Carbon dioxide exhalation temporarily increases during electroconvulsive therapy

SHINOBU SAKURAZAWA, SHIGERU SAITO, MAKIKO YAMADA, FUMIO NISHIHARA, and FUMIO GOTO

Department of Anesthesiology, Gunma University Graduate School of Medicine, 3-39-22 Showamachi, Maebashi 371-8511, Japan

Abstract

Electroconvulsive therapy induces hypermetabolism and elevates oxygen and energy demands, while more carbon dioxide is produced than usual. The purpose of the present study was to determine the elevated carbon dioxide exhalation and the adequate ventilation volume during electroconvulsive therapy. Carbon dioxide exhalation during an electrically induced seizure was continuously monitored by capnography and spirography in 15 patients with endogenous depression. A laryngeal mask airway was used to measure the airway gas flow. Data were collected during a total of 80 electroconvulsive therapy trials. The carbon dioxide exhalation at 1 min after electrical stimulation was higher than the control value (2.8 ± 0.4 versus 2.3 ± 0.3 ml·min^{-1}·kg^{-1}, mean ± SD; $P < 0.05$). The ventilation volume was increased for 3 min after the electrical stimulation to maintain the end-tidal carbon dioxide partial pressure at 35–40 mmHg. The results showed that increasing the ventilation volume by approximately 20% may be necessary to compensate for the increased carbon dioxide exhalation during electroconvulsive therapy.

Key words Indirect calorimetry · Capnogram · Electroconvulsive threrapy · Hypermetabolism · Laryngeal mask airway

Introduction

Seizure induces hypermetabolism and elevates oxygen and energy demands, while more carbon dioxide (CO_2) is produced than usual [1]. During an electrically induced seizure in electroconvulsive therapy (ECT), in which the convulsive movement is restricted to limited distal muscles, CO_2 production may also increase, and adjustment of the ventilation volume might be neces-

sary to maintain the targeted arterial CO_2 partial pressure [2, 3].

Until now, however, there has been no study that has continuously monitored CO_2 production and examined the appropriate ventilation volume during the electrically induced seizure. Although experienced physicians who have ample knowledge of ECT practice can accomplish this goal without any special monitor or airway device, it may not be easy for inexperienced physicians to control CO_2 tension without capnogram data or guidelines regarding ventilation management [4].

The purpose of the present study was to determine the CO_2 exhalation level during electroconvulsive therapy using capnography and spirography. Airway gas flow was precisely measured by use of a laryngeal mask airway.

Subjects and methods

Informed consent was obtained from the patients or, where necessary, from an appropriate relative. This study protocol was approved by the local Clinical Study Committee, which considers the ethics and legal aspects of clinical investigations. ECT was prescribed for 15 patients (3 men and 12 women) suffering from endogenous depression. The patients ranged from 27 to 74 years of age and were in good physical heath. No patient had cardiovascular or cerebrovascular complications or any drug allergies. All patients were treated more than six times (three times per week at 2-day intervals), and data were collected from the second ECT trial and thereafter. The total number of trials from which data were collected was 80.

To avoid an unfavorable parasympathetic reflex, atropine (0.01 mg·kg^{-1}) was given intramuscularly as premedication. Arterial blood pressure (BP) was measured continuously at the right radial artery using a tonometric BP monitor (CBM-7000; Colin, Komaki,

Japan). General anesthesia was induced with propofol (1 mg·kg^{-1}). Propofol was administered intravenously over 15 s through an indwelling catheter. After loss of consciousness, succinylcholine chloride (1 mg·kg^{-1}) was administered and ventilation was assisted using a face mask and 100% oxygen. Following the completion of muscular fasciculation, a laryngeal mask was inserted. Absence of air leakage up to 20 cmH$_2$O airway pressure was confirmed immediately after mask insertion by auscultation at the neck. Then, ventilation at 10 ml·kg^{-1}, 15 times per min was started. In the ECT procedure, end-expiratory CO$_2$ partial pressure (end-tidal CO$_2$) and expiration volume were monitored continuously at the joint of the laryngeal mask and airway circuit by a respiration monitor (Cosmo Plus 8100; Nova Metrics, Wallingford, CT, USA). Anesthetists were advised to maintain end-tidal CO$_2$ tension at 35–40 mmHg by readjusting the ventilation. Ventilation was not halted during the electrical stimulation and was continued until the recovery of spontaneous breathing. The monitor was placed beside the face of each patient for easier viewing by the anesthetists. In all of the patients, arterial blood oxygen saturation (Sp$_{O_2}$) was measured at the left index finger, and the Sp$_{O_2}$ value was kept above 98% by manual ventilation assistance throughout the therapy.

One minute after mask insertion, an electrical current was applied bilaterally for 5 s at the minimal stimulus intensity, which had been determined during the first ECT trial by stepwise increases in electrical intensity. The electrical stimulus was delivered bilaterally by a trained psychiatrist using an ECT stimulator (CS-1; Sakai Iryo, Tokyo, Japan). The efficacy of electrical stimulation was determined by the so-called tourniquet technique, that is, by observation of convulsive movements of the distal leg, around which an inflated tourniquet was set to block the distribution of muscle relaxant. The tourniquet was deflated after the completion of measurement.

The data are expressed as means ± SD. Data were compared by one-way analysis of variance, and post hoc testing was performed by using the Scheffe method (StatView 5.0TM; SAS Institute, Cary, NC, USA) with a P value < 0.05 considered statistically significant.

Results

Demographics of the 15 patients in this study were as follows: 57 ± 17 years in age, 159 ± 11 cm in height, 50 ± 12 kg in weight, 105 ± 8 V electrical stimulus (mean ± SD). According to their histories, patients in this study had been prescribed multiple psychiatric medications at various doses. However, they were nonresponsive to drug therapies, and the medications were interrupted at least 1 day before the start of the ECT sessions. In all cases in this study, the insertion of the laryngeal mask airway was completed atraumatically within 15 s.

The CO$_2$ exhalation at 1 min after electrical stimulation was higher than the prestimulus value. The value gradually returned to the baseline value within 5 min (Fig. 1) ($P < 0.05$). Ventilation volume was significantly increased at 1 (6.3 ± 0.8 l), 2 (6.5 ± 0.7 l), and 3 (6.4 ± 0.6 l) min after the electrical stimulation compared with the prestimulus value (4.6 ± 0.5 l) to compensate for the increased CO$_2$ exhalaton ($P < 0.01$). As a net result, end-tidal CO$_2$ was stable throughout the ECT.

Mean blood pressure increased by approximately 20% until 3 min after the electrical stimulation (21 ± 9% at 1 min), as observed in our previous study using a laryngeal mask airway [5]. Heart rate did not change significantly throughout the ECT session. Muscular seizure duration and electroencephalogram seizure duration were 37 ± 12 and 43 ± 18 s, respectively. No patients showed postictal excitement or complained of sore throat after ECT.

Discussion

For the precise measurement of CO$_2$ production, trapping expired gas without leakage is mandatory. A properly positioned laryngeal mask enables physicians to collect the gas with minimal intervention. Although the mask may be dislodged during muscle contractions, intermittent positive pressure ventilation is usually possible with this device. In our previous study, we showed
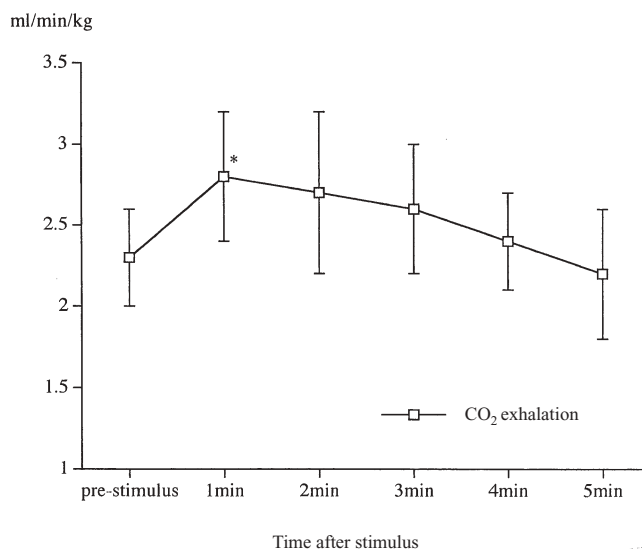

Fig. 1. Carbon dioxide (CO$_2$) exhalation during electrically induced seizure. CO$_2$ exhalation was monitored by a respiration monitor, which measured end-expiratory CO$_2$ partial pressure and ventilation volume. Data are means ± SD (*, $P < 0.05$)

that a laryngeal mask is beneficial for ensuring ventilation during ECT, especially when face-mask fitting is difficult [6]. Since the laryngeal mask in the present study was used for less than 10 min, no patients in the present study complained of laryngeal discomfort after ECT.

The gas analyzer used in this study was originally designed for respiratory management of patients in an intensive care unit [7]. The CO_2 content of the expired gas is calculated by multiplying the CO_2 partial pressure by expiration volume measured by the near-infrared and mainstream method. The measurement error is reported to be less than 5%. Changes in metabolism alter CO_2 production, which changes the CO_2 content of the expired gas. Since the produced CO_2 is diluted by the reserved gas in the lungs, rapid alteration of the level in the blood is buffered for some period of time. Ventilation conditions prior to monitoring, and hemodynamic changes after the electrical stimulation, also possibly contributed to the data obtained in this study. Therefore, the data obtained in this study reflect temporary aspects of changing values. Also, the machine used in this study measures CO_2 content breath by breath and averages the data every minute. This averaging process possibly blunts rapid alterations during a seizure, which is completed within a few minutes. A more quickly responding instrument might reveal the abrupt metabolic changes more precisely.

In the present study, CO_2 exhalation prior to electrical stimulation was $2.3 \pm 0.3\,\mathrm{ml \cdot min^{-1} \cdot kg^{-1}}$. This value is smaller than the standard value ($3–5\,\mathrm{ml \cdot min^{-1} \cdot kg^{-1}}$) obtained from human volunteers (manufacturer's information). This discrepancy may be explained by the fact that patients in this study had already been anesthetized when data collection was started. General anesthetics, including propofol, suppress metabolism and CO_2 production [8]. During electroconvulsive therapy, several organs, including the brain and muscles, produce more CO_2 than usual. Brain metabolism is elevated during an electrically induced seizure. Brodersen et al. [9] demonstrated a CO_2 increase in jugular vein blood. Szirmai et al. [3] showed an elevation in CO_2 content in the internal jugular vein following seizure. It is believed that excessive expenditure of O_2 and hyperproduction of CO_2 immediately after an electrical current are compensated for by a concomitant augmentation in cerebral blood flow [5]. The excess CO_2 is quickly washed out of brain tissue, flows into systemic circulation, and is expired from the lungs.

In the modified ECT, where muscle relaxant is used, muscle contraction is only provoked in limited muscles.

However, fasciculation provoked by succinylcholine produces extra CO_2, and facial muscles are forced to contract by the application of electrical current. When the tourniquet technique is used for monitoring muscular seizure duration, the isolated muscles produce CO_2 during the convulsive movement (although the produced CO_2 flows into systemic circulation following the tourniquet release). The elevated CO_2 exhalation observed in this study was considered to be a net result of these phenomena during ECT. Further study in which nondepolarizing muscle relaxant is used and muscular seizure is not monitored by the tourniquet technique may minimize the contribution of muscles to CO_2 production.

In the present study, we showed that CO_2 exhalation was increased by approximately 20% immediately after ECT. However, by increasing ventilation volume adequately, the physician can prevent accumulation of CO_2.

References

1. Meric P, Barrere B, Peres M, Gillet B, Berenger G, Beloeil JC, Seylaz J (1994) Effects of kainate-induced seizures on cerebral metabolism: a combined 1H and 31P NMR study in rat. Brain Res 638:53–60
2. Gaines GY, Rees DI (1992) Anesthetic considerations for electroconvulsive therapy. South Med J 85:469–482
3. Szirmai I, Boldizsar F, Fischer J (1975) Correlation between blood gases, glycolytic enzymes and EEG during electroconvulsive treatment in relaxation. Acta Psychiatr Scand 51:171–181
4. Saito S, Kadoi Y, Nishihara F (2003) End-tidal carbon dioxide monitoring stabilized hemodynamic change during electroconvulsive therapy. J ECT 19:26–30
5. Saito S, Yoshikawa D, Nishihara F, Morita T, Kitani Y, Amaya T, Fujita T (1995) The cerebral hemodynamic response to electrically induced seizures in man. Brain Res 673:93–100
6. Nishihara F, Ohkawa M, Hiraoka H, Yuki N, Saito S (2003) Benefit of laryngeal mask for airway management during electroconvulsive therapy. J ECT 19:211–216
7. Bhavani-Shankar K, Philip JH (2000) Defining segments and phases of a time capnogram. Anesth Analg 91:973–977
8. Reves JG, Glass PSA, Lubarsky DA (2000) Nonbarbiturate intravenous anesthetics. In: Miller RD (ed) Anesthesia. 5th ed. Churchill Livingstone, Philadelphia, pp 228–272
9. Brodersen P, Paulson OB, Bolwig TG, Rogon ZE, Rafaelsen OJ, Lassen NA (1973) Cerebral hyperemia in electrically induced epileptic seizures. Arch Neurol 28:334–338