Editorial Views

The Central Organization of the Baroreceptor Reflexes

The significance of the baroreceptor reflexes in the homeostatic control of blood pressure has long been recognized by both the physician and the basic scientist. Nevertheless, while the cardiovascular events initiated upon baroreceptor activation are generally well understood, our lack of knowledge of the central components of this important reflex arc is truly astounding. The site of the inhibition of central sympathetic nervous discharge induced by baroreceptor stimulation has not been determined. Both a medullary and a spinal site of inhibition have been proposed. The site of origin of the cardiomotor vagal neurons responsible for a major component of baroreceptor-induced reflex bradycardia is in dispute. An increasing body of evidence strongly suggests that the preganglionic cell bodies of the cardiac vagus may be located within an area of the ventrolateral medullary reticular formation, namely the nucleus ambiguus, rather than in the dorsal motor nucleus of nerve X.

Just as disconcerting is our limited knowledge of the basic synaptic processes involved in both baroreceptor-induced inhibition of central sympathetic outflow and vagal activation. This arises not only from the confused anatomic picture, but also from the difficulty in identifying and recording successfully from single "cardiovascular" neurons of the medullary reticular formation. Reports dealing with areas of the brain stem which respond to electrical stimulation of the carotid sinus nerve have just begun to appear in the literature.

Only recently, we have become fully aware that the medullary components of the baroreceptor reflexes are subjected to both inhibitory and facilitatory influences from more rostral areas of the brain. In our laboratory experiments on spinal cats have demonstrated that vagal bradycardia evoked by electrical activation of the carotid sinus nerve can be inhibited by stimulation of the posterior portions of the hypothalamus. Stimulation of the posterior hypothalamus in intact preparations yields marked hypertensive responses associated with significant tachycardia. It seems evident that tachycardia associated with the pressor response evoked by hypothalamic stimulation results not only from an increase in cardiac sympathetic nerve activity but also from inhibition of baroreceptor-induced cardiac vagal activation. Facilitation of baroreceptor-induced vagal bradycardia upon electrical activation of certain forebrain sites within the area preoptica, area septalis and anterior hypothalamus has also been demonstrated.

Perhaps of greater concern to the anesthesiologist is our ignorance of the pharmacology of the central components of the baroreceptor reflexes. Many anesthetics are known to prevent EEG activation induced by stimulation of the brain-stem reticular formation. This effect is observed even when transmission over specific sensory pathways is little impaired or not affected at all. Thus, it has been suggested that a rather specific depressant action of certain drugs on the reticular-activating system.

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is responsible for the behavioral and EEG changes which occur in anesthesia. Yet, the actions of anesthetics on descending reticular systems subserving cardiovascular function have hardly been approached. In this regard, the studies presented by Skovsted and Price in this issue of the journal are most welcome. Skovsted and Price suggest that diethyl ether and fluroxene block baroreceptor-induced inhibition of central sympathetic nervous outflow at a medullary site. This proposal is based on three major observations. First, administration of the anesthetics enhanced central sympathetic outflow in the “intact” cat to that level observed in the absence of the anesthetics, but following denervation of the carotid sinus and aortic arch baroreceptors. Second, the anesthetics failed to alter preganglionic sympathetic nervous activity significantly in the baroreceptor-denervated preparation. Last, the anesthetics blocked the reflex fall in blood pressure and decrease in centrally-emanating preganglionic sympathetic nervous discharge evoked by electrical stimulation of the aortic depressor nerve. Since the anesthetics lowered blood pressure only after baroreceptor denervation, Skovsted and Price further proposed that central blockade of the depressor reflex compensated for the direct vascular depressant effects of these agents in the normal cat. Algebraic summation of the central and peripheral effects would explain the stable hemodynamic condition usually associated with diethyl ether and fluroxene anesthesia.

If it were not for the fact that the authors demonstrated blockade of the sympathoinhibitory responses evoked by stimulation of the aortic depressor nerve, the results of their studies could be easily explained by a peripheral vascular effect of the anesthetics. Peripheral vasodilatation would be sensed by the arterial baroreceptors. This would lead to a reflex increase in medullary sympathetic outflow, which would maintain mean blood pressure at or near the control level by increasing cardiac output. Under these conditions, preganglionic sympathetic discharge also might reach that level observed in the untreated, baroreceptor-denervated preparation. Thus, blockade of inhibition of preganglionic sympathetic nervous activity induced by stimula-

References


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**Obstetrics and Pediatrics**

**BLOOD GLUCOSE** Blood glucose in scalp capillaries was measured in 38 fetuses prior to, during and after labor in 12 normal pregnancies, 11 with toxemia, nine with Rh-immunization and ten after prolonged pregnancy. Results showed no significant difference in fetal or maternal blood glucose between normal and abnormal pregnancies. Prior to labor, fetal glucose averaged 62 mg/100 ml, maternal 88 mg/100 ml. By the second stage of labor these values had increased to means of 73 mg/100 ml in the fetus and 106 mg/100 ml in the mother. At delivery, umbilical vein glucose was 78 mg/100 ml; maternal, 110 mg/100 ml. Labor and delivery tend to be associated with increasing fetal and maternal blood glucose. (Ravio, K. O., and Teramo, K.: Blood Glucose of the Human Fetus Prior to and during Labor, Acta Paediat. Scand. 57: 512 (Nov.) 1968.)

**BRONCHOPULMONARY DYSPLASIA** Patent ductus arteriosus was found in three premature infants with severe respiratory distress syndrome who were treated with prolonged positive-pressure ventilation (16, 19, and 59 days, respectively). In one patient, surgical ligation of the ductus was followed by clinical improvement, enabling cessation of assisted ventilation. One infant had spontaneous delayed closure of the ductus. The third infant died after 19 days of assisted ventilation, with a widely patent ductus arteriosus. All three infants had signs of the recently described syndrome of “bronchopulmonary dysplasia.” Careful auscultation in infants with respiratory distress syndrome is essential to discover an associated patent ductus arteriosus with left-to-right shunt, which may aggravate symptoms attributed to “bronchopulmonary dysplasia.” (Siassi, B., and others: Patent Ductus Arteriosus Complicating Prolonged Assisted Ventilation in Respiratory Distress Syndrome, J. Pediat. 74: 11 (Jan.) 1969.)