POSTTRAUMATIC OULOCARDIAC SYNDROME FROM A NEURO-SURGICAL POINT OF VIEW

REPORT OF A CASE

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In cases of head injury the differential diagnosis of an intracranial hematoma is important. A useful diagnostic criterion is the slowing down of the pulse rate caused by increased intracranial pressure; this sign in a case of head injury is considered almost pathognomonic of an intracranial hematoma.

However, trauma of the eye or orbital region may cause a retardation of the heart rate without any existing intracranial hematoma. This phenomenon is produced by the oculocardiac reflex, which is well known therapeutically in cases of paroxysmal tachycardia, in which pressure on the eyeball may abort the attack.

In the present paper a case is reported in which a marked oculocardiac reflex followed a blow to the eye.

CASE REPORT

J.Nr. 620/1946. B.P., a 25-year-old man, was admitted with a diagnosis of intracranial hematoma.

About 4 hours previously he had been involved in a fight and received a blow to his left eye. He tumbled down, bumped his occiput against the pavement and fainted. After some minutes of unconsciousness he recovered, and was taken to the out-patient department of another hospital. On examination there, about ½ hour after the accident, he was slightly confused but well oriented. On the left side a small subconjunctival hematoma and a slight mydriasis were found. The left pupil reacted sluggishly to light. There were no other signs of injury. Roentgenograms of the skull did not disclose any fracture.

The pulse rate was 60 per min., and the B.P. was 130/70. Shortly afterwards the pulse rate dropped to 44, and during the next 2 hours it became progressively slower until it reached 38 per min. The B.P. was unchanged and the patient was fully conscious and mentally unaffected.

The left subconjunctival hematoma and the palpebral edema had increased, and the left pupil was more dilated. The clinical picture gave suspicion of an intracranial hematoma and the patient was transferred to our clinic.

Examination, 4 hours after the accident. The patient showed no signs of slow cerebration. He was mentally unaltered and without any retrograde amnesia.

The pulse rate was 36 per min. B.P. was 120/80. Respiratory rate was 20 per min.

The left “black eye” showed considerable palpebral edema, and a subconjunctival hematoma was found. The movements of the eyeball were impaired, with a partial paresis of the rectus inferior and oblique muscles. The left pupil showed mydriasis with diminished reaction to light. The vision was 1.5 bilaterally. The media were clear and the left fundus showed a slight retinal edema. The intraocular pressure measured 12 mm. in both eyes; it was thus within normal limits, but on the threshold of a bulbar hypotonia.

Course. The clinical picture was ascribed to an exaggerated oculocardiac reflex elicited by the intraorbital hematoma and edema, as a thorough neurological examination had not revealed any other signs of an intracranial hematoma. His course was uneventful. On the day after admission the pulse rate increased to above 40 and the patient did very well. On the 4th day he left the hospital on his own volition. He still had a slight left-sided subconjunctival hematoma and a slow pulse rate.

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Electrocardiographic Examination. On the 2nd day after admission electrocardiography was performed. The tracings showed a bradycardia with a rate of about 40 per min., but were otherwise normal (Fig. 1 A).

After closing the eyelids digital pressure was exerted upon the eyeballs.

Pressure on the left, injured eye provoked an extreme bradycardia. After a latent period of 15 sec., an arrhythmia ensued, with considerable lengthening of the compensatory period. After a heart beat of 0.7 sec. there was a pause of 3.6 sec. It may be presumed that this prolonged compensatory period was the beginning of an asystolia. The electrocardiogram revealed that the pressure against the orbital content also provoked a nodal rhythm (Fig. 1 B), which disappeared immediately on relieving the pressure.

On the other hand, the same or even a stronger pressure exerted against the right healthy eye could not provoke more than a slight slowing down of the pulse rate, without any other pathological findings on the electrocardiogram. Of course, too strong a pressure was not exerted, as there might have been a risk of retinal damage.

The trauma caused by the blow thus seemed to have sensitized the left eye, eliciting a spontaneous oculocardiac reflex.

Control Examination. The patient was again examined 1 ½ years after the accident. Neurological findings and both eyes were normal. The pulse rate at rest was slightly above 60 per min. Pressure on either eyeball caused a slowing down of the pulse to a rate of 50 per min. The electrocardiogram did not reveal any pathologic changes of the kind observed at the acute stage of the accident.

DISCUSSION

The oculocardiac reflex is often called the “Aschner-Dagnini reflex” as these authors,1,7 in 1908, independently described a reflex that was elicited by pressure on the eyeball and provoked a slowing down of the pulse rate.