

Beware of “curare cleft” like changes during unilateral capnothorax

Manila Singh · Kapil Chaudhary · Rajeev Uppal · Sachin Jain

Received: 8 May 2013 / Accepted: 9 October 2013 / Published online: 17 October 2013
© Springer Science+Business Media New York 2013

Abstract Capnography is a standard monitoring tool during general anaesthesia. Diaphragmatic movement with the weaning of muscle relaxant effect produces the characteristic “curare cleft” on capnography. Various artefacts can mimic this trace intraoperatively. Cautious interpretation and identification of these is essential to avoid any undue overdosing of the patients with muscle relaxants. We report “curare cleft” like artefact during ventilation with a single lumen tube in a patient with unilateral capnothorax undergoing minimally invasive esophagectomy.

Keywords Unilateral capnothorax · Single lumen tube · Prone · Curare cleft · Capnography

1 Introduction

The characteristic square wave capnographic trace is the result of exhalation of CO₂ from the alveoli to mouth and subsequent inhalation of CO₂ free gases. This has been found to be identical in all humans with healthy lungs [1]. Any variation from this warrants proper evaluation as it may provide relevant information related to patient, anesthesia machine, anesthesia technique or surgical procedure.

Valuable evidence based data related to capnography has been obtained from case reports like ours, which has helped in patient management strategies in specific scenarios. The written informed consent for publication of data was taken post-operatively from the patient as the findings were observed intra-operatively in the anesthetised patient.

2 Case report

A 65 year old male, weighing 65 kg was scheduled for minimally invasive esophagectomy (MIE) for carcinoma oesophagus (gastro-esophageal junction) in prone position. Routine pre-anaesthesia check up and all biochemical and radiological investigations were normal. The patient did not have any other co-morbidities.

Anaesthesia was induced, following epidural catheter placement in T₈-T₉ interspace, with fentanyl 2 mcg/kg, midazolam 2 mg and propofol 100 mg. Intubation with a single lumen endotracheal tube internal diameter 8.5 mm (Portex, Smiths Medical ASD, Keene, USA) was facilitated by muscle relaxation with 50 mg rocuronium. Patient was ventilated using Datex Ohmeda S/5 Avance (Datex Ohmeda Inc., Madison, USA) at a tidal volume of 500 ml and an intermittent positive pressure ventilation (IPPV) rate of 12 in a closed circuit. The patient was positioned prone thereafter. Routine monitoring which included ECG, pulse oximetry, invasive blood pressure, temperature (Dräger Infinity Delta XL, Draeger Medical System, Telford, USA), capnography and agent analysis (Dräger four oxi plus, Dräger Medical, Lübeck, Germany) were applied. Anaesthesia was maintained with rocuronium infusion at 0.3 mg/kg/h, Isoflurane 0.6–1.0 % in nitrous-oxide oxygen mixture and epidural infusion of 0.25 %

M. Singh (✉) · K. Chaudhary · R. Uppal · S. Jain
Department of Anaesthesia and Intensive Care, G.B. Pant
Hospital, E-269, East of Kailash, New Delhi, India
e-mail: dr.manilasingh@gmail.com

K. Chaudhary
e-mail: kapsdr@yahoo.com

R. Uppal
e-mail: drrajeevuppal@gmail.com

S. Jain
e-mail: drsachinjain.jain@yahoo.com

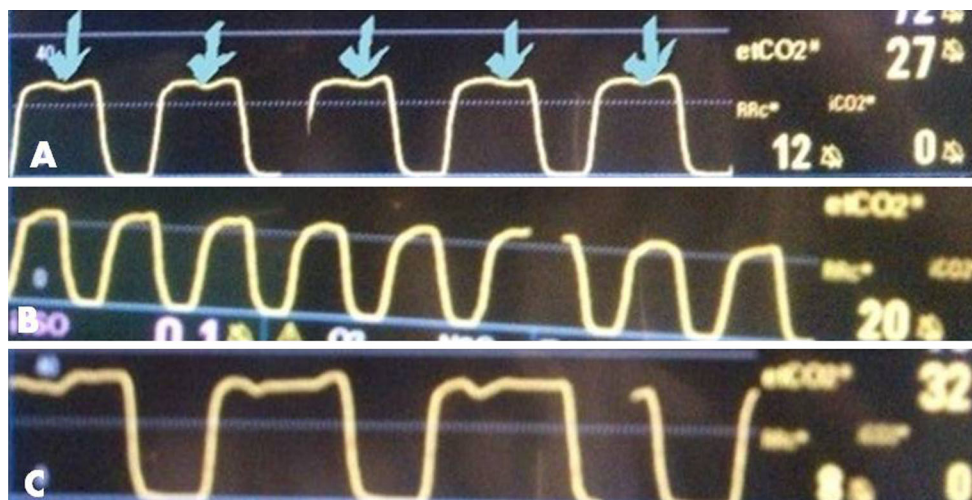


Fig. 1 Arrow depicting “curare cleft” like artefact in the alveolar plateau phase of the capnograph trace (a). The cleft disappeared with an increase in the IPPV rate (b) and reappeared with a decrease in the IPPV rate (c)

bupivacaine with 4 mcg/ml of fentanyl at the rate of 8 ml/h. The capnography trace showed normal square wave pattern.

About 35 min after induction, on creation of right sided capnothorax, a cleft in the alveolar plateau phase similar to the “curare cleft” was observed (Fig. 1a). A TOF count of one on giving a TOF stimulus (TOF Guard INMT, Oregon) indicated adequate muscle relaxation. The surgeon was asked to stop surgery so as to check for any artefact due to diaphragmatic movement. The PETCO₂ analyser and circuit were checked for any disconnections. The ventilator relief valve movements were checked for any malfunction. All these were absent as further evidenced by an equal inspired and expired tidal volume. The PETCO₂ was also within normal limits. The airway pressure tracings also did not suggest any inspiratory effort (Fig. 2a). The surgeon was to dissect around the aorta and heart. A bolus of 10 mg rocuronium was administered considering the clinical situation and some amount of residual diaphragmatic muscle activity to be the cause. Subsequent TOF stimuli gave a TOF count of zero. However, the cleft still persisted. A repeat TOF stimulus again revealed a count of zero. The cleft persisted despite another bolus dose of 5 mg rocuronium. An increase in IPPV rate caused immediate disappearance of the cleft which again became conspicuous on decreasing the IPPV rate (Fig. 1b, c). Thoracoscopic visualisation of the diaphragm at all times revealed complete immobility. Once the capnothorax was drained with a chest tube and underwater seal leading to right lung re-expansion, the cleft on the capnographic trace immediately disappeared.

Similar findings were noted in a subsequent case of MIE conducted in prone position using an SLT, after creation of a unilateral capnothorax.

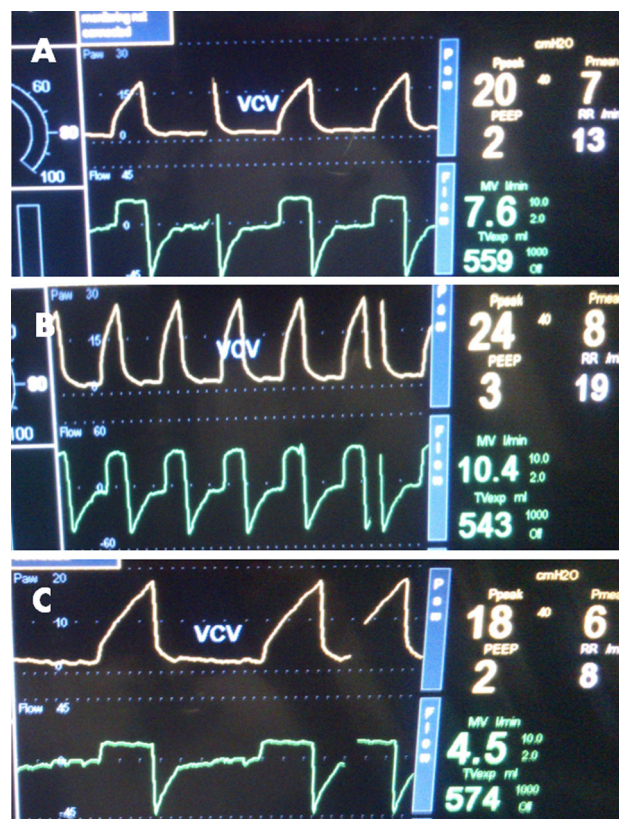


Fig. 2 The airway pressure tracings showed absence of any spontaneous effort when capnograph showed “curare cleft” like artefact (a). The pressure tracings remained same on increasing (b) and decreasing (c) the IPPV rate

3 Discussion

Capnographic detection of diaphragmatic movement is an important tool to assess the requirement of muscle relaxant supplementation [2]. Phase III of capnogram consists of an

alveolar plateau, representing CO₂ rich gas from pulmonary alveoli [3]. Any movement of the diaphragm may cause a cleft in this plateau phase commonly appreciated as the “curare cleft” by the anaesthesiologists. Various intraoperative events like partial disconnection of CO₂ transducer, stuck ventilator relief valve or differential lung emptying noted in lung transplant recipients can be confused with this [4–6]. Inadequate muscle relaxation, subtle diaphragmatic movement, stuck ventilator valve and disconnection of capnography transducer and circuit were ruled out in this case. The PETCO₂ was always within normal limits. Also there was no apparent cause of diaphragmatic movement like surgical manipulation which would persist in every square wave.

During thoracoscopy induced capnothorax and concomitant use of a single lumen endotracheal tube, there is some ventilation of the collapsed lung despite positive intrapleural pressure induced by the CO₂ gas during the inspiratory phase of IPPV. Emptying of this lung occurs very rapidly during the expiratory phase due to the same amount of positive intrapleural pressure and zero end expiratory pressure. The cleft in the capnography trace during the plateau phase in this case probably resulted from an increased emptying of CO₂ from the relatively hypercapnic and hypoventilated lung followed by gradual emptying from the well ventilated lung. This differential emptying of the expanded lung after relatively complete emptying of the collapsed lung could have led to the “curare cleft” like artefact on capnograph in an adequately paralysed and anaesthetised patient. Decreasing the IPPV rate may have exaggerated the same, thus making it more prominent. Whereas, increasing the IPPV rate might have caused the preferential filling of the more compliant expanded lung due to a decrease in inspiratory time which probably obscured this artefact. The immediate disappearance and reappearance of cleft on an increase and decrease in IPPV rate, respectively, in the presence of normal PETCO₂ at all times ruled out any stimulation of ventilation from elevations in CO₂ resulting from capnothorax.

It may be argued by some that the cleft observed by us was not as deep as the classical “curare cleft”. However, in the absence of facilities for obtaining capnograms and the paucity of literature on quantitative estimation of curare cleft, we relied on qualitative assessment of capnography traces after ruling out the artefactual causes described in literature. Also, it must be noted that a typically deep “curare cleft” is observed when the muscle relaxant effect is almost totally weaned off and the patient movements (coughing, bucking, limb movements) may be suddenly observed if not promptly treated. In order to avoid such situations, it is a common practise among anaesthesiologists to administer muscle relaxants after ruling out the

causes mimicking “curare cleft”, even when the clefts are qualitatively not as deep as those published in literature. The need for complete patient relaxation by the surgeon, given nearby vital structures (e.g. heart and aorta), prompts the anaesthesiologists to give neuromuscular blocker when no other obvious cause of such cleft is observed. Waiting for the typically deep “curare cleft” on qualitative assessment, which probably indicates return of spontaneous efforts to a large extent was unwarranted in this surgery. Also, though the spontaneous respiratory efforts were absent on airway pressure tracings at all times (Fig. 2), in authors experience the tracings may not be able to adequately detect early recovery of muscle activity in many cases. This led us to take prompt action and administer the muscle relaxant on observing this artefact even when the cleft was not deep enough. However, the findings of TOF count being zero at almost all stages of mediastinal dissection, absence of any movement on visual inspection of the diaphragm during surgery and persistence of the artefact despite administration of muscle relaxant subsequently confirmed complete muscle relaxation during the maintenance of capnothorax and “curare cleft”.

To the best of our knowledge, the findings in this case report have not been previously reported. Since the described procedure demands complete relaxation and avoidance of any movement especially when dissecting around vital structures, such artefacts may be aggressively treated with muscle relaxants. It is thus important for anaesthesiologist to be aware of such artefacts, especially in settings where facilities for neuromuscular monitoring are not available. This emphasizes the need for further studies to quantitatively estimate the “curare cleft”, which appears to offer greater clinical utility than its qualitative assessment. Until studies quantifying the curare cleft are available, careful direct visualisation of the diaphragm may be a simple tool to rule out inadequate muscle relaxation in such scenarios.

In summary a “curare cleft” like artefact on capnography trace can be observed in a patient with unilateral capnothorax, being ventilated with a SLT. We suggest cautious interpretation of qualitatively subtle changes in the plateau phase of a capnograph so as to avoid the undue administration of muscle relaxants and possible sequelae.

Conflict of interest None.

References

1. Kalenda Z. Mastering infrared Capnography. The Netherlands: Kerkebosch-Zeist; 1989.
2. Bissinger U, Lenz G. Capnographic detection of diaphragm movements (“curare clefts”) during complete vecuronium

- neuromuscular block of the adductor pollicis muscle. *Anesth Analg.* 1993;77:1303–4.
3. Bhavani Shankar K, Kumar AY, Moseley H, Hallsworth RA. Terminology and the current limitations of time capnography. *J Clin Monit.* 1995;11:175–82.
 4. Tripathi M, Tripathi M. A Partial disconnection at the main stream CO₂ transducer mimics “Curare-cleft” capnograph. *Anesthesiology.* 1998;88:1117–9.
 5. Hensler T, Dhamee MS. Anesthesia machine malfunction simulating spontaneous respiratory effort. *J Clin Monit.* 1990;6: 128–31.
 6. Benumof JL. *Anesthesia For Thoracic Surgery*, vol. 2. Philadelphia: W B Saunders Company; 1995. p. 245–50.