(10) Cerebral Hemodynamics and Cerebrospinal Fluid in Dyspnea

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Cerebral hemodynamics in respiratory failure were studied. From these data the relationship between the lung and the brain in dyspnea was discussed.

Cerebrospinal fluid (CSF) acid-base balance was measured in normal young men and the patients who complained of dyspnea.

Methods:

Studies were performed on 20 normal male volunteers ranging in age from 18 to 29 years and 29 hospitalized patients with dyspnea.

Cerebral blood flow (CBF) was measured by a modification of the Kety and Schmidt's nitrous oxide method. Blood O_2 and CO_2 contents, as well as N_2O concentration, were determined manometrically in a Van Slyke apparatus.

After a control resting determination, the subjects breathed several gas mixtures containing 5% CO_2 plus air, 10% O_2 plus N_2 , 5% CO_2 and 10% O_2 plus N_2 . At the end of 5 minutes inhalation of gas mixtures paired blood samples were withdrawn from indwelling needles placed in the femoral artery and in the internal jugular bulb. Changes of CBF during gas inhalation was estimated by the arterial-jugular venous O_2 differences.

CSF was obtained anaerobically by lumbar puncture, and the pH, CO_2 and O_2 tensions were determined by the electrodes using an Radiometer micro gas-analyzer immediately after withdrawal.

Results:

1. The effects of hypercapnia on cerebral circulation

In normal young subjects there was a significant correlation between rising $PaCO_2$ and increasing CBF, and the regression was linear ($r=0.78$, $P<0.01$). The relationship between $PaCO_2$ and PO_2 of the jugular blood (PvO_2) was significant ($r=0.87$, $P<0.01$). When the $PaCO_2$ increased above 45 mm.Hg, PvO_2 elevated over the normal range (35.7 ± 6.89 , $P=0.01$).

There was a significant correlation between $PaCO_2$ and PCO_2 of the internal jugular blood ($PvCO_2$). In spite of the widely scattered values for $PaCO_2$ (20 to 60 mm.Hg), the $PvCO_2$ was remained in the normal range (49.5 ± 9.31 , $P<0.01$).

The increased cerebral blood flow caused by carbon dioxide is a protective mechanism to control the cerebral CO_2 tension within the normal range.

2. The effects of hypoxia on cerebral circulation

The inhalation of 10% O_2 caused a marked rise in cerebral blood flow and a con-

siderable drop in cerebrovascular resistance. These changes occurred despite a fall in mean arterial blood pressure and the slight reduction of PaCO_2 .

The relationship between PaO_2 and CBF was not linear. The CBF was markedly increased at tensions below 50 mm.Hg of PaO_2 and probably was increased moderately at tensions between 50 and 70 mm.Hg.

The CBF was constant at tensions between 70 and 110 mm.Hg of PaO_2 . In spite of the widely scattered value for PaO_2 (30 to 110 mm.Hg), the PvO_2 remained in the normal range.

The increment of CBF caused by hypoxia is a protective mechanism to control the cerebral O_2 tension within the normal range.

3. Cerebral circulation in dyspnea

Reduction in PaO_2 and elevation of PaCO_2 commonly develop in respiratory failure. The combined effects of hypoxia and hypercapnia on cerebral blood flow were studied in normal young men. The data lead to the conclusion that simultaneous hypoxia and hypercapnia have additive dilator effects on the cerebral vasculature. These cerebrovascular responses to combined hypoxia and hypercapnia prevent the development of cerebral hypoxia in respiratory insufficiency.

It was reported that the threshold cerebral venous O_2 tension for EEG slowing was 18.95 ± 2.55 mm.Hg.

From these data, the possibility that cerebral hypoxia may play a main role in the pathogenesis of pulmonary encephalopathy is very rare.

On the other hand, it is well known that severe respiratory insufficiency may be accompanied by CO_2 intoxication or CO_2 narcosis. Meyer and Gotoh suggested from experimental CO_2 narcosis that the factor responsible for cerebral dysfunction was cerebral cellular acidosis. In human study, they reported that EEG slowing regularly appeared when the pH of internal jugular blood fell below a threshold of 7.087.

In cerebrovascular diseases, the cerebrovascular reactivities to the inhalation of 5% CO_2 or 10% O_2 were reduced. For these reasons, subjects with severe cerebral circulatory disturbance are more prone to cerebral acidosis.

In thirteen subjects with respiratory insufficiency the mean values were PvO_2 30.5, PvCO_2 59.75 and H^+ 46.73 n M./L. These data reflected the tendency of cerebral acidosis in this state. In dyspnea, the measurement of jugular venous blood PO_2 , PCO_2 and pH is an important method to predict the impending pulmonary encephalopathy.

4. Acid-base balance in cerebrospinal fluid

It has been proposed by Lassen, Loeschcke, and more recently by Mitchell et al., that changes in composition of CSF exert their effects on pulmonary ventilation via "medullary respiratory chemoreceptors" which are specifically sensitive to hydrogen ion concentration.

If this should be a proper inference, then the study of acid-base balance in CSF might be important to understand the mechanism of dyspnea. In thirteen normal young male subjects, the values of CSF were compared with the values of the arterial

and jugular venous blood.

The mean pH was more acid in CSF (7.282 ± 0.0346) than in arterial blood (7.404 ± 0.0332) and jugular blood (7.357 ± 0.0190). The PCO_2 of CSF (50.4 ± 5.59) was higher than the arterial value (40.1 ± 5.37), but was very close to the jugular blood PCO_2 (49.5 ± 6.57). $[\text{HCO}_3^-]$ of CSF (20.92 ± 2.549 mEq./L.) was lower than arterial $[\text{HCO}_3^-]$ (23.75 ± 2.518) and jugular blood $[\text{HCO}_3^-]$ (26.24 ± 3.348). CSF PO_2 (41.9 ± 8.84) was lower than arterial PO_2 (94.9 ± 8.84), but was slightly higher than jugular PO_2 (35.7 ± 4.91).

In normal subjects, the correlation between CSF PCO_2 and blood PCO_2 was statistically significant. About $[\text{H}^+]$ and $[\text{HCO}_3^-]^2$, there were relationships between the values of CSF and blood.

Brief inhalation of 5% CO_2 and hyperventilation did not cause any change in the composition of CSF.

Twenty-nine subjects with dyspnea were divided into two groups.

In group 1 (13 cases), the arterial PO_2 was under 70 mm.Hg. The mean arterial values were PO_2 54.0, PCO_2 48.68, $[\text{H}^+]$ 41.96 and $[\text{HCO}_3^-]$ 27.85. The means of CSF were PO_2 44.0, PCO_2 56.02, $[\text{H}^+]$ 55.94 and $[\text{HCO}_3^-]$ 21.54. Jugular venous blood mean values were PO_2 30.5, PCO_2 59.79, $[\text{H}^+]$ 46.73 and $[\text{HCO}_3^-]$ 29.95. In this group, the elevation of CSF PCO_2 and CSF H^+ , as well as arterial values, were observed.

In group 2 (16 cases), the arterial PO_2 was over 60 mm.Hg. The mean arterial values were PO_2 85.6, PCO_2 37.6, $[\text{H}^+]$ 39.81 and $[\text{HCO}_3^-]$ 22.68. The mean values of CSF were PCO_2 47.2, $[\text{H}^+]$ 54.92 and $[\text{HCO}_3^-]$ 18.91. The means of jugular blood were PO_2 36.6, PCO_2 52.5, $[\text{H}^+]$ 46.36 and $[\text{HCO}_3^-]$ 26.73. In this group a significant fall of CSF $[\text{HCO}_3^-]$ and slight increase of CSF $[\text{H}^+]$ were observed.

In dyspnea, the CSF acid-base balance should be measured in the cases without any change of arterial blood contents.

Conclusion:

The cerebrovascular response to hypoxia and hypercapnia was studied. In dyspnea, the clinical significance of the measurement of jugular venous O_2 and CO_2 tension and pH was stressed. The acid-base balance of cerebrospinal fluid was measured in normal subjects and the patients with dyspnea. The increase of hydrogen ion concentration in CSF was postulated as a factor responsible for dyspnea.