Experimental In Vivo Microcirculatory Dynamics in Brain Trauma

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Trauma to the central nervous system has long been one of the major problems encountered in the practice of neurosurgery in both the military and the civilian environments. Massive open or penetrating wounds to the central nervous system, while posing great problems in clinical management, entail a rather well-defined pathophysiological problem. However, closed head trauma with concussion, direct damage, contre-coup injuries, and compromise of major vessels and compromise of major vessels results in a wide variety of pathological changes which are poorly understood. For example, the concept of concussion, that is, physiological dysfunction without gross anatomical changes, has long been debated, particularly as to its distinction from actual contusion or laceration. But many excellent studies have established beyond doubt that microscopic degenerative neuronal changes can occur in trauma without gross or microscopic evidence of tissue disruption.

The first description of petechial perivascular hemorrhages and diffuse cerebral swelling secondary to closed head trauma, without focal damage, is a part of the oldest medical records. In 1831, Bright's excellent studies documented this finding in postmortem material from clinical and laboratory sources. Photomicrographs have revealed microscopic distention and leakage of vessels at the capillary level with resultant, microhemorrhagic changes.

Recently we have seen several patients who died secondary to closed head injuries but in whom no gross contusions, laceration, or bleeding was noted. Diffuse cerebral edema was present as well as microscopic foci of perivascular hemorrhages. In an effort to clarify the underlying pathophysiology of such changes, we have undertaken the present study. Although postmortem changes have been widely documented, there has been no previous report of the in vivo microcirculatory dynamics that occur following brain trauma.

Materials and Methods

Ten animals comprised the experimental series for this study, consisting of 4 mongrel dogs, 3 Rhesus monkeys (Macacus mulata), and 3 baboons (Papio porcarius). All animals were subjected to identical operative procedures. They were anesthetized by intravenous pentobarbital in a dosage of 30 mg/kg of body weight. The baboons required the pre-anesthetic administration of 40 mg of phencyclidine hydrochloride to make safe removal from their cages possible. After induction of anesthesia, a slow intravenous infusion was continued in order to maintain a route should further anesthesia be required. The airway was maintained by endotracheal intubation. The Harvard pump respirator was available should ventilatory assistance be required, but this was seldom used.

The head was closely clipped and immobilized in a stereotaxic ear bar head holder to eliminate any possible movement. The operative site was prepared with a thimerosal solution and appropriately draped. The scalp was opened in a midline linear incision extending from the glabella to the inion by the cutting current of the Bovie electrocautery unit, which minimized scalp blood loss. The temporalis muscles were detached by incising the fascial and pericranial attachments along their superior margins and stripping these away from the cranial surfaces. Self-retaining retractors were used to retract the

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† The principles of laboratory animal care, as promulgated by the National Society for Medical Research, were observed during this study.
Fig. 1. Pre-trauma appearance of artery and vein over cortical gyrus. Baboon brain, ×16.

Fig. 2. Immediate post-traumatic cortical appearance that shows the perivenous flush. Baboon brain, ×40.

Fig. 3. Early petechial formation at capillary venous junctions with a persistent flush around the vein after 4 minutes. Baboon brain, ×25.

Fig. 4. Petechial formation is more advanced and the flush is less prominent after 10 minutes. Baboon brain, ×25.

Fig. 5. Petechial formation is most marked at the venous capillary junction at 20 minutes. Baboon brain, ×40.