DYNAMIC AXIAL BRAIN-STEM DISTORTION AS A MECHANISM EXPLAINING THE CARDIO-RESPIRATORY CHANGES IN INCREASED INTRACRANIAL PRESSURE*

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For the past half century the generally accepted theory of the mode of action of increased intracranial pressure on cardiorespiratory mechanisms has been based on the experimental work of Harvey Cushing. As a result of his and other experimental studies it has been conceded that the mechanism whereby increased intracranial pressure produces cardiorespiratory changes was that of anemia of the medullary centers. Bailey in his book on intracranial tumors illustrates this theory (Fig. 1). It was Cushing's contention that in order for medullary anemia to exist there must be a suppression of blood flow into the medulla. This occurred at an intracranial pressure in excess of the systolic pressure of blood. These cardiorespiratory changes consist of slowing of the pulse, increase in pulse pressure, elevation of blood pressure, and slowing of respiration with terminal respiratory arrest and final cardiovascular failure.

In applying Cushing's experimental findings to increased intracranial pressure in the human being, most students and investigators have had difficulty in accepting increased intracranial pressure as high as the systolic pressure of blood in the clinical patient. For it is the clinical observation of most investigators that these cardiorespiratory changes usually attributed to increased intracranial pressure occur at levels of intracranial pressure below that of the systolic pressure of blood. Therefore, a search has been underway to find a mechanism whereby these cardiorespiratory changes can be explained at lower levels of intracranial pressure more compatible with those levels found in clinical cases. It is our feeling that this mechanism responsible for cardiorespiratory changes in increased intracranial pressure is an acute dynamic axial distortion of the brain stem affecting the conductivity of the pontomedullary centers for respiration and cardiovascular activity.

Long before the experiments of Cushing in 1901, increased intracranial pressure and the mechanisms by which it acted on the experimental animal and patient were subjects of numerous investigations. As early as 1811, Ravina studied increased intracranial pressure after inserting glass windows in the skull. In 1824, Astley Cooper found that crude pressure against the...
Fig. 1. The mechanism of cardiorespiratory effects of increased intracranial pressure. (Reproduced from Bailey)

surface of the brain with the finger produced slowing of the pulse in animals and, with marked pressure, a diminished state of consciousness occurred. von Bergmann in 1880, working with increased intracranial pressure on animals, produced a slow pulse and slow respirations. He determined that when the intracranial pressure was equal to the systolic blood pressure in the carotid artery death would ensue. In 1892, Spencer and Horsley attributed the medullary response to mechanical pressure on the bulbar centers. Hill was one of the first to ascribe the medullary symptoms to a vascular origin and presumed that the stimulus to the medullary centers was anemia, whereas late effects were caused by exhaustion. Tsubura, working with increased pressure on the medullary centers, described distortion of the stem but then returned to the ischemic theory of medullary failure. While clinical application of the work in Kocher’s laboratory was questioned, it was the outstanding papers of Browder and Meyers that critically attacked the clinical application of experimental work that had been done. They recommended that Kocher’s scheme of the various clinical stages of increased intracranial pressure be abandoned.

Meyers, working on dogs and human beings, concluded that there must be factors other than that of increased intracranial pressure per se to explain these cardiorespiratory changes normally attributed to increased intracranial pressure. In 1951, Evans with co-workers was able to increase the intracranial pressure above that which is found clinically without appreciable changes in the cardiorespiratory mechanisms. He, therefore, concluded that some factor other than pressure must be responsible for these changes. Ryder, Evans, and associates attempted to develop a theory of intracranial pressure based upon the pressures and flows of the various vascular components. Rodbard in 1952, studying increased intracranial pressure, concluded that anemia was not the responsible factor for medullary stimulation. He proposed a pressure-sensitive center in the medulla similar to the barore-