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Cerebral Thrombosis and Oral Contraceptives

SIR,—Dr. M. P. Vessey and Professor Richard Doll (14 June, p. 651) find that oral contraceptives increase sixfold the risk of cerebral thrombosis in women aged 16–40 who have no known predisposing cause. It is good that despite the small numbers they have now been able to reach a definite conclusion, based on such a careful statistical inquiry. But some of the supporting evidence quoted comes from less well-controlled studies which call for scrutiny. Bergeron and Wood¹ reported an increase in angiographically verified vascular occlusions in 1966 as compared with 1960, not in hospital admissions for cerebral thrombosis; their 55 and 58 patients with "non-occlusive disease" included those with aneurysms, tumours, haematomas, etc. Three features of this report are surprising: the similar number of cases investigated in these two years during which most hospitals greatly increased their turnover, the fact that only one patient was pregnant, and the fact that no case had internal carotid occlusion.

In our study of 146 patients aged 15–45 who had carotid angiograms and who were diagnosed on discharge as suffering from carotid territory ischaemia, the proportion of non-pregnant women was similar in recent as in former years.² The number of annual referrals more than doubled during this period (1956–65), but this was exactly balanced by more men in the same age group. Of the 42 non-pregnant women, 40 never had oral contraceptives. More than a third of the women in this age group with ischaemic strokes were pregnant or puerperal, and the mortality rate was three times as great as in the non-pregnant women. Our experience accords with that of Dr. Vessey and Professor Doll in that cerebral ischaemia was usually due to arterial rather than venous occlusion, and this applied equally to the pregnant and puerperal patients, although the distribution of lesions was different. The middle cerebral artery was more often affected in the pregnant group and the internal carotid in the neck in the non-pregnant group.³ It is to be hoped that eventually statisticians will compare the

risk of pregnancy with the risk of contraception in relation to this particular complication, both as regards incidence and mortality.

Dr. Vessey and Professor Doll allude to the difficulty of establishing the diagnosis and apologize for the term "cerebral thrombosis." The term cerebral ischaemia would seem preferable as it does not anticipate the pathological mechanism involved. It would seem wise to make clear in all reports whether or not the diagnosis has been based on angiography, because it is becoming increasingly recognized that other intracranial pathology can mimic vascular lesions. In this regard it is not absolutely clear whether the patients with an "uncertain diagnosis" (Table VIII) were those who had a normal angiogram or who did not have an angiogram at all.

The disparity in findings between different series may rest on definitions such as this and on the criteria used in collecting cases. Thus Dr. Vessey and Professor Doll excluded all patients with a predisposing cause for thromboembolic disease, while Salmon *et al.*,⁴ reviewing the literature, found that such factors operated in 52 patients developing strokes in association with oral contraceptives; they were a feature of 26% of the Glasgow series. This latter series excluded vertebrobasilar disease, which made up 26% of Salmon's series.—I am, etc.,

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Hazards from Raw Fish

SIR,—In the answer to the question on the health hazards from eating raw fish (28 June, p. 812), risks due to bacteria have not been mentioned. Two important diseases of bacterial origin are botulism and the so-called Japanese fish-poisoning.

Outbreaks of botulism, especially those due to *Clostridium botulinum* type E, have been associated with the consumption of unheated or partially heated fish or fish products which have been smoked, dried, salted, or fermented. Outbreaks have been reported from several countries where such products form an important part of the normal diet, and certainly occur in Japan.¹ Raw or preserved fish of either marine or fresh water origin may be involved, and home processed fish or fish products are a special risk.²

Botulism is an intoxication and not an infection. *Cl. botulinum* type E can multiply and form lethal toxin in fish at low temperatures (4–5° C.), and the practice of prolonging the shelf life by storing raw fish in vacuum-packed plastic bags at temperatures between 5–10° C. may be potentially dangerous.

Japanese fish-poisoning is an acute gastroenteritis arising from the consumption of raw sea fish or fish products, and the causative organism is a halophilic vibrio, now known as *Vibrio parahaemolyticus*.³ This type of food-poisoning is, so far, exclusive to Japan, and occurs mainly in summer. Indeed, *V. parahaemolyticus* is probably the commonest cause of bacterial food-poisoning in Japan at present.⁴

Outside Japan, attempts in Britain and elsewhere to find this organism in outbreaks associated with uncooked shellfish such as oysters have failed. However, other halophilic marine vibrios have been incriminated in gastroenteritis syndromes in certain parts of the world.

Cholera may be another hazard from eating semi-raw shell-food preparations such as "alamang," a food paste made from fresh-