The Sequence of Alterations in the Vital Signs During Acute Experimental Increased Intracranial Pressure*

JAVAD HEKMATPANAH, M.D.

Division of Neurological Surgery, The University of Chicago Pritzker School of Medicine, Chicago, Illinois

REduced pulse and respiratory rate, dilatation of the pupils, increased arterial pressure, and alteration of the electroencephalogram are well known phenomena in acute increased intracranial pressure. The sequence of their development, however, is not well known and should be of value. In a series of experiments actually carried out to evaluate the cerebral circulation and perfusion during rising intracranial pressure, it was noted that the alteration of vital signs occurred in a sequential pattern, which we are reporting in this paper.

Technique

The cats were anesthetized by intraperitoneal injection of sodium pentobarbital (Dialbutal), 20 mg/kg. Tracheostomy was performed in all animals to prevent any chance of airway obstruction. Two supratentorial burr holes were made, one on each side near the midline of the skull. Through each burr hole a small collapsed balloon connected to a polyethylene tube was inserted into the epidural space, one for inflation and the other for monitoring intracranial pressure. Two small drill holes were made on each side behind the burr holes; steel screws were driven through these to serve as dural leads for electroencephalography. A polyethylene catheter was inserted through the femoral artery into the thoracic aorta to monitor the central arterial pressure. A Harvard pneumograph bellows was used to monitor the respiration. The epidural balloon monitoring intracranial pressure, the central arterial catheter, and the pneumograph bellows were each connected to the Beckman dynograph through strain gauges. The burr holes were filled with dental cement to seal the openings.

Before inflation of the balloon with saline, the strain gauges monitoring the arterial and intracranial pressures were standardized against a mercurial monometer. The amplification of the EEG electrodes was calibrated and the resistance of electrodes checked. A baseline recording was made for a period of 30 minutes to 1 hour.

Results

The records of 15 experiments suitable for study of the alterations in vital signs were analyzed. Although there were slight variations in different animals which will be discussed later, there were astonishing similarities.

The baseline intracranial (epidural) pressures were usually at or below zero mercury (Fig. 1). As the balloon was inflated with small increments of saline (usually 0.2 ml, occasionally 0.1 ml), the intracranial pressure rose. Depending on the size of the animal, the first few increments were not usually associated with a sustained rise in intracranial pressure. Although the pressure rose momentarily, it soon returned to or near its previous level. As the intracranial pressure was raised with additional increments, the vital signs altered in the following order.

Electroencephalogram. The first noticeable change occurred in the EEG recording of the side ipsilateral to the compression (Fig. 2). In three animals there were slight reductions of the respiratory rates simultaneous with the EEG. The electroencephalographic changes consisted of a declining amplitude and frequency.

Respiration and Pulse. As the first increments were added, the respiratory rate decreased (Fig. 3). In a few animals the pulse rate slowed a few beats but not significantly. Further inflation of the balloon was associated with a definite reduction of the pulse rate and marked slowing of respiration.

Blood Pressure. When the intracranial...
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Fig. 1. Baseline recordings showing the intracranial pressure (ICP), systemic blood pressure (BP), respirations (Resp), and the electroencephalogram (EEG) from left (L) and right (R) side. The recording paper is run with a speed of 25 mm/min. Periodically the paper is run with the speed of 25 mm/second to see the details (the lighter portion of the recordings). The pupils in this and the following figures are the tracings of actual photographs. Both the intracranial and the arterial pressure are measured in millimeters of mercury. Note that the former is registered at zero and the latter at about 140 mm Hg.

pressure reached about 50 mm Hg (680 mm H₂O) there was a periodic change of 2 to 3 mm Hg in the blood pressure. The blood pressure declined with respiration and rose in between (Fig. 3), giving a sinusoid appearance. Identical waves with a lesser amplitude were seen concomitantly in the intracranial pressure recordings. These sinusoidal waves were synchronous with the respirations and were present even before the cerebral compression. As the respirations became slower, with the increasing intracranial pressure, their amplitude increased.

When the intracranial pressure approximated the diastolic arterial pressure, the respiration became markedly slow and both pupils gradually became enlarged. The EEG slowing and flattening were now marked and bilateral, frequently displaying a rhythm synchronous with the heart beats. Usually at this stage an increment of 0.1 or 0.2 ml was sufficient to give a sudden parallel rise of the intracranial and arterial pressures. Sometimes arterial and cranial pressures rose spontaneously after a long stationary period. With this sudden parallel rise of pressures the respiration became short and shallow associated with periods of apnea. Both pupils now became larger and the electroencephalogram became flat.

The heart rate, however, usually increased as the arterial pressure rose. If the balloon was not deflated the intracranial and the arterial pressures declined in a few minutes, the pulse rate decreased significantly, and long periods of apnea occurred. The intracranial and systemic arterial pressure gradually declined to zero or just above it. Respirations ceased and finally the heart stopped; the pupils remained dilated (Fig. 3).

If the balloon was deflated the intracranial and the arterial pressures suddenly dropped, the former to zero or below it, the latter to a
level near its original. However, the animals usually died if the balloons were deflated after the peak in cranial pressure had been reached and when the dilated pupils and flat EEG had fully developed.

**Discussion**

The rise of the arterial pressure following the cerebral compression in animals was described by Cushing in 1901 and recently by Langfitt, et al., and Ishii, et al. The study of the records in our experiment reveal, as in Fig. 3, that the rise in arterial pressure occurs only late when the intracranial pressure is markedly elevated. We can, therefore, rightly conclude that the rise in arterial pressure was a late and not a useful sign for warning. Actually, the blood pressure may decline temporarily while the intracranial pressure is rising (Figs. 3 and 4).

It is a familiar fact that in patients with increased intracranial pressure the arterial pressure often is not elevated. Browder and Meyers doubted the reliability of the "classical pattern of signs" of increased intracranial pressure. Jackson found that the rise of the blood pressure was one of the late signs in patients with head injuries, and he pre-
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Fig. 3. The entire recording, excluding those few minutes with faster speed. Note the sequential alteration of the EEG, respiration, pupils, and blood pressure.

ferred to rely on direct measurement of the cerebrospinal fluid pressure through lumbar puncture. Fremont-Smith and Merritt found that "there was no definite change in the blood pressure with an increase of less than 800 mm in the cerebrospinal fluid pressure," in 122 cases of a high intracranial pressure due to increased intracranial content. In patients with brain tumors, also, Frazier and Wilson did not find elevated blood pressure.

In our experiments the alteration of the respiration occurred early and soon after the alteration of the ipsilateral electroencephalographic changes. If the percentage variation of the respiration, pulse, and blood pressure is charted against the rising intracranial pressure (Fig. 4), it will be noted that the percentage reduction from the original rate is more significant in the respiration. The pulse rate, although slowed initially after the reduction of the respiratory rate, rose again as the arterial pressure increased. The marked reduction of the pulse rate was

Fig. 4. The percentage variation of the blood pressure, pulse, and respiration plotted against the rising intracranial pressure. Note that respiratory change occurs earlier; its percentage variation is also more conspicuous. The blood pressure decreases somewhat before it rises. The pulse is slowed but becomes faster as the arterial pressure rises.
noted in the terminal stage when the cranial and arterial pressures declined.

Five animals died in spite of deflation of the balloon after the intracranial pressure was markedly raised. Since these animals and those who did not die were perfused at the end for evaluation of the cerebral perfusion, the mortality rate cannot be calculated. Mead, however, in 20 dog experiments designed to study this matter found that 19 died within 24 hours if the balloon was deflated later than 3 minutes after the electroencephalogram had become flat.

Except for the slight depression of the blood pressure in the majority of the animals in the earlier stages of rising intracranial pressure, the alteration of the blood pressure is consistent with the findings of previous investigators. In our experiment, however, there was not a "rebound" of the intracranial pressure as described by Langfitt, et al., and Ishii, et al. who performed a series of experiments on dogs to evaluate decompression in experimental head injuries, noted that there was a rebound phenomenon following the deflation of the balloon. But Moody's impression was that the rebound phenomenon was seen in those animals who were given artificial respiration.

Summary

Acute increased intracranial pressure in cats caused by inflation of an extradural balloon is associated with a sequential alteration in vital signs. The first noticeable change occurs in the ipsilateral EEG. This is soon followed by a reduction in respiratory rate. Although the pulse rate may be slightly reduced, the significant reduction occurs later, and marked reduction occurs only in the terminal condition when the intracranial and arterial pressures decline. The blood pressure rises quite late, when the intracranial pressure approximates the diastolic pressure. The ipsilateral pupil frequently dilates before the rise of the arterial pressure, and both pupils are dilated when there is sudden and sustained rise in arterial pressure. At this stage the electroencephalogram is usually flat bilaterally.

References