PHYSIOLOGY OF HEAD INJURY IN THE EARLY STAGE

(EXPERIMENTAL STUDY ON THE SO-CALLED
INITIAL SHOCK AND APNEA)

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SO-CALLED INITIAL SHOCK

It is well known that the most cases which die in the early stage after severe head injuries give rise to shock-like symptoms. However it has been some question whether these symptoms derive from the real traumatic shock itself. In our Department of Surgery, the clinical and experimental studies on the trauma have been studied using three shock indicators, i.e. the increased amino-N of arterial blood, decreased pH and CO₂ content in the traumatic or electric shock. The past ten year's research in our Department has brought the following conclusion about the traumatic or electric shock.

1. The electric shock is a traumatic shock.
2. The electric current of more than 8000 Joule which is applied to the medial aspect of bilateral thigh of rabbits always induces the electric shock.
3. These electric shock does not occur when the animal is anesthetized, or, its spinal cord or visceral nerve is incised before the electric current is applied.
4. From these experiments the increase of amino-N and the decrease of pH and CO₂ in arterial blood are proved to be the suitable indicator to evaluate the traumatic shock.

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5. The above mentioned three major indicators of arterial blood in most patients who sustained severe trauma in some parts of body except the head show the specific pattern of shock indicator. However, those of patients who sustained the heavy head injury do not show the pattern of the traumatic shock.

Method:

The experimental head injury was produced by dropping a lead-made weight from a height on the head of rabbit through the pipe which is fixed on the surface of anterior portion of specimen's skull.

By the head blow to the animal it results in typical neurological signs as the clinical head injury does; the pathological and EEG findings show the evidence of the sufficient head injury.

Results:

1. The animal which sustained above mentioned experimental head injury does not show the traumatic shock from the standpoint of three shock indicators.

2. When more than 8000 Joule electric current is applied on the medial aspect of both upper thighs soon after the experimental head injury, one group of animals died in the early stage shows the severe traumatic shock pattern and the other group does not show the shock pattern and survives for longer time. Namely, the head injury applied prior to the electric current inhibits the appearance of electric shock, but sometime both effects of head injury and electric current overlap the shock on the animal.

The similar result is obtained from our clinical data as shown in Table 1. These four cases sustained severe head injury as well as the severe trauma on the other part of body, two of them show the typical traumatic shock pattern but other two cases do not.

3. When more than 8000 Joule electric current is applied to the medial aspect of upper thighs about 30 to 40 minutes after the experimental head injury, they survive for longer time and do not show the traumatic shock pattern. Namely, the head injury applied sufficiently long time prior to the electric current application has inhibited the traumatic shock in the animal.

As mentioned above in the experimental head injury the animal shows symptoms similar to those of traumatic shock from the clinical standpoint, but they do not show any characteristic shock picture of trauma, from our measurement using three major shock indicators.

However, in the head injury accompanied with other injuries might sometimes appear the characteristic shock pattern. It might be concluded that the

Table 1
Chemistry of Blood in Cases of Head Injury, Accompanied with Other Injury

		Time when blood with-drawn after injury	Blood pressure	Arterial blood		Amino-N	Ht.	P. P.	Prognosis
				CO ₂	O ₂				
1. ■	Head injury, fracture of lumbar spine	25 min	110/70	41.3	10.7	8.38	37	6.5	Died within one hour and 55 minutes
2. ■	Fractures of left thigh and clavicle, head injury	17 hr	110/58	44.4	10.1	8.5	24	6.4	Recovered
3. ■	Head injury, contusion	55 min	100/50	20.0	10.9	10.7	40	7.2	Died within twenty minutes
4. ■	Contusions of forehead and face	25 min	70/50	44.8 ↓ 17.7	11.2 ↓ 10.6	7.88 ↓ 8.7	39	5.6	Died within four hour and fifty minutes

damage in the brain may inhibit the shock just like the general anesthesia which was proved in our past series and also if the injury is applied to the other part of body it is much heavier than that of the brain, the shock is not inhibited by the damage of the brain.

RESPIRATION AND HEART BEAT IN THE EARLY STAGE OF HEAD INJURY

It is well known fact that the direct cause of death in the early stage of head injury is the cease of respiration, i.e. in the clinical head injury as well as the experimental one, the heart beat of patient and animal continues to work for longer time even after the respiration stops and if they receive the artificial respiration for a while they can restore the respiration. Therefore this type of apnea in the early stage of the head injury seems to be the reversible phenomenon.

This mechanism of the early cease of respiration in head injury was discussed by several experiments as well as the heart function.

Method:

The blood pressure cuff is wound around the chest of animal and a tube is connected with the tambour which is arranged to transmit the respiratory movement on the kimogram.

A fine polyethylene tube was inserted into the femoral artery and blood pressure was recorded on the same kymogram. In the particular group ECG was taken under the complete relaxation of muscles using curare or succine with artificial respiration and sufficient oxygen supply. After the animal dies the head of specimen is removed and fixed in the formalin solution, then the scalp is removed to evaluate the intracranial damage. The histopathological examination was done by the hematoxylin-eosin staining from the five frontal section at each portion of brain.

Results:

1. Respiration

The respiration immediately after the head injury is classified as five main types (A-E) as shown in Fig. 1. Each type has appeared with almost same frequency when the 140 cm/800 gm head blow was applied. However, this frequency changes under several conditions as follows.

Fig. 1 Patterns of respiration.

1) Pathological findings: Histopathological findings of animal revealed damages of pons and medulla which were apparently less than that of the cerebrum and even the case which died immediately after the head blow under the E-type apnea did not show particularly the heavy damage in the pons and medulla.

2) General anesthesia: If the animal is anesthetized by the sodium penthotal intravenous administration before the head blow, the occurrence of the E-type is remarkably inhibited. Namely the animal is hardly died from the apnea immediately after the head injury under the general anesthesia.

3) Chlorpromazine: The E-type of respiration does not appear when the chlorpromazine is given intravenously before the head injury.

4) Repeated head injury: If the light head blow is given repeatedly before the standard heavy blow, the occurrence of E-type apnea is remarkably inhibited.

5) Cérébri isolé: In the *cérébri isolé* specimen the E-type apnea never happens even by the heavier head blow than the standard one and the animal continues the calm and regular respiration like A-type.

2. Heart beat and blood pressure

By the heavy head blow the blood pressure elevates soon after the head blow always with the typical tonic cramp and falls gradually then expires. If the blow is not heavy enough, the blood pressure does not elevate or elevates little and then continues to maintain the initial level.

This type of the blood pressure is classified in eight types as Fig. 2. ECG shows temporary interruption of the heart beat followed by arrhythmia immediately after the head blow.

Fig. 2 Patterns of blood pressure after head injury.

1) General anesthesia: The animal does not have tonic cramp and also has no elevation of the blood pressure at all. One case which had the elevation of the blood pressure had the tonic convulsions at the same time.

2) Chlorpromazine: The animal has no tonic cramp as well as the elevation of blood pressure.

3) Repeated head injury: After the repeated head injury the standard heavy blow does not bring the elevation of the blood pressure and tonic cramp just like the anesthetized animal.

4) Cérébri isolé: The elevation of blood pressure is inhibited remarkably and the blood pressure is inclined to have a tendency to drop immediately after the blow.

5) Banthine and vagotomy: When banthine is given or both vagus are incised before the head blow the post-traumatic interruption of heart beat does not occur at all.

6) Curare and succine: The animal to which enough dosis of curare or succine was given for complete muscle relaxation under the artificial respiration with enough oxygen was used for investigation.

CSF PRESSURE IN HEAD INJURY

Some people have believed that the cerebral concussion is due to the rapid elevation of CSF pressure but the hypothesis has been denied by the several neurophysiologists.

In our series the CSF pressure was investigated at the time of head blow and particular method was invented to produce the head injury by producing the rapid change of CSF pressure.

1. CSF pressure in head injury.

Cisternal space was punctured with a needle which is connected to the manometer with fine polyethylene tube and CSF pressure at the time of head blow was investigated. Right after the head blow, there happens remarkable shaking in CSF pressure as shown in all cases (Fig. 3), this shaking does exist in the cisternal space of the carcass of the rabbit when the same head blow is given.

Fig. 3 Pattern of CSF change.

After this shaking some cases do not have any change in CSF pressure, some have gradual elevation and some show rapid elevation to the unlimited.

heights. The last always has massive intracranial bleeding which comes out through the manometer.

2. New method of producing the head injury without the damage in the brain.

A hard rubber-made balloon was connected with cisternal space using polyethylene tube and the balloon with the tube were filled with normal saline solution. A weight which was used in the previous experiment was dropped on the balloon through the pipe which was fixed on the middle of balloon. After the blow on the balloon the animal shows just the same symptoms, respiration and blood pressure pattern as did in the previous experimental head injury.

It was proved that water in the balloon did not go into the cisternal space by the fact that the dye injected into the balloon did not go into the cisternal space by the blow on the balloon.

SUMMARY AND DISCUSSION

1. The apnea immediately after the head injury is a reversible phenomenon and is not necessary to accompany the severe lesion in pons and medulla where the respiratory center is supposed to be.

2. It has been reported by the neurophysiologist that the several areas of the cerebrum in frontal and temporal lobes play an important inhibiting role for the respiration. This type of the respiratory inhibition must have very important role in the apnea after the head blow i.e. the inhibitory areas are stimulated by the direct head blow and inhibitory impulse comes down to stop the respiration.

The reason why the apnea is inhibited in the *cérébri isolé* animal and anesthetized or chlorpromazine-administrated animal or by the repeated head blow must be explained rationally by the fact that the inhibitory center is blocked by the intracollicular incision, paralyzed by the anesthesia and chlorpromazine; and damaged by the repeated head blow.

3. The temporary elevation of blood pressure is mainly due to the tonic cramp following to the head blow, because the blood pressure rather tends to drop under the complete muscle relaxation by curare and succine.

4. The temporary interruption of heart beat is seemed to be due to the direct excitation of vagal center and the cerebral cortex does not play an important role in apposition to the respiration cease. The fact that the heart function is more stable than the respiration is the very important characteristic feature of the head injury.

5. The elevation of CSF pressure is not necessary factor for the cease of respiration, but rapid and much amount of intracranial bleeding causes the rapid stop of respiration.

6. A new type of head injury which has no damage of the brain substance is produced by the balloon method. The cause of apnea is supposed to be the direct mechanical impulse which is produced by the rapid change of CSF pressure or vibration.

CONCLUSION

1. In the early stage of head injury three indicators do not show the shock pattern if the individual has no trauma in other parts of body unless it has superimposed head injury, the latter shows the shock pattern easily.

2. Heavy damage in the brain apparently inhibits the shock even if the enough dosis of the electric current is given to produce the shock.

3. In the apnea of the early head injury respiratory inhibition from the cerebral cortex plays rather important role for the apnea than the damage in the respiratory center in pons and medulla themselves, therefore the occurrence of this reversible type of apnea is inhibited by the general anesthesia, chlorpromazine, *cérébri isolé* as well as in the repeated head injury.

4. Temporary interruption of the heart beat happens right after the head injury and this is due to the direct impulse to the vagal center.

5. The temporary elevation of the blood pressure immediately after the head blow is due to the tonic cramp and the blood pressure rather drops under the complete relaxation of muscles.

6. There is a typical shaking of CSF pressure right after the head blow in all cases; but animal may expire under the E-type apnea without the elevation of CSF pressure following to the shaking.

7. Experimental head injury without damage in the brain is produced by the new balloon procedure which makes the vibration or rapid change in CSF pressure by blowing the balloon connected to the cisternal space.

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