EFFECT OF ADRENOBLOCKERS OF VASCULAR RESPONSES DURING CAROTID SINUS PRESSURE REFLEXES

V. G. Mal'tsev and M. D. Gaevyi

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In acute experiments on cats using local anesthesia, listhenon, and artificial respiration, the blood pressure was recorded in the common carotid artery and the tone of the cerebral and femoral vessels was measured by a resistographic method. An increase in pressure in the isolated carotid sinuses was accompanied by a decrease in arterial pressure and tone of the femoral and cerebral vessels. When the pressure in the carotid sinuses fell, the arterial pressure and tone of the femoral and cerebral vessels rose. In both cases the response of the cerebral vessels was less marked than that of the femoral vessels. Blocking α-adrenoreceptors with phenatolamine considerably weakened or completely abolished these responses; they were not affected by β-adrenergic blockade with obsidan. The role of the carotid baroreceptors in the regulation of the cerebral circulation during orthostatic collapse is discussed.

KEY WORDS: cerebral circulation; adrenoblockers; carotid sinus reflexes.

In clinical practice adrenergic blockade is often accompanied by orthostatic collapse. This points to a disturbance of the compensatory mechanisms of regulation of the cerebral circulation, an important place among which is ascribed to the carotid sinuses. However, their role in self-regulation of the cerebral circulation has not yet been explained [7-9]. All that is reliably known is that the carotid sinus baroreceptors play an important part in the regulation of the systemic arterial blood pressure and create optimal conditions for the blood supply to the brain and other organs.

The object of this investigation was to study the effect of phentolamine and propanolol (obsidan) on reflex changes in arterial pressure and tone of the cerebral and peripheral vessels during changes in pressure in the carotid sinuses.

EXPERIMENTAL METHOD

Experiments were carried out on 25 cats weighing 2.5-4 kg under local procaine anesthesia together with a muscle relaxant (listhenon) and artificial ventilation. The vascular responses of the brain and hind limb were recorded by resistography. For perfusion of the vessels, one channel of the resistograph was connected with a three-way tube to the two internal maxillary arteries. The branches of the external carotid arteries supplying the extracranial tissues, and the vertebral arteries were ligated. The second channel of the resistograph perfused the femoral artery by Khayutin's method [6]. The third channel was connected through a three-way tube to the two carotid sinuses, isolated by the "blind sac" principle [1]. By regulating the output of the pump of the third channel the pressure in the carotid sinuses could be varied from 20 to 200 mm Hg. The perfusion pressure and the systemic arterial pressure (in the carotid artery) were recorded by mercury manometers. Heparin was used as anticoagulant.

Carotid sinus pressure reflexes are accompanied by considerable falls of arterial pressure. Consequently, if the pressure at the input of the resistograpy is suddenly changed, a definite error could be introduced into its readings; a special device stabilizing the pressure at the input of the resistograph accordingly was used [3].

In each experiment several tests were carried out: rapidly raising or lowering the pres-

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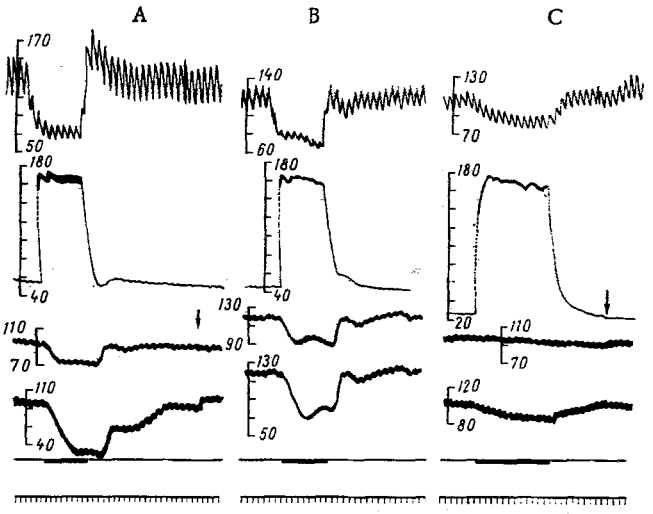

Fig. 1. Response of arterial pressure and cerebral and femoral vessels to increase of pressure in carotid sinuses. A) Control, B) after propranolol, C) after phentolamine. From top to bottom: systemic arterial pressure, pressure in carotid sinuses, perfusion pressure in cerebral vessels, perfusion pressure in femoral vessels, marker of stimulation (change in intracarotid pressure), time marker (5 sec). Arrows indicate stopping tape-winding mechanism for 1-2 min.

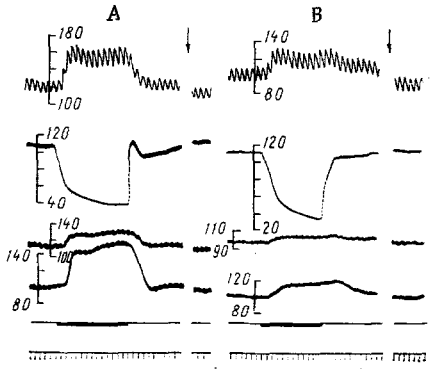

Fig. 2. Responses of arterial pressure and of cerebral and femoral vessels to a decrease in pressure in carotid sinuses. A) Control, B) after phentolamine. Remainder of legend as in Fig. 1.

sure in the carotid sinuses for 1-3 min before and after adrenergic blockade. The α-adrenoreceptors were blocked by phentolamine (1-3 mg/kg) and β-adrenoreceptors by propanolol (1-3 mg/kg). The adrenolytics were injected intraarterially (into the perfusion system) by means of a microinjector. The experimental results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

The initial level of the systemic arterial pressure was 122.1±5.0 mm Hg and the perfusion pressure in the cerebral vessles 91.2±1.4 mm Hg and in the femoral vessels 97.3±0.9 mm Hg. After administration of the adrenolytics, especially phentolamine, the arterial pressure fell; it was often necessary to maintain it by intravenous drip infusion of dextran.

In control tests (before adrenergic blockade) raising the pressure in the carotid sinuses

to 170-200 mm Hg led to a significant ($P<0.001$) fall in tone of the cerebral and femoral vessels and of the systemic arterial pressure by 14.4±1.0, 36.4±3.7, and 50.6±1.5% respectively (Fig. 1A). With a fall in pressure in the carotid sinuses from 140 to 20-10 mm Hg a significant ($P<0.001$) increase was observed in the tone of the cerebral and femoral vessels and in the arterial pressure by 13.2±2.9, 36.3±9.2, and 54.0±13.1% respectively (Fig. 2A). Consequently the responses of the cerebral and peripheral vessels and also of the arterial pressure were in the same direction, although the cerebral vessels gave the weakest response. Only in two experiments did the cerebral vessels respond in the opposite direction, and in two other experiments they gave no response, although the femoral vessels and arterial pressure continued to respond.

These observations fully confirm the results obtained by Krasil'nikov, under the same experimental conditions, and of other workers [2, 5].

It will be noted that the vascular responses were in the same direction, i.e., vasodilation in response to an increase and vasoconstriction to a decrease in the intracarotid pressure, observed in 84% of these experiments. This type of response for the cerebral vessels casts doubts on the role of the carotid vessels in self-regulation of the cerebral circulation during changes in arterial pressure, in cats at least. Their role in the regulation of the cerebral circulation can most probably be reduced to maintenance of a stable arterial pressure.

After blockade of the α-adrenoreceptors by phentolamine, elevation of the pressure in the carotid sinuses caused a decrease in the systemic arterial pressure by 28.6±1.8% ($P<0.001$), in the tone of the cerebral vessels by 0.5±0.7% ($P>0.1$), and in the tone of the femoral vessels by 10.0±3.3% ($P<0.05$). In response to a decrease in the intracarotid pressure accompanied by α-adrenergic blockade the arterial pressure rose by 16.0±5.6% ($P<0.05$) and the tone of the cerebral and femoral vessels rose by 2.2±0.8% ($P<0.05$) and 5.9±3.3% ($P<0.05$) respectively. After blockade of the β-adrenoreceptors with propanolol, in response to elevation of the pressure in the carotid sinuses the systemic arterial pressure fell by 52.8±2.4% ($P<0.001$) and the tone of the cerebral and femoral vessels fell by 14.2±1.4% ($P<0.001$) and 43.0±3.4% ($P<0.001$) respectively. The decrease in the intracarotid pressure under these conditions caused a significant ($P<0.001$) increase in the arterial pressure and tone of the cerebral and femoral vessels by 24.6±2.1, 8.2±1.3, and 17.8±3.0% respectively.

Blockade of the α-adrenoreceptors thus considerably weakened or completely abolished the responses of the arterial pressure and cerebral and peripheral vessles to changes in pressure in the carotid sinuses (Figs. 1C and 2B). β-adrenergic blockade did not significantly change the response of the cerebral vessels and systemic arterial pressure, but potentiated the responses of the femoral vessels to elevation of the pressure in the carotid sinuses somewhat (Fig. 1B). With a decrease in pressure in the carotid sinuses, all the responses tested were maintained but were weaker.

It can be concluded from these results that orthostatic disorders of the cerebral circulation during administration of adrenolytics take place chiefly because of blockade of the α-adrenoreceptors of the peripheral vessels. Under these conditions the carotid sinus baroreceptors (and also, possibly the aortic) cannot exert effective control over the arterial pressure during exposure to gravitational loads. The question of the role of the carotid baroreceptors in self-regulation of the cerebral circulation still remains unanswered.

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