OPHTHALMOLOGICAL HYDROSTATIC PRESSURE SYNDROME

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ESCAPE from military aircraft in flight by means of an ejection seat exposes the occupant to prolonged high decelerative forces which may be injurious or lethal at supersonic speeds. In experiments conducted by Stapp (1), a rocket-propelled sled on rails, decelerated by a braking system that reproduces the forces encountered in supersonic escape from aircraft, has been used to determine voluntary tolerance limits of exposure on human subjects. Criteria for tolerance are incipient reversible injuries, as well as the subjective evaluation. By this means it has been experimentally established that a human subject seated facing forward, exposed to deceleration perpendicular to the long axis of the body, can sustain a rate of onset of 1,500G per second or less, a peak magnitude of 50G, and total duration of forces higher than 25G but less than 50G of not more than one second. In 2 of the 76 human experiments accomplished to date, signs and symptoms suggestive of a syndrome have been found which correspond to those found in 3 cases of accidental exposure under widely different circumstances. In these 5 cases the syndrome is caused by high decelerative forces of abrupt onset applied from the rear to the front of the head while the blood vessels of the head and face are congested by high hydrostatic pressures. Abrupt rise in intrathoracic pressure, due to (1) displacement of abdominal viscera against the diaphragm or (2) abrupt compression of the abdomen or lower chest, can be transmitted through the blood vessels to the head and face, causing rise of hydrostatic pressure. Simultaneous application of the decelera-

tive force results in signs of cerebral concussion with confusion, retrograde amnesia, circulatory shock, temporary loss of vision, retinal hemorrhages, subconjunctival hemorrhages, ecchymosis of the eyelids, and periocular edema. The paranasal sinuses are congested and even hemorrhagic.

Similar fundus changes have from time to time been noted by other observers such as Purtscher (2), who noted the appearances of hemorrhages, exudates, and edema after a compression type of injury to the trunk. Vogt (3) reported a similar case, except that the macular area was free from exudate, in contrast to one of the cases reported here. Other similar cases have been reported by Stokes (4), and one similar case by Urbanek (5), in which actual sections of the retina containing the white exudate were taken, revealing collections of transudated fluid.

Of these 5 cases, 2 are the first instances of producing this syndrome under experimental conditions in which the forces were measured, corroborating the findings in the supersonic bailout here reported. Two other cases confirm that the combined hydrostatic and decelerative force application described above produces the syndrome, rather than external wind pressures against the face which occurred to the pilot in the supersonic escape when his helmet blew off during ejection.

EXPERIMENTAL OCCURRENCE OF SYNDROME

The first case of this syndrome occurred during experimental exposure to more than 25G at 500G per rate of onset within a quarter of a second. Injuries were limited to petechial hemorrhages of the sclera, retinal vascular spasms, superficial retinal hemorrhages, and signs of concussion manifested by severe frontal headache for thirty-six hours. This experiment was performed at Edwards Air Force Base in June, 1951. It consisted of a linear deceleration on a rocket sled with the subject seated facing forward, restrained by shoulder straps, lap belt, and inverted "V" leg straps, with the head bare and bowed as far forward as possible. The head swung through an arc of 55 degrees during deceleration. A windshield excluded windblast.

The second instance occurred during a rocket sled deceleration experiment in December, 1954, at Holloman Air Force Base. The

subject was in the same position, and, in addition to the restraints described above, a chest belt was drawn tightly enough around the lower part of the thorax to completely stop rib movements in respiration. Head and face were completely enclosed in a protective helmet which was lashed to the headrest, limiting the forward motion of the head. Exposure to an initial force of 35G applied at a 600G per second rate of onset was followed by a plateau of 25 to 27G lasting for .4 of a second and a second peak of 40G of less than .1 second duration followed by a plateau of approximately 25G for the remainder of a total duration of 1.1 seconds. Facial congestion was extreme, and eye signs were: appearance of lateral subconjunctival hemorrhages which later became confluent, forming complete hemorrhagic blebs, along with periocular edema and hemorrhage; initial inability to see, then foggy light perception, with rapid clearing and ability to count fingers. Cerebral signs were not severe. The protective helmet excluded windblast from the head and face (see Figure 1).

OCCURRENCE OF SYNDROME IN SUPERSONIC ESCAPE

The third instance, in February of 1955, was incurred during escape by ejection seat from an aircraft (F-100) at a velocity of 1,160 feet per second at an altitude of 6,500 feet, with the craft in an 80 degree dive. G.S., the test pilot, was protected by helmet and visor until shortly after the exit. Shoulder straps were loose, and he was retained by his lap belt only during ejection. For the initial wind drag linear deceleration, the pilot was seated with his face on his knees, so that in addition to the compression of the lower abdomen with the lap belt and the setting up of a hydrostatic column from hips to head level, there was an impingement of the abdominal viscera against the diaphragm. This, and possibly other jolts to the abdomen prior to separation from the seat, resulted in traumatic perforation of the terminal ilium. The patient was seen within an hour after the incident, and the head and face were extremely congested. Vision in both eyes was limited to counting fingers, and external examination revealed extensive periorbital ecchymosis. There were complete subconjunctival hemorrhages so extensive that no sclera could be seen, and there appeared to be a mild bilateral proptosis of the eyes, although no exophthalmometer

readings were available at the time. Both pupils reacted well to light and accommodation, and there appeared to be no abnormalities of the extraocular muscles. Both corneas and anterior chambers appeared to be clear. Fundus examination at the time revealed normal clear media with no involvement of the discs. There was a mild arteriospasm present, and a few small questionable hemorrhages in the periphery.

On the second day, a few retinal hemorrhages began to appear accompanied by cotton wool patches. The first hemorrhages were small and flame shaped, but later the same day several round and one large subhyaloid hemorrhages appeared.

On the third day, the hemorrhages were more pronounced, with new ones occurring all the time. At this time a medical artist was called in, and complete fundus drawings were made, showing the extensive hemorrhages in both eyes. The large subhyaloid hemorrhages in the left eye are well illustrated in Figure 3, and they appear to be venous in nature, occurring along the branch of the superior nasal vein and superior temporal vein. The edema patches and smaller hemorrhages are also well illustrated in this drawing. Figure 2 illustrates the many smaller hemorrhages and edema occurring in the right eye during the third day of hospitalization.

By the fourth day, the patient was able to read large print, but new hemorrhages were still appearing all the time, together with more generalized retinal edema, giving the macular areas a cherry red appearance.

On the fifth day, the patient's vision had improved to 15/30 in each eye, and the entire process appeared more stationary. During this entire period, the external appearance of the eyes remained much the same.

From the fifth day on, there was a continuous improvement in vision, with slow disappearance of the hemorrhages and exudates. No new hemorrhages appeared after this time, although the eventual clearing was quite slow, especially in the left eye. Figure 4 illustrates this, and represents a drawing of the left fundus about two weeks after the initial injury, still showing the extensive hemorrhages and edema patches. The last drawing, Figure 5, shows the left fundus six weeks after the injury. Here can be seen the organization of the nasal hemorrhage and the complete disappearance

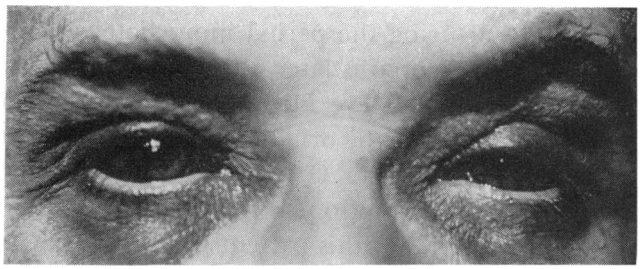

FIGURE 1. SUBJECT OF EXPERIMENT 2 AT 48 HOURS

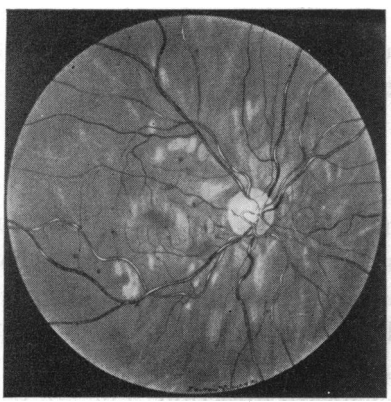

FIGURE 2. TEST PILOT G.S. AT 48 HOURS, O.D.

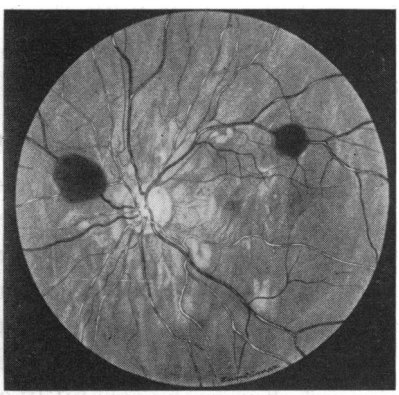

FIGURE 3. TEST PILOT G.S. AT 48 HOURS, O.S.

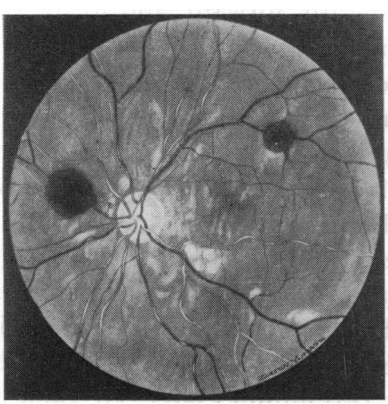

FIGURE 4. TEST PILOT G.S. AT TWO WEEKS, O.S.

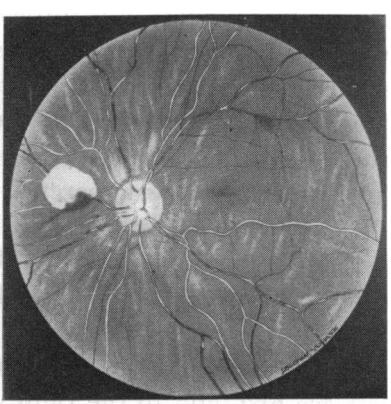

FIGURE 5. TEST PILOT G.S. AT SIX WEEKS, O.S.

of the large temporal hemorrhage. Practically no retinal edema remains at this time. During the period immediately following the injury, and until three months later, there appeared to be a small central scotoma in the left eye identifiable with the 1/1000 test object. Three months after the original injury, the patient's vision was 20/20 O.D. and 20/20–2 O.S. Color vision, stereopsis, biomicroscopy, and depth perception were all within normal limits.

It has been estimated that this patient was subjected to 40G for .31 seconds, and the patient's head accelerated to a peak of 60G for .09 of a second, accounting for the severity of the retinal hemorrhages. This, plus the pooling effect on the vascular system (2) when the human body is subjected to great G forces, probably accounts for the signs observed above.

OCCURRENCE OF SYNDROME IN LOW VELOCITY ESCAPE

The fourth instance, in March, 1955, occurred to Goodyear Company test pilot L.E. in an experimental flight in a sail plane, investigating the Sierra Wave phenomena near Bishop, California. At an altitude of 15,000 feet and an airspeed of 60 miles per hour, he entered an area of turbulence under a lenticular cloud formation. The left wing of the sail plane, stressed to 8G, was violently sheared off near the cockpit in a downward direction. The tail section broke off next. The cockpit went into violent tumbling in an outside loop attitude. The pilot, in winter clothing, including winter flying boots, was thrown upward, forward, and to the left with such violence that the left shoulder strap (1,500-pound tensile strength) was broken, and the canopy was knocked off by his head. He wore a crash helmet. Apparently his feet were caught in the rudder pedals by the winter flying boots. He sustained temporary loss of consciousness, apparently related to the stunning blow against the canopy, and did not recall the subsequent violent tumbling gyrations. Eventually he was able to pull the ripcord of his parachute. He was aware of loss of vision lasting about ten minutes, from the time the wing sheared off until he was able to see his wrist watch again during the parachute descent. Vision returned to the left eye first, and to the right eye two minutes later. He came to earth with no further injury. His boots were found still stuck in

the rudder pedals in the wreckage of the cockpit. Physical examination showed severe contusions of shoulders and hips on strap impingement areas, periocular congestion, subconjunctival hemorrhages, mild signs of concussion, and mild circulatory shock at the time of the first examination. There were, however, no signs of retinal hemorrhages. Recovery was complete and uneventful in three weeks. Tumbling with an axis of rotation through the feet resulted in high negative G, and resultant hydrostatic pressure rise in the subject's head. The low speed of unpowered flight precluded windblast as a factor.

The fifth instance of this syndrome occurred during a takeoff crash of an F-84F jet in August, 1955, in which the pilot either intentionally or accidentally ejected himself through the canopy. The aircraft, at stalling speed, struck and broke four power lines at a height of between 38 and 52 feet. In succession the aircraft struck a house, a 24-inch diameter pine tree, and a second house, in a path 326 feet beyond the first impact. At some point along this path the pilot ejected through the canopy. The seat collided, footrest first, with the base of a tree, coming to rest six feet from this final point of impact. The pilot was found in the ejection seat lying on his right side, at a point 347 feet from where the aircraft struck the ground. The pilot was conscious, but in a state of shock.

Injuries included fracture of all four extremities and a head injury, the latter consisting of cerebral concussion and contusion with retrograde amnesia, a mental confusion and transient disorientation. The pilot has referred to a normal take off roll, but it is not determined if this refers to the flight in question or to a prior flight. Memory is clouded and unreliable to about forty-eight hours prior to the accident and there is still considerable confusion.

The initially observed dusky flushing of the face, later appearance of lateral sub-conjunctival hemorrhages which later became confluent, forming complete hemorrhagic blebs, along with the peri-ocular edema and hemorrhage; also the petechial hemorrhages and bruises from shoulder straps; also the inability to see then foggy light perception, with rapid clearing and ability to count fingers—all these bear a striking and fascinating similarity to the sequence described by Lt. Colonel Stapp, in his experiments with abrupt deceleration at Holloman Air Force Base.[1]

[1] Reported by Major Eugene Leiter, USAF(MC) Flight Surgeon.

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DISCUSSION

DR. DONALD J. LYLE. My contribution to this presentation is extremely meager. I first met Col. Stapp and examined his eyes a number of years ago, when he was stationed at the Areo Medical Laboratory, Wright-Patterson Air Base, Dayton, Ohio. At that time he was engaged in crash experiments to determine the limit of human tolerance, using rocket sleds.

In the experiment resulting in retinal hemorrhages, he had decelerated from 154.8 m.p.h. to 34.4 m.p.h. in 31.2 feet with measured force of 46G. There was no blackout or grayout at the end of the run. Retinal vascular spasm was evidently present, followed by retinal edema and serous exudates and, within four hours, by fogging of vision in both eyes. The next day, dimness remained in the right eye, with paracentral and peripheral scotomata. The vision in the left eye had cleared, although a peripheral hemorrhage producing scotoma existed. This history was obtained, and examination made, at the time, and immediately following the experiment. One month later, when I first saw the condition, the hemorrhages were still present. Vision at that time, corrected, was 20/20 in each eye. The scotomata persisted, with slight annoyance to vision in the right eye.

Col. Stapp was last examined by me about a year ago, at which time his corrected vision was 20/20 in each eye. The scotomata were still present but were clearing with rapid improvement, following the last experiment, when he was projected 632 m.p.h., stopping in 1.4 seconds. However, Col. Stapp confided in me that he could not conscientiously recommend this procedure as routine treatment for retinal hemorrhage or residual scotomata.

One might possibly expect, after 29 experiments, brain damage such as is found in a "punch drunk" pugilist. The contrary is the case, as any one can testify after a few minutes' conversation with Col. Stapp.