Analysis of the cerebrospinal fluid pulse wave in intracranial pressure

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The configuration of the intracranial pressure (ICP) pulse wave represents a complex sum of various components. Amplitude variations of an isolated component might reflect changes in a specific intracranial structure. Fifteen awake patients suffering from hydrocephalus, benign intracranial hypertension, or head injury underwent ICP monitoring through a ventricular catheter and were subjected to three standardized maneuvers to alter the intracranial dynamics: head elevation, voluntary hyperventilation, and cerebrospinal fluid (CSF) withdrawal. A 12° head elevation and fractionated CSF withdrawal caused a mild ICP drop and a proportionate amplitude reduction of all the wave components. Voluntary hyperventilation caused a comparable fall in ICP, and a disproportionate reduction in the amplitude of the wave components, especially the P2 component. It is postulated that the decrease in amplitude of the P2 component reflects the reduction of the cerebral bulk caused by hyperventilation. Head elevation and CSF withdrawal caused a decrease of global ICP but no specific changes in any intracranial structure, and consequently the configuration of the pulse wave remained unchanged. The establishment of relationships between anatomical substrate and particular wave components is promising since potentially it could be useful for monitoring conditions such as vasoparalysis, impaired cerebrovascular reactivity, and cerebral edema.

KEY WORDS - benign intracranial hypertension • cerebrospinal fluid pulse wave • head injury • hydrocephalus • intracranial pressure • intracranial pulse-pressure wave
FIG. 1. The cerebrospinal fluid (CSF) pulse wave has three main components: the percussion wave (P₁), the tidal wave (P₂), and the dicrotic wave (P₃). The arrow indicates the dicrotic notch, between P₂ and P₃. It corresponds to the dicrotic notch of the arterial pulsation that originates the CSF pulse wave.

The bed by 12°; 2) voluntary hyper- and hypoventilation for as long as necessary to reduce the ICP, but the maneuver was abandoned if no drop was verified by the end of 3 minutes. Some patients were also requested to hold their breath until the ICP rose; and 3) CSF withdrawal in samples of 2 ml at a time until a drop in ICP was recorded.

Results

Spontaneous Variations

There was no consistent similarity between wave patterns related to the patients’ diagnosis, nor was there any concordance between pattern and ventricular size. There were slight changes of the distal components of the pulse wave during the respiratory cycle. At the beginning of the expiration, the P₃ component was elevated, but it became depressed at the beginning of inspiration. These variations have been previously interpreted as the result of retrograde transmission of

FIG. 2. The spontaneous increase in intracranial pressure (ICP) is accompanied by a disproportionate elevation of the cerebrospinal fluid pulse wave components P₂ and P₃, resulting in changes of the shape of the pulse wave. It first becomes rounded and, at higher ICP values, it acquires a pyramidal shape.

FIG. 3. Cerebrospinal fluid pulse wave recorded from a hydrocephalic patient. Voluntary hyperventilation causes a decrease in intracranial pressure (ICP) and changes the shape of the pulse wave by a disproportionate decrease of P₂ and P₃ components. Voluntary breath-holding reverses the process. Head elevation in the same patient causes a similar drop in ICP but no changes in the pulse wave configuration.
Cerebrospinal fluid pulse wave changes

venous pressure changes from intrathoracic pressure variations with breathing. For each individual patient the configuration of the CSF pulse wave was related to the mean ICP. If the ICP values were low, the pulse wave presented a descending saw-tooth appearance, with a clearly distinct P1 component. As the mean ICP rose spontaneously, there was a progressive elevation in the magnitude of P2 and to a lesser extent of P3. On the other hand, P1 changed only mildly. As a result of the disproportionate increases in its components, the pulse wave became rounded. At high mean ICP's, as at the highest points of plateau and B-waves, the appearance was pyramidal (Fig. 2). Lowering of the mean ICP resulted in a reversal of these trends.

Induced Variations

Head elevation and CSF withdrawal caused a drop in the mean ICP of the order of 5 mm Hg, but there were no clear changes in the pulse wave configuration (Figs. 3 and 4). It is possible that greater reductions in ICP would have induced the gradual variations of the pulse wave configuration observed during spontaneous ICP changes. However, after using hyperventilation to induce minor reductions in ICP of about 5 mm Hg, notable changes in the configuration of the pulse wave were observed. There was a clear decrease in the relative height of P2 and to a lesser extent of P3, whereas P1 was relatively unaffected. In two cases, a decrease in P2 amplitude was observed when there was no ICP reduction. Breath-holding led to a reverse phenomenon, with significant elevation of P2 as ICP rose (Figs. 3 and 5).

Discussion

The mechanisms responsible for the generation of the CSF pulse wave are not clearly understood. It is assumed that the pulsations of the choroid plexus play a major role. Choroid plexectomy as well as occlusion of the anterior choroidal artery cause a significant reduction in the CSF pulse wave. However, it has been suggested that in general only the initial components of the pulse wave are dependent upon the arterial pulsations, while the later components are the result of retrograde venous pulsations. Portnoy and collaborators reported that the state of the intracranial components can modify the final wave configuration.

If we assume that hyperventilation can alter the bulk of the brain but not the choroid plexus, since hyperventilation did not change P1, our findings are compatible with an origin of the pulse wave in the choroidal plexus. It is also possible that the pulsations of the major conductive intracranial arteries contribute to the P1 component, since their pulse is probably concomitant with those of the choroidal arteries.

Fig. 4. Reduction in intracranial pressure (ICP) induced by removal of 2 ml of cerebrospinal fluid (CSF). No change is observed in the configuration of the pulse wave.

Fig. 5. Tracings in a patient with benign intracranial hypertension (BIH). The reduction in intracranial pressure caused by hyperventilation is accompanied by a decrease in the relative amplitude of P2 and P3 (upper tracing). The reverse is observed after hypoventilation (lower tracing).
The configuration of the CSF pulse wave after reduction in ICP caused by hyperventilation differed from that following comparable reduction in ICP caused by head elevation and CSF withdrawal. In the latter two instances, there was a decrease in the magnitude of the pulse wave but the configuration of the wave remained unchanged. On the other hand, following hyperventilation there was a decrease in the pulse pressure as well as a reduction in the relative amplitude of the P2 component (Fig. 6). Since it is known that hyperventilation reduces ICP by vasoconstriction of arterioles and subsequent shrinkage of the bulk of the brain, the P2 component may be dependent upon the cerebral compliance. This assumption is reinforced by the observation of the height of the P1 component (Fig. 6). Here again, the increasing magnitude of the P2 component is proportionally bigger than all the other components. Studies by Portnoy and collaborators in dogs and cats also suggested that the CSF pulse wave configuration could reflect changes in the cerebral compliance.

The CSF pulse wave can be broken down into its wave components, the temporal sum of which provides its final configuration. The magnitude of each component depends not only on the state of the different structures in the cranium, but also on the ICP at its onset. The baseline pressure affects all components. Therefore, with an elevated ICP, a certain increase in the components of the pulse wave would be expected, but, since P2 also depends on the bulk compliance, one would expect its amplitude to be more affected. This would explain the variation of the pulse wave configuration as the ICP rises, from a descending saw-tooth appearance to a rounded one. Since a relative change in amplitude of one of the components is observed during changes of the ICP, one has to assume that the state of the structure responsible for that component has been altered.

Conclusions

The interpretation of changes in the CSF pulse wave configuration is complex, since possibly several factors are involved in its formation. In this series of patients, we searched for a definite pattern of change. No correlation was observed between a specific wave slope and the etiological diagnosis or ventricular size. Similar observations have been made by other workers. However, following acute manipulation of the intracranial dynamics, the findings pointed to the possibility that different components of the pulse wave might be caused by different areas of the intracranial compartment. The P1 component could result from pulsations originating at the choroid plexus and large intracranial conductive vessels, while the P2 component may reflect variations in the cerebral bulk compliance. The origin of the later components was not clear.

The suggestion that variations in the amplitude of specific components of the CSF pulse wave might reflect changes in the structures of the brain has important clinical implications. For instance, vasospasm of conductive intracranial vessels could potentially be monitored by the observation of the height of the P1 component. Furthermore, if P2 reflects the bulk compliance of the brain, it could be used to indicate microcirculatory vasoparalysis, cerebral swelling, and edema.

References

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