Sympathetic Activity in Cerebral Embolism

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Summary

Responses of the right cervical sympathetic nerve and the systemic arterial pressure to vertebral and carotid embolism were examined in rabbits anesthetized with α-chloralose and the following results were obtained:

1. Vertebral embolism resulted in a marked rise of blood pressure, while carotid embolism resulted a fall.
2. Hypertension produced by vertebral embolism was converted to hypotension by carotid embolism, whereas hypotension produced by carotid embolism to hypertension by vertebral embolism.
3. Hypertension produced by vertebral embolism disappeared on administration of hexamethonium or tetraethyl-ammonium chloride immediately after the rise of blood pressure, whereas it became more marked following the administration of the latter after several minutes indicating increased circulating vasopressor substances. After administration of either hexamethonium or phenoxybenzamine, vertebral embolism caused only a slight rise in blood pressure.
4. The action potentials of the cervical and renal sympathetic nerves increased usually at the onset of hypertension in vertebral embolism, whereas they decreased at the onset of hypotension in carotid embolism.

In conclusion, the increased sympathetic activity participated in hypertension produced by vertebral embolism, while the decreased sympathetic activity in hypotension produced by carotid embolism. It was supposed that sympathetic function was dominant in the portions supplied by the vertebral arteries, whereas sympathetico-inhibitory function in the portions supplied by the internal carotid arteries.

Additional Indexing Words:
Vertebral embolism Carotid embolism Effects of ganglion blocker and α-blocker Sympathetic nerve activity

The vertebral arteries, according to Kunz, Wechsler and others, supply the blood to the posterior and medial hypothalamus and caudal brain stem; while the internal carotid arteries supply the blood to the cerebrum, preoptic area, anterior, medial and posterior hypothalamus. The blood from these two supplying systems meet with each other in the posterior...
communicating arteries and form a dead point.\textsuperscript{5)}

There are both vasopressor and depressor areas supplied by the vertebral arteries. The areas pointed out as vasopressors are the fovea inferior of the medulla,\textsuperscript{21,14)} posterior hypothalamus,\textsuperscript{20)} nucleus points, basilar and tegmental portions of the pons,\textsuperscript{13)} central grey\textsuperscript{23)} and the vermis of the cerebellum.\textsuperscript{6)} On the other hand, the areas pointed out as vasodepressors are the area postrema of the medulla,\textsuperscript{12,14,22)} tegmental portion of the pons,\textsuperscript{13)} posterior hypothalamus in dogs,\textsuperscript{4)} the pyramidal tract and its surroundings,\textsuperscript{13)} posteromedial lobe of the cerebellum\textsuperscript{21)} and so on. There are also both vasopressor and depressor areas supplied by the internal carotid arteries. The areas pointed out as vasopressors are frontal cortex (in cats) and medial or lateral hypothalamus.\textsuperscript{3)} On the other hand, the areas pointed out as vasodepressors are frontal cortex\textsuperscript{7,13,15)} preoptic area,\textsuperscript{8,13,23)} lateral and rostral parts of anterior hypothalamic area,\textsuperscript{13)} and so on.

The locations of the vasopressor and depressor areas and their nourishing arteries have been thus clarified to some extent. However, it has still been obscure whether the mosaic supplied by either system responds as a whole as vasopressor or vasodepressor to a certain stimulus.

The present experiments were carried out to elucidate these problems by examining the responses of the sympathetic nerve and blood pressure to either vertebral or carotid embolism.

\textbf{Methods}

Sixty rabbits were used for the present experiments. The rabbits were anesthetized with \(\alpha\)-chloralose (100 mg./Kg. to 120 mg./Kg.) injected intravenously. The respiration was maintained with an endotracheal tube attached to a respirator pump. A catheter was inserted through the right common carotid artery into the right subclavian artery. The distal of the subclavian artery and its branches except the right vertebral artery were ligated. Another catheter was inserted retrogradely into the left external carotid artery with its tip at the orifice of the internal carotid artery. The impulses were led off from the right cervical sympathetic nerve by the small bipolar electrodes, amplified for display on an oscilloscope, and were recorded. The femoral arterial pressure was recorded by a Statham strain gauge on a pen-oscillograph in some, while by a mercury manometer on a smoked drum in the others.

About 10 mg. of the plastic beads 50 to 80 \(\mu\) in diameter suspended in 0.1 ml. of Ringer’s solution were injected slowly into the right vertebral artery or the left internal carotid artery through the respective catheters.

The relationship between the response patterns of the arterial pressure and distribution of the emboli injected into the vertebral and internal carotid artery was examined in 13 and 10 rabbits, respectively. The effects of carotid embolism on hypertension produced by vertebral embolism were examined in 3 cases, and those
of vertebral embolism on hypotension produced by carotid embolism in 4 cases. The effects of tetrathylammonium chloride and hexamethonium on hypertension produced by vertebral embolism were studied in 3 and 2 cases, respectively. The responses of the right cervical sympathetic nerve to vertebral and carotid embolism were studied in 10 and 9 cases, respectively. The efferent renal nerve activity was recorded in 3 rabbits with occluded renal and adrenal nerve, and the effects of vertebral embolism were examined. The effects of preceding administration of an α-blocker, phenoxybenzamine, on the responses of blood pressure to vertebral embolism were studied in 6 cases.

**Results**

Vertebral embolism resulted in hypertension in all cases (Fig. 1). The arterial pressure remained elevated for several tens of minutes in 4 cases, returned nearly to the control levels after hypertension subsided in 3 cases, fell to lower levels than the control after hypertension of short duration in 6 cases. In the cases in which blood pressure remained elevated, the emboli were found in...

![Fig. 1. Typical response of the arterial pressure to vertebral embolism. The emboli were injected at the point of arrow. The emboli were found in the basilar artery and its branches in this case.](image1)

![Fig. 2. The response pattern of blood pressure to vertebral embolism.](image2)
the basilar artery and its branches; in the cases in which blood pressure returned nearly to the control levels, the emboli were distributed as far as the junctions of the internal carotid arteries with Willi's ring; and in the cases in which blood pressure fell to lower levels, however, the emboli were distributed over the anterior communicating arteries into the anterior cerebral arteries (Fig. 2 and 3). On the other hand, carotid embolism resulted in hypotension in 8 of 15 cases (Fig. 4, 5 and 6). The low blood pressure continued for several tens of minutes and then returned gradually to the control levels in 2 cases; in the other 6 cases, however, death occurred in the course of hypotension. In these 8 cases, the emboli were distributed as far as the posterior com-

![Diagram](image)

**Fig. 3.** The relations of the distribution of the emboli to the response patterns of the arterial pressure in vertebral embolism.

![Graph](image)

**Fig. 4.** Responses of the arterial pressure to carotid embolism. The emboli were injected at the points of arrows. The emboli were found in the area supplied by the internal carotid artery, but not in the basilar artery and its branches in this case.
Fig. 5. The response patterns of the arterial pressure to carotid embolism.

Fig. 6. The relationship between the distribution of the emboli and the response patterns of the arterial pressure in carotid embolism.

municating arteries. In 7 of 15 cases, on the contrary, blood pressure rose up transiently and then fell to lower levels. The emboli were distributed over the posterior communicating arteries into the basilar artery in 2 of these cases examined (Fig. 5 and 6).

Hypotension produced by carotid embolism was converted to normoten-
sion or hypertension by vertebral embolism in all 4 cases (Fig. 7). On the other hand, hypertension produced by vertebral embolism was converted to normotension or hypotension by carotid embolism in all 3 cases (Fig. 8). The intravenous injections of hexamethonium during hypertension caused by vertebral embolism resulted in normotension or hypotension (Fig. 8). Tetraethyl-
ammonium chloride reduced the hypertension when administered immediately after embolization, whereas it caused a further rise in blood pressure when administered in several minutes after embolization (Fig. 9).
Fig. 7. Effects of vertebral embolism on hypotension produced by carotid embolism.
C: carotid embolism.
V: vertebral embolism.

Fig. 8. Effects of hexamethonium and carotid embolism on hypertension produced by vertebral embolism.

Fig. 9. Effects of tetraethylammonium chloride on hypertension produced by vertebral embolism.
T: tetraethylammonium chloride.
Fig. 10. Effects of hexamethonium on renal nerve activity and blood pressure.

Ev: vertebral embolism.
RNA: renal nerve activity.

Fig. 11. The changes of the arterial pressure following the injection of the emboli into the vertebral artery and the abdominal aorta after administration of phenoxybenzamine under occlusion of the renal and adrenal veins.

Ev: vertebral injection of the emboli.
Ea: aortic injection of the emboli.
NA: noradrenalin.
After administration of either hexamethonium or phenoxybenzamine, vertebral embolism caused only a slight rise of blood pressure in 6 rabbits in which the renal and adrenal veins had previously been occluded (Fig. 10 and 11).

Cervical sympathetic nervous activity increased at the onset of hypertension in vertebral embolism in all 10 rabbits (Fig. 12). On the contrary, it decreased at the onset of hypotension in carotid embolism in 5 of 9 cases in
which spontaneous discharge was recorded (Fig. 13). In the other 4 cases, in which a transient rise preceded hypotension, however, the nervous activity increased with the rise in blood pressure.

**DISCUSSION**

In the present experiments, vertebral embolism caused an increase in cervical sympathetic nervous activity followed by a marked rise in blood pressure. This marked rise in blood pressure, however, was not observed after the administration of either hexamethonium or phenoxybenzamine. These findings indicate that the increased sympathetic activity participated in producing the hypertension and that the sympathetico-excitatory function of the vasopressor areas supplied by vertebral arteries overcame the sympathetico-inhibitory function of the vasodepressor areas supplied by the same arteries. Carotid embolism resulted in a decrease in sympathetic nerve activity followed by a fall in blood pressure in the rabbits in which the emboli examined microscopically were distributed as far as the posterior communicating arteries, presumably indicating that the sympathetico-inhibitory function of the vasodepressor areas overcame the sympathetico-excitatory function of the vasopressor areas. The emboli were found in the basilar artery in several rabbits of carotid embolism, while they were found in the Willi's ring or anterior cerebral arteries in several rabbits of vertebral embolism. These differences of distribution might be due to the experimental conditions such as injecting power and control blood pressure.

Uramoto noted that administration of ephedrine, KCl or nicotine into the vertebral arteries caused more easily a rise in blood pressure, but that the administration into the carotid arteries more easily a fall. Dickinson, Katsuki, and Ueda & Uchida observed that the injections of synthetized angiotensin II into the vertebral artery produced a higher rise in blood pressure than that produced by injecting it into the internal carotid artery or thoracic aorta. Also, Ueda observed that vertebral injection of this substance into the left vertebral artery in men caused a higher rise in blood pressure than that caused by injecting the same dose into the descending thoracic aorta and that heart rate increased following vertebral injection, whereas it decreased following aortic injection. Katsuki noted that chronic hypertension was produced by occluding the basilar artery. It is supposed, from the observations mentioned above, that sympathetico-excitatory function is dominant in the portions supplied by the vertebral arteries, but that sympathetico-inhibitory function is dominant in the portions supplied by the internal carotid arteries.

In carotid embolism, a transient rise in blood pressure with a concomitant
increase in sympathetic nervous activity was observed in several cases in which the emboli were spread over the posterior communicating arteries into the basilar artery. It is supposed that the sympathetico-excitatory function of the vaso-pressor areas embolized overcame initially the sympathetico-inhibitory function of the vasodepressor areas, and vice versa in the later stage.

References