

Second Impact Syndrome: Concussion and Second Injury Brain Complications

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Second impact syndrome was first described in 1973 by Richard Schneider in 2 young athletes who experienced initial concussive syndromes and subsequently died after a relatively minor second head injury.¹ Saunders and Harbaugh coined the term *second impact syndrome* in their 1984 description of a 19-year-old college football player who suffered a head injury with brief loss of consciousness, returned to play, reported a headache, and on the 4th day collapsed, became unresponsive, and died.² Postmortem examination revealed no space-occupying hematoma and extensive cerebral edema. It is the second collision impact, absence of space-occupying hematoma, and subsequent rapid and profound brain swelling³⁻⁷ that identify and mark the second impact syndrome. The severe brain swelling and absence of impact hematoma are identical to first head injury findings duplicated in head injury laboratory investigations⁸⁻¹⁴ and identified in clinical series of severe head injury patients.¹⁵⁻²³

Statistics as to the occurrence of second impact syndrome do not exist and the actual prevalence and incidence remain unknown. Second impact syndrome has always had proponents and detractors. Multiple head injury experts have discussed this phenomenon as a worrisome corollary in virtually every article or chapter on sport-related head injury.²⁴⁻²⁸ However, others have been suspicious that the pure form is rare, and some have called into question its existence, commenting “. . . it is fear of this entity that underpins concussion guidelines regarding return to sport.”²⁹ The latter statement prompted a strong rebuttal from a long-term and fully fledged head injury investigator who noted that, although the syndrome might be uncommon, its existence is a reality and is identifiable at some level virtually every year. In addition, it would be naïve to suggest return to play guidelines are solely in place as a consequence of possible second impact syndrome, when there are multiple reasons to protect a concussed athlete from re-exposure to play too early.²⁸ Second impact syndrome is only a small determinant for return to play guide-

lines, and other factors, such as reported concussions are endemic in contact play and sport and are probably underreported in high school football players³⁰; laboratory evidence⁵ and clinical series of concussed athletes reveal an increased susceptibility to additional concussions³¹; repetitive concussions have prolonged neuropsychiatric effects^{32,33}; and recurrent concussions are related to substantial late-life cognitive impairment.³⁴ Return to play guidelines after sport-related traumatic brain injury are beyond the scope of this article, but are founded and invested in athletic safety by assuring that the celerity of thought and clarity of mentation have returned to normal so that athletic mental and physical reflexes are not still impaired, which would make the athlete more susceptible to any subsequent injury; and recognizing that concussed athletes are more susceptible to a second concussion and that permanent sequelae might result. The reader is referred to return to play guidelines and their continued evolution, for which there are multiple sources.³⁵⁻³⁹

Head injury research during the last century has clinically and experimentally produced clear evidence that severe first impact head injury—induced cerebral autoregulatory failure, with simultaneous catecholamine-induced marked blood pressure elevation, leads to rapid and often fatal malignant brain swelling.⁸⁻²³ It is highly probable that the same events occur in the second impact syndrome as well, only in tandem sequence with lesser energies and generally in young people and children, but with the same devastating results.

PATHOPHYSIOLOGY OF HEAD INJURY

Autoregulation

Cerebral autoregulation is best described as the “tone” the arterial tree assumes to either uniformly dilate or constrict to keep cerebral blood flow constant under normal conditions between a mean arterial blood pressure of 50 to 150 mmHg.⁴⁰⁻⁴² It is primarily an arterial smooth muscle-mediated phenomenon, as originally described by Bayliss (Bayliss effect),⁴³ although there can be minor arterial endothelial paracrine influences coupled with metabolic tissue needs. Regardless, every arterial system in the body autoregulates blood pressure to the recipient organ. Brain arteries are extremely sensitive autoregulators of arterial blood pressure, but the importance remains puzzling. Pressure auto-

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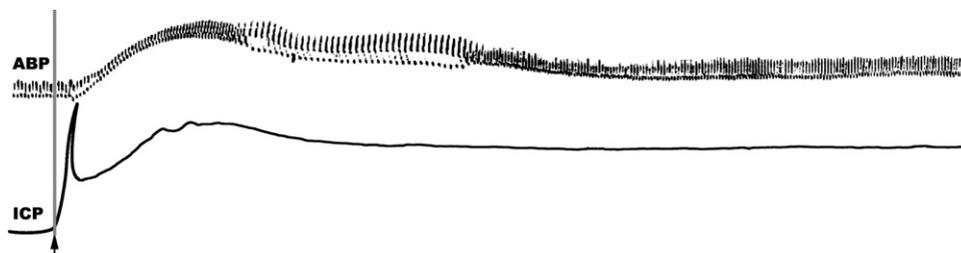


Figure 1. Actual tracing of an anesthetized rat undergoing severe fluid percussion head injury. The arrow depicts the injury. Note immediate arterial autoregulatory failure clearly documented by the mean arterial blood pressure (ABP) wave transmission to the intracranial pressure (ICP). The 2 pressure waves are superimposable.

regulation can be disturbed by cranial surgery, trauma, even pharmaceuticals, and yet even its absence does not correlate well with head injury severity or outcomes.⁴⁴⁻⁴⁸ However, there is ample evidence that traumatic brain injury can induce dysfunctional or absent autoregulation for brief periods (hours or days) and, in severe cases, prolonged time periods (weeks).⁴⁷ This absence of tone predisposes the brain to extremes in blood pressure; hypotension is poorly accommodated with vasodilation, so the brain will be at increased susceptibility to ischemia; and hypertension is poorly accommodated by vasoconstriction, so that substantially elevated mean arterial blood pressure is rapidly transmitted to the intracranial pressure by distention of the arterial tree, producing marked vasodilation and cerebral blood volume engorgement. This failure of pressure autoregulation occurs in a linear fashion, with increasing frequency proportional to increasing severity of head injury. Clinical studies reveal mild traumatic brain injury produces autoregulatory failure in 20% to 30% of patients,^{49,50} and severe head injury can elicit autoregulatory failure in up to 80% of patients.^{46,47} The results are the same, an inability to respond to blood pressure gradient changes in a normal manner.

Stress-induced catecholamine surge

Catecholamine surge with profound and sudden elevation of heart rate and blood pressure is an irrefutable stress response to traumatic head injury and occurs in a linear response, ie, mild head injury can produce mild responses and severe head injury induces dramatic and massive responses. Pronounced catecholamine responses to head injury were first noted by Polis⁹ as immediate effects, and have been documented as brainstem-mediated by Walker and colleagues⁴⁸ and several other investigators during the past century.^{8,10-18} In fact, sympathetic discharge of catecholamines with resultant marked and rapid elevation of blood pressure has proved to be a consistent and immediate response of all mammalian head injury models.^{12,14} Epinephrine and norepinephrine elevations with severe head

injury have been documented in numerous laboratory and clinical series of severely head-injured patients. The response is dose-dependent, so that minor head injury might produce a small response, and severe head injury produces a robust response, but the catecholamine release and effects occur rapidly, within seconds of the head injury itself.

Failure of cerebral autoregulation combined with traumatic catecholamine surge

In the setting of head-injury-induced cerebral arterial blood pressure autoregulatory failure, the arterial tree is instantly paralyzed with impact and becomes atonic, unable to respond to sudden and rapid escalation of blood pressure. Traumatic brain injury also rapidly induces systemic catecholamine surge, with elevation of blood pressure directly correlating in a linear fashion, so that a “malignant storm” can occur, producing rapid and profound brain engorgement that can be fatal. Figure 1 reveals an actual animal in our traumatic brain injury laboratory^{51,52} in which this malignant storm occurs; instantaneous severe brain injury (3 atmospheres fluid percussion injury) induced autoregulatory failure and concomitant massive stress induced catecholamine surge. The result reveals that the arterial blood pressure wave and intracranial pressure wave are superimposable, with massive and rapid escalation of intracranial pressure leading to rapid cardiovascular collapse and death of the animal. Figure 2 reveals a CT scan of a 17-year-old severe head injury patient involved in a high-speed motor vehicle rollover and found in coma with fixed and dilated pupils and no brainstem reflexes minutes after the accident. This CT scan obtained 30 minutes after injury reveals a massively swollen brain without space-occupying hematoma. This is the identical CT scan depiction described in numerous cases of second impact syndrome.

Case summaries

Previous documented cases have been summarized.²⁹ What separates the second impact syndrome from expanding in-

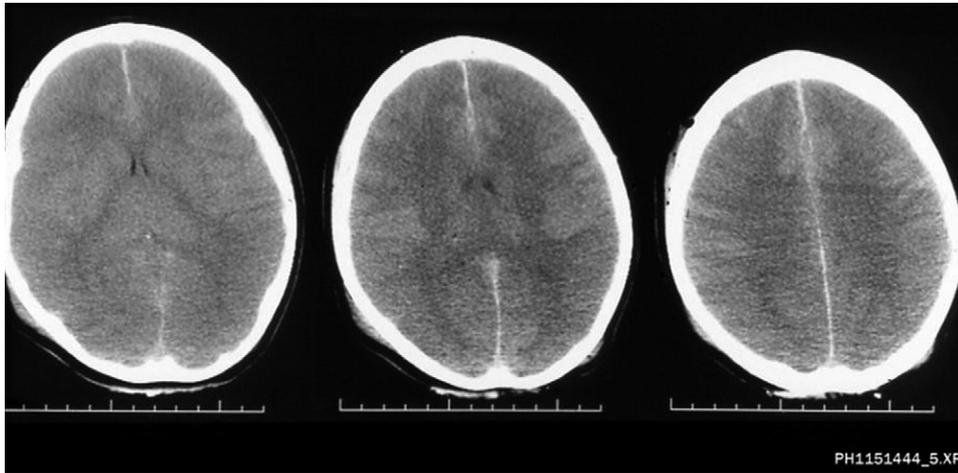


Figure 2. Severe head injury from a motor vehicle accident with malignant brain swelling. A 17-year-old severe head injury patient 30 minutes after accident. The image depicts the CT description of massive brain swelling described in second impact syndrome, which is probably due to the same phenomenon depicted in Figure 1.

tracranial hematoma due to head injury and rapid patient deterioration is the absence of any substantial hematoma. The patient rapidly deteriorates and only massive brain swelling with fatal brain herniation is identified on CT scan or at autopsy. Younger patients, particularly children or young adults, might be more susceptible, as others have discussed.⁷ A case report with CT scan and autopsy findings will serve as an illustrative case.⁵³

A 17-year-old male high school football player was concussed in a game and reported a headache during the next week. During the next game, 1 week after his concussion, he was tackled by helmet-to-helmet contact, stunned, but then continued to play. After several minutes, he collapsed and during several minutes progressed into coma with fixed and dilated pupils. A CT scan revealed a diffusely swollen brain without space-occupying hematoma (see Fig. 2 for similar description). Attempts at salvage failed. At autopsy, a massively swollen and engorged brain without space-occupying hematoma was the only discernable cause of brain death.

Massive brain swelling has been a clinical feature of severe head injury patients for decades. It is not surprising that the same features are identically described in patients with second impact swelling. The only difference is that second impact syndrome patients suffer a mild concussive head injury that produces cerebral autoregulatory failure. A second impact anywhere on the body, but particularly a second head injury, can produce a systemic stress-induced catecholamine surge and rapid blood pressure elevation that produces the same devastating results of acute and massive brain swelling.

The coupling of these 2 pathophysiologic events can produce devastating results. However, the necessary foundation is cerebral autoregulatory failure. The scenario of massive brain swelling with severe head injury is not uncommon because both autoregulatory failure and massive catecholamine surge are directly related to the amount of energy traumatically delivered to the brain and occur simultaneously. The mild concussive energy delivery to the brain in the first phase of the second impact syndrome induces the cerebral autoregulatory failure, which can last hours or days, but it is the subsequent stress-inducing second impact, which ironically can be produced by any systemic injury, that causes catecholamine release, rapid blood pressure elevation, and massive brain swelling. This scenario is considerably less frequent because the energy delivered is less, and lesser degrees of stress-induced catecholamine surge occur as a corollary. However, under infrequent conditions, mild head injury—induced autoregulatory failure, followed by any second stress-inducing injury, produces the same concatenation of events with devastating effects.

Author Contributions

Study conception and design: Wetjen, Pichelmann, Atkinson
 Acquisition of data: Wetjen, Pichelmann, Atkinson
 Analysis and interpretation of data: Wetjen, Pichelmann, Atkinson
 Drafting of manuscript: Wetjen, Pichelmann, Atkinson
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