Blood pressure does not predict lacunar infarction

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SUMMARY We studied the relation between blood pressure and type of cerebral infarction (large or lacunar) in 134 patients with acute hemispherical infarcts that were detectable by computed tomography and that could not be attributed to causes other than atherothrombotic arterial disease. Lacunae were present in 26 patients, and systolic blood pressures were higher in this group than in the 108 patients with large infarcts. The overlap was so wide, however, that large infarcts predominated at every level of blood pressure. The presence of a lacune can be inferred only from the combination of clinical signs and, most important, computed tomography.

Lacunae are small infarcts in the deep regions of the brain or brain stem, caused by occlusion of single perforating arteries or their branches.^{1,2} They are considered a hallmark of hypertension.³ Indirect evidence suggests that most brain infarcts in patients with diastolic pressures above 110 mm Hg are of the lacunar type.⁴ Thus, the level of the diastolic blood pressure often helps to differentiate at the bed-side between patients with atheromatous lesions of extracerebral arteries and patients with disease of small vessels.⁵ This dichotomy has been extended to the management of transient ischaemic attacks; some investigators exclude hypertensive patients—as defined above—from carotid angiography or anticoagulant treatment, unless there is a local bruit.⁶ For the past few years we had adopted this policy in our department, although epidemiological studies indicate that hypertension is common in all types of stroke.⁷ Since the advent of computed tomography (CT) we have found not only large cortical infarcts in patients with severe hypertension, but we have also been surprised to find lacunar infarcts in some normotensive patients. This experience has been confirmed from several centres.^{8,9}

We have been prompted by these inconsistencies to review the blood pressure readings in a large series of patients with acute brain infarcts due to arterial disease.

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Patients and methods

From February 1977, CT has been standard practice in patients with cerebrovascular disease who were admitted to our service. In May 1980 the records of the department of radiology contained 860 cases of cerebral infarction. We limited our analysis to the 134 patients who fitted the following criteria:

(1) unequivocal evidence of cerebral infarction on CT in the supratentorial compartment. Brain-stem infarcts are difficult to detect on CT, and may also directly affect the blood pressure,¹⁰ (2) acute onset of corresponding clinical features, preceding CT by not more than 10 days. Beyond this period, small haemorrhages might have resolved and resemble infarction. In addition, old infarcts might have been asymptomatic, or the blood pressure might have changed in the mean time, (3) no referrals from other hospitals, because these cases were highly selected, (4) no possible cause other than atheroma or small-vessel disease; we excluded patients with atrial fibrillation, valvular heart disease, recent myocardial infarction, hypotension or hypoxia, vasculitis, polycythaemia, migraine, use of oral contraceptives, ruptured aneurysm, meningitis, eclampsia, cerebral vein thrombosis, or a co-existent malignant tumour.

Computed tomography was performed with an EMI-1010 machine, in standard 13-mm sections. Measurements were made from transparent films, with correction for the reduction in format. Volumes of deep infarcts were estimated according to the method of Nelson *et al.*:⁸ largest horizontal dimension of radiolucent area multiplied by dimension at right angles to it multiplied by depth of the lesion (derived from the number of sections on which the lesion was visible), this product then being divided by two (fig 1).

Blood-pressure values were available for all the 134 patients with cerebral infarction. The readings had been obtained with an ordinary sphygmomanometer, by

Fig 1 CT scan of a 49-year-old man, six days after onset of sudden weakness of the left arm and leg: radiolucent area in the posterior limb of the right internal capsule, presumably infarction (not visible on other slices; estimated volume 0.4 ml).

resident physicians or nursing staff. For each patient, mean systolic and diastolic pressures were calculated from the first three recorded measurements in the first two days after admission. In nine cases only two recordings were available before antihypertensive therapy had been started. A control group for patients with non-lacunar or "large" infarcts (defined below) was formed by matching each of these with a subject of the same age and sex, admitted during the same year for a neurological disorder other than cerebrovascular disease (excluding conditions that could directly affect the blood pressure). In this control group, only the first measurement of the blood pressure was used.

Table Age, sex and mean blood pressures in patients with large brain infarcts, in their paired controls, and in patients with lacunar infarcts

	n	Age (mean $\pm$ SD)	% males	Blood pressure (mean $\pm$ SD)	
				Systolic	Diastolic
Large infarcts	108	62.5 $\pm$ 11.8	66	160.1 $\pm$ 27.9	95.4 $\pm$ 14.8
Controls	108	62.4 $\pm$ 11.8	66	145.0 $\pm$ 19.9 p < 0.001	89.1 $\pm$ 11.1 p < 0.001
Lacunar infarcts	26	61.8 $\pm$ 9.4	68	172.3 $\pm$ 25.4	99.8 $\pm$ 12.5
Large infarcts	108	62.5 $\pm$ 11.8	66	160.1 $\pm$ 27.9 p < 0.05	95.4 $\pm$ 14.8 p < 0.20

Statistical tests Student *t* tests were used to compare two groups with respect to blood pressures. The difference between patients with large infarcts and their matched controls was assessed with the *t* test for paired observations. We chose the *t* test for independent samples for comparing the "lacunar group" with the patients who had large infarcts.

Results

Of the 134 patients with recent supratentorial infarcts on CT, 96 had a cortical infarct (territory of the middle cerebral artery in 85, of the posterior cerebral artery in eight, and of the anterior cerebral artery in three). In the other 38 patients the infarct was restricted to subcortical structures: in every case the internal capsule or corona radiata, with or without involvement of the basal ganglia or thalamus. The largest recorded infarct resulting from occlusion of a single perforating artery² measured approximately 5 ml. Therefore, only the 26 deep infarcts of less than 5 ml were considered lacunae (range 0.1-2.5 ml, mean 0.8 ml). The clinical correlate of these lacunar infarcts was a pure motor hemiparesis¹¹ in 22, a sensorimotor stroke^{12,13} in two, and hemiparesis with transient motor aphasia^{2,13} in another two cases. The 12 capsular infarcts of more than 5 ml (mean volume 12.5 ml) were attributed to disease of main arteries ("large infarcts"), together with the 96 cortical infarcts.

The mean blood pressures in the three groups of patients (lacunar infarcts, large infarcts and non-vascular neurological disease) are shown in the table. Patients with large infarcts had higher systolic and diastolic values than their controls. On comparing the "lacunar group" with the patients who had large infarcts, of comparable age and sex, the mean blood pressure was higher in the patients with lacunar infarcts, the difference being statistically significant for systolic values. Nevertheless, the distribution of individual systolic pressures showed a wide overlap between the two infarct groups (fig 2). Eleven of the 26 lacunae occurred with pressures that were below the mean of the group with large infarcts (160 mm). At higher systolic values, lacunar infarcts

Fig 2 Systolic pressures (steps of 10 mm Hg) in patients with lacunar brain infarcts or with large infarcts.

are relatively more numerous, but still outnumbered by large infarcts at every level of pressure. The mean volume of the lacunes was exactly the same on either side of the dividing line of 160 mm Hg.

Discussion

Brain infarcts have many causes. If emboli from the heart, blood diseases, arteritis, migraine and oral contraceptives have been ruled out, "degenerative" arterial disease is likely. At that stage, it is important to distinguish between atheroma of large extra-cranial vessels and changes in small penetrating arteries to the internal capsule and basal ganglia. In small-vessel disease angiography is not warranted, and anticoagulant treatment may be dangerous because of co-existent micro-aneurysms.¹⁴

Our results show that the level of the blood pressure is a poor guide for making this distinction, at least for determining whether an infarct is large or lacunar. In both groups the blood pressures were higher than in controls, and large infarcts predominated in the entire range of pressures. Apparently, hypertension induces brain infarction mainly by enhancing atherosclerosis of large vessels,¹⁵ and to a lesser extent by damaging perforating arteries. Unfortunately, clinical features alone are also unreliable as a guide to the pathology. A pure motor hemiparesis, for instance, can be the result of large infarcts,^{8 13 16} or even of small haemorrhages.^{16 17} Conversely, lacunar infarcts occasionally present with a sensorimotor stroke^{12 13} or with hemiparesis and transient motor aphasia.^{2 13} This was confirmed in our series.

Computed tomography provides the most reliable evidence for a lacunar infarct during life. Some lacunes are probably too small for detection by

CT,^{8 16} and the combination of a normal scan and a pure motor hemiplegia or other appropriate clinical signs^{11 18-20} also suggests a lacune. We should make the reservation that the presence of a lacune is not quite synonymous with primary disease of small perforating arteries, as these vessels are not exempt from embolism. Carotid angiography may show an operable atheroma,⁸ and in our series we also found carotid ulceration in one patient, against four cases with normal angiograms. On the other hand, it is not possible at present to incriminate such lesions with certainty, and Fisher's meticulous necropsy study² demonstrated local abnormalities of the feeding vessel in seven of nine patients with lacunes. In conclusion, the knowledge that an infarct is lacunar should at least influence the decision whether or not to perform angiography or to give anti-coagulant treatment. Such knowledge cannot be obtained from the blood pressure, but only from the combined information of clinical signs and CT.

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