Ocular Venous Air Embolism (OVAE): A Review
Supplemental Report 2

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1. Monitoring End-Tidal Carbon Dioxide (ETCO₂)
Niels Hauff, MD and Robert Morris, MD

Since 2010, the American Society of Anesthesiologists (ASA) has included exhaled end-tidal carbon dioxide (ETCO₂) monitoring in its Standards for Basic Anesthetic Monitoring. ETCO₂ monitoring in the operating room setting is most commonly performed utilizing capnography (monitoring of the partial pressure of carbon dioxide in the respiratory gases) via a side-stream sampling line during general anesthesia. Using capnography, the quantitative value and waveform of exhaled carbon dioxide (CO₂) can provide diagnostic information regarding cardiopulmonary pathology.

ETCO₂ monitoring allows for objective assessment of ventilation (the circulation and exchange of gases in the lungs). When air lock of the right ventricle occurs due to rapid air entrainment, as in OVAE, venous blood return to the lungs drops suddenly, exchange of CO₂ ceases despite continued air circulation. A precipitous decline in ETCO₂ is seen.

The type of airway device used to conduct ETCO₂ monitoring impacts the value of the information provided. When non-invasive airway devices, such as a nasal cannula or simple facemask, are used to conduct ETCO₂ monitoring, the quantitative value is difficult to interpret. In this setting, the anesthesia provider often treats the monitoring as binary, with either the presence or absence of CO₂, to ensure airway patency, assess ventilatory effort, and quantify the patient’s respiratory rate. Since regional anesthesia is commonly utilized for vitrectomy, the value of ETCO₂ (as a very early sign of OVAE) during such surgery is lessened.

When invasive airway devices, such as a laryngeal mask airway (LMA) or endotracheal tube, are utilized, the quantitative value of CO₂ obtained during capnography is more informative. Changes in the quantitative value of ETCO₂ and
the shape of the waveform can indicate intrinsic heart or lung disease, as well as assist in diagnosis of pathologic changes in the patient’s cardiopulmonary status (such as OVAE) during anesthesia.

The differential diagnosis for a sudden decrease in ETCO₂ includes sudden increases in dead space ventilation (pulmonary embolism or decreased cardiac output), airway or circuit obstruction, extubation, or equipment malfunction.² When monitoring ETCO₂ with an invasive airway device, sudden decreases in ETCO₂, paired with hemodynamic instability, should immediately make the clinician suspicious of pulmonary embolus.

When an operation involves the use of pressurized air infusion into a body cavity or an organ (the eye), venous air embolism (VAE) is the likely source of such pulmonary embolus, and the patient should be emergently managed with cessation of air infusion and cardiopulmonary support.

2. **Acute Respiratory Distress Syndrome (ARDS)**
Niels Hauff, MD and Robert Morris, MD

Acute Respiratory Distress Syndrome (ARDS) is the result of a sudden insult to the lungs that leads to inflammation and fluid accumulation which impairs gas exchange. ARDS can occur as a result of a number of pathologic processes that ultimately lead to lung inflammation and non-cardiogenic pulmonary edema. Three common causes are sepsis, pneumonia and, less commonly, VAE.³ The diagnosis of ARDS is made by identifying diffuse lung injury within one week of an apparent clinical insult; ruling out a cardiac etiology or volume overload; and documenting a decreased ratio of arterial partial pressure of oxygen to inspired partial pressure of oxygen (PaO₂/FI₉O₂). The severity of ARDS is diagnosed based on the degree of impairment of gas exchange as measured by the PaO₂/FI₉O₂ ratio. Treatment of ARDS is largely supportive. It focuses on identification and treatment of the underlying etiology and is followed by implementation of a lung protective strategy, which often requires controlled ventilation by intubation. This strategy aims to minimize further lung injury by avoiding large tidal volumes and providing adequate positive end expiratory pressure (PEEP).⁴ **Extracorporeal life support (ECLS) may ultimately be required to avoid a fatal outcome such as that seen in Case 13.**
3. Analysis of the Four OVAE Survivors
Robert Morris, MD

Of the four patients who survived OVAE, one likely had extremely rare entrainment through a tiny central retinal vein after a direct laceration of the optic nerve head during a penetrating eye injury (Case 2). 5

Of the three apparent vortex vein entrainment survivors, one (Case 12) survived due to the vitrectomy team already being on high alert during choroidal melanoma vitrectomy resection, armed with knowledge of a similar prior OVAE fatality (Case 4). 6 The air infusion was stopped immediately upon noticing an ETCO₂ decrease. The patient then recovered uneventfully.

The second vortex vein entrainment survivor was “dead” after 87 minutes of unsuccessful CPR (Case 5). 7 Six minutes after emergency cardiopulmonary bypass was achieved by percutaneous cannulation of the common femoral artery and vein, cardiac arrest resolved. Extracorporeal life support continued for five days and the patient was removed from a respirator seven days postoperatively and recovered. The third survivor of air entrainment through vortex veins was the only patient who developed choroidal detachment intraoperatively and in whom the surgeon DID NOT immediately respond by raising infusion pressure (Case 1). 8 Accidental suprachoroidal air infusion, leading to intraoperative choroidal detachment and torn vortex veins, can follow slippage of an unsutured cannula, commonly used since approximately 2005 (Figure 1). In this case we believe that because of the relatively low infusion pressure only a single vortex vein was torn, perhaps on the small side of the variation in vessel size commonly seen (see Supplemental Video). This allowed recovery even after an estimated four minutes of gradual air entrainment.

![Figure 1. 25G infusion cannula inserted into a transconjunctival, trocar cannula. Note the unsecured trocar cannula with the inserted infusion cannula has slipped outward, predisposing the eye to suprachoroidal air infusion.](image-url)
Additional OVAE Comments

Skepticism or denial in the face of an iatrogenic, fatal air embolism has been seen in many affected fields of surgery. This attitude is enabled by the silent, invisible, and transient nature of air embolism, making VAE an ephemeral clinical diagnosis. Even at autopsy, VAE is not easily discovered unless special investigative measures are requested.

Litigation, or the threat of litigation, also appears to discourage OVAE reporting. Only one of five OVAE cases known to have occurred in the United States (the leading country in health care related litigation) has been promptly reported (Case 8). One of these cases was actually discovered through litigation, and finally reported in 2017, more than 20 years after its occurrence (Case 7). In addition to a PubMed search, we found OVAE cases during extensive discussions with colleagues, by review of the American Society of Anesthesiologists closed claims database, by contact with the (anesthetist) sister of an OVAE decedent who had heard of our work, and by learning by chance of the first (at the time of its occurrence) known intraoperative OVAE fatality. The latter inspired our investigations and our search for unreported cases. Coincidentally, the world’s leading authority on VAE (Maurice Albin, MD, now deceased) had retired to our community and greatly encouraged our studies.

Morris et al showed that a lethal dose of air can likely be entrained through torn vortex veins within one minute through a 25-Gauge (G) infusion cannula slipped into the suprachoroidal space, at an infusion pressure of 40 mm Hg (Supplemental Video). As little as 200 cc of air rapidly entrained can be fatal. During OVAE, that volume of air can entrain in less than 30 seconds.

The earliest reliable sign of OVAE is a sudden decrease in ETCO₂, but it is less reliable in most vitreoretinal cases since they are performed without general anesthesia. Before signs other than decreasing ETCO₂ fully develop — hypotension, cardiac arrhythmias, decreased oxygen saturation, and a mill wheel murmur - cardiovascular collapse can become difficult to reverse. Consequently, seconds count when life-threatening air entrainment begins.

OVAE fatalities occur at an average age of 56 years, and do not often occur in a setting of preexistent, life-threatening heart or lung disease. These patients enter cardiogenic shock from a transient insult, while being monitored, and have optimal potential for life-saving rescue by temporary extracorporeal life support (ECLS - a rapidly deployable form of cardiopulmonary bypass via the femoral vessels). ECLS can thus provide a bridge to survival for OVAE patients who otherwise have a substantial life expectancy.
But ECLS technology is only gradually spreading beyond cardiovascular surgery suites and intensive care units.

To prevent accidental suprachoroidal air infusion leading to (usually fatal) OVAE, we’ve suggested use of specific language within the vitrectomy team to confirm cannula position in the vitreous cavity before beginning air infusion. The vitrectomy team, consisting of all surgical personnel in the room, must make a special effort to apprise the anesthetist of beginning and continuing infusion.

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