FAILURE OF ENDTIDAL CARBON DIOXIDE TO CONFIRM TRACHEAL INTUBATION IN A NEONATE WITH A SINGLE VENTRICLE AND SEVERE PULMONARY STENOSIS

- Case Report -

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A 12 day old baby girl (body weight 3.5 kg) with dextrocardia and single ventricle, both great arteries arising from single ventricle, severe valvular and subvalvular pulmonary stenosis and hypoplastic pulmonary artery, and non restrictive atrial septal defect, was scheduled for Blalock-Taussing shunt under general anesthesia.

On the day of surgery, heart rate (HR) was 139 bpm, noninvasive blood pressure (BP) 93/31 mmHg, SpO2 81% on room air, and temperature 36.5° C. Patient was preoxygenated with 100% O2, which increased SpO2 to 99%.

Anesthesia was induced by face mask with sevoflurane in oxygen with gradual increase in concentration to 6%. Immediately after venous access was obtained, propofol 2 mg/kg was administered. Patient was easy to ventilate and saturation was 99% when intubation was attempted for the first time. Upon laryngoscopy, vocal cords could be visualized and uncuffed endotracheal tube (3.0 mm) was inserted easily.

Although the patient was ventilated, and bilateral air entry by auscultation was detected, no endtidal carbon dioxide (ETCO2) tracing on the monitor could be seen, and the patient started developing hypoxia (SpO2 60%), and BP decreased to around 40/25 mmHg. Intubation was considered esophageal and a repeated attempt of intubation with direct visualization of the tube passing through the vocal cords, showed no improvement in the above findings. Also, patient developed bradycardia (HR 85 bpm), and was given atropine 10 µg/kg. Following a transient increase of HR to 120 bpm and saturation to 75%, both values dropped again, and still no ETCO2 could be detected. Capnograph was checked and no deficiency could be found. However, the chest rise observed and bilateral air entry by auscultation confirmed correct placement of the tube. Since hypotension and bradycardia persisted, a bolus injection of epinephrine 10 µg/kg was given. BP increased to 70/35 mmHg, HR to 115, and SpO2 to 79%, and low ETCO2 tracing (20 mmHg) could be detected. When circulation improved, surgery was carried out.

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Discussion

When CO₂ is absent as measured by capnograph, it means either the endotracheal tube is in a wrong position (esophageal) or there is absent/decreased presentation of CO₂ to the lungs. False negative results can occur in many situations, where ETCO₂ is not detected, even though the tube is properly placed in the trachea. Gas sampling problem, such as disconnection of the tracheal tube from breathing apparatus, apnea, equipment failure, a kinked or obstructed tracheal tube, unintentional PEEP to a loosely fitted or uncuffed tube, and dilution of proximal sampling by fresh gas flow in Mapelson D systems and Dryden absorber may be misinterpreted as absent waveform caused by esophageal intubation. A marked decrease in pulmonary blood flow will increase alveolar component of the dead space; this occurs with low cardiac output, hypotension, pulmonary stenosis, pulmonary embolism, tetralogy of Fallot, and kinking or clamping of the pulmonary artery during pulmonary surgery¹.

Propofol use in children with congenital heart disease (CHD) may decrease systemic vascular resistance and lead to a change in the ratio between systemic and pulmonary flow²,³. Also, propofol produces bradycardia by its action on the sinoatrial node and attenuation of the β adrenergic receptors at the level of the ventricular myocytes. Even a relative decrease in HR may be deleterious in infants and young children who depend on HR to maintain cardiac output as they cannot increase stroke volume. Previous study found that induction with sevoflurane decreases BP which returned to baseline values in few min after reducing concentration of inhaled anesthetic⁴.

In our neonate, severe pulmonary stenosis and single ventricle are expected to produce low ETCO₂ values on capnography. Sevoflurane induction combined with propofol may have caused a further reduction in the pulmonary blood flow secondary to reduced systemic vascular resistance, and bradycardia. As a consequence, the cardiac output dropped resulting in an increase of the arterial/ETCO₂ gradient and subsequently alveolar dead space. This caused a completely absent CO₂ waveform on capnography (false negative result). Restoration of the circulation resulted in the reappearance of the waveform.

Repeated attempts of intubation can be complicated in cyanotic neonates by deleterious hypoxia and cardiovascular decompensation. The apneic episodes during repeated attempts of laryngoscopy can result in rapid desaturation because of the low functional residual capacity and high rate of oxygen consumption of the neonate. Also, the basal low saturation places the neonate with cyanotic congenital heart disease in the steep part of the oxyhemoglobin dissociation curve.

In summary, ETCO₂ in neonates with cyanotic CHD associated with pulmonary stenosis and single ventricle consistently underestimates the true arterial CO₂ level. Any additional decrease in pulmonary flow such as that induced by sevoflurane-propofol combination may render ETCO₂ waveform an inadequate tool to confirm tracheal intubation.

Keywords: Congenital heart disease: pulmonary stenosis, single ventricle; Capnography: entidal carbon dioxide; Anesthesia: pediatric.

References