Continuous Monitoring of Jugular Bulb Oxygen Saturation in the Management of Patients with Severe Closed Head Injury

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Introduction

Continuous monitoring of intracranial pressure (ICP) is essential in the management of the patients with severe head injury, and various methods to decrease ICP have been utilized [1]. Hyperventilation, however, may occasionally cause inadequate cerebral perfusion, resulting in secondary brain damage in patients with increased ICP [2]. We have performed experimental and clinical studies using simultaneous monitoring of ICP and jugular bulb oxygen saturation (SjO₂) with a fiberoptic catheter to evaluate dynamic changes in cerebral perfusion and cerebral metabolic rate, with the aim of finding an appropriate treatment strategy for patients with severe head injury [3]. On the basis of our previous data [3] and a review of the literature [4,5], it was considered that a value for SjO₂ less than 50% indicated hypoperfusion and a value more than 80% suggested hyperemia. We present herein our early experience of the management of severely head-injured patients by using simultaneous monitoring of ICP and SjO₂ and discuss the treatment protocol we have developed.

Material and Methods

Patient Selection

This series consisted of 15 comatose patients with severe head injury in whom a simultaneous measurement of ICP and SjO₂ was made continuously. Immediately after each patient's arrival, resuscitative treatment and neuro-

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logical assessment were made. The patients were examined by a computed tomography (CT) scan as soon as possible and assessed using the CT criteria advocated by Marshall et al. [6]. In patients with diffuse brain injury type II and III, continuous monitoring of $SjO_2$ and ICP was employed. When a large extraparenchymatous hematoma was found, the mass was evacuated before employing this monitoring. The following patients were precluded: patients in whom the monitoring systems did not work properly for various reasons, and patients who had pulmonary injury or severe systemic injury, shown by abnormal laboratory data such as hypoxia or severe blood loss.

**Measurements of $SjO_2$ and ICP**

A fiberoptic 4Fr catheter system (Opticath and Oximetric III, Abbott Laboratories, Abbott Park, IL, USA) was used for continuous measurement of $SjO_2$. This catheter tip was placed in the right jugular bulb at the level between the skull and the top of the C-2 vertebra; placement was confirmed with a plain X-ray film. The system was calibrated in vivo using blood slowly withdrawn from the catheter. During continuous monitoring of $SjO_2$, the system was recalibrated every 8h. The saturation of the systemic arterial blood ($SaO_2$) was monitored by pulse oximetry at the patient’s finger.

ICP was measured with a subdural pressure monitoring system (Camino kit, digital pressure monitor model 420, Camino Laboratories, San Diego, CA, USA) inserted through a burrhole made on the frontal convexity in most patients. In the patients from whom masses had been evacuated, this pressure monitoring system was placed through the craniotomy. Increased ICP was defined as more than 20 torr.

In a few patients, local cerebral blood flow (LCBF) and brain temperature were monitored simultaneously by using a laser Doppler flowmeter in addition to continuous monitoring of $SjO_2$ and ICP.

**Illustrative Case Presentations**

**Case 1**

A 22-year-old man had a road traffic accident and was transferred to our hospital immediately after the injury. The initial Glasgow Coma Scale score (GCS) was 8 (E1, V2, M5). A CT scan showed a high-density spot in the posterior part of the corpus callosum, but no signs suggesting increased ICP. Laboratory examination including a complete blood count and a blood gas analysis showed no abnormalities. He was treated conservatively under the simultaneous monitoring of ICP and $SjO_2$ (Fig. 1). The initial values were within normal range. However, 14h later, $SjO_2$ decreased to less than 50%, although ICP remained normal and other vital signs were stable. These data were highly suggestive of dehydration. Soon after administration of physiological saline, the $SjO_2$ returned to normal. The patient recovered uneventfully and returned to his previous life-style.