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SHORT COMMUNICATION

On Cerebrovascular Deaths in Middle-aged Men during a 7–10-year Follow-up

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Between 1970 and 1973 all men born in 1920-1924 in the city of Uppsala were invited to a health examination. In all 2.322 men participated and 446 men did not participate. Those with hypertension, hyperlipidaemia and reduced glucose tolerance were treated.

The death causes up to September 1980, during an average follow-up of 8.5 years (range 7-10 years) were examined. The number of cerebrovascular death (CVD) among participants was 9 (0.4 %) and among non-participants 4 (0.9 %). Thus the annual incidence of CVD was 45.6 and 105.5 per 100 000 men, respectively. The corresponding figure in all Sweden was 50.2 (1). As the number of CVD was small the differences in incidence should be interpreted with caution.

The proportion of different subdiagnoses within the group of CVD was surprising (Table below).

	Participants	Non-participants	Total
Subarachnoid haemorrhage	7	1	8
Intracerebral haemorrhage	1	1	2
Encephalomalacia	1	2	3
Total	9	4	13

All except one of the 13 men had had autopsy. The non-autopsied man had no hypertension and a clinical diagnosis of subarachnoid haemorrhage (SAH).

The ratio between deaths from SAH and other CVD was 3.5:1 among the participants. The same ratio in the male 50-59 year age group of the whole of Sweden was 1:3.9. There were 7 deaths in SAH in Uppsala (2.5 were expected) and 2 deaths in other cerebrovascular causes (7.4 were expected). The number of deaths due to SAH was significantly ($p < 0.05$) greater than expected.

Hypertension was known in two men - one with cerebral haemorrhage and one with encephalomalacia. The first man had a known hypertension since the end of the 1950s with an unsatisfactory blood pressure reduction most of the time. One more man, a non-participant, had a cerebral haemorrhage. Thus, cerebral haemorrhage is a rare cause of death in middle-aged men if they are treated for hypertension. No participant with SAH had hypertension at the health examination.

The main observation in the present follow-up was that the ratio between deaths due to SAH and due to other cerebrovascular lesions was greater in Uppsala than in Sweden. In fact, the incidence of SAHs was significantly greater in Uppsala than in Sweden. The reason for this is not known. In other mortality statistics, SAH has not decreased in spite of a reduced overall incidence of CVD (1, 2).

The occurrence of SAH should not be influenced to any great extent by an improved antihypertensive treatment which is considered to be the most likely explanation of the reduction of other CVD. SAH is generally caused by rupture of a congenital aneurysm. Therefore, it is possible that any further reduction of CVD in middle-aged men is limited by the lack of reduction of the incidence of SAHs.

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