Complications of Lymphography

SIR,—Dr. R. J. A. Jackson (14 May, p. 1203) has written a useful article on complications of lymphography, but from my experience with over 120 cases comprising diagnostic, colour, and therapeutic lymphographies, I feel that the dangers have been exaggerated and inadequately discussed.

Complications in my series included transient pyrexia, which has been reported by various workers, delayed wound healing—most of the cases belonging to the group of lymphomas—and one instance of haemoptysis in a patient with thrombophlebitis, which occurred on the fifth day of lymphography unaccompanied by any other symptoms. Whether the latter was a result of lymphography or his original condition could not be clearly understood, as the post-lymphographic and fifth-day chest films revealed no evidence of fat embolization.

Dr. Jackson concluded his article with a grim warning about the quantity of contrast medium to be used in this investigation, but I firmly believe that the rate of injection has a much more important bearing on the causation of pulmonary complications than the amount of injected contrast medium. About 9 ml. has been estimated to be required to fill the nodes without overflowing the thoracic duct. Invariably this limit is exceeded, and amounts of 18 to 24 ml. of media are injected without any complications, while in some cases with less than 5 ml. of radioactive Lipiodol there has been high lung uptake when injected at a faster rate. Leakage of oily medium into the venous channels results in three ways: (1) overflow from thoracic duct; (2) by opening up lymphatic-venous communications with raised intra-lymphatic or intrasinusoidal pressure, and, finally, (3) by accidental injection into the venules, which at times resemble lymphatics. This is an easily avoidable mistake if one is careful and not over-confident.

Discussion is incomplete without the mention of well-known contraindications like iodine sensitivity, and pre-existing real or hepatic functional impairment.

In the light of my experience I have been convinced that lymphography is a safe and a potentially diagnostic tool and an effective means of drug delivery in cancer therapy, and that it carries no extra risk than any other investigative procedures if done carefully.—I am, etc.,

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Salivary Gland Enlargement and Phenylbutazone

SIR,—We would like to draw attention to an interesting case of salivary gland enlargement that has recently come under our care.

A male, aged 44 years, presented in April 1965 just after a short febrile illness with symmetrical enlargement of both parotid and submandibular glands, associated with profound xerostomia. These symptoms were suggestive of an attack of mumps, but the complement fixation test for mumps S and V antibodies excluded this diagnosis. He made a spontaneous recovery

and we saw nothing more of him until a year later, when he again presented complaining of nausea and feeling generally unwell. He was febrile and again there were symmetrically enlarged salivary glands with marked xerostomia. Haematological examination was normal, except that he had a sedimentation rate of 51 mm. in the first hour.

On close questioning an interesting fact emerged; on each of the two occasions that he had experienced salivary gland swelling his illness had started two days after commencing a course of phenylbutazone, which his medical practitioner had prescribed for "tennis elbow." It was found that the salivary swellings had begun to subside when he stopped the tablets and had disappeared within two days. The first attack was more severe and on that occasion he had been taking a higher dose of phenylbutazone. There was no history of any other exposure to the drug.

It seemed to us that this might be a rare side-effect produced by phenylbutazone similar to the case reported by Løkkegaard.¹ Dryness of the mouth occurs in nearly 20% of patients on phenylbutazone,² and Meyler³ records oedema of the face twice in a series of 452 patients on phenylbutazone. It is recognized that the plasma volume may increase up to 50% in patients on the drug and oedema may occur.⁴

In the above case we feel there is a strong possibility that the patient's symptoms were caused by phenylbutazone. The alternative explanation is that the sialadenitis was produced by a virus infection other than mumps. Parotitis is reported in association with Cocksackie, E.C.H.O., and also lymphocytic choriomeningitis viruses.⁵ Our patient's clinical picture was quite unlike recurrent parotid swellings, as there was associated general illness and the submandibular glands were also involved. A subsequent sialogram showed no alteration in gland architecture and rapid emptying.

We would be grateful for any comments from your readers that might shed more light on this interesting case.—We are, etc.,

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Home Treatment of Pulmonary Tuberculosis

SIR,—In reply to Dr. Kenneth M. Citron (7 May, p. 1170) my attitude to home treatment evolved from my own experience and from the reports of the Madras experiment. Fox,¹ writing of the latter, commented . . . "The fact that the time-honoured virtues of . . . good accommodation, combined with isolation, proved remarkably unimportant, is a tribute to the power of effective chemotherapy." The Madras experiment^{2,3} also demonstrated that in the conditions of the experiment the risk to contacts is no greater if the patients are treated in their homes than if they are removed to hospital. The majority

of the patients lived under grossly overcrowded conditions.

For the past 10 years I have performed annual tuberculin testing of children in all primary schools in my area, with follow-up of reactors. No new converter has been traced to a smear-positive case undergoing ambulatory chemotherapy.

If a patient on domiciliary treatment were infectious to others in the home infection would most likely occur during daytime in the living- and dining-rooms. Accepting this opinion to be correct a separate bedroom would appear to be illogical.

It is doubtful if any of the prophylactics used against other conditions are 100% effective, but Dörner *et al.*⁴ have shown that isoniazid may reach this target if the drug is regularly taken.—I am, etc.,

Mullingar, Ireland. MICHAEL P. FLYNN.

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Treatment of Clubfoot

SIR,—The cure of this deformity stubbornly resisted my efforts, using the orthopaedics of the 1920's. Frustration caused me to say, "Start manipulation at the third stage of labour and at two weeks add splintage (plaster-of-Paris) and pursue it for a few years." Stern handling and tenacity are indispensable principles in establishing a cure of this "invincible," ugly, and seriously crippling lesion.

Paediatricians and patients are indebted to the reasoned elucidation of the current methods of Sir Denis Browne and of Kite given in your correspondence column last week (21 May, p. 1298).—I am, etc.,

Bristol 8

A. WILFRID ADAMS.

Mild Smallpox

SIR,—Your leading article on "Mild Smallpox" (21 May, p. 1248) brings to memory the prolonged outbreak of this disease which I experienced in West Ham during 1928.

The symptoms then do not appear to differ from those of the present, and, while every endeavour was then made to check contacts by the usual procedures as the epidemic advanced, this became well nigh impossible, and the question eventually arose as to whether or not the precautions taken justified the "man hours" involved.

As you state, in contrast to modified classical smallpox variola minor breeds true in that only the same mild disease is produced in contacts. Furthermore, in 1928 it was believed that an attack of variola minor immunized one against variola major.

If such is the case, would it not be better in this age of a low vaccination rate to let the present epidemic take its course uninterrupted with the minimum of supervision, and so ensure a greater protection against the