CORRESPONDENCE

Circulatory Isolation of the Head

To the Editor.—The article by Price, Linde and Morse [1] in your journal provides an interesting new approach towards solving the problem of the cause of halothane induced hypotension. However, there is one point which appears to have been overlooked in the technique that was used.

Perfusion of the cephalic circulation was obtained by retrograde perfusion through the ligated axillary artery, whilst the flow in the brachiocephalic and omocervical arteries was obstructed. Blood flow through the left vertebral artery was prevented by obstructing the left subclavian artery. Blood was then flowing from the perfusion apparatus through both carotid arteries and the right vertebral artery to the head. No mention was made of the right internal thoracic and the right costocervical arteries,[2] both of which are particularly difficult to approach (in my experience) without removing the first rib. These arteries were probably also receiving blood from the perfusion. As the right costocervical artery supplies blood to the right stellate ganglion it seems likely that this ganglion was being perfused, and it is possible that the left stellate ganglion also received blood from the perfusion by retrograde flow through the left vertebral and left costocervical arteries, and possibly the anastomosing intercostal arteries as well.

In a series of experiments in this laboratory in which the stellate ganglion was perfused by a technique remarkably similar to that described by Price and his colleagues, it was possible to show that halothane can produce ganglionic block in the stellate ganglion of cats. It is possible that stellate ganglion block occurred in the experiments described by Price and his colleagues.

Consequently the effects they describe (namely a reduced heart rate, a reduction in myocardial contractile force and mean arterial blood pressure) could still be the result of ganglionic block uncomplicated by central effects or a combination of both.

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REFERENCES

1. Price, H. L., Linde, H. W., and Morse, H. T.: Central nervous actions of halothane affecting the systemic circulation, ANESTHESIOLOGY **24**: 770, 1963.
2. Miller, M. E.: Guide to the dissection of the dog, ed. 3. Ann Arbor, Mich., Edwards Brothers Inc., 1958, p. 147.

To the Editor.—Dr. Purchase's comment emphasizes the problems involved in attempting to secure circulatory isolation of the head. Our technique was not described in full detail in the original publication because of its complexity. A further description is given below.

After the sternum was split the internal thoracic arteries were ligated bilaterally. The numerous branches of the brachiocephalic artery beyond the origin of the right vertebral artery, including the costocervical, omocervical, and internal thoracic arteries, were ligated under direct vision, using both a thoracic and an axillary approach. It was not necessary to remove a rib. The left vertebral artery was identified and a ligature passed around it. This artery was clamped in several cases after head perfusion had begun, but in most experiments the left subclavian artery was ligated instead just beyond the origin of the vertebral artery, thus eliminating the access of blood to the costocervical, omocervical, axillary, and internal thoracic arteries on the left. Subsequently, the left subclavian artery was also clamped below the origin of the vertebral artery. For these reasons the stellate ganglia were not perfused in the manner suggested by Dr. Purchase.

On the other hand, we recognize that the idea of circulatory isolation is chimerical so long as any normal structures joining the separated areas remain intact. Even bone is perfused with blood. Consequently, it is conceivable that the stellate ganglia were affected by halothane in our experiments. The only way to be certain would have been to test the response of the ganglia by direct electrical stimulation in each experiment; this was technically impracticable.

There are four pieces of indirect evidence which suggests that the results observed were not caused by blockade of the stellate ganglia. First, the administration of halothane to the pump caused the arterial perfusion pressure to decline below that present in the systemic circulation, thus favoring (assuming that a leak was present) perfusion of the head and neck by systemic arterial blood containing no halothane rather than the other way around. Second, in unreported experiments we were able to duplicate the published findings when halothane was perfused only through the vertebral arteries. Third, not all the effects observed in our study (reduced heart rate, reduced systemic arterial pressure, reduced cardiac output, reduced cardiac contractile force, and reduced total peripheral resistance) can be produced by bilateral blockade of the stellate ganglia. In fact, in normal man at rest none of these changes occurs (Price *et al.*, J. Clin. Investigation, 41: 604–610, 1962). Finally, blockade of canine stellate ganglia by anesthetic concentrations of halothane has not been shown, to my knowledge.

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SYMPATHETIC BLOCKS Most frequent indications are for treatment of causalgia, early thrombophlebitis, arterial embolism and severe arterial spasm. Less frequent use is stellate ganglion block in intracranial arterial occlusion and dorsal ganglion block in intractable angina pectoris. Less well defined effects result from splanchnic and celiac ganglion blocks in chronic pancreatic disease, biliary "dyskinesia" and ureteral colic. Surgical sympathectomy is required in the majority of well established causalgias. Sympathetic blocks of all types have been found dangerous in patients under effective anticoagulant therapy. (*Hunter, J. A., Najafi, H., and Julian, O. C.: Sympathetic Blocks in Surgery, Surg. Clinics N. Amer. 44: 81 (Feb.) 1964.*)

ELECTROENCEPHALOGRAM AND PAIN Painful stimuli (dental pulp) in awake men induce surface potentials in the extracranial electroencephalogram of 50–200 msec. with an amplitude of 4–20 microvolts and peak latency of 75–120 msec. if the pain is perceived by the person. If it is not perceived the potentials have a duration of 60 msec. with a latency of 50 msec. Application of a simultaneous acoustical stimulation of 90 decibels for 200 microseconds suppresses the pain response in the EEG and induces a potential characteristic for the acoustical stimulus. Slight to medium pain can be suppressed by simultaneously applied acoustical stimuli subjectively. (*Spreng, M., and Ichioka, M.: Slow Cortical EEG Potentials after Painful Stimuli in Man, Pflueger Arch. Ges. Physiol. 279: 122 (March) 1964.*)