Severe Increase of Intracranial Pressure after Deflation of a Pneumatic Tourniquet

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The pneumatic tourniquet as first described by Cushing1 in 1904 to obtain a bloodless field is frequently used in orthopedic surgical procedures. Anesthesia for multiply injured patients is also a common occurrence.

We report a case of an acute severe increase in intracranial pressure after release of a pneumatic tourniquet for lower extremity surgery in a patient with multiple trauma including closed head injury.

REPORT OF A CASE

A 27-yr-old man involved in a motor vehicle accident was found unresponsive at the scene of the accident with dilated and unresponsive pupils, but with stable vital signs. The patient was transported to a nearby local hospital where his Glasgow Coma Score was 8 upon admission to the emergency room. His trachea was intubated, and hyperventilation instituted. After stabilization, which included iv administration of mannitol, he was transferred to our hospital. Injuries on admission included closed head injury, multiple lacerations and abrasions, right scapular fracture, right clavicular fracture, right second and third rib fractures, left humerus fracture, right tibia and fibular fractures, right hemo-pneumothorax, and hemo-peritoneum. On admission, the patient localized to pain, opened his eyes in response to

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speech, but would not follow commands. His blood pressure was 127/81, heart rate 117, pupils were equal and reactive, hematocrit was 36%, and computerized tomography (CT) of the head showed evidence of right parietotemporal contusion, but no hematomas or midline shifts. The patient was brought to the operating room for exploratory laparotomy, open reduction and internal fixation (ORIF) of his lower extremity fractures, repair of multiple lacerations, and insertion of an epidural fiberoptic intracranial pressure monitor. Mechanical ventilation with oxygen was continued, and anesthesia was induced with thiopental 150 mg, lidocaine 100 mg, and sufentanil 100 μg. Monitors included esophageal stethoscope, electrocardiograph, radial artery catheter for continuous blood pressure, pulse oximeter, end-tidal carbon dioxide (ETCO₂), temperature, and urine output. Anesthesia was maintained with oxygen, air, isoflurane 0.25%, sufentanil, and pancuronium. Initial arterial blood gas (ABG) showed pH 7.44, PaO₂ 30, PaCO₂ 132; ETCO₂ was 26 mmHg. During laparotomy to repair liver lacerations, a Ladd™ fiberoptic epidural transducer for intracranial pressure (ICP) monitoring was inserted with an initial ICP of 15 mmHg. After the laparotomy was completed, a right lower extremity tourniquet was inflated to 350 mmHg and the ORIF started. Throughout the course of the surgery it became necessary to make small increases in minute ventilation to keep the ICP below 20 mmHg. The patient was hemodynamically stable throughout with mean arterial blood pressure (MAP) equal to 80 mmHg. After 1 h 58 min the ORIF was completed, and the pneumatic tourniquet was deflated. Within 15 s the ETCO₂ increased 7 mmHg; coincident with this was an abrupt rise in the ICP from 17 to 58 mmHg. At the same time the MAP fell to 68 mmHg, leaving the effective cerebral perfusion pressure (CPP) equal to 10 mmHg (CPP = MAP – ICP). Immediately, vigorous manual hyperventilation was instituted and within 1 min the ETCO₂ was brought back to previous levels, and the ICP was reduced to less than 20 mmHg. Phenylephrine 40 μg iv was given to increase MAP, and, consequently, CPP. The remainder of the case and postoperative period were uneventful. Approximately 23 h after the injury, and 3 h after completion of surgery, the patient awoke in the intensive care unit, was following commands, was neurologically intact, and eventually recovered completely.

**DISCUSSION**

Several authors have demonstrated a sustained increase in ETCO₂ and PaCO₂ after the release of a leg tourniquet.2,3 This increase is presumably the result of an efflux of hypercapnic blood from the ischemic limb.4,5 Decreases in blood pressure have also been seen and may be due to a combination of a decreased peripheral resistance after tourniquet deflation, posts ischemic reactive hyperemia, and a decreased level of noxious stimulation caused by the tourniquet.

This case demonstrates the effects of increasing CO₂ tension in a patient with poor intracranial compliance. The presence of an intracranial monitor alerted us to the fact that this patient’s ICP was dangerously high, and that his CPP was inadequate. The information provided by the ICP monitor allowed us to rapidly institute measures to treat and correct a problem which we might have overlooked had the ICP monitor not been in place. A 7-mmHg rise in ETCO₂ most likely would have prompted an adjustment in minute ventilation with gradual return to former levels, but such gradual compensation might have left our patient at risk for several minutes.

This case emphasizes the importance of hyperventilation as a treatment modality in patients with suspected poor intracranial compliance. This case also illustrates that deflation of a pneumatic tourniquet can dramatically affect intracranial pressure in patients with poor intracranial compliance. We recommend that vigorous manual hyperventilation be instituted in anticipation of deflation of pneumatic tourniquets to keep ETCO₂ at former levels, thereby hoping to prevent acute severe elevations of intracranial pressure.

**REFERENCES**