EDITORIALS

Role of Respiratory Responses to Changes in Arterial Blood Pressure

Activation of pressoreceptors in the aortic arch and carotid sinuses is known to result in three striking changes: vasodilatation, decrease in heart rate and inhibition of respiration (1). Therefore, a rise in blood pressure in the intact mammal would be expected to cause inhibition of breathing through sino-aortic mechanisms, and a fall in blood pressure would be expected to elicit hyperpnea. During hemorrhage (2) or vasodilatation (3) respiration is stimulated; while during the rise in blood pressure produced by internal occlusion of the descending aorta (4) or by the injection of vasoconstrictor agents (1) respiration is inhibited. Thus it is clear, both that stimulation of the afferent fibers from pressoreceptors causes inhibition of breathing and that procedures which cause a sudden increase or decrease in blood pressure will tend to cause an opposite change in the rate and depth of breathing.

The greater part but not all of the respiratory stimulation elicited by hemorrhage or vasodilatation is dependent upon sino-aortic reflexes (2, 3). Of the two types of receptors in the sino-aortic zones, chemoreceptors and pressoreceptors, decreased activity of the latter probably is more important in eliciting the hyperpnea; however, this is not definitely established. Also, there are alterations in blood flow through the respiratory center during changes in arterial pressure. In general the flow varies with the arterial pressure, hence a fall in pressure would favor an accumulation of carbon dioxide in the center and a rise in pressure would tend to cause a lowering of carbon dioxide tension. Thus, the changes in CO₂ tension produced by changes in blood flow through the respiratory center are such as to cause inhibition of breathing during a rise in blood pressure and stimulation of breathing during a fall in blood pressure.

It seems that the teleological significance of the reflex effects on respiration from the pressoreceptors has not been envisaged or, if so, any suggestions which have been made have not become generally known. Schmidt (5) stated in 1941 that the reflex effects on respiration from pressoreceptors have no known physiological purpose, and Aviado and Schmidt (1) recently wrote, “Inhibitory effects on respiration from stretch receptors are encountered only in the carotid-aortic zones (type I) and in the lungs (types 2-B and 2-C). The apnea of type I serves no known physiological purpose and perhaps it only represents a now useless survival of a primitive organization for con-
serving energy in a gill-breathing animal when blood pressure rises as part of the flight reaction." Admittedly, if one focuses his attention on the ventilatory function of the respiratory movements no advantage of apnea during a rise in blood pressure or of hyperpnea during a fall can be visualized. There is no apparent beneficial effect of lowered oxygen tension in arterial blood when arterial hypertension occurs; and, since oxygenation of hemoglobin approaches 100 per cent at normal alveolar ventilation (at barometric pressure of 760 mm. of mercury), there could be no significant effect of increased pulmonary ventilation when blood flow through the lungs or the systemic circuit decreases as in hemorrhage or when vasodilation occurs.

Since it appears that no advantage to the organism can accrue from changes in pulmonary ventilation during alterations in arterial blood pressure, it is logical to turn to a consideration of a nonrespiratory function of the breathing movements. The concept of the "respiratory pump" was introduced by Donders in 1859. During inspiration the pressure in the abdomen rises while that in the thorax decreases, and movement of blood toward the right atrium is accelerated (6). The valves in the leg veins prevent reflux into them during inspiration. Breathing is particularly important in maintaining venous return when gravity is counteracting it. Action of leg muscles promotes flow of blood into the venous system in the abdomen, and the respiratory pump forces blood on into the thoracic portion of the inferior vena cava.

When arterial blood pressure decreases as a result of inadequate venous return, which is the case following either hemorrhage or severe vasodilatation, the arterial pressure could be restored or the fall counteracted through reflex vasoconstriction (which does not result in an improvement in blood supply to an organ) or through increased cardiac output. An increased cardiac output can not be achieved simply through cardiac stimulation, but venous return also must be accelerated. This is one of the effects of the increased respiratory activity during arterial hypotension. Thus, each of the triad of cardiorespiratory reflexes elicited by a fall in pressure in the sino-aortic zones has importance in counteracting the ill effects of either excess capacity of the circulatory system or inadequate volume of blood. The respiratory pump accelerates the venous return, cardiac stimulation causes the heart to pump more blood than it would at a given venous pressure level in the absence of such stimulation, and resistance is increased in the vascular beds of those organs which can best withstand a decrease in blood flow.

The respiratory inhibition during a rise in blood pressure, especially in a person in the orthostatic position, would promote pooling of blood in the splanchnic region so that venous return would be decreased, thus cardiac output would be lowered and the rise in blood pressure would be counteracted.

This interpretation of the role of the respiratory responses to
changes in blood pressure does not in any way negate explanations of the buffer role of the pressoreceptor reflexes. On the other hand, it supplements this view in that it adds a mechanism on the venous side of the circulatory system. According to the view presented here, the pressoreceptor reflexes have a unitary role, namely, the stabilization of arterial blood pressure. The characteristics of these reflexes are such that they are useful in counteracting sudden changes of brief duration; they are not capable of counteracting chronic influences.

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REFERENCES
5. Schmidt, C. F.: Macleod's Physiology in Modern Medicine, P. Bard, Editor, ed. 9, St. Louis, Missouri, C. V. Mosby Company, 1941, page 579.

An Author's Best Friend

Fifteen years ago I chanced upon an associate reddened with anger. He was staring at a heavily pencilled manuscript with numerous comments written upon the margins. I learned that the Professor had just returned one of my friend's proposed contributions to medical literature. The recommendations and comments were detailed, forthright and frequent. My associate complained loudly that he had published many papers and that his former chief, a busy clinical professor, had always returned his papers with praise and few corrections; his present chief did not recognize ability. Being a neophyte, I sympathized with my associate. It appeared incredible that a physician and member of a medical school faculty should have his reasoning questioned and his grammar corrected as if he were a high school student.

Today, I realize that my sympathy was ill-founded. Neither of us realized then how fortunate we were in having a chief who would read our reports carefully and give us the benefit of his experience. Nor did we recognize that the other professor had been too busy to read another's work carefully.

The preparation of a report for publication is a laborious task. The pressure of an impending deadline is often present. Time appears