"DEAD POINT" DYNAMICS IN BRAIN SURFACE ARTERIAL ANASTOMOSES DURING AN ACUTE RISE OF BLOOD PRESSURE

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In response to an acute rise of blood pressure (BP) produced by closure of the aorta, injection of a bolus of autologous blood under high pressure, or in various forms of chemically induced arterial hypertension (noradrenalin, hypertensin II, bicuculline, and so on), the failure of the response of cell-regulation of the cerebral blood flow (CBF), expressed as a primary increase in permeability of the blood-brain barrier relative to plasma proteins and its subsequent damage, is most marked in normal animals in the zones of mixed blood supply [1, 2, 7]. The same rule of greatest vulnerability of the zone of mixed blood supply in hypertensive crises has also been observed in autopsy material in cases of acute hypertensive encephalopathy [2]. The causes of increased vulnerability of the blood vessels of brain tissue in zones of mixed blood supply are not yet clear. The great importance of the geometry of the pial vessels has been suggested: arteries and anastomoses with a straight course are more vulnerable than vessels with a winding course [1], that the adrenergic innervation of the most distant branches of the pial vessels is poorly developed [6], and that local monoamine-secreting cells, present in the greatest density in these same regions of the vascular system of the brain, may play an important role [5].

The aim of this investigation was to study the state of the blood flow in anastomoses of the pial arteries appearing during an acute rise of BP, in order to establish any possible differences in the responses of the afferent arterial branches and arterio-arterial anastomoses formed by them. Special attention was paid to the study of responses of those portions of the anastomoses in which, under normal conditions, the "dead point" of the blood flow, the point of equilibrium of two counterflows of blood, is located.

EXPERIMENTAL METHOD

Experiments were carried out on eight adult chinchilla rabbits under urethane (1.0 g/kg) or pentobarbital (40 mg/kg) anesthesia, with monitoring of the arterial blood pH and gas concentration and recording of the averaged EP. Microfilming of the pial vessels in the zone of mixed blood supply between branches of the middle and anterior cerebral arteries was carried out through a "window" formed in the skull by the method in [4]. The speed of filming was 16 frames/sec, on type LN-8 motion picture color film with optical magnification of 28. Before and during an acute rise of BP produced by injection of noradrenalin (100 μg/kg in physiological saline, intravenously) the value of the "dead point" of the blood flow in a test anastomosis was recorded during the first 20 sec and after establishment of a high BP for the first 2-3 min. The diameters of the anastomoses and of the arteries included in them also were recorded. The microfilms were analyzed on a montage table, with variable projection speeds of up to 16 frames/sec. The diameters of the different parts of the vascular bed studied were determined by frame analysis, using the RZD-DO semiautomatic linear dimensions recorder (Leitz, West Germany) on a Leitz-Orthoplan microscope.

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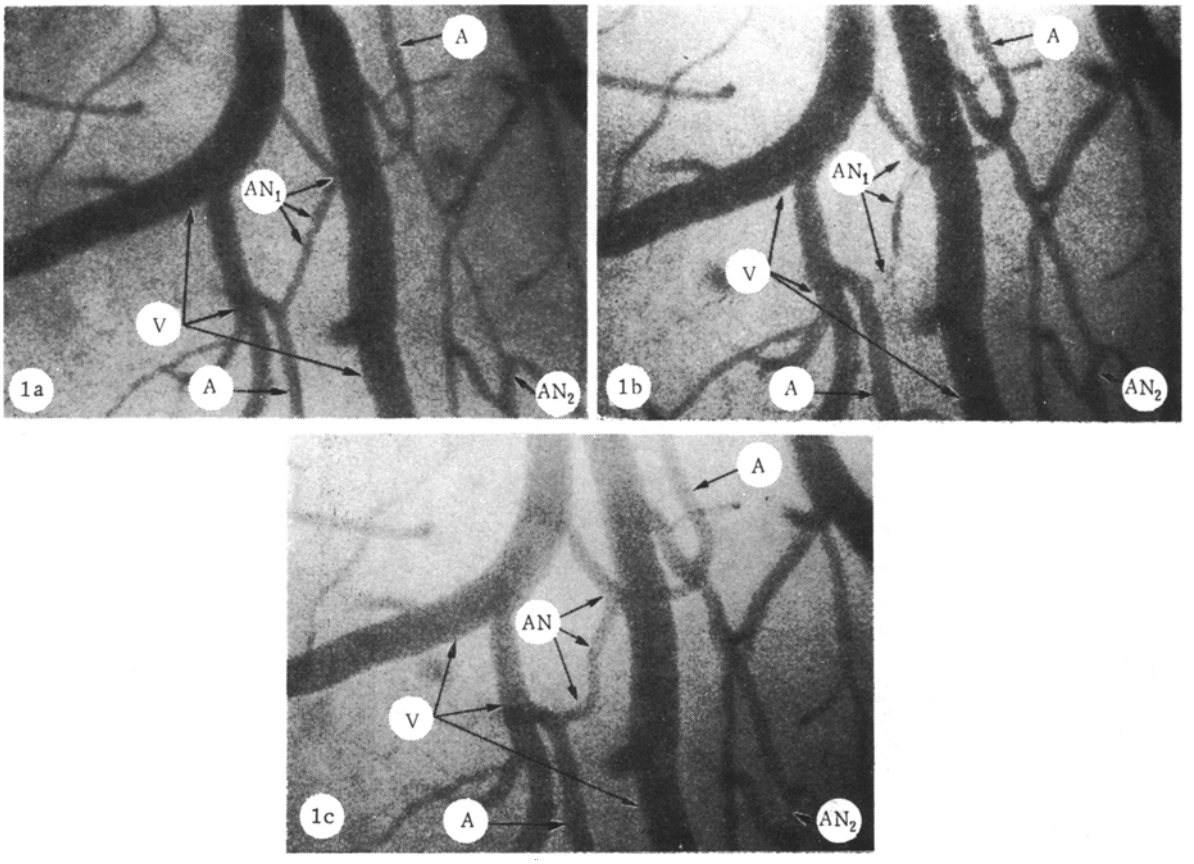

Fig. 1. Filling of an anastomosis (AN_1) with blood plasma because of marked widening of the "dead point" of the blood flow. a) Background; b) 20 sec after beginning of injection of noradrenalin on reaching maximal BP level; c) movement of "dead point" of blood flow and filling of another anastomosis (AN_2) with plasma. A) Arteries; V) vein. Cineangiography through a "window" in the rabbit's skull. 28 ×.

EXPERIMENTAL RESULTS

Under the initial experimental conditions before injection of noradrenalin the region of the "dead point" of the blood flow in the anastomoses had the appearance of a narrow disk consisting purely of blood plasma, with no cells. After various time intervals (on average once or twice a minute) movement of this plasma disk was observed alternately to one side and then to the other, along the length of the anastomosis with no change in its size. The diameter of the vessels also remained unchanged, and as a result the movement of the plasma disk seemed difficult to explain. It can be tentatively suggested that these displacements of the "dead point" corresponded to a fine redistribution of the blood flows in neighboring arteries, connected with changes in the cerebral metabolism and the changes in tone of the various intracerebral vascular basins accompanying it.

Microcineangiography of the brain surface during an acute rise of BP revealed some particular features of the behavior of the "dead point" of the blood flow both during the rise of BP, i.e., in the course of 20-30 sec, and after its establishment on a high, steady level. The most dynamic changes in the region of the "dead point" of the blood flow occurred in the period of rise of BP, when frequent movements of it were observed, every 2-3 sec, i.e., significantly more often than under normal conditions, but at the same time, rather indeterminately, for each change in the position of the "dead point" of the blood flow occurred after different time intervals. The qualitative change in the state of the "dead point" of the blood flow was particularly important. The narrow plasma disk which was the normal appearance of the "dead point" became wider during an acute rise of BP. Its extent along the length of the anastomosis continually varied. In some time intervals the whole of the anastomosis under observation could be filled only with blood plasma (Fig. 1a, b). The boundaries of such a greatly widened "dead point" could be located at the points where the intracerebral arteries were given off, in which the blood flowed in opposite directions, and during that

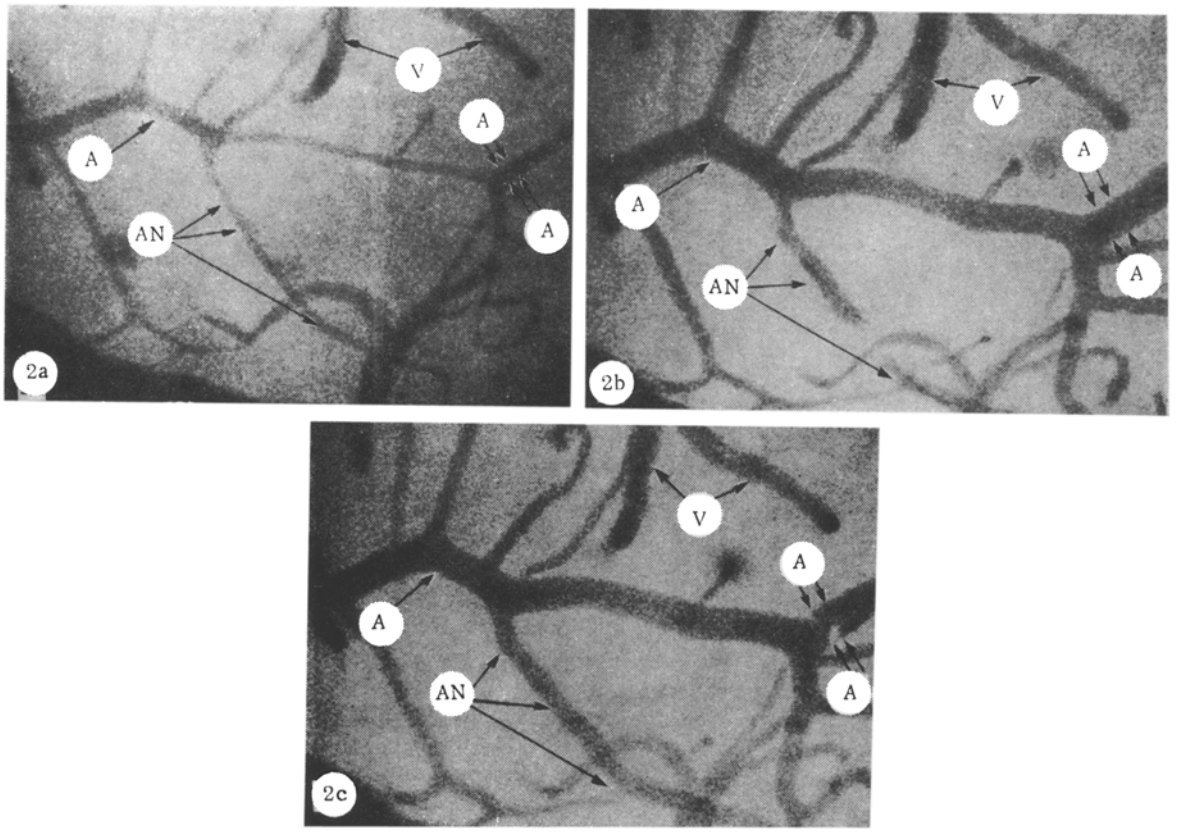

Fig. 2. Sudden widening of "dead point" of blood flow in anastomosis (AN) of staircase type with marked scarcity of blood cells in this segment. c) Movement of "dead point" of blood flow into mouth of one of the arteries forming anastomoses (two parallel arrows). Remainder of legend as to Fig. 1.

time the plasma in the anastomosis did not move. After 1-2 sec there was a sudden movement of the boundaries of the region filled entirely with blood plasma, and one intracerebral artery or more could be included in the anastomosis. It will be clear from Fig. 1b that a segment of blood vessel filled entirely with blood plasma disappears from the field of vision on the photographs and can be seen only on microfilming on account of movement of the blood cells on its boundaries. It must be emphasized that filling of quite long segments of anastomoses and the anastomoses forming them with blood plasma during an acute rise of BP frequently changes the location (Fig. 1b, c) and direction of the blood plasma flow. This was also characteristic of anastomoses of the so-called "staircase" type and of other types (Fig. 2).

With a rise of BP followed by its stabilization at high level, at the end of the 1st minute and during the next 2 min, when the increases in BP were quite considerable, dilatation of the vessels on the brain surface was observed, and is usually interpreted as failure of the response of self-regulation of the cerebral blood flow at its upper limit against the background of the action of noradrenalin.

The change in diameter of the vessels during an acute rise of BP during the first 20-30 sec differed in the anastomoses and in the pial arteries. In 10 of the 12 anastomoses, during the first 10-15 sec the most frequent reaction was dilatation, amounting to 18.2 ± 5.3%. In two cases the anastomoses during this time interval were constricted.

By contrast with this, a very small change in diameter of the pial arteries was established during the first 20-30 sec. The smaller pial arteries (under 30 μ in diameter), incidentally, were constricted in all cases studied (M = −7.5 ± 1.9%). The larger arteries (up to 50 μ in diameter) were very slightly dilated (M = 4.4 ± 1.1%). Differences in the response of the anastomoses and pial arteries were significant ($p < 0.05$).

At the height of the rise of BP and its stabilization at a high level during the first 2 min all vessels observed on the brain surface underwent dilatation. Dilatation was weakest

in the larger pial arteries. The smaller arteries at this time were distinctly dilated (on average by 85.1 ± 15.3%). Maximal dilatation was observed in arterio-arterial anastomoses (194.1 ± 45.6%), and in individual anastomoses it reached 400% or even 600% of the original diameter.

However, it must be emphasized that because of the situation of the "window" in the skull above the zone of mixed blood supply, it was impossible in this investigation to compare the response of arterio-arterial anastomoses in the zone of mixed blood supply between branches of the main arteries of the brain, and anastomoses located in the territory of distribution of each of them. For this reason no final conclusion can be drawn as to whether anastomoses in the zone of mixed blood supply constitute the most "unsteady" part of the vascular bed by comparison with other anastomoses or whether they are so only by comparison with the arteries forming them.

The response of the blood flow described above and changes in the diameters of the blood vessels during an acute rise of BP suggest that the change in tone and intravascular pressure of different branches of the same artery and of branches of the anterior and middle cerebral arteries under these conditions takes place unevenly and that the peripheral resistance in the territories of distribution of the arteries forming anastomoses also changes unevenly. If the phenomenon described above is assessed as an important characteristic of responses of the arteries to injection of noradrenalin and to a rise of BP, it can be concluded that the increase in peripheral resistance takes place at different rates in different vessels, as a result of which the "dead point" of the blood flow moves first in one direction, then in the other, in the anastomoses, and under these circumstances the segment of the vessel which it occupies changes. As already pointed out, an important role in dilatation of the pial vessels, which can be regarded as failure of the response of self-regulation of the cerebral blood flow at its upper limits, is evidently played by the frequent change of direction of the blood flow with movement of the widened "dead point".

The increased vulnerability of the zone of mixed blood supply between branches of the chief arteries of the brain during an acute rise of BP may thus be connected with yet another factor, revealed by the method of microcineangiography of the pial vessels. The dynamics of the "dead point" of the blood flow described in this paper and the more marked changes in the diameters of the anastomoses may be limited among the known factors which evidently determine the greater vulnerability of anastomoses than of other vessels. In our view, a possible disturbance of metabolism of the vessel wall in contact only with blood plasma in certain time intervals plays a role in the increased vulnerability of anastomoses. The effect of repeated changes in the direction of the blood flow may also perhaps be important, for it was shown previously that the blood vessels of the brain are sensitive to changes in the rheologic properties of the blood and its flowability [3]. Unevenly developing changes in tone of the arteries composing the anastomosis, revealed by repeated changes in direction of the blood flow in them and displacement of the "dead point" of the blood flow may also have a variable influence on the tone of anastomosis itself, leading to a disturbance of its function. Such vessels evidently ought to withstand a high or rising BP less effectively, and they may actually be the most vulnerable part of the vascular system of the brain.

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