Effect of Blood Pressure on Prevalence of Carotid Atheroma


SUMMARY In an attempt to elucidate the mechanism by which hypertension acts as a risk factor for major non-haemorrhagic stroke, its effect on the prevalence of carotid atheroma has been studied angiographically. The carotid angiograms of 269 patients with cerebral tumours have been reviewed for evidence of coincidental atheromatous change. The prevalence of signs of arterial disease at the carotid bifurcation proved to be greater amongst patients with an elevated casual blood pressure reading, though the effect was small and confined to subjects over 50 years of age.

It is argued that other factors must contribute to the connection between hypertension and the risks of cerebral infarction.

CEREBROVASCULAR ACCIDENTS in hypertensive subjects may be due to cerebral hemorrhage or infarction. Cerebral hemorrhage is almost always associated with elevated arterial blood pressure except in those with primary haematological disorders. It is believed to be due to rupture of Charcot Bouchard aneurysms which are only common in hypertensive individuals and match in distribution the sites of major hemorrhage.

Cerebral infarction is also more common in hypertensive subjects. The prospective data from the Framingham Survey show that hypertension is the most important risk factor for the development of "atherothrombotic brain infarction" (by which was meant stroke with no blood-staining of the cerebrospinal fluid). This is partly due to small deep lacunar infarcts occurring in hypertensive subjects as a consequence of lipohyalinosis of small perforating arteries exposed to chronically elevated blood pressure. However, large infarcts, as encountered in most non-haemorrhagic strokes are also associated with hypertension and this has traditionally been attributed to an increase in the prevalence or severity of atheroma in the presence of high blood pressure.

We have investigated this hypothesis by examining the carotid bifurcation on angiograms not performed for cerebrovascular disease but for cerebral tumour to find whether evidence of vessel wall disease is related to the patient's blood pressure.

Methods

Patients undergoing angiography for investigation of cerebral tumour at the National Hospitals Queen Square and Maida Vale, and at The Middlesex Hospital were considered. The prevailing blood pressure on admission was extracted from the case notes and therefore represents a single "casual" reading. The mean arterial blood pressure was also calculated. The patients' sex and age were also noted.

The angiograms were reviewed by one or both authors and the neck films studied. A decision was taken as to whether they showed any evidence of irregularity of the vessel wall likely to represent the effect of atheromatous change. The blood pressure levels were unknown at the time of the reading of the angiograms.

Results

The angiograms from 269 patients were studied (174 males; 95 females). Fifty-eight showed changes indicating atheromatous disease at the carotid bifurcation (34 males; 24 females). Mean values for the mean arterial blood pressure are shown in figure 1. Although the levels are higher in the groups with angiographic abnormalities, the differences are not statistically significant.

The prevalence of angiographic lesions in those with a blood pressure of over 150 mms Hg. systolic and of over 90 mms Hg. diastolic was however greater (figure 2) due to an effect in those over 50 years of age where 39% of hypertensive but only 17% of normotensives had angiographic abnormalities (Chi square 9.2 p < 0.01).

A regression analysis (fig. 1 for males; fig. 2 for females) showed wide scatter of individual results, and little evidence of separation of those with angiographic abnormality. The slope for males with abnormal angiograms is the only one that differs from the others, but the difference is only of one standard deviation.

Only 7 patients had angiograms showing narrowing of the lumen of between 20 and 60 per cent. Their mean blood pressures ranged from 93 to 140 and they represent too small a group for separate analysis. All 7 were over 50 years old.

Discussion

The risk of ischaemic heart disease and cerebrovascular accidents increases progressively with increasing levels of arterial blood pressure. In stroke due to cerebral hemorrhage and lacunar infarction, the mechanisms seem well established. Larger cerebral infarcts occurring in hypertensive patients have been assumed to result from more severe atheroma provoked by hypertension. Experimentally, cholesterol feeding of rabbits causes increased lipid incorporation into the wall of the aorta if the animals are also made hypertensive. Monkeys rendered hypertensive by surgical coarctation of the aorta develop severe atheroma on a high cholesterol diet, the atheroma extending to involve smaller arterial branches than are affected in normotensives. A similar mechanism has been inferred for man. Atheroma is more severe and more
FIGURE 2.  Relationship between age and mean arterial blood pressure (male subjects). Continuous regression line for patients with angiographic signs of atheroma (△): broken regression line for patients with angiographically normal carotid bifurcation (○).

extensive in the presence of hypertension.\textsuperscript{10, 11} Carotid occlusion or embolism of atheromatous or thrombotic material from the internal carotid artery are frequent causes of stroke and visual loss.\textsuperscript{12} Potentiation of carotid atheroma by hypertension would be expected to increase the risk of strokes. The present angiographic data, however, reveal only a small influence of hypertension on carotid artery disease. Human post-mortem data also showed only a small increase in aortic atheroma attributable to hypertension.\textsuperscript{13} If stroke victims rather than patients with no symptoms of cerebrovascular disease are considered, there is still little evidence of increased neck vessel disease in the presence of hypertension.\textsuperscript{14-16}

If the effect of hypertension on carotid artery disease is small, what is the explanation of the link with non-haemorrhagic stroke? One factor is undoubtedly the occurrence of small deep lacunar infarcts related to small vessel change.\textsuperscript{6} Another factor may be the effect of blood pressure on atheroma of intracranial vessels which has been demonstrated at autopsy,\textsuperscript{11, 17, 18} but did not appear to account for intracranial angiographic abnormalities in patients with multi-infarct dementia.\textsuperscript{19}

Other possibilities are that hypertension increases stroke risk through an effect on cardiac function or on the tendency to thrombus formation. Hypertension might play a causal role in triggering thrombosis in diseased vessels by increasing the risk of intramural hemorrhage.\textsuperscript{20}

It is noteworthy in passing that only 7 of the 269 patients had carotid stenosis. This highlights the pathological significance of stenosis in patients with transient cerebral ischemic attacks where it is found in 40 to 50%.\textsuperscript{21, 22}

Acknowledgement

We are grateful to Prof. G. du Boulay and Dr. B.E. Kendall of the X-Ray Departments of the three hospitals and to Prof. J. Marshall for their assistance.

References


FIGURE 2. See legend to figure 1. Female subjects.
Effect of blood pressure on prevalence of carotid atheroma.
M J Harrison and L A Wilson

Stroke. 1983;14:550-551
doi: 10.1161/01.STR.14.4.550

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