Cerebral Hemodynamics and Metabolism Following Experimental Head Injury*

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The pathogenesis of cerebral concussion has long been debated. The present experiments were undertaken to clarify the nature of acute cerebral disorders resulting from head injury.

Concussion was defined by Denny-Brown as a "transitory and reversible nervous reaction with immediate onset following physical stress of sufficient violence and brevity, and characterized by progressive recovery thereafter."96 There are two main theories concerning the pathogenesis of concussion: the excitation theory of Walker, et al.,43 and the paralytic theory of Denny-Brown and Russell. These two theories, which postulate opposite mechanisms, will be reviewed.

Walker and his associates43 observed the appearance of fast activity in the electroencephalogram (EEG) with little change in amplitude immediately after a compressive impact applied to the exposed dura and brain in experimental animals. This was followed by "extinction." The EEG changes were frequently accompanied by tonic extension movements of the extremities. They suggested that this type of concussion resulted from excitation of the central nervous system.

An opposite view was proposed by Denny-Brown and Russell. Based on experimental observations of concussion produced by a pendulum striking the freely moving head (acceleration concussion), they concluded that this type of concussion was due to temporary paralysis of nervous function.

In man, concussion is characterized by transient loss of neural function, accompa-

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nied by loss of consciousness. In animals, concussion can only be defined by accompanying cardiorespiratory changes, loss of the corneal reflex of varying duration, and according to some,43 a convulsive spasm. Because of difficulties in measuring loss of consciousness and amnesia in animals, criteria for experimental concussion that are generally accepted are loss of the corneal and tendon reflexes, changes in cardiorespiratory function, and abnormalities in the EEG.

Little information is available concerning changes in cerebral blood flow (CBF) and metabolism following experimental brain injury in animals, and this is often conflicting.¹,8,14,26,37

The present investigation was designed to provide continuous measurements of CBF and cerebral oxygen consumption (CMRO₂) following experimental brain trauma. Measurements were also made of cerebral lactate and pyruvate production. These measurements were correlated with the excitation type of concussion described by Walker, et al., and the paralytic type described by Denny-Brown and Russell. Cerebral hemodynamics and metabolic changes were also correlated with cerebral contusion and laceration of the brain stem.

Methods

We conducted 64 experiments in 58 baboons weighing 3 to 8 kg. The animals fasted for 24 hours before the procedure. Anesthesia was induced by the intravenous injection of sodium pentobarbital in doses of 5 to 10 mg/kg body weight plus inhalation of ether and by local infiltration of 0.5% lidocaine at all operative sites. Atropine sulfate (0.4 mg) was given intramuscularly.

Body temperature was maintained between 35° to 37°C with a heating pad. Tracheostomy was performed, but artificial respiration was not employed since cardio-
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respiratory changes are important criteria for estimating the effects of concussion.

End-tidal CO₂ was monitored with a Beckman infrared gas analyzer, and if the carbon dioxide was not within the range of 30 to 42 mm Hg prior to injury, the data was discarded. Systemic blood pressure was recorded with a Statham pressure transducer connected to a catheter inserted into the abdominal aorta. Blood samples for measuring cerebral arteriovenous metabolic differences were taken from the femoral artery and a catheter inserted into the torcular.

A burr hole was made in the region of the occipital protuberance, and a small (0.9 mm in outer diameter) catheter was inserted into the torcular for sampling the cerebral venous blood. Another burr hole was made in the parietal region for inserting a small rubber balloon for recording intracranial pressure. A third burr hole was made in the opposite parietal region into which was screwed the muzzle of an airgun covered with a rubber membrane for providing the concussive blow. After adjusting the apparatus, each hole was tightly sealed with methylmethacrylate plastic cement.

Both internal jugular veins were exposed and their cervical branches ligated. The vertebral and external jugular veins were all exposed and ligated. Cerebral venous outflow was measured continuously by two electromagnetic flow meters whose probes were applied about each internal jugular vein. To calculate CBF in ml/100 gm brain/min, a correction was made for the sinus blood removed for metabolic measurements. The scalp and temporal muscles were incised and widely reflected, and six electrodes were inserted through drill holes in the frontal, parietal, and occipital regions for recording the EEG. The electrocardiogram (EKG) was also recorded.

The compressive blow was delivered by a specially constructed airgun.* The firing pressure was held constant at 40 lbs per sq inch, but the duration was varied between 10 ms and 300 ms. The longer the duration of the blow the more severe the concussion or contusion. The monkey was placed in the supine position with the neck slightly extended and the head fixed. Brain stem laceration was performed by thrusting a small scalpel into the exposed brain stem in the region of the pons, but care was taken to avoid laceration of the basilar artery and its major branches.

Cerebral arteriovenous oxygen differences (cerebral A-VO₂ differences) were monitored by the use of a Guyton A-VO₂ oxygen analyzer. Mean values for cerebral A-VO₂ difference obtained from the Guyton recorder agreed within 2% with the mean values obtained by the standard Van Slyke manometric method. The CMRO₂ was calculated from the product of CBF and cerebral A-VO₂ difference, and it was expressed as ml/100 gm brain/min.

Arterial (a) and cerebral venous (CV) values for pH were monitored with Beckman pH electrodes, values for oxygen tension (PO₂) with a Clark type electrode, and carbon dioxide tension (PCO₂) with a modified Severinghaus electrode mounted in a cuvette maintained at 37°C. The animals were given intravenous heparin prior to these procedures.

Blood samples for lactate and pyruvate measurements were drawn from the femoral artery and from the torcular, and estimated by the colorimetric method of Hochella and Weinhouse for lactate, and by the fluorimetric method of Segal, et al., for pyruvate. Cerebral lactate and pyruvate production (CMR lactate and CMR pyruvate) were calculated from the product of the CBF, and the cerebral arteriovenous lactate and pyruvate differences were expressed as mg/100 gm brain/min. Excess lactate formation by the brain was calculated from the formula of Huckabee expressed as mg/100 ml.

All recordings were graphed on a Grass Model 5 polygraph and a Grass EEG machine. Mean arterial blood pressure (MABP) was expressed in mm Hg (torr) by adding one-third of the pulse pressure to the diastolic pressure. Cerebral vascular resistance (CVR) was calculated by dividing MABP by CBF and expressed as mm Hg/ml/100 gm brain/min. At the end of each experiment, the brain was weighed and serial sections were examined.

The data were subjected to statistical analysis.

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analysis by means of the paired and unpaired "t" tests.

**Results**

Results were categorized into five groups according to the nature and the severity of the blows as follows:

*Group 1.* Subconcussion resulted from blows of less than 10 ms duration.

*Group 2.* Mild concussion from blows of 50 ms duration.

*Group 3.* Severe concussion from blows of 150 ms duration.

*Group 4.* Contusion from blows of 300 ms duration.

*Group 5.* Brain stem laceration from the thrust of a small scalpel in the pons.

A blow was considered to be concussive whenever cardiorespiratory changes appeared, even if EEG changes were minimal and loss of the corneal reflex was uncertain. There were no measurable effects obtained from subconcussive blows.

In the group with concussion, no changes were noted in the brain at necropsy except in three out of 18 cases, all with severe concussion that showed slight subarachnoid hemorrhages in the cerebellar tonsils.

Following cerebral contusion, petechial hemorrhages were regularly seen around the zone of impact and in the cerebellar tonsils, and the brain was diffusely swollen.

After brain stem laceration, brain swelling with tonsillar and tentorial herniation was noted.

A few cases of "fatal concussion" were observed without evidence of damage to the brain at necropsy. Following the fatal blow, the EEG became isoelectric, blood pressure fell to zero, accompanied by a rapid decrease of CBF, CMRO₂, end-tidal CO₂, and CvpH.

**Effects On Corneal, Tendon, and Limb Reflexes.** The corneal reflex was usually lost for about 3 to 5 sec following a concussive blow, 20 sec being the longest interval. In some cases of mild concussion (5 out of 19), tonic and clonic extension movements of all four extremities were noted for some 5 to 60 sec after the blow. Sometimes the tendon reflexes were reduced following the blow. Convulsive movements were seen less frequently (2 out of 18) in the group with severe concussion, and none was noted with contusion.

**Respiratory Changes.** Respiratory changes occurred immediately after all types of brain injury. These consisted of irregular gasps for about 20 sec, or transient apnea followed by hyperventilation for some 1 to 2 min. The mean duration of respiratory arrest was 5 sec for mild concussion, 13 sec for severe concussion, 11 sec for contusion, and 20 sec for laceration. These respiratory disturbances caused a transient decrease in the partial pressure of arterial oxygen (PaO₂) and an increase in the partial pressure of arterial carbon dioxide (PaCO₂). Following brain stem laceration, respiratory disturbances usually occurred some minutes after the injury and caused marked decreases in PaO₂ and increases in PaCO₂.

**Blood Pressure and EKG Changes.** Concussion or contusion usually resulted in a rapid decrease in blood pressure followed by a rapid rise above resting levels for about 5 to 15 sec, or a return to normal over an interval of 2 to 5 min. In some cases, the blow produced a rapid increase in blood pressure without an initial fall and returned to normal over the ensuing 4 to 5 min. Cerebral contusion usually resulted in a small decrease in MABP following the blow. Brain stem laceration was accompanied by a precipitous fall in BP followed by severe to moderate hypertension which persisted until the experiments were terminated.

Marked slowing and irregularities in the EKG were commonly noted after almost all types of brain injury associated with the changes in blood pressure. The pulse rate and rhythm returned to normal within a few minutes, except in some cases of brain stem laceration in which irregular rhythm often persisted for 15 to 30 min.

**Cerebral Vascular Resistance.** The CVR increased in all groups following the blow. In concussion, the mean increase was 10%, in contusion the increase was 34%, and in brain stem laceration, the increase was 57%. In general, the more severe the blow, the more the CVR was increased.

At the moment of impact, the intracranial
pressure (ICP) showed a sudden rise followed immediately by a fall below the initial level and a return, thereafter, to levels that were normal or elevated. Then, after a mild concussive blow, the ICP became elevated for some minutes but gradually returned to control values within 15 min. After severe concussion, the ICP remained increased for over 15 min. Following contusion, the ICP became progressively and severely increased. Laceration of the brain stem produced the most profound and progressive increase (over 200%), and this sometimes terminated in herniation of the brain at the tentorium and foramen magnum.

Cerebral Hemodynamics and Oxygen Consumption Correlated with EEG Changes. In all groups, the CBF was decreased immediately after the blow for 1 or 2 sec, and this was usually followed by a brief increase above control levels. Following mild concussion, the CMRO₂ was transiently increased after the blow (Fig. 1) which correlated in 10 of 19 cases with a burst of low voltage, fast activity at

![Graph](image-url)

**Fig. 1.** The mean metabolic effects of a mild concussive blow calculated for the series of 19 experiments. CMRO₂ transiently increased immediately after the blow. MABP also increased. There was a small increase of CBF which gradually decreased.
14 to 20 cps in the EEG. In the remainder, the concussive blow was followed by no changes, or "extinction" \(^1\) to transient slowing of the EEG, but no spike discharges were seen (Fig. 2). Arterial and cerebral venous pH showed no changes.

On the other hand, severe concussion significantly decreased both CBF and CMRO\(_2\) for about 15 min (Fig. 3). The reduction of CMRO\(_2\) was accompanied by temporary slowing in the EEG at 5 to 7 cps with or without an increase in amplitude (Fig. 4). Changes in PaO\(_2\), arterial pH (apH), and PaCO\(_2\) appeared to result from irregularities of respiration. There were no changes in cerebral venous (CV) pH or PO\(_2\).

Cerebral contusion, like severe concussive blows, decreased both CBF and CMRO\(_2\), but unlike concussion, these tended to progress following the blow without recovery to normal levels (Fig. 5). The reduction in CMRO\(_2\) correlated with EEG changes consisting of persistent slowing in the 5 to 7 cps range without recovery (Fig. 6). Cerebral venous PO\(_2\) and pH both decreased after cerebral contusion.

Shortly after brain stem laceration, CMRO\(_2\) was reduced significantly by 25\%, accompanied by a decrease in CBF but not of the same magnitude as the reduction of CMRO\(_2\) (Figs. 7 and 8). The cerebral A-VO\(_2\) difference was decreased at this time, and cerebral venous PO\(_2\) increased by 18\%. Within 5 min after injury to the brain stem, respiratory irregularity caused increases in arterial PCO\(_2\) accompanied by decreases in arterial PO\(_2\) and pH so that cerebral venous PO\(_2\) eventually decreased. Cerebral venous pH became significantly decreased. The EEG showed slowly progressive but diffuse slow activity beginning 1 to 2 min after injury to the brain stem (Fig. 9) and often became isoelectric after 15 or more min.\(^2\) The EEG changes were well marked within 10 to 15 min and correlated well with the depression in CMRO\(_2\).

**Cerebral Lactate and Pyruvate Production.** Following concussive blows, there were no changes in either CMR lactate or pyruvate (Fig. 10).

Following cerebral contusion, CMR lac-

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**Fig. 2.** The EEG effects of a mild concussive blow. Note the appearance of a burst of low voltage fast activity at 14–20 cps. The EEG change was accompanied by an increase in CMRO\(_2\) and tonic movements of the extremities.
**Fig. 3.** The mean effects in 18 experiments of a severe concussive blow. CBF decreased along with the reduction of CMRO$_2$ by 9%. Thereafter, CMRO$_2$ gradually returned to control levels during the next 15 min. The CVR became increased shortly after the blow. There were no changes in CvpH or CvPO$_2$.

tate started to increase 5 min after the blow and became significantly increased within 20 min (Fig. 11). CMR pyruvate showed little or no increase, so that there were increases in excess lactate. Increases in cerebral lactate production were even more remarkable following brain stem laceration, CMR lactate becoming significantly increased within 5 min after the trauma (Fig. 12). There was no change in CMR pyruvate; hence there was a profound and significant increase in excess lactate which correlated with the decrease in cerebral venous pH and cerebral venous PO$_2$.\textsuperscript{33,41}

**Discussion**

**Methodology.** There are two different methods for producing experimental concussion. "Acceleration concussion" is one method whereby a mass moves at a known velocity to strike the freely movable head. "Compression concussion" is the other more widely used method by which concussion is produced by compression of the brain with the head fixed. Compression concussion has the advantage of being readily reproducible and produces less disturbance to apparatus attached to the head and neck. Although the resulting EEG changes are similar by the
two methods, cardiorespiratory effects are different. Arterial hypertension and respiratory disturbances are more marked following acceleration concussion, and bradycardia is more severe in compression concussion. Despite these technical differences, identical physiological results can be produced by either method.\textsuperscript{6,10,42}

In the present series of experiments, in accordance with reports of Gurdjian, \textit{et al.},\textsuperscript{11} the parameters measured varied according to the duration of the impact. Measurement of the CBF using electromagnetic flow probes placed about the carotid arteries has been justly criticized in the past because of the inevitable extracerebral contamination. In the present study, the CBF was measured by recording the cerebral venous outflow which has been shown to be free of this objection.\textsuperscript{30,31}

In the present series of experiments, the mean control value for CBF was slightly lower than that reported previously\textsuperscript{31} in rhesus monkeys, probably because of differences in the depth of anesthesia and possible differences in species.

Following cerebral trauma, changes in CBF were rapid within the first few seconds following the blow, but tended to become stable within the ensuing 30 sec. In the statistics of the present study, therefore, calculation of CBF was started 1 min after the trauma, when CBF became stable and a steady state was achieved. Values for CBF and cerebral A-V\textsubscript{O}\textsubscript{2} differences were obtained using mean values calculated during intervals of every 20 sec.

\textit{Criteria For Concussion}. The corneal reflex was not always absent after a concussive blow, even when there were changes in vital signs and EEG. Thus, the corneal reflex was not a reliable index of cerebral concussion.

Respiratory disturbances arising from the head injury correlated well with the intensity of the blow. In every group, the PaCO\textsubscript{2} was

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig4}
\caption{As shown in this typical illustration, a severe concussive blow produced slow activity (5–7 cps) in the EEG which gradually returned to control frequencies over the next 15 min.}
\end{figure}
The mean hemodynamic and metabolic effects of cerebral contusion in 18 experiments. Cerebral contusion was accompanied by a decrease in both CBF (by 19%) and CMRO₂ (by 17%). The CVR showed a marked increase, and CvPO₂ gradually decreased.

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Cerebral Hemodynamics Following Head Trauma. Changes in MABP were not identical in each type of head injury. It has been said that in mild concussion, excitation of the vasomotor center in the brain stem may lead to peripheral vasoconstriction.¹⁴ In severe or fatal concussion and contusion in the present experiments, it appeared that there was paralysis of the vasomotor center, and the tone of peripheral vessels appeared to be decreased or lost. Following brain stem laceration, however, the BP increased, which correlated with the progressive increase in ICP. The increase in BP was probably due to the direct effect of trauma and distortion of the brain stem with ischemia of the vasomotor center,¹⁴ resulting in peripheral vasoconstriction.

The increased intracranial pressure after elevated during the interval of respiratory disturbance occasioned by the blow. The hyperventilation which usually followed was presumably caused by the hypercarbia stimulating the chemoreceptors in the medulla.²⁵ It was concluded that the cardiorespiratory disturbances immediately following the blow were due to transmitted effects on the medullary centers which are known to vary with the severity of the blow.¹²,⁴²,⁴³
cerebral trauma is known to be due to increased cerebral blood volume, cerebral vasodilatation in the presence of a raised blood pressure, and capillary-venous injury with resulting swelling of the brain.22-24,39,40

The increase in CBF following mild concussion was attributed to the fact that cerebral vascular tone was decreased immediately following cerebral trauma. This was due to loss of autoregulation, since it occurred before any change in PaCO₂. Hence, cerebral blood volume increased along with the simultaneous increase in BP, transmitting the high perfusion pressure directly to the capillaries and veins, resulting in brain swelling and increased ICP and CVR.44,45

The consistent and progressive increase in CVR following severe concussion and contusion also appeared to result from impaired autoregulation immediately after the blow, resulting in cerebral congestion and transmission of perfusion pressure directly to cerebral capillaries and veins, causing cerebral swelling, perivascular hemorrhage, and edema.

In fatal concussion, the cause of death was attributable to acute neurogenic circulatory failure.10

Following brain stem laceration, it appeared that progressive ischemia of the brain stem caused an increase in blood pressure, and this, together with loss of autoregulation, resulted in cerebral edema and a profound increase in ICP, later complicated and worsened by respiratory disturbances resulting in cerebral hypoxia, carbon dioxide retention, and cerebral metabolic acidosis.27

Cerebral Metabolism After Head Injury.

The transient increase in cerebral metabolism resulting from a mild concussive blow, associated with desynchronization of the
EEG, appeared to indicate brief cerebral excitation induced by the blow. Presumably, the transmitted force acted as a stimulus to the brain stem reticular formation depolarizing its nerve cells with widespread excitation of the nervous system. In contrast to this, after severe concussion, contusion, or laceration, the CMRO₂ was decreased and there was slowing of the EEG. This is believed to indicate paralytic injury to the reticular formation with inhibition of its normal excitatory “drive,” producing a state of diaschisis. In general, the greater the blow the greater the depression of CMRO₂, although the site of injury was important, since the greatest depression of CMRO₂ resulted from a laceration or injury limited to the brain stem.

After laceration of the brain stem, the CBF was reduced relatively less than the marked decrease in CMRO₂. Thus, a “luxury perfusion syndrome” existed for a few minutes after trauma, wherein the CBF was in excess of cerebral metabolic requirements. Hence, cerebral venous oxygen tension rose, as cerebral A-VO₂ difference decreased. The decrease in cerebral venous pH was attributed to the increased cerebral lactate production, and this would further increase the cerebral venous PO₂ by the Bohr effect.

It is well known that an early step in glycolysis by the brain is conversion of glucose to pyruvic acid by phosphorylation. In the presence of adequate oxygen, pyruvic acid is oxidized further to carbon dioxide and water through the tricarboxylic acid cycle. As demonstrated by the pioneer work of Gurdjian, et al., either the absence of sufficient oxygen for cerebral metabolism or the effects of trauma to the brain impair this pathway with increased lactic acid formation. These authors suggested that such biochemical

* The Bohr effect is the effect of pH on the dissociation of oxygen from hemoglobin. The more acid the blood, the greater the dissociation of oxygen and hence the higher the plasma PO₂.
changes in areas of contusion may be the result of a combination of both of these factors, i.e., direct injury to the cells and later anoxia resulting from vascular damage. The present data confirm this view.

Some have questioned whether lactate is able to diffuse from nerve cell membranes into the cerebral venous blood. However, many investigators now agree that both pyruvate and lactate cross cell membranes, and blood levels, may be used as an indicator of intracellular events.

Cahn, et al., were among the first to demonstrate increased production of lactate in the cerebral venous blood due to impaired oxidative metabolism of the brain resulting from anoxia. Others have shown that besides trauma and anoxia, increased lactate production may be measured in the cerebral venous blood in such non-injurious conditions.

FIG. 8. The mean effects on cerebral hemodynamics and metabolism in nine experiments of laceration of the brain stem. After laceration of the brain stem, CMRO₂ became greatly reduced without any significant change in CBF. Hence the cerebral A-VO₂ difference became significantly decreased, CvPO₂ increased and CvpH decreased, indicating "luxury perfusion syndrome." About 5 min after the injury, CvPO₂ and CvpH became reduced, accompanied by increased cerebral lactate production.
Fig. 9. Typical EEG changes caused by laceration of the brain stem. Following laceration of the brain stem, the EEG showed progressive slowing and often became isoelectric.

Fig. 10. Following severe concussive blows, there were no significant increases in lactate production in 18 experiments, despite the temporary decrease in CMRO₂.
as infusions of glucose, pyruvate, insulin, or epinephrine. Presumably, these substances, when infused, increase total glycolysis.

Values for excess lactate were calculated in the present series of experiments and were also found to be greatly increased, confirming that there was an increase in lactate production. The increase in cerebral lactate production resulted from anaerobic glucose metabolism, which in turn was caused by traumatic or hypoxic damage to the normal aerobic pathway using the Krebs cycle.

It will be noted that despite the reversible depression of CMRO$_2$ in severe concussion, cerebral lactate production was not increased. However, in the prolonged depression of CMRO$_2$ resulting from contusion and brain stem laceration, lactate production was increased.

Correlation of the timing of depressed CMRO$_2$ with the appearance of increased lactate production in contusion and brain stem laceration showed that the lactate appeared within the cerebral venous blood after a delay of several minutes. The delay in the appearance of lactate may indicate that it takes several minutes to diffuse across the membranes in quantities sufficient to be measured by the method used, or, more likely, that prolonged decrease in oxygen delivery to the brain was necessary before cerebral lactate production increased sufficiently to be measured.

Summary

Cerebral hemodynamics and metabolism were measured in baboons following concussion, contusion, and brain stem laceration. A mild concussive blow of short duration caused temporary excitation of the central nervous system, manifested by EEG desynchronization and convulsive movements of the extremities and by an increase of...
cerebral oxygen consumption (CMRO₂).
Severe concussive blows caused transient slowing in the EEG accompanied by transient decrease in CMRO₂, indicating reversible paralysis of the central nervous system.

Contusion caused by blows of longer duration produced more progressive and permanent slowing of the EEG accompanied by corresponding decreases in CMRO₂ and cerebral blood flow (CBF). Following cerebral contusion, there was a significant increase in lactate production by the brain.

Shortly following brain stem laceration, a "luxury perfusion syndrome" was noted since CMRO₂ was greatly reduced while CBF was decreased less. At a later interval, CMRO₂, CBF, cerebral venous oxygen tension (CvPO₂), and cerebral venous pH (CvpH) became reduced, associated with marked slowing in or loss of the EEG. There were marked increases in cerebral lactate production following brain stem injury.

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