorly placed placentae. Other workers have detected placentae equally well irrespective of the depth of the placenta from the surface scanned. This, I think, is the key to the problem. When a breast is scanned, the thermograph records surface changes of temperature caused by local abnormal metabolism or local differentiation of vascular flow, and independent of capillary tone. This is one distinct and recognized bodily process. I do not believe that this process contributes much to placental localization or that the thermographs of the pregnant abdomen are merely a simple matter of heat flow.

It has been suggested that a lumbar disc lesion may be demonstrated thermographically. This clearly cannot be a simple matter of heat flow. It becomes necessary to postulate that a separate process occurs, in that surface changes in a chilled subject are due to local vasoconstrictive response, and may be indicative of interruption of the reflex arc.

It is therefore insufficient merely to place a cold damp towel on the abdominal wall. One must ensure a vasoconstrictive response; "goose pimples" are a fair indication of this. This vasoconstriction removes many problems as Millar presents them:

1. Ambient temperature becomes less important. Perfectly adequate thermographs may be obtained at an ambient temperature of 25° C. in my experience, and pictures can