Transient and repetitive rises in blood pressure synchronized with plasma catecholamine increases after head injury

Report of two cases

TADAHITO SHIOZAKI, M.D., MAMORU TANEDA, M.D., MASANOBU KISHIKAWA, M.D., ATSUSHI IWAI, M.D., HISASHI SUGIMOTO, M.D., TOSHIHARU YOSHIKA, M.D., AND TSUYOSHI SUGIMOTO, M.D.

Departments of Traumatology and Neurosurgery, Osaka University Medical School, Osaka, Japan

The authors report two patients with repetitive episodes of acute transient rise in blood pressure synchronized with increases in plasma catecholamine after severe head injury. In both cases, the paroxysmal hypertension occurred suddenly on the 2nd day posttrauma, and its frequency declined gradually, disappearing with time. The pathophysiological basis of this peculiar clinical manifestation is discussed.

**KEY WORDS** • paroxysmal hypertension • head injury • catecholamine

It is well known that hypertension frequently develops after severe head injury and follows one of two patterns: persistent or labile. Many reports have revealed that continuous sympathetic overactivity causes persistent hypertension. However, there are only a few clinical reports on labile hypertension, and the etiological mechanism remains unclear.

We describe two cases of repetitive episodes of acute transient rise in blood pressure after severe head injury, and demonstrate that the fluctuations in blood pressure were synchronized with plasma catecholamine levels.

**Case Reports**

**Case 1**

This 58-year-old man was admitted to our department following a traffic accident, in which he had sustained a severe head injury and a left tibial fracture. On admission, he was deeply comatose, his blood pressure was 154/94 mm Hg, and his pulse rate was 80 beats per minute.

A computerized tomography (CT) scan obtained 8 hours after injury showed a small hematoma in the left dorsal midbrain and a small hematoma deeply situated in the right hemisphere adjacent to the internal capsule (Fig. 1). A skull x-ray film was normal. The right pupil was dilated and the left was in midposition; both were responsive to light. He had conjugate deviation of the eyes to the left. He was intubated, hyperventilated, and treated with fluid restriction for 7 days. An intraventricular catheter was inserted for continuous monitoring of intracranial pressure (ICP). The ICP remained between 5 and 25 mm Hg during this period.

Fluctuations in blood pressure occurred suddenly on the 2nd day after injury. The patient showed no neu-

![Fig. 1. Computerized tomography scans obtained 8 hours after injury demonstrating a small hematoma in the left dorsal midbrain (left) and a small hematoma deeply situated in the right hemisphere adjacent to the internal capsule (right).](image-url)
The patient's medical history was unremarkable for hypertension and other endocrine disorders, including pheochromocytoma. To rule out the possibility of masked pheochromocytoma, laboratory studies and abdominal CT were performed. An abdominal CT scan revealed neither an adrenal gland pheochromocytoma nor adrenal hemorrhage. Laboratory studies demonstrated no evidence of pheochromocytoma.

The patient was transferred to rehabilitation hospital on Day 22 with moderate neurological recovery. After discharge, no paroxysmal hypertension was observed.

Case 2

This 52-year-old man suffered from myasthenia gravis 10 years prior to admission and underwent thymectomy 1 year later. At the time of the accident, however, he was in good health and had no history of hypertension after the operation. The patient was struck by a train and was deeply comatose on arrival at our hospital. He had an open frontal fracture with prolapsed brain tissue and right forearm fractures. His blood pressure was 110/80 mm Hg and his pulse rate was 76 beats per minute.

A CT scan on admission (Fig. 3) showed a depressed open fracture of the right orbitofrontal bone and mul-

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**TABLE 1**

<table>
<thead>
<tr>
<th>Measured Variable</th>
<th>Normal Values</th>
<th>Timing of Measurement*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Point A</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Case 1</td>
</tr>
<tr>
<td>catecholamine (ng/ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>norepinephrine</td>
<td>0.07–0.31</td>
<td>0.93</td>
</tr>
<tr>
<td>epinephrine</td>
<td>&lt; 0.10</td>
<td>0.09</td>
</tr>
<tr>
<td>dopamine</td>
<td>&lt; 0.10</td>
<td>&lt; 0.10</td>
</tr>
</tbody>
</table>

hemodynamics

| cardiac index (liter/min/sq min) | 3.16 | 2.60 | 4.66 | 4.57 | 3.14 | 2.79 | 4.68 | 4.44 | 3.08 |
| stroke volume (ml/beat)         | 62.5 | 50.0 | 76.4 | 56.8 | 63.4 | 51.4 | 74.1 | 57.3 | 61.0 |
| total peripheral resistance (dynes-sec-cm⁻²) | 1042 | 1182 | 1115 | 1317 | 1039 | 1132 | 1128 | 1359 | 1086 |

* Each time point corresponds to the same letter in Fig. 2 (Case 1) and Fig. 4 (Case 2). – = measurement not available.
Paroxysmal hypertension after head injury

Fig. 4. Case 2. Graph demonstrating the fluctuations in arterial blood pressure (BP) and intracranial pressure (ICP) during the paroxysmal hypertension. Note that the ICP is never greater than 10 mm Hg, although the ICP rises synchronously with elevation in the BP. Points A, B, C, D, and E indicate time points of measurement and correspond to the points in Table 1.

Multiple bilateral orbitofrontal contusions. The left pupil was in midposition, but was unresponsive to light; the right pupil could not be examined because of the right orbitofrontal contusion. The patient breathed spontaneously and blood gas levels were within normal limits. An emergency right frontal craniotomy was performed. The crushed region of the brain and the detached pieces of skull were removed. An intraventricular catheter was inserted for continuous monitoring of ICP. Postoperatively, the patient received continuous hyperventilation and was treated with fluid restriction for 7 days. Laboratory study data minimized the possibility of a pheochromocytoma.

Fluctuations in blood pressure developed suddenly on the 2nd day after head injury. No neurological improvement was observed during this period. The hypertensive phase (190/80 mm Hg) and the normotensive phase (110/50 mm Hg) appeared alternately at intervals of 2 or 3 minutes (Fig. 4). The ICP was accompanied by elevations in blood pressure; however, the ICP was never greater than 10 mm Hg after the operation. The clinical features of the paroxysmal hypertension in this patient were the same as in Case 1. On Day 9, the paroxysmal hypertension subsided spontaneously. The plasma catecholamine levels and the hemodynamics during the paroxysmal hypertension are shown in Table 1. As observed in Case 1, these data also indicated the elevated plasma norepinephrine and epinephrine levels and the hyperdynamic state during the hypertensive phase.

The patient's postoperative course was complicated by a brain abscess and pneumonia. On Day 21 evacuation of the necrotized brain tissue was performed, and the patient was discharged to a rehabilitation hospital on Day 60 with moderate neurological recovery. After discharge, he had no paroxysmal hypertension.

Discussion

Development of hypertension after head injury is not uncommon and has been estimated to occur in 11% to 15% of head-injured patients.7,8 Many clinical reports have revealed that in the acute phase of head injury the hemodynamic characteristics consist of a hyperdynamic state including hypertension, tachycardia, and increased cardiac output. Moreover, it has been demonstrated that this hyperdynamic state correlates well with plasma catecholamine levels.6,11

Recently, it has been recognized that there were two patterns of hypertension after head injury: persistent and labile. There have been many clinical reports of the former; however, only two cases have previously been reported of the latter.1,4 The two cases in our report demonstrated that the hyperdynamic state was accompanied by increases in the plasma norepinephrine and epinephrine levels. As the plasma norepinephrine level reflects the degree of sympathetic nervous activity,7 this sympathetic overactivity seems to have occurred during the paroxysmal hypertension in our cases. Sympathetic overactivity with labile hypertension has been found to occur in patients with head injury,10 intracranial hypertension,10 spinal cord injury,10 and pheochromocytomas.1 In our cases, the physical examination and spine x-ray films ruled out spinal cord injury, and both the laboratory studies and abdominal CT excluded the possibility of a pheochromocytoma. Intracranial hypertension after head injury did not appear to play a pathogenetic role in this phenomenon because the ICP was lower than 10 mm Hg during the paroxysmal hypertension (Fig. 4). Therefore, injury to the regulatory centers of blood pressure in the brain seems to have been the cause.

Blood pressure is controlled by the interlaced neural connections, including the hypothalamus, thalamus, septum, amygdala, orbitofrontal cortex, and brain-stem regions such as the nucleus ambiguus and nucleus tractus solitarii.4,10 Theoretically, therefore, fluctuations in blood pressure may occur when any part of these connections is damaged. Injuries to the brain-stem regions (Case 1) and the orbitofrontal cortex (Case 2) may have been the cause of the labile hypertension in our patients (Figs. 1 and 3).

Evans, et al.,3 reported that a tumor in the brain stem caused hypertension similar to that observed in pheochromocytoma. On the other hand, the orbitofrontal cortex is believed to inhibit sympathetic activity and to promote parasympathetic excitation.4,10 Thus, traumatic lesions in the brain stem or in the orbitofrontal cortex may be proposed as the cause of the sympathetic overactivity after head injury. It is difficult to understand why there have been only a few reported patients with labile hypertension after head injury as the orbitofrontal cortex is one of the most vulnerable areas in head injury. We speculate that labile hypertension may occur only when these neural linkages are injured incompletely in several regions and therefore cannot completely perform the integrating functions as a control system. The sympathetic overactivity would diminish spontaneously with reintegration of the injured brain, as the frequency of the fulminating hypertension declined gradually and then disappeared with time in our cases.

References

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