

CASE REPORT

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# Electroencephalogram evaluation of accidental cerebral congestion during unexpected superior vena cava clamping: a case report

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## Abstract

**Background:** Intraoperative superior vena cava (SVC) clamping causes hypotension and cerebral congestion. There is no established method for monitoring brain function during cerebral congestion. We encountered a case of cerebral congestion caused by unexpected SVC clamping.

**Case presentation:** A 64-year-old man underwent SVC clamping during lung tumor resection. The entropy and electroencephalogram monitoring values decreased with SVC clamping and increased in response to the release of congestion by phlebotomy and SVC declamping.

**Conclusions:** Because entropy sharply reflects brain viability during cerebral congestion, it was considered helpful in evaluation of the monitoring of cerebral congestion.

**Keywords:** Electroencephalography monitoring, Brain wave, Cerebral congestion, Superior vena cava clamping

## Introduction

Unscheduled superior vena cava (SVC) clamping during surgery is life-threatening and challenging for anesthesiologists. Acute clamping of the SVC during surgery causes a decrease in the cardiac output and hypotension due to reduced venous return to the heart and reduces cerebral perfusion flow and cerebral congestion owing to increased central venous pressure (CVP). We need to decide on a case-by-case basis. There are no established management guidelines for cerebral congestion caused by unexpected SVC clamping, and appropriate monitoring of cerebral viability is necessary to avoid irreversible cerebral dysfunction. However, there is no consensus on monitoring cerebral perfusion or cerebral function during cerebral congestion.

Entropy™ (GE Healthcare, Helsinki, Finland) and Bispectral Index® (BIS; Medtronic-Covidien, Dublin, Ireland), which are processed electroencephalogram (EEG) monitors, are routinely used to evaluate the depth of anesthesia. With the entropy module, we can know the state entropy (frequency range from 0.8 to 32 Hz) calculated from the EEG, the response entropy (frequency range from 0.8 to 47 Hz) obtained by adding the electromyogram component to the SE, and the BSR value (%) indicating the ratio of flat EEG per minute. EEG has also been reported to be useful as an indicator of cerebral ischemia and cerebral hypoperfusion, and the entropy value calculated from EEG is expected to monitor cerebral function during cerebral congestion [1].

We report a case of cerebral congestion during an unexpected SVC clamping. This case showed that entropy is useful for evaluating brain viability and function during cerebral congestion. Written consent for publication was obtained from all patients.

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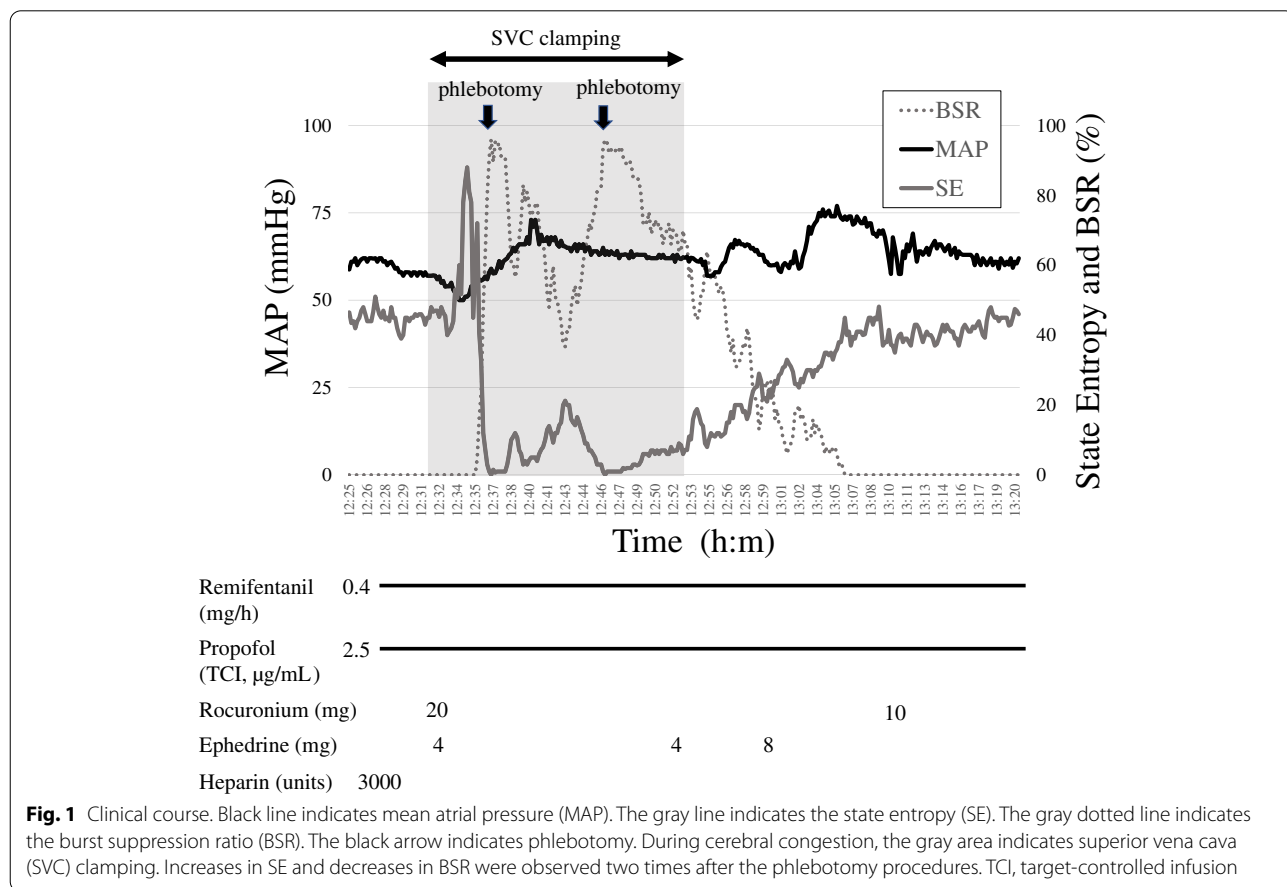
**Case description**

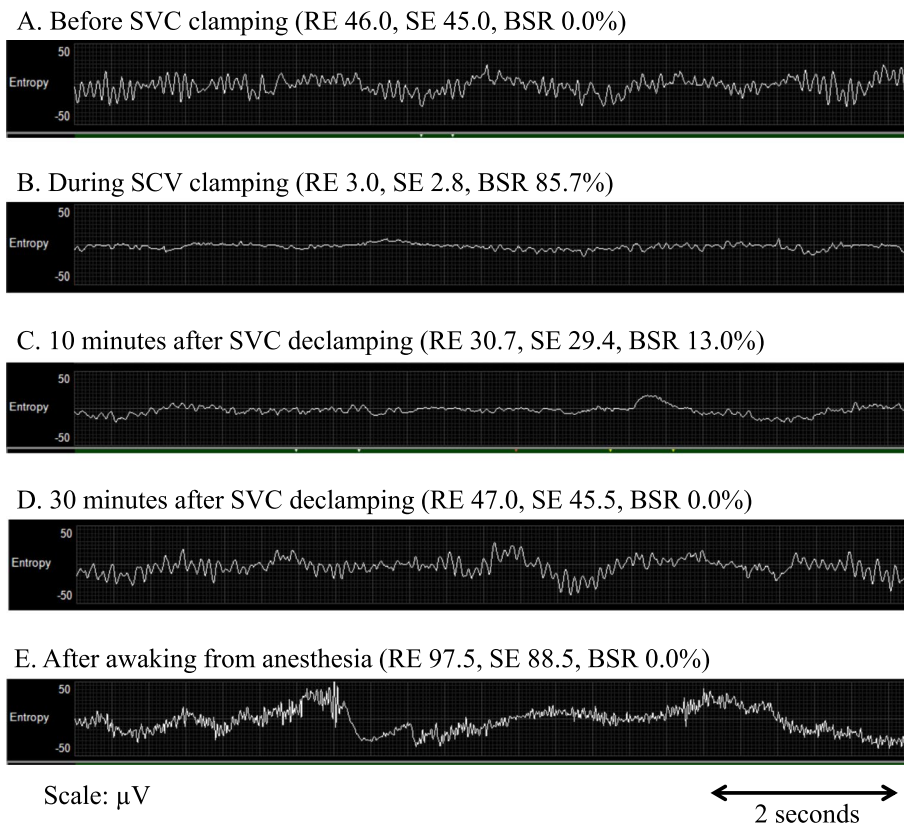
A 64-year-old man underwent video-assisted thoracic surgery, right upper lobectomy combined with bronchoplasty, and SVC plasty for right lung cancer. General anesthesia was induced and maintained with propofol and remifentanil combined with paravertebral nerve block. After induction of anesthesia, propofol was administered at 2.5 µg/mL with a target-controlled infusion system (TCI pump TE-371TM; Terumo, Tokyo, Japan), and remifentanil was administered at 0.4 mg/h. Before clamping the SVC, the state entropy remained around 50 (Fig. 1). Because the tumor had invaded the SVC, it was necessary to clamp the SVC for resection. After administration of 3000 units of heparin, the SVC was clamped on the cranial and caudal sides of the tumor, and the invading tumor was excised simultaneously with the SVC. Immediately after clamping, the mean arterial pressure (MAP) dropped from 60 to 50 mmHg. Blood pressure quickly recovered to its previous level after administration of 4 mg of ephedrine. State entropy rose temporarily for 4 min after clamping and then steeply slowed down to a value less than 10, and the burst suppression ratio (BSR) increased from 0 to 93% (Fig. 2A and B). The flattening of

EEG was thought to be caused by cerebral congestion and reduced cerebral perfusion; however, the clamp could not be released because the SVC was already incised. We performed phlebotomy by releasing the clamp on the cranial side to relieve SVC congestion. Time course of an increase of MAP, phlebotomy, increase of state entropy (SE), and decrease of BSR should be correctly described. SE increased after declamping SVC (Fig. 1). We performed phlebotomy with reference to the improvement in entropy EEG. Phlebotomies were performed twice during a total SVC clamping time of 21 min, for a total volume of 600 mL. State entropy gradually increased after SVC declamping and fully recovered 30 min after declamping (Fig. 2C, D, and E). After the surgery time of 7 h and 12 min, he awakened from anesthesia and was extubated in the operating room. The patient was discharged without neurological sequelae.

**Discussion**

In the present case, unplanned SVC clamp during lung cancer surgery caused cerebral congestion and flattened EEG waves. Since the hemodynamic fluctuations after the SVC clamp were mild, we considered that cerebral





**Fig. 2** Electroencephalogram before and after superior vena cava (SVC) clamping. This figure shows the electroencephalogram before and after the SVC clamping. The vertical axis indicates amplitude ( $\mu\text{V}$ ). The horizontal axis indicates the time, and the arrow scale means 2 s. **A** Before SVC clamping;  $\alpha$  waves and  $\Theta$  waves are mixed. **B** During SCV clamping; brain waves disappear and flatten. **C** Ten minutes after SVC declamping; brain waves remain flat. **D** Thirty minutes after SVC declamping; the brain wave recovers, and the waveform is the same as before the clamp. **E** After awaking from anesthesia;  $\beta$  wave, which is an awakening wave, appears. The values of response entropy (RE), state entropy (SE), and burst suppression ratio (BSR) are shown

congestion was also mild. However, as the EEG waves flattened rapidly, we needed to get rid of cerebral congestion. Considering the urgency, we tried phlebotomy instead of bypass surgery. As Fig. 1 shows, improvement in entropy (increased SE value and decreased burst suppression ratio) was consistent with the two phlebotomy procedures. These trends were not associated with improvement in hypotension with hypertensive drugs. In addition, propofol and remifentanyl were administered continuously, and their infusion rate was not changed. From these facts, we determined that the change in entropy reflects the state of cerebral congestion caused by the SVC clamp.

In some animal model studies, brain function during SVC clamping can be maintained for a relatively long time if cerebral perfusion is maintained. Urayama et al. reported that neurological impairment was not observed after 120-min SVC clamp despite reduced amplitude and prolonged latency of somatosensory-evoked potentials during clamping in dogs [2]. Masuda et al. demonstrated

no abnormalities in EEG with reduced regional cerebral blood flow to about 80% by SVC clamping for 60 min in monkeys [3]. Even from clinical data, it is considered that brain damage does not occur if the SVC clamp is within 45 to 60 min [4]. In a small-scale multicentric study, postoperative neurological complications did not occur with a median of 40-min clamping (including 100 min or more) [5]. Many studies have shown that cerebral congestion with SVC clamping is reversible, and cerebral viability can withstand long-term clamping if cerebral circulation is maintained.

For scheduled SVC clamping, it is common to decompress the SVC by veno-veno bypass to avoid cerebral congestion, which is used in cases of high venous pressure [6]. In accidental SVC clamping, the anesthesiologist must determine whether the patient can tolerate cerebral congestion. The measurement of CVP and regional oxygen saturation ( $r\text{SO}_2$ ) has been reported to evaluate cerebral congestion due to SVC clamping, but there is no established evaluation or management method.

Furthermore, the assessment of cerebral congestion using these methods is indirect and does not evaluate brain viability. Both BIS and entropy are calculated based on EEG. Frequency of EEG waves is decreased in response to mild ischemia [7, 8], which is also induced by cerebral congestion [6], suggesting that entropy also decreased by cerebral congestion.

In a report using BIS in an SVC clamping, the value of BIS after declamping was proportional to MAP [1]. However, this report does not discuss the relationship between cerebral congestion state and BIS values. In the present case, the gradual increase in entropy after the release of cerebral congestion indicated an improvement in brain function. In the present case, a rapid decrease in entropy and a sudden rise in BSR were observed approximately 4 min after SVC clamping. We increased the blood pressure with ephedrine and depressurized CVP by phlebotomy. Increases in entropy and decreases in BSR were observed with two phlebotomy procedures. The timing of phlebotomy and recovery of entropy were the same. This clinical course assumes that entropy clearly reflects cerebral perfusion flow and brain viability during cerebral congestion due to SVC clamping. Raising blood pressure may be effective against cerebral congestion, but we cannot assess the effectiveness of raising blood pressure in this case because we did not attempt to raise the MAP above the normal range.

Entropy, the processed EEG monitor, has the possibility of reflecting brain viability during cerebral congestion and could be useful for evaluating cerebral congestion monitoring.

#### Abbreviations

SVC: Superior vena cava; CVP: Central venous pressure; EEG: Electroencephalogram; BIS: Bispectral index; MAP: Mean arterial pressure; BSR: Burst suppression ratio; rSO<sub>2</sub>: Regional oxygen saturation; TCI: Target-controlled infusion.

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#### Authors' contributions

HM, this author helped conceive/design the case report, write the manuscript, and analyze the data. RN, this author helped with data collection and editing the manuscript. SO, this author helped with data collection and editing the manuscript. YN, this author helped with the anesthesia procedure and helped with data collection and editing the manuscript. NS, this author helped with the anesthesia procedure, obtaining patients consent, data collection, and editing the manuscript. YMT, this author helped conceive/design the case report and editing the manuscript. The authors read and approved the final manuscript.

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#### Availability of data and materials

The data used in this case report are available from the corresponding author on reasonable request.

## Declarations

#### Ethics approval and consent to participate

Not applicable.

#### Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

#### Competing interests

The authors declare that they have no competing interests.

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