Ocular pneumoplethysmography in head-injured patients

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Severe head injury is frequently associated with multiple trauma. In the comatose patient, endotracheal intubation and ventilator support are often required, if there is associated dysynchronous spontaneous effort. The latter is managed with therapeutic (drug) paralysis. An elaborate life-support and monitoring system coupled with controlled paralysis limits the mobility of the patient for diagnostic procedures, and a continuing reevaluation of neurological status is difficult. Under these circumstances the ocular pneumoplethysmograph provides a simple rapid noninvasive assessment of ocular blood flow, and this reflects cerebral blood flow and alterations in brain compliance. Alterations in the therapeutic regimen can be based on these observations.

KEY WORDS • ocular pneumoplethysmography • head injury • brain compliance • hemodynamics • blood flow

Over the past decade, an intense effort has been made to improve the assessment and management of the head-injured patient. An important observation in this regard has been the recognition of a clear delineation between primary brain damage as a direct result of the head injury and brain damage secondary to associated ventilatory problems and altered intracranial fluid dynamics. In the area of altered intracranial fluid dynamics, epidural, subdural, intracerebral, and other hemorrhages have long been recognized as the principal sources of acute and subacute fluid changes. Assessment of intracranial hemorrhage, especially since the advent of computerized tomography (CT), is straightforward, and clearly defined principles of management have been established. However, another major source of secondary brain damage due to altered intracranial fluid dynamics is the elevation of the intracranial pressure (ICP) as a result of brain edema. It has been established that there must be considerable brain edema before an elevation in the ICP is noted, but that little further increment in brain edema results in a precipitous rise of the ICP.

This volume-pressure response has stimulated the search for some means of determining degrees of brain edema, with its associated loss of brain compliance, in order that therapy might be directed at its control, prior to a precipitous rise of the ICP. We are reporting a prospective study in 81 patients, with the intent of reviewing ocular pneumoplethysmography (OPG) in head-injured patients for evidence of OPG changes related to suspected loss of brain compliance. Also, the data from the calculations of ocular blood flow in these 81 patients is compared to data from 767 patients without head injury.

Clinical Material and Methods

The mechanics of the instrument (OPG-Gee) used in this study have been described elsewhere. These mechanics have been briefly summarized in the companion paper to this report. The 767 control patients without head injury have been the subject of a previous report, in which the ophthalmic systolic pressure data reflecting the status of the 1534 carotid/ophthalmic arterial hemisystems were described. The ocular blood flow (OBF) data in these 767 patients have not been previously reported. The method for calculation of these data is contained in the companion paper to this report. Briefly, a calculation of OBF (ml/min) results from a product of the pulse rate, the millimeter amplitude of the maximum ocular pulse wave as measured by the OPG, and a fixed factor (0.0016). This latter factor is a product of the ratio of the instrument calibration (l cu mm/10 mm pen deflection, 1/10), the conversion ratio of milliliters to cubic millimeters (1/1000), and the ratio of the surface area of the eye to that portion of the surface area covered by the instrument eyecup (16/1). Arteriography in these 767 patients demonstrated total occu-
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TABLE 1
Ocular blood flow data for 767 control patients and 81 head-injured patients*

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>No. of Eyes</th>
<th>OBF (ml/min)</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>no head injury</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>no carotid lesion</td>
<td>979</td>
<td>1.45</td>
<td>0.56</td>
</tr>
<tr>
<td>carotid lesion</td>
<td>555</td>
<td>1.15</td>
<td>0.48</td>
</tr>
<tr>
<td>head injury</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>survived (47)</td>
<td>94</td>
<td>1.16</td>
<td>0.51</td>
</tr>
<tr>
<td>died (34)</td>
<td>68</td>
<td>0.64</td>
<td>0.51</td>
</tr>
</tbody>
</table>

* OBF = ocular blood flow; SD = standard deviation.

Direction or stenosis of hemodynamic consequence (75% or greater, cross-sectional area) in 555 of the 1534 carotid arteries. The remaining 979 carotid arteries contained no lesions of hemodynamic consequence. The OBF data for these two groups of carotid arteries are presented in Table 1. The mean of the OBF in the 555 eyes ipsilateral to carotid lesions of hemodynamic consequence is only 79% of that in the 979 eyes ipsilateral to carotid arteries without such lesions (p < 0.0001).

A normal finding in recordings of peripheral arterial pressure or blood flow is a notch in the catacrotic (descending) limb of the wave, which results in a dicrotic wave. The dicrotic wave reflects the combined effects of aortic valve closure and of peripheral resistance to blood flow. A dicrotic wave is seen in peripheral plethysmography also. However, the OPG wave normally has no catacrotic notch. It has the character of a sine wave, which reflects the extremely low intracranial arterial resistance and the damping effect of the carotid siphon. Not only does this latter reduce intracranial arterial systolic pressure, but it also prevents reflection of the effect of aortic valve closure, as there is no reversal of blood flow in the internal carotid artery during cardiac diastole. It is rare to see dicrotic ocular pulse waves in patients other than those with head injury. It is presumed that the development of a dicrotic ocular pulse wave reflects increased intracranial resistance. Diffuse intrinsic arteriopasm, mass lesions (including hematomas), and brain edema (with associated loss of compliance) are all possible causes of increased intracranial arterial resistance.

As the study progressed, it became obvious that a grading system for the severity of ocular pulse wave catacrotic notching was necessary. Five stages were established: absent, mild, moderate, severe, and critical. Figure 1 is an example of severe catacrotic notching. Every ocular pulse wave from both eyes is affected. The slow variation in the baseline of both ocular pulse tracings reflects the respiratory cycle. During any one of these cycles, the ocular pulse amplitude varies with the cardiac stroke output changes associated with inspiration and expiration. The moderate form of dicrotic ocular pulse wave is identical in appearance to this severe form, but it is intermittent. However, it is usually observed in the same period of every respiratory cycle. The mild form of catacrotic notching is the same in appearance as that noted in Fig. 1, but it is not seen in every respiratory cycle, and it is usually observed only when the ocular pulse waves have achieved maximum amplitude. Figure 2 demonstrates the critical form of catacrotic notching. The ocular pulse waves associated with the early period of a respiratory cycle appear almost square-topped. The magnitude of the ocular pulse waves in this example is exceptional, as the critical form of dicrotic notching is usually seen in patients with much lower ocular pulse amplitudes.

**Results**

Among the 81 patients in the head-injured group, there were 47 survivors and 34 deaths. The mean of the OBF's in the two groups is as noted in Table 1. The difference between the two groups, and the difference between each group and the figure noted for patients without head injury or carotid lesions of hemodynamic consequence (pilot group) were all statistically significant (p < 0.0001). The mean OBF of the survivors was reduced 21% compared with the pilot group. The mean OBF of those in the head-injured group who died was reduced 56% compared with the pilot group.

Dicrotic ocular pulse waves were noted in 64% of the survivors and 74% of those who died. Twelve of the 81 patients died on the day of admission or on the second hospital day. Each of these 12 patients had only one OPG study. The remaining 69 patients had serial studies. The distribution of grade of severity was quite different in the two groups. Mild or moderate catacrotic notching was seen in 32% (15) of the 47 survivors but in only 12% (four) of the 34 who died. Severe catacrotic notching was seen in 23% and 35% of the patients who survived and died, respectively. Only four of the 47 survivors (8.5%) had the critical form of catacrotic notching, whereas this was noted in 26% (nine) of the 34 who died. The absence of catacrotic notching had a different connotation in the two subgroups. Nine of the 34 patients who died had no dicrotic ocular pulse waves noted. However, in each of these studies the ocular pulse wave amplitudes were so low that catacrotic notching could not be appreciated. Of the 47 survivors, 17 (36%) had no catacrotic notching noted in any of the serial studies. In none of these studies was the ocular pulse amplitude so low as to preclude appreciation of catacrotic notching.

The data from both subgroups (survivors and those who died) were reviewed collectively. There were 20 patients in whom the OBF was bilaterally reduced to less than 0.53 ml/min. Only one of these patients survived, and this patient has persisted in a vegetative state for over 2 years. Thus, a reduction of bilateral OBF to less than 37% of the mean of normal was lethal, with regard to cerebral survival, in 100% of patients. Only one of these patients had catacrotic notching of mild or moderate degree. Twelve of the 20 patients had
catacrotic notching of severe or critical degree. Seven patients had no dicrotic ocular pulse waves.

Among the 47 survivors, there were only three patients in whom catacrotic notching persisted on serial studies. One of these three patients suffered a severely depressed left temporal skull fracture with extensive destruction of the underlying cerebral hemisphere. In spite of considerable debridement of this area, there was moderate recovery of right upper and lower extremity function and of speech. Computerized tomography scans were not performed. Thus, there is no evidence for the suspected cause of persistent dicrotic ocular pulse waves. However, in the other two patients, serial CT scans of the head demonstrated the probable cause of persistent catacrotic notching in both. One of these two patients was the single survivor of the 20 patients in whom bilateral OBF of less than 0.53 ml/min was encountered. Serial CT of the head demonstrated progressive bihemispheric atrophy, associated with the development of large lateral ventricles. This was initially thought to represent hydrocephalus. However, at the time of ventricular shunting, no increased ventricular pressure was noted. The second of these two patients has also persisted in the vegetative state. This was one of the very few patients with a critical degree of catacrotic notching who survived. Serial CT of the head demonstrated progressive diffuse brain atrophy (Fig. 3). In multiple serial CT scans of the head this patient never exhibited any lesions suggestive of acute subdural hematomas. Although the appearance of the final two studies suggests bilateral hygromas secondary to chronic subdural hematomas, the serial CT and the OPG evidence indicates that ischemic brain atrophy preceded the fluid accumulations. In both patients the replace-

![Figure 1](image-url)

**Fig. 1.** Four-channel ocular pneumoplethysmograph (OPG-Gee) is obtained at a paper speed of 10 mm/sec. The *two short arrows* on the electrocardiogram (ECG) channel encompass 15 seconds of recording, during which 26 QRS complexes are noted. Thus, the pulse rate is 104/min. The *arrow* extending from the beginning of the first right eye pulse demonstrates that this occurred at an ophthalmic systolic pressure of 101 mm Hg. The *arrow* extending from the beginning of the first left eye pulse demonstrates that this occurred at an ophthalmic systolic pressure of 99 mm Hg. The *first double-ended arrow* connects the maximum ocular pulse amplitudes which are 9 mm in both eyes. The *second double-ended arrow* connects the volume calibrations of the respective ocular pulses, in which a 10-mm pen deflection represents a 1.0 cu mm ocular pulse volume change. The product of the pulse rate (104) and the respective maximum ocular pulse amplitude (9) is 936. The product of 936 and 0.0016 results in an ocular blood flow of 1.50 ml/min, which is above the demarcation of 0.53 ml/min, as defined in the text. The ophthalmic systolic pressures in this patient were unequal by less than 5 mm Hg, and both were above the lower limit of normal for a brachial systolic pressure of 130 mm Hg, as measured in this patient immediately after the test by arm cuff and auscultation. The only adverse aspect of this test is the catacrotic notching of all of the ocular pulse waves. Subsequent studies in this patient demonstrated progressive restoration to normal, characterized by the complete absence of dicrotic notching. The patient recovered from the head injury and presumed brain edema.
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ment of brain tissue by fluid is the presumed source of the decreased compliance, as indicated by the persistent continuous dicrotic ocular pulse waves.

Discussion

In a collaborative study for appraisal of the criteria of cerebral death, coma was listed with cerebral non-response, apnea, dilated pupils, absent cephalic reflexes, and electrocerebral silence; all five criteria must be present for 30 minutes at least 6 hours after the onset of coma and apnea. In addition, this study proposed as a safeguard a confirmatory test relating to cerebral blood flow. The OPG is a readily portable device, and bedside testing can be done with ease. The test has a duration of 30 to 45 seconds, and can be repeated at short intervals (every 20 minutes). Its technical application is simple, and interpretation of the record is straightforward. It provides a direct measure of bilateral simultaneous ophthalmic systolic pressures and OBF's.

It appears that bilateral reduction of OBF below 0.53 ml/min is incompatible with cerebral survival, as all 20 patients in this study in whom this finding was observed either died (19) or persisted in a vegetative state (one).

Twenty-four of the 81 patients had continuous ICP measurements using cranial bolts. Although the data were insufficient for meaningful analysis, a consistent relationship between ICP measurement and dicrotic ocular pulse waves was noted. Absent or intermittent catacrotic notching (Grades 0 to 2) was noted in patients

FIG. 2. The first ocular pulses are noted at a vacuum of 260 mm Hg, and both ophthalmic systolic pressures are equal to 103 mm Hg. At a brachial systolic pressure of 170 mm Hg, both ophthalmic systolic pressures should be above 112 mm Hg. The pulse rate is 136/min. The maximum right ocular pulse amplitude is 8 mm, and the respective ocular blood flow is 1.74 ml/min. The maximum left ocular pulse amplitude is 7.5 mm, and the respective ocular blood flow is 1.63 ml/min. Both ocular blood flows are well above the demarcation of 0.53 ml/min. However, all ocular pulse waves have catacrotic notching, and the waves in the midsegment of each respiratory cycle are nearly square-topped in appearance. ECG = electrocardiographic recording.

FIG. 3. Serial computerized tomography scans of the head on the 38th day after injury. Similar studies on the day of injury, and 3 and 17 days after injury demonstrated no evidence of acute subdural hemorrhage. The study on the 17th day post-injury did show an anterior pole rim suggestive of a small subdural fluid collection, with probable indication of some cerebral atrophy. The scans shown here demonstrate atrophy with fluid replacement in the anterior, middle, and posterior cranial fossae on the 38th day.
with ICP's ranging between 0 and 15 mm Hg. Continuous (Grades 3 and 4) catacrotic notching was noted in patients with ICP's over 15 mm Hg. However, in the latter category (high ICP's), absence of catacrotic notching was also noted. This was invariably in patients in whom very low ocular pulse amplitudes precluded any appreciation of catacrotic notching. The most important conclusion to be drawn from these observations is that a progressive loss of brain compliance can be detected by serial noninvasive testing before critical levels (> 30 mm Hg) of ICP are reached. Progression from intermittent to continuous catacrotic notching was used as the indication for the institution of a treatment regimen designed to improve brain compliance. Another group of investigators has made similar observations using a different noninvasive technique, common carotid artery velocity waveform analysis. That study was directed at differentiating the brain-injured patient from the brain-dead patient. Although it included only eight patients in each group, for a total of 16 patients, the data were carefully analyzed and the conclusions appear to be both valid and clinically useful.

Finally, the head-injured patient frequently presents with additional injury to other structures. Ventilator support is often required. Therapeutic (drug) paralysis is commonly necessary, either to limit self-destructive combative ness or ablate respiratory efforts dysynchronous with ventilator support. Once a life-support and monitoring system is in place, it is difficult to move the patient to other areas of the hospital for further diagnostic or therapeutic procedures. Also, therapeutic paralysis limits serial reassessment of neurological status. If serial OPG studies demonstrate deterioration of oculovascular physiology compatible with severe brain ischemia, therapeutic paralysis is discontinued. This permits an assessment of the standard criteria for brain death. If brain death is documented, and if the patient is a possible organ donor, the therapeutic regimen can shift to that which is ideal for organ maintenance.

References


