Neurosurg. Rev.
21 (1998) 98–101

Secondary insults during intrahospital transport of neurosurgical intensive care patients

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Abstract

Secondary insults occuring after injury have been prospectively assessed in seven head-injured patients who required intrahospital transfer to a computerized tomography unit for re-evaluation of their brain injury. During transportation the intracranial pressure, blood pressure, and arterial blood gases were monitored. A significant increase in intracranial pressure was observed during transport ($p < 0.01$). The conclusion is that patients should be ventilated and have appropriate sedation and analgesia. This could provide some protection against secondary insults.

Keywords: Head injury, intracranial pressure, transportation of patients.

1 Introduction

Patients at the Neurological Intensive Care Units (ICU) need to be transported within the hospital to various diagnostic facilities such as Computerized Tomography (CT) and Magnetic Resonance Imaging (MRI). Provided ventilation is adequate, critically ill patients can be transferred safely [2]. Various studies have shown that transportation generally causes secondary ischemic brain insults in many patients [1]. Hypotension, hypoxemia, and intracranial hemorrhage ICH can result in ischemic brain injury in head injured patients during intrahospital transport. They can be considered secondary insult. It was shown that on arrival at hospital up to 40 % of severely head injured patients have hypoxia and hypotension, which also occur in a further 33 % of patients in the ICU and are associated with a significant increase in mortality and morbidity.
More than three-quarters of patients who die from head injury have hypoxic-ischaemic brain damage which emphasises the importance of secondary insults to the traumatized brain [1, 2, 9].

2 Materials and methods

We have investigated the effects of intrahospital transportation on the intracranial pressure of patients with a Glasgow Coma Score (GCS) of 8 and below, and compared our results with those of previous studies.

This study is a prospective investigation of patients admitted to Uludag University, School of Medicine, Department of Neurosurgery. Seven patients with a GCS of 8 and below (APACHE II score) were included in the study. Six of the seven patients had a head-injury, one had a spontaneous intracerebellar hematoma. None of the patients had visceral injury. The patients ages ranged from thirty-five to fifty-seven; mean age was forty-six. Intracranial pressure (ICP), invasive blood pressure (IBP), central venous pressure (CVP), and electrocardiogram (ECG) were monitored continuously. The peripheral oxygen saturation (SpO2) was measured pulsoxymetrically and arterial blood samples were obtained for arterial blood gas analysis.All patients were managed by a standard ICU regimen [5]. All had external ventricular drainage sets (EVDS) and were sedated with propofol 150–200 μgr/kg/min. Muscle relaxation was provided by a bolus injection of (0.1 mg/kg) vecuronium bromur followed by an infusion of 1–2 μgr/kg/min. An infusion of fentanyl (0.05–0.2 μgr/kg/min) was used for analgesia. The patients were ventilated with an Ohmeda CPU-I ventilator to maintain PaO_2 at ≥ 100 mmHg, and

$PaCO_2$ at 27–30 mmHg. When patients had to be transported for investigations, all the previously mentioned parameters were monitored. During transportation, patients were ventilated manually with fresh oxygen flow 6 lt/min and FiO_2:1, ICP was monitored with an ICP monitor (Camino laboratories, San Diego, California) IBP and pulsoxymetry was recorded by a monitor (Protocol systems, INC., Beaverton). Sedatives and muscle relaxants were administered with infusion pumps rather than by repeated bolus injection during this period.

During transportation, the patient's head was kept at midline in a neutral position and elevated to 25°–30°° degrees. All patients were transported on their own beds. A special board was used to move the patients on to the CT examination table. The measurements were taken four times; before transportation, in the elevator, at the site of investigation, and finally upon return to the ICU. The average transport distance (go and return) was 300 meters, and the average duration of transport was 45 minutes. For statistical analysis a paried t-test was used.

3 Results

Average values were taken into account for comparison. For all cases, the initial values before transportation were averaged and compared with the average of three values recorded during transportation (in the elevator, at the site of investigation, upon return). According to these comparisons, a 27 % increase was noted in ICP (Figure 1). The highest ICP was recorded during CT examination.

The values of $PaCO_2$ was recorded to be lower than expected at the moment of stopping the ventilator and beginning of manual ventilation (Figure 2). But the levels were the lowest in the recordings in the elevator, during investigation, and during transport. Although the mean decrease of $PaCO_2$ was 16 %, the $PaCO_2$ levels were increased 42 % at certain times (Figure 3).

An average increase of 6.25 % was observed in BP (Figure 4). Variations in BP and arterial blood gases were not found to be statistically significant, while variations in ICP were significant ($p < 0.01$).

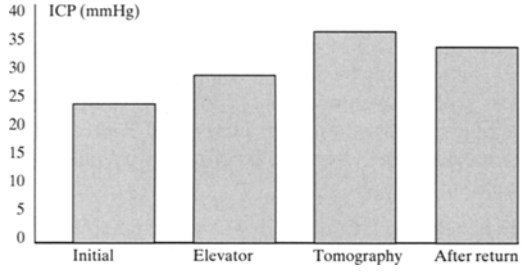

Figure 1: Changing values of ICP during transportation

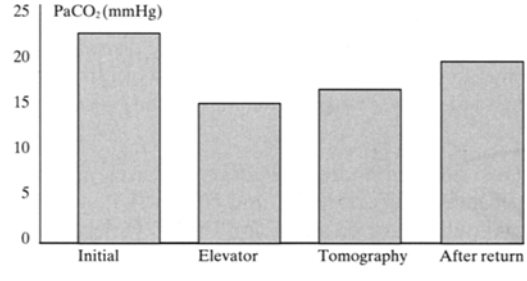

Figure 2: Changing values of $PaCO_2$ during transportation

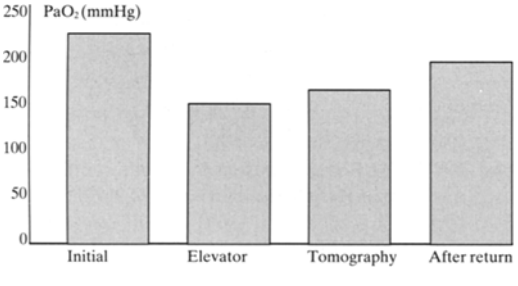

Figure 3: Changing values of PaO_2 during transportation

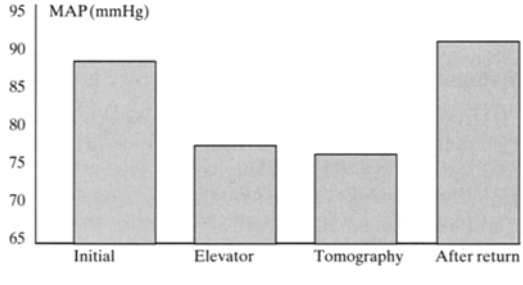

Figure 4: Changing values of BP during transportation

4 Discussion

It is well known that transportation of patients with brain injuries to x-ray departments presents considerable dificulties. Studies point out that in 30 % of fatal head-injuries secondary insults such as hypoxia and hypotension play a role and avoidable factors such as uncontrollable seizures and delay in evacuation of hematomas further contributes to the outcome [3, 11–13, 16]. In some studies a negative contribution of 40 to 50 % to the outcome is mentioned [4, 10]. Since it was technically not possible to monitor pre-hospital transportation, we monitored our patients during intrahospital transportation.

The major problem related to the transportation of ICU patients arises from their cardiovascular instability and fluctuations in arterial blood gas values [1]. As reported for inter-hospital transfer of critically ill patients, hypotension and cardiac arrhythmias are associated with changes in arterial blood gases [7, 17, 18]. The provision of life-support equipment and adequate assessment, resuscitation, and monitoring are important factors [12, 13, 15, 16].

Variations of hemodynamic parameters can be associated with intracranial hypertension in patients running the risk of intracranial hypertension. Both pain and agitation can also increase ICP. For this reason analgesia and sedation must be provided. Intracranial hypertension can be treated by raising the head of the bed and thus facilitating the venous outflow.

Durward et al studied the effect of head elevation at 0° (horizontal), 15°, 30°, and 60° on ICP, systemic and pulmonary pressure, and CPP in patients with intracranial hypertension. They concluded that 15° or 30° of head elevation significantly reduced ICP while maintaining CPP and cardiac output [6]. Feldman et al. reported that head elevation of 30 ° significantly reduced ICP in the majority of the 22 patients without reducing CPP or CBF [8]. Rosner et al. emphasised the importance of adequate CPP and provided physiological and clinical evidence suggesting that CPP is maximal when patients are in the horizontal position, even though ICP is usually higher in this position [14].

Probably the inability to provide elevation during CT examinations resulted in elevation of in ICP during this particular examination, as noted in our study. An increase in ICP was noted in 60 % of the patients during intrahospital transportation and the mean increase was 27 % [1].

The aim is to have a controlled artificial ventilation by maintaining the values of PaO_2 at ≥ 100 mmHg, and $PaCO_2$ at 27–30 mmHg in craniocerebral traumatic cases. But artificial ventilation can increase intrathorasic and intracerebral pressure by affecting venous outflow. For this reason besides sedation and analgesia; muscle relaxation must be provided. Our patients were provided with controlled artificial ventilation in the ICU. But during transport they were ventilated manually because of technical reasons; we did not have a ventilator for transport.

Even if ventilation remains constant, stimulation of the patient can cause an increase in metabolic rate, so that ventilation may then become inadequate.

Thus at the end of a period of inadequate therapy the patient may be left in a compromised state with less reserve to compensate [1]. In spite of adequate analgesia and sedation the increase of PaO_2 and the decrease of $PaCO_2$ can be explained by irregular manual ventilation.

In conclusion ECG, IBP, ICP, and peripheral O_2 saturation should be monitored during intrahospital transportation of ICU patients with craniocerebral injury. Furthermore patients must be provided with adequate sedation and analgesia, head elevation 30° and with a controlled artificial ventilation.

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Submitted October 21, 1996. Revised June 17, 1997. Accepted August 29, 1997.

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