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16. Abstract

This paper presents clinical observations, physiological data and pathological findings that have been collected on a series of baboons exposed to controlled occipital impacts under local anesthesia. This acute experimental trauma study was accomplished to establish trauma mechanisms and compare the relative effects of impact with supported heads and heads capable of responding to impact in a controlled horizontal A-P plane. Fourteen laboratory-conditioned test subjects were surgically instrumented to obtain EEG, bilateral carotid blood flow and aortic and venous blood pressures. Standard ECG and pulse and respiration rates were recorded in addition to the clinical observations of pupillary responses and corneal reflexes that were documented with high-speed movies. Pre-impact and post-impact data baselines were continuously recorded with the impact event. Occipital blows were delivered in the midsagittal plane (parallel to the Frankfort plane) with a pneumatic piston containing load cells and accelerometers. The piston was programmed for a stepped-impact profile of 42 to 52 feet per second velocity.

In all tests with the supported and mobile heads, there was apparent reversible stunning, mydriasis, bradycardia, absence of corneal reflexes and EEG slowing. Anterior (opposite) surface contrecoup trauma was observed in all tests with controlled-head motion and contrecoup trauma was produced with the supported-head tests. A unique degenerative nerve cell change was found in all traumatized subjects.

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to contact the dura. Sterile normal saline solution was placed extradurally in both preparations to maintain a fluid interface with the transducer. The transducers were aligned in an anterior-posterior plane five millimeters parasagittally to the left. An array of two miniature accelerometers (Endevco 2000 Series) was aligned and mounted as a unit midsagittally in the post-orbital ridge area to measure anterior-posterior and check vertical accelerations during the impact sequence.

Although difficult to accurately measure without more sophisticated gas or isotope techniques, attempts to measure cerebral blood flow were made with Biotronex BL flow transducers. Flow rates were measured by placing a transducer at the distal part of each common carotid artery through a single, anterior midline incision on the neck. Both external carotid branches were ligated but no attempt was made to occlude the paired vertebral artery source. ECG measurements were obtained with a standard array of three needle electrodes and stabilized by taping the leads.

Arterial and venous blood pressures were measured with Konigsberg catheter type (P model) pressure transducers positioned in the thoracic portion of the descending aorta and inferior vena cava respectively. These implants were accomplished by passing the catheter through the right femoral artery and vein via accessory branches. Entry through and subsequent ligation of the accessory vessels were made to avoid interruption of the primary limb circulation. A catheter tube was likewise placed via the left femoral artery for an external Statham type pressure transducer as a backup measurement of arterial blood pressure. The left femoral vein was fitted with a cannula to draw internal blood samples for analysis with a Radiometer, Model BMS–3 acid-base analyzer. Dual, small-diameter air tubes were positioned on the head restraint to direct air flow into the eyes as a continuous stimulus for eye reflexes.

Test Procedures

All test procedures were arranged and executed in a standardized, chronological sequence to assure compatibility with test control requirements of critical procedural time limits. Two days prior to the test, a set of head and neck x-rays, shaving of the surgical surface areas and preparations for restraint sizing were completed. The intervening day was required to permit the subject to completely recover from the effects of phencyclidine hydrochloride and sodium pentabarbital used in preparation procedures.

Surgical preparation of the test subject was accomplished as the first procedure the following day. An initial 75 mg sodium methohexital solution was administered intramuscularly with atropine prior to surgical anesthesia, then tracheal intubation was performed and anesthesia was accomplished with a nitrous oxide-oxygen mixture with halothane (per cent ratio of 6:4:2). A maintenance dosage rate was continued with decreased halothane volume of one-half to one per cent. After completion of all implant procedures, the subject was positioned and secured in the restraint system. Anesthetic agents were immediately withdrawn and the subject allowed to regain consciousness with continued oxygen support. The subject was monitored until all vital signs and responses were considered normal, then placed in the test position. Following a brief clinical evaluation, the endotracheal tube was removed and bioinstrumentation connected to start baseline monitoring and recording. Instrumentation continuity checks were made with viable signals for amplifying and recording equipment; then data sampling at five-minute intervals began at 30 minutes before impact with activation of the automatic switching timer for camera, lights and impact countdown. At five minutes pre-impact, recorders were set on continuous run for data sampling until 30 minutes post-impact. Euthanasia solution was administered following this monitoring period through the venous catheter to terminate the subject.

Within one hour after death, a comprehensive necropsy protocol was conducted to identify and describe gross trauma of all head and neck structures. All observations were recorded on tape and by color photography. Autopsy procedures were completed with the removal of the intact brain and cervical cord for histological preparation and study. A standardized plan of brain and cord sectioning was followed to assure comparison of similar sections in all test subjects. Typical fixing procedures with buffered formalin solutions were followed for routine staining with hematoxylin-eosin and galocyanin blue agents. Since autopsy procedures were initiated within

the hour after death, additional immediate fixing of the brain and cord was not considered necessary to inhibit autolysis.

III. Results

Summaries of test results for both test variables of supported- and moving-head series are outlined in Tables 1 and 2. Difficulties in achieving mechanical stability of instrumentation implants during impact resulted in certain data artifacts and, in some instances, data loss. The continuity of EEG electrode and carotid flow transducer interfaces was momentarily disrupted in all tests with artifactual or partial data loss. Although EEG signals did recover in part as stable signals, both flow transducers failed to stabilize for meaningful data records. Similarly, signal output from the transducers measuring intracranial pressure appeared to be artifactual and will not be considered for analysis in this paper. The design limits of accelerometers bonded to the anterior calvarium were exceeded by impact velocities and were not used thereafter. Blood gas values obtained were not considered significant for the purposes of this study. Although post-impact monitoring continued for as long as one hour in isolated cases, data analysis for this paper will consider only the first 30-minute period following impact.

Supported-Head Series

Subject CP 110. Pupil dilation occurred bilaterally and returned to normal in 14 seconds on only the right side. Conversely, only the left corneal reflex was functional after 35 seconds following bilateral loss. EEG patterns demonstrated slowed activity post-impact. No significant changes in the ECG pattern were observed, but both pulse rate and arterial pressure levels dropped after impact and recovered to baseline values at 40 and 30 seconds respectively. Gross autopsy findings were subcutaneous hemorrhage at the impact site, a simple non-depressed linear fracture from the occiput parasagittally into the foramen magnum and contused areas intradurally in the inferior-frontal, temporal and occipital areas and the cerebellum. Subarachnoid hemorrhages were histologically confirmed in the same areas and a few isolated hemorrhages were found deep in the temporal and cerebellar substance. Degenerative nerve cell changes were observed among normal, healthy cells in the frontal and occipital areas. These cell changes were manifested as shrunken and pyknotic with loss of nuclear detail.

Subject CP 111. Both pupil dilation and corneal reflex loss were bilateral with return except for continuous dilation of the left pupil. Corneal reflexes were active after 46 seconds. EEG activity was briefly slowed and ECG demonstrated a continuous bradycardia and loss of P wave. Both pulse rate and arterial pressure levels were depressed post-impact and did not return to normal baseline values. Coincident with a small laceration and subcutaneous hemorrhage, a complex, fragmented fracture pattern was centered around the impact site with occipital and post-parietal involvement. Occasional subarachnoid hemorrhages in the frontal, occipital and cerebellar areas were confirmed histologically, but deep micro-hemorrhages were found only in the occipital portion of the cerebrum and the cerebellum. In the frontal, temporal and occipital portions of the cerebrum, some of the large nerve cells were shrunken and misshapen and presented altered staining reaction. Their nuclei were indistinct with absence of chromatin and nucleoli. Cells in the occipital portion showed more intense degenerative changes with pyknotic nuclei or indistinct structure and increased number of glial elements.

Subject CP 112. Pupils dilated bilaterally with normal return in 60 seconds. Corneal reflexes were extinguished and became functional after 12 and 20 seconds post-impact. EEG activity slowed intermittently and the ECG pattern changed significantly with distinct bradycardia and premature ventricular contractions. Pulse rate and arterial pressure increased briefly, then decreased to remain below baseline levels. Pathologically, a scalp laceration with subcutaneous hemorrhage and a multi-fragmented fracture pattern was centered around the impact site. Gross examination revealed isolated areas of contusion on the inferior surfaces of the frontal lobes, temporal, occipital and posterior cerebellum. Subarachnoid hemorrhages were observed in microscopic sections from the frontal and temporal areas and the cerebellum. The only internal hemorrhages were located throughout the cerebellum. A majority of the large nerve cells presented the same degenerative changes as described in the other test subjects. Although

nerve cells in the cervical cord section were largely well formed with distinct nuclei, Nissl substance appeared to be absent.

Subject CP 121. Pupil dilation was bilateral with return on the right side only at 35 seconds. Bilateral corneal reflex loss was very brief for four seconds. EEG showed a delayed, but brief, slowed activity and bradycardia with premature ventricular contractions disappearing by the end of the monitoring period. Pulse rate and arterial pressure decreased and recovered at 40 and 35 seconds, respectively. A drop in the carotid flow rates was observed for a few seconds, then became unstable with neck movement. Isolated hemorrhages were found subcutaneously at the impact site, superficial to the origin of a single linear fracture that continued into the foramen magnum. Gross contusions were observed within the dura over the temporal, pons, cerebellar and post-cervical areas. Subarachnoid hemorrhages were scattered along the surfaces of the temporal lobes, posterior cerebellum, pons and the cervical cord. This is the only test subject that did not demonstrate degenerative nerve cell changes.

Subject CP 122. Post-impact pupil dilation was bilateral with one of the greatest durations of 120 seconds. Conversely, the bilateral loss of corneal reflexes was only two seconds. Both the EEG slowing and ECG patterns of bradycardia and premature ventricular contractions remained for only 45 seconds. The pulse rate stabilized after 55 seconds and arterial pressure and carotid flow rates returned to baseline after 180 seconds from depressed levels. A small subcutaneous hemorrhage was confirmed at the impact site, but there was no fracture in the cranial vault. This was the only subject without fracture. Two small areas of dural contusions were found over the inferior aspect of the frontal lobes and posterior surface of the occipital lobes. Subarachnoid hemorrhages were confined to the occipital and pons areas and internal hemorrhage only in the substance of the cerebellum and superior cervical cord. A number of large nerve cells in all cerebral areas were typically degenerative in nature, most without nuclear detail and Nissl substance. The Purkinje cells in the cerebellum were not remarkable but few ganglion cells in the cervical cord demonstrated degenerative changes. None of the cells possessed Nissl substance.

Subject CP 124. Bilateral pupil dilation remained for 43 seconds post-impact and corneal reflexes were functional within 16 seconds. The EEG patterns typically demonstrated slowed activity. Premature ventricular contractions were consistent throughout the monitoring period, but only a brief interval of bradycardia appeared immediately post-impact. Pulse rate, arterial pressure and carotid flow rate decreased immediately after impact, then recovered to normal values after 20 seconds. A subcutaneous hemorrhage overlying a single non-depressed occipital fracture was coincident with the impact site. The entire anterior surface areas of the frontal lobes were contused, unlike all other subjects. Subarachnoid hemorrhages were found in the sub-frontal and inferior temporal surfaces and along the posterior aspects of the occipital lobes and cerebellum. Internal hemorrhages were located in the pons and deep throughout the cerebellum and typical degenerative neural alterations were confirmed in the temporal and occipital areas.

Moving-Head Series

Subject CP 113. Bilateral pupil dilation and loss of corneal reflex occurred immediately post-impact with recovery within 20 seconds. Slowed EEG activity occurred briefly and the ECG patterns were not remarkable. Arterial pressure increased for 20 seconds with similar carotid flow shifts, but the pulse rate depressed 120 seconds. Typical scalp laceration and subcutaneous hemorrhage were associated with a short linear fracture of the occipital bone at the impact site. Contused areas were found on the inferior surfaces of the frontal lobes, anterior aspects of the temporal lobes and posterior occipital areas. Diffuse subarachnoid hemorrhages were confined to corresponding frontal and temporal areas and a single area of internal hemorrhage was located in the right occipital lobe. Among normal cells, degenerative changes were found in the frontal, temporal and occipital areas.

Subject CP 114. Pupil dilation and corneal reflex loss were bilateral with short term recovery time at 12 and 2 seconds respectively. As in Subject CP 113, the ECG patterns were not remarkable and only a brief few-second slowed activity was evident. Both pulse rate and arterial pressure were depressed for 25 and 80 seconds respectively. Local subcutaneous hemor-

rhage was coincident with the occipital impact site and the origin of a linear fracture passing parasagitally into the foramen magnum. Areas of intradural hemorrhage were found on the anterior aspect of the temporal lobes, posterior aspects of the occipital poles and cerebellum. Subarachnoid hemorrhages were confirmed in the frontal and temporal areas and deep hemorrhages were revealed in the substance of the pons and cerebellum. Typical degenerative nerve cell changes were manifest, with some difference. A few well-formed cells showed only indistinct nuclear detail with some loss of chromatin, but many more of the severely affected cells presented satellitosis and early garyolysis. Most of these changes were confined to the cerebrum.

Subject CP 115. Bilateral pupil dilation occurred and corneal reflexes extinguished without recovery of either. EEG waves were flattened and slowed and pulse rate, arterial pressure and carotid flow all depressed without return to normal baseline levels. A scalp laceration and subcutaneous hemorrhage attended a complex, multi-fragmented fracture pattern of the occipital and parietal bones. Depressed fragments were the cause of sagittal sinus lacerations and subsequent loss of a large volume of blood. This subject was terminated after a 15-minute monitoring period without regaining consciousness. Gross contusion areas were observed over the anterior temporal lobe, occipital and cerebellar areas. Subarachnoid hemorrhage was extensive in the temporal, occipital, pons, cerebellum and cervical areas. Degenerative nerve cell changes were confined to the occipital and cerebellar areas.

Subject CP 116. There was no pupil dilation and bilaterally extinguished corneal reflexes returned after two seconds. There was a questionable area of slowed EEG activity and pulse rate and arterial pressure depressed for only 12 and 25 seconds respectively. A small subcutaneous hemorrhage and short linear fracture were found at the impact site. Although dural congestion was evident on the anterior surfaces of the temporal lobes, occipital and cerebellar surfaces, subarachnoid and deep hemorrhages were confined to the cerebellum. Conversely, degenerative nerve cell changes were found in all histological sections with the exception of the pons.

Subject CP 117. The bilateral pupil dilation did not fully return to normal; however, both corneal reflexes were extinguished for only two seconds. Slowed activity was suggested in the EEG patterns, but the ECG was not remarkable. Pulse rate was depressed for 39 seconds and arterial pressure returned to normal after increasing for 25 seconds. The inferior frontal, occipital and cerebellar areas showed dural contusions; however, only the cerebellum had both subarachnoid and deep hemorrhages. An isolated temporal area of subarachnoid hemorrhage was confirmed. Degenerative nerve cell changes were found in the frontal, temporal, occipital and cerebellum areas. There appeared to be gradations of changes with slightly misshapen and altered nuclear detail in many cells.

Subject CP 118. Although bilateral pupil dilation never recovered on the right side, and the left remained dilated for 113 seconds, both corneal reflexes were active after only two seconds. EEG activity slowed and the P wave in the ECG was depressed. The pulse rate remained elevated for 220 seconds and, conversely, the arterial pressure depressed for only 60 seconds before recovery to normal baseline level. A simple linear fracture in the occipital bone was found with superficial subcutaneous hemorrhage and scalp laceration. Grossly observed areas of contusion were located along the anterior temporal, occipital and cerebellar surfaces. Degenerative nerve cell changes were confined to the temporal, occipital and cerebellar sections with clear zones surrounding these misshapen cells to suggest edema. Intense areas of subarachnoid hemorrhages were found in the front and temporal portions and deep micro hemorrhages only in the cerebellum.

Subject CP 120. Similar to CP 117, there was no recovery of normal pupil diameter but corneal reflexes returned bilaterally within 13 seconds. Slowed EEG activity is suggested and P wave remained depressed for most of the monitoring period. A depressed pulse rate recovered after 25 seconds post-impact and arterial pressure decreased to baseline levels after 80 seconds. A complex, multiple fracture pattern in the occipital bone and parietals was not depressed and associated with the typical lacerated scalp and subcutaneous hemorrhage. Gross contusion areas of the dura were most extensive in this group

covering the sub-frontal, temporal, occipital, cerebellar areas. Subarachnoid hemorrhage was extensive in the frontal, temporal, occipital, cerebellar and cervical surfaces with deep hemorrhage only in the occipital and cerebellar sections. Shrunken and misshapen cells with pyknotic nuclei were again found among normal cells in the frontal, temporal, occipital and cerebellar areas.

IV. Discussion

The relatively small number of subjects in this initial test series precludes a detailed statistical analysis of data values; however, trends of certain response phenomena are distinct and appear to be predictable. The general nature of clinically observed and measured acute responses to direct occipital impact was not unlike systemic responses that have been observed and documented by others. The results do suggest that certain functional and structural changes are occurring under these particular impact conditions which may not conform to the more popular explanations of injury mechanisms or which have not been experimentally produced by others. In general, the relatively higher levels of shock input resulting in the supported-head series produced more intense injury and more pronounced changes in systemic response than found in the test with controlled-head motion. Although there was an average difference in impactor velocity of 4 ft./sec. faster and relatively higher inputs to the heads of the moving-head series, the type and intensity of cranial vault fracture were not significantly different, with two exceptions. In Subject CP 122 there was no fracture in a predicted worse case condition and, ironically, multiple depressed fractures occurred in a moving-head subject (CP 115) that made artifactual most of the physiological response data.

Loss of corneal reflexes and pupil dilation occurred as expected and typically confirmed by other researchers such as Denny-Brown and Russell (1941), McCullough (1971) and Tindall (1972). The duration of the reflex loss was longer in the supported-head series and the pupil dilation without recovery is attributed to the more severe shock input based on histological evidence. There also appears to be a significant difference in the ECG responses with distinct bradycardia and premature ventricular contractions occurring in five of the six supported-head tests and only a marked P wave depression occurring in two subjects of the moving-head series. This phenomenon seems consistent with the relative intensity of altered physiological responses occurring with supported heads. These levels are also consistently higher than produced in other relatable work and cannot be effectively compared.

Of greater significance, examination of pathological findings reveals unusual combinations of trauma that seem to contradict established conventions of injury mechanisms. The conformation of such trauma by histological analysis was not completely unexpected in some instances; however, the distinct and intense hemorrhage in the frontal and temporal areas of all subjects with supported heads as well as those in the moving-head series are not regarded as typical findings under these experimental conditions. By conventional definition these trauma areas are defined as contrecoup injuries and should have only occurred in heads that were free to move with the occipital blow. This definition of contrecoup was well documented by LeCount and Apfelbach (1920), Vance (1927) and Munro (1938) with confirmed experimental and human data. These same observations were confirmed experimentally by Denny-Brown and Russell (1941), among others, who failed to produce contrecoup injury with supported-heads at impact, but at lower levels of shock input.

It is interesting to note that while contrecoup injuries and loss of consciousness were produced in both moving and supported heads with translational motion-force orientation, the theory of Holbourn (1943) and experimental evidence by Ommaya (1966), Hirsch (1970) and Gennarelli (1972) demonstrate that rotational, not translational, motion is the primary component associated with concussion and brain injury for brief shock inputs. Whether or not contrecoup trauma can be expected to occur predictively under these and similar conditions must await further experimental evidence. Since this study phase did not consider the rotational component as an experimental variable, the relative significance of the translational role in producing unconsciousness under other conditions is not known.

Similarly, the extensive histological analysis has revealed a degenerative neurological phenomenon associated with impact events of both test conditions. With only one exception, all traumatized subjects in both test series demonstrated alterations of the large nerve cells and elements as shrunken, misshapen and pyknotic nuclei and classic loss of nuclear detail. It is usually agreed that such degenerative changes require a number of hours to manifest following insult. These cell changes occurred within 60 minutes or less after impacts in this study. Considerations of viral problems and other predisposing factors were not confirmed after a lengthy and detailed analysis. Control subjects, both laboratory-bred and fresh-colony imports of different origin, were analyzed for possible sources of artifacts with negative results and a re-evaluation of potential test conditions for sources of anoxia had the same result. In a detailed analysis of over 300 other histological surveys of traumatized baboons without direct-head impact, no degenerative changes similar to those were found. Only two other sources of experimental work, by Snyder (1972) and Portnoy (1970), have confirmed similar findings and were, in fact, produced under similar impact conditions.

V. Conclusions

On the basis of these initial tests, there appear to be certain combinations of physiological responses and associated injury mechanisms that may be either contradictory in part to current theories now held by a number of investigators or may actually indicate additional phenomena not previously documented. Of greatest significance, the contrecoup injury has been produced by direct occipital impact to heads supported and not free to respond kinematically to the impact force. Further, this type of indirect injury has been produced by only translational forces in both the supported heads and heads free to move in a controlled anterior-posterior plane with relatively similar force inputs. Degenerative nerve cell changes have also been consistently produced under the conditions of acute head impact in less than one hour and, without benefit of further experimental evidence, appear to be trauma related. It is suggested that the experimental evidence presented here is sufficient to seriously question the absolute adequacy of current injury tolerance data that are being offered and used to establish tolerance standards. Additional experimental testing will be necessary to statistically evaluate these potentially conflicting theories and provide realistic tolerance values for design standards.

REFERENCES

1. Abernethy, John: *Surgical Observations on Injuries of the Head*, 3rd ed., London, Longman (and others), 1825.
2. Bergmann, E. Von: Die Lehre von dem Kopfverletzungen, in Billroth and Lueke, DEUTSCHE CHIRURGIE, Lief., 30, Stuttgart, 1880.
3. Denny-Brown, D., W. R. Russell: Experimental Cerebral Concussion, BRAIN, 64:93–164, 1941.
4. Gennarelli, T. A., L. E. Thibault, A. K. Ommaya: Pathophysiologic Responses to Rotational and Translational Accelerations of the Head. *Proceedings of the 16th Stapp Car Crash Conference*, SAE, NY, 1972.
5. Haynes, A. L., H. R. Lissner: Experimental Head Impact Studies. *Proceedings of the Fifth Stapp Automotive Crash and Field Demonstration Conference*, University of Minnesota, 1961.
6. Hirsch, A. E., A. K. Ommaya: Protection from Brain Injury: The Relative Significance of Translational and Rotational Motions of the Head After Impact. *Proceedings of the 14th Stapp Car Crash Conference*, SAE, NY, 1970.
7. Holbourn, A. H. S.: Mechanics of Head Injury, LANCET, 245:438–441, 1943.
8. Hutchinson, Jonathan: *Illustrations of Clinical Surgery*, London, J. & A. Churchill, 1875–84.
9. Le Count, E. R., C. W. Apfelbach: Pathologic Anatomy of Traumatic Fractures of Cranial Bones and Concomitant Brain Injuries. J. AMER. MED. ASSN., 74:501–511, 1920.
10. McCullough, D., K. Nelson, A. K. Ommaya: The Acute Effects of Experimental Head Injury on the Vertebrobasilar Circulation: Angiographic Observations. JOUR. OF TRAUMA, 11 (5), 1971.
11. Munro, Donald: *Cranio-Cerebral Injuries. Their Diagnosis and Treatment*, New York, Toronto & London, Oxford Press, 1938.
12. Ommaya, A. K., A. E. Hirsch, J. Martinez: The Role of 'Whiplash' in Cerebral Concussion. *Proceedings of the 10th Stapp Car Crash Conference.* SAE, NY, 1966.
13. Portnoy, H., D. Benjamin, M. Brian, L. McCoy, B. Pince, R. Edgerton, J. Young: Intracranial Pressure and Head Acceleration During Whiplash. *Proceedings of the 14th Stapp Car Crash Conference.* SAE, NY, 1970.
14. Snyder, R. G.: Personal communication, 1972.
15. Tindall, G. T.: Personal communication, 1972.
16. Vance, B. M.: ARCH. SURG., 14:1022, 1927.

TABLE 1. Summary of Supported-Head Impact Responses

POSTIMPACT CONDITIONS	CP 110	CP 111	CP 112	CP 121	CP 122	CP 124
PUPIL DILATION	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral
PUPILS NORMAL	R=14 sec L= None	R=90 sec L= None	Both at 60 sec	R=35 sec L= None	Both at 120 sec	Both at 43 sec
CORNEAL REFLEX OUT	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral
CORNEAL REFLEX IN	R= None L=35 sec	Both at 46 sec	R=20 sec L=12 sec	Both at 4 sec	Both at 2 sec	R=16 sec L=13 sec
EEG	Slowed	Slowed	Slowed	Slowed	Slowed	Slowed
ECG		Bradycard No P Wave	Bradycard PVC	Bradycard PVC	Bradycard PVC	Bradycard PVC
PULSE RATE	Down:Norm at 40 sec	Down: No Return	Up-Down: No Return	Down:Norm at 40 sec	Down:Norm at 55 sec	Down:Norm at 20 sec
ARTERIAL PRESSURE	Down:Norm at 30 sec	Down: No Return	Up-Down: No Return	Down:Norm at 35 sec	Up:Norm at 180 sec	Down:Norm at 20 sec
CAROTID FLOW				Decrease	Increase	Decrease
GROSS: CRANIAL FRACTURE	Simple: Occipital	Complex: Occipital	Complex: Occipital	Simple: Occipital	None	Simple: Occipital
GROSS: BRAIN CONTUSION AREAS	Inf Front Temporal Occipital Cerebellum	Occipital Cerebellum	Inf Front Temporal Occipital Cerebellum	Temporal Occipital Cerebellum	Inf Front Occipital	Frontal Temporal Occipital Cerebellum
HISTOLOGY: EXT BRAIN HEMORRHAGE	Frontal Temporal Occipital Cerebellum	Frontal Occipital Cerebellum	Frontal Temporal Cerebellum	Temporal Pons Cerebellum Cervical	Occipital Pons	Frontal Temporal Occipital Cerebellum
HISTOLOGY: INT BRAIN HEMORRHAGE	Temporal Cerebellum	Occipital Cerebellum	Pons Cerebellum	Pons Cerebellum	Cerebellum Cervical	Pons
HISTOLOGY: NERVE CELL CHANGES	Frontal Occipital	Frontal Temporal Occipital	Frontal Temporal Occipital	None	Frontal Temporal Occipital	Temporal Occipital
IMPACTOR VELOCITY	43.7 ft/sec	43.7 ft/sec	43.0 ft/sec	41.9 ft/sec	43.5 ft/sec	42.0 ft/sec
IMPACTOR LOAD			975 lbs	1025 lbs	930 lbs	750 lbs
IMPACTOR PEAK g			1260 g	1200 g	1100 g	900 g

TABLE 2. Summary of Moving-Head Impact Responses

POSTIMPACT CONDITIONS	CP 113	CP 114	CP 115	CP 116	CP 117	CP 118	CP 120
PUPIL DILATION	Bilateral	Bilateral	Bilateral	None	Bilateral	Bilateral	Bilateral
PUPILS NORMAL	Both at 20 sec	Both at 12 sec	None	Bilateral	None	R= None L=113 sec	None
CORNEAL REFLEX OUT	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral
CORNEAL REFLEX IN	Both at 20 sec	Both at 2 sec	None	Both at 2 sec	Both at 2 sec	Both at 2 sec	R=13 sec L=10 sec
EEG	Slowed	Slowed	Slowed	Slowed	Slowed	Slowed	Slowed
ECG						P Wave Depressed	P Wave Depressed
PULSE RATE	Down:Norm at 120sec	Down:Norm at 25 sec	Down: No Return	Down:Norm at 12 sec	Down:Norm at 39 sec	Up:Norm at 220sec	Down:Norm at 25 sec
ARTERIAL PRESSURE	Up:Norm at 20 sec	Down:Norm at 80 sec	Up-Down: No Return	Down:Norm at 25 sec	Up:Norm at 25 sec	Down:Norm at 60 sec	Up:Norm at 80 sec
CAROTID FLOW	Increase	Decrease	Decrease	Increase			Decrease
GROSS: CRANIAL FRACTURE	Simple: Occipital	Simple: Occipital	Complex: Occipital Parietal	Simple: Occipital	Complex: Occipital Parietal	Simple: Occipital	Complex: Occipital Parietal
GROSS: BRAIN CONTUSION AREAS	Inf Front Ant Temp Occipital	Ant Temp Occipital Cerebellum	Ant Temp Occipital Cerebellum	Ant Temp Occipital Cerebellum	Inf Front Occipital Cerebellum	Ant Temp Occipital Cerebellum	Inf Front Temporal Occipital Cerebellum
HISTOLOGY: EXT BRAIN HEMORRHAGE	Frontal Temporal	Frontal Temporal	Temporal Occipital Pons Cerebellum Cervical	Cerebellum	Temporal Cerebellum	Frontal Temporal	Frontal Temporal Occipital Cerebellum Cervical
HISTOLOGY: INT BRAIN HEMORRHAGE	Occipital	Pons Cerebellum	Frontal Temporal Occipital Pons Cerebellum Cervical	Cerebellum	Cerebellum	Cerebellum	Occipital Cerebellum
HISTOLOGY: NERVE CELL CHANGES	Frontal Temporal Occipital	Frontal Temporal Occipital	Occipital Cerebellum	Frontal Temporal Occipital Cerebellum Cervical	Frontal Temporal Occipital Cerebellum	Temporal Occipital Cerebellum	Frontal Temporal Occipital Cerebellum
IMPACTOR VELOCITY	43.6 ft/sec	45.3 ft/sec	50.2 ft/sec	43.7 ft/sec	45.7 ft/sec	48.8 ft/sec	51.6 ft/sec
IMPACTOR LOAD	700 lbs	925 lbs	400 lbs	1125 lbs	800 lbs	875 lbs	850 lbs
IMPACTOR PEAK g	840 g	1100 g	800 g	1325 g	900 g	1050 g	1020 g