

of atheromatous change in the coronary arteries. This latter observation could well be found significant in a prospective study to relate the use of oral contraceptives and coronary thrombosis.

Oral contraceptives may contain both oestrogens and progestogens. The effects of each should be considered.

Premature withdrawal of natural oestrogens has been shown to increase as much as seven-fold the incidence of coronary thrombosis.<sup>1,2</sup> Stander has demonstrated that there is a significant increase in serum cholesterol in women who have been castrated or who experience an early menopause. Davis<sup>3</sup> has further demonstrated that oestrogen-replacement therapy reduces the degree and rate of increase of serum cholesterol in such women. Current evidence suggests that oestrogens will reduce rather than increase the risk of coronary thrombosis.

There is also some evidence of the possible effect of progesterone or progestogens on the coronary arteries. In 1960 Watson and his colleagues<sup>4</sup> were able to present 24 cases collected from the literature of coronary thrombosis occurring in young pregnant women and added two cases of their own. These are very small numbers and suggest that the incidence of coronary thrombosis is rare compared with venous thrombosis in pregnancy. There is evidence<sup>5</sup> that progestogen therapy is associated with an increase in some coagulation factors which could increase the clotting tendency. There is no information, as far as I am aware, to relate progestogens alone with coronary thrombosis, though these hormones have been used for some years.

On the whole I feel that Dr. Naysmith could be advised that there is at present insufficient evidence to show that oral contraceptives increase the risk of coronary thrombosis. The relationship could as easily be casual as causal, and the use of oral contraceptives should not be withheld on the grounds of increasing the risk of coronary thrombosis.—I am, etc.,

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#### "Epidemic Collapse"

SIR,—I read with interest the article by Drs. G. T. Pollock and T. Morrison Clayton (26 December, p. 1625), and the suggestion by Dr. A. Frame (9 January, p. 127) that the symptoms could be attributed to the syncope following modified Valsalva manoeuvres.

I consider the outbreak unlikely to be due to Valsalva manoeuvres. During a misspent boyhood I regret to admit that my young colleagues and I engaged in this experiment. Loss of consciousness usually supervened, occasionally accompanied by convulsions. Recovery was usually complete within minutes. Vomiting and upper abdominal pain did not usually occur, and the feeling of faintness did not persist. These symptoms were prominent in the outbreak so carefully described by Drs. Pollock and Morrison

Clayton, and persisted for some while in children, who presumably were kept under continuous observation. If one recalls the applied physiology of a modified Valsalva's manoeuvre, one will find it unlikely for resulting symptoms to persist for so long afterwards.

I suggest, therefore, that the outbreak must be attributed to a virus infection or possibly an unknown toxin. The latter may even have been self-administered.—I am, etc.,

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#### New Type of Accident

SIR,—Dr. Alan J. Byron's letter (New Type of Accident) (16 January, p. 192) prompts me to report a similar case seen in the casualty department here some time back.

The patient was a boy of 17, an apprentice mechanic at a local factory, and he was working with an electric hand-drill which had a built-in fan to cool the system. After working for a while he felt very hot, and started perspiring, and decided to cool his forehead with the fan in the drill, which he held near his forehead. In so doing he caught a lock of his front curls in the fan and jerked his head away from it. A wedge of hair, 2 in. by 2 in. (5 cm. by 5 cm.), was avulsed but no scalp removed. The wound was dressed and it healed satisfactorily in due course.

Whilst I quite agree that the legislation regarding wearing protective hair nets should be extended to men as well, I wonder if it is only the length of hair, which was normal in this case, or whether it is the ignorance of the hazards of fast machinery amongst the apprentices that matters as well.

My thanks are due to Mr. F. W. T. Davies, F.R.C.S., Orthopaedic Surgeon, for permission to publish this case.

—I am, etc.,

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#### Idiopathic Splenomegaly

SIR,—The paper by Dr. P. D. Marsden and his colleagues (9 January, p. 89) describing a valuable investigation on tropical or idiopathic splenomegaly in Uganda drew attention to the possible causative role of long-standing infections with *Plasmodium malariae* as shown by the more frequent finding of parasites in their patients when compared with "controls" and also by the results of fluorescent-antibody tests in the two groups. The authors wisely stated that the relationship between splenomegaly, lymphocytic infiltration of the hepatic sinusoids, and quartan malaria should be regarded as a working hypothesis. Two points raised in this excellent paper should be stressed, as they need further investigation.

First, that in tropical Africa the term "splenomegaly" should be always related to the age of the patient or of the examined group. Moreover, when referring to adult Africans living in a malarious area "splenomegaly" must be understood in its relative, clinical meaning—namely, the presence of an unusually large and palpable spleen. This "petitio principii" is not as absurd as it sounds!

Based on the traditional simple spleen-palpation carried out in the standing or recumbent position the "spleen census" of a malaria survey of the unselected indigenous population of tropical Africa shows usually the classical results of a high (over 75%) spleen rate in children and a low (about 15%) spleen rate in adults. A biometric study of spleen weights based on 4,560 necropsies in Lagos, Nigeria,<sup>1</sup> showed that the apparently low frequency of enlarged spleens found on palpation of the adult Africans living in a malarious area is deceptive. In fact at least 80% of these "not-palpable" adult spleens are enlarged to nearly twice the "normal" size, since their average weight is 265 against 160–170 g. of the non-African spleen. The epidemiological conclusions of this investigation are not relevant for the purpose of the present letter, but I suggest that more biometric studies of the data obtained from the routine work of morbid anatomy should be carried out in teaching hospitals and departments of pathology in Africa.

The second point refers to Marsden's and his colleagues' conclusion that infections with *P. malariae* in Africa are much more common than it was thought previously. This is true not only for a selected sample from a hospital but also for a larger, unselected sample of overall population—especially in rural areas of Africa. It has been shown in Nigeria that careful and repeated blood examinations carried out during a longitudinal survey of asymptomatic adult carriers of malaria parasites will reveal that the true rate of *P. malariae* infections is 20% as compared to about 2% of the single blood examination.<sup>2</sup>

Apart from its importance for our better understanding of the natural history of holoendemic malaria this finding indicates the inadequacy of our present routine methods for reliable detection of malaria parasites when the parasites are very scanty.

It is not improbable that further development of new immunological and other methods may be of much help in this field. But that is another story!—I am, etc.,

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#### REFERENCES

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#### Behçet's Syndrome

SIR,—I would like to make a comment about the Clinicopathological Conference (6 February, p. 357). The discussion concerning the occurrence of sinus thrombosis in relation to Behçet's syndrome attracted my attention, since I reported a similar case to the Clinical Section of the Royal Society of Medicine in 1959.<sup>1</sup>

The case that I demonstrated was a man aged 29, and sinus thrombosis had occurred one week after a moderately intensive 18-day course of prednisone. The difficulty was that there might have been hypercoagulability of the blood after the cortisone-analogue therapy,<sup>2</sup> which could have accounted for the event. The case described in the Clinicopathological Conference had no prednisone until 16 months after the episode of dural sinus thrombosis, so that it must be disease-related.