DISCUSSION

It is generally considered that in steady-state situations, $V_O_2$ at rest remains roughly constant even when cardiac output decreases because of the compensatory increase in arteriovenous oxygen difference. However, in cases of extreme cardiac output decrease such as in severe heart failure\(^1\) or in septic shock,\(^2\) $V_O_2$ becomes lower than normal. Whether a decrease in systemic blood flow can also result in a change in $V_O_2$ in nonsteady-state situations such as graded exercise remains unknown. In particular, it is unknown whether in such cases, the peripheral extraction of oxygen within the muscle can increase sufficiently and in time to permit $V_O_2$ to continue to increase.

In this article, four cases are described in which either an arrhythmia or acute mitral regurgitation resulted in a transient decrease in pulmonary $V_O_2$ while the workload, and thus metabolic demand of the peripheral muscles, continued to increase. Although cardiac output was not directly measured, it is highly likely that the decrease in $V_O_2$ was secondary to a decrease in cardiac output, and not to a decrease in the arteriovenous oxygen difference. In these cases, the decrease in $V_O_2$ in the lungs probably signifies that the arteriovenous oxygen difference has not increased in time to compensate for the decrease in cardiac output. These observations demonstrate the interdependence of $V_O_2$ and pulmonary blood flow during exercise.

CONCLUSION

Transient hemodynamic disturbances may cause a reduction in $V_O_2$ during exercise, probably secondary to a decrease in cardiac output. These observations illustrate the dependence of $V_O_2$ on oxygen supply in nonsteady-state conditions as well as the potential of respiratory gas monitoring during exercise to detect the hemodynamic and metabolic consequences of events resulting in transient changes in systemic blood flow, especially in patients with cardiac failure or arrhythmias.

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REFERENCES


Transtachael Oxygen Catheterization With Pneumomediastinum and Sudden Death*

David A. Kristo, MD, FCCP; J. F. Turner, MD, FCCP; and Ruth Hugler, RN, CNS

A 69-year-old man with hypoxemic COPD underwent placement of a transtachael oxygen (TTO) catheter. At 3 months, the catheter tract appeared mature with minimal erythema and no evidence of infection at the catheter site. The patient and his spouse were taught to remove and reinsert the catheter but were told to delay beginning the procedure due to erythema at the stoma site. Despite instructions not to remove the catheter for cleaning, the spouse removed the TTO catheter and attempted to reinsert it using the flexible metal cleaning rod. Subsequently, the patient suffered an acute episode of subcutaneous air and hemodynamic collapse resulting in death. Necropsy revealed a false catheter tract occluded by clotted blood and a defect in the platysma muscle where oxygen had dissected into the mediastinum. The patient died due to pneumomediastinum and cardiac tamponade.

(CHEST 1996; 110:844-46)

Key words: cardiac tamponade; pneumomediastinum; transtachael oxygen

Abbreviation: TTO=transtachael oxygen

Patients with hypoxemic COPD have decreased mortality after institution of continuous oxygen therapy.\(^1\) Supplemental oxygenation by nasal cannula or transtachael oxygen (TTO) catheterization can correct chronic hypoxemia. Compared with nasal cannula oxygenation techniques, TTO catheterization results in improved cosmeses and compliance with decreased oxygen demands, cost, and work of breathing.\(^2-7\) Complications of TTO catheterization are rare but include cellulitis, mucus plug formation on the catheter tip, subcutaneous air, pneumomediastinum, cephalic displacement of the catheter, granulation tissue, and tracheal perforation.\(^8-10\) There is one prior reported death due to a TTO catheter associated with a mucus plug on the catheter tip with subsequent airway occlusion.\(^11\) This report describes a patient with a mature TTO catheter tract who developed a false catheter lumen following the traumatic use of a flexible metal cleaning rod to remove and reinsert the catheter at home. Due to the malpositioned catheter, there was a defect in the platysma muscle. The patient died suddenly as a result of pneumomediastinum and cardiac tamponade.

*From Pulmonary/Critical Care, Landstuhl Regional Medical Center, U.S. Army, Germany (Dr. Kristo); Pulmonary/Critical Care Department, Fitzsimons Army Medical Center, (Dr. Turner and Ms. Hugler), Aurora, Colo.

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Reprint requests: Maj David A. Kristo, MC, USA, Box 1342, CMR 402, APO AE 09186

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CASE REPORT

A 69-year-old man suffered from hypoxicemic COPD. A TTO system was recommended to improve his comfort and cosmeses while reducing his oxygen requirement. After informed consent, the patient underwent uneventful placement of a SCOOP 1 (Transtracheal Systems; Englewood, Colo) TTO catheter. The tract was mature 3 months after initial placement; at that time, there was no swelling, drainage, discomfort, or resistance to catheter replacement or removal when the patient was examined in the clinic. Consequently, at the 3-month visit, the patient and his wife were given an initial training session on home removal and cleaning of the catheter per our protocol. During the initial training, the patient’s wife verbalized understanding and changed the catheter successfully without any difficulty. A small area of erythema around the stoma was seen, but there was no evidence of infection or subcutaneous bleeding. The patient was asked to abstain from initiating removal of the catheter at home for 1 week to insure the site was not infected and to call or return in person for questions or problems with the TTO catheter. Both the patient and his wife were admonished at all times not to remove the catheter if they “were not completely comfortable with the catheter change procedure and to return for further teaching if desired.” However, the following week in the clinic, the patient’s wife stated “the catheter didn’t seem to want to come out” while performing home catheter removal, cleaning, and reinsertion despite our previous request to leave the catheter in place. The stoma site at the time was not infected upon reexamination. The patient and his wife were asked to return for further education on home catheter removal and cleaning the following week, and they were again asked not to change the catheter in the interim period.

Before the scheduled appointment and despite the previous admonition to leave the catheter in place, the wife decided to perform a routine catheter cleaning and removed the catheter without difficulty. She later stated that the catheter was difficult to reinset and “kept bending,” prompting the wife to use a flexible metal cleaning rod to replace the catheter. The patient complained of discomfort throughout the catheter replacement. That night the patient felt dyspneic while using TTO. He turned off the TTO and used his nasal cannula. The next morning, the wife removed the TTO catheter and found two “kinks” in it and replaced the catheter with a new one. The patient then restarted TTO. Approximately 2½ h later, he suddenly developed chest discomfort and within seconds had subcutaneous air all over his head, arms, and chest. The patient was extremely dyspneic, prompting the wife to increase the oxygen flow to the TTO catheter. The patient collapsed unconscious. Paramedics arrived in 4 min and performed bilateral needle decompression of each hemithorax without hemodynamic improvement. The patient was intubated and brought to the emergency department with an idioventricular rhythm with occasional ventricular fibrillation leading to asystole. All attempts at resuscitation were unsuccessful, and he was declared dead.

Necropsy showed massive mediastinal, intraperitoneal, and subcutaneous emphysema. The TTO stoma site had a blood clot with local subcutaneous hemorrhage. The stoma led to a stenotic tracheal perforation where the TTO catheter had been placed. The TTO catheter stoma site also was contiguous with two 3-mm hemorrhagic areas on the right external surface of the platysma muscle and a 3-mm defect in the platysma muscle with irregular and discolored borders (Fig 1). This defect led to an open space between the platysma muscle and the trachea. It appeared that blood had clotted around the false tract, preventing the oxygen from tracking along the catheter and out of the patient. Consequently, the oxygen dissected subcutaneously and through the platysma muscle leading to pneumomediastinum with secondary cardiac tamponade and sudden death. The portable oxygen source had no mechanical defect; however, these units are not designed with a pressure-release device. Consequently, the oxygen flowed into the mediastinum until the supply was turned off by the paramedics.

DISCUSSION

This is the first case known to us of sudden death resulting from pneumomediastinum and cardiac tamponade from a TTO catheter. A nonfatal case of pneumomediastinum in a TTO patient with a mature tract was previously described in a patient who attempted to replace a TTO catheter (Heimlich MicroTrach; Life Medical Technologies; Salt Lake City, Utah) inadvertently dislodged after 4 months of use.12 Our patient experienced rapid decompression at home, making the prospects for successful resuscitation unclear; however, when the wife increased the flow of oxygen to the TTO catheter rather than turning off the oxygen supply, death was imminent. The lack of cellulitis at the stoma site indicates that the false catheter tract was created by aggressive misdirected replacement of the catheter while using the cleaning rod and not catheter dissection through infected tissue.

The SCOOP I TTO catheter may require frequent catheter changes due to blockage by mucus. The dangers of replacement by a nonprofessional who does not follow correct procedures, as occurred in this case report, are evident. Other TTO catheters (eg, Heimlich MicroTrach) do not obstruct...
with mucus, and therefore require changing, at most, once a month. The replacement of catheters such as a Microtrach, therefore, can be conveniently and safely done in the physician's office.

Patients and clinicians must be aware of the risk of pneumomediastinum and cardiac tamponade with TTO catheters. We recommend the following precautions regarding TTO catheter placement and reinsertion:
1. Unexplained dyspnea or the presence of subcutaneous air is reason to immediately turn off the TTO supply and switch to a nasal cannula source.
2. A TTO catheter should never be advanced against resistance.
3. Flexible metal cleaning rods should be used solely for catheter cleaning and not as an aid in catheter placement by patients or home-care providers.
4. Ideally, a pressure-release device on portable oxygen sources could be developed to prevent ongoing oxygen delivery to the patient in the event of a displaced catheter.
5. Use of TTO catheters that do not obstruct with mucus facilitates, at most, a monthly catheter change performed safely in the physician's office. Consequently, frequent and potentially dangerous catheter removal and cleaning by nonprofessionals at home is eliminated.

We believe that numerous studies have shown the overall safety and efficacy of TTO in certain patients. The potential for serious complications, however, still exists as this report demonstrates.

REFERENCES

Atrial Septal Aneurysm Plus a Patent Foramen Ovale*

A Predisposing Factor for Paradoxical Embolism and Refractory Hypoxemia During Pulmonary Embolism

Philippe Estagnasi, MD; Kanel Djedaini, MD; Geneviève Le Bourdelles, MD; François Coste, MD; and Didier Dreyfuss, MD

We report three consecutive cases of patients who had refractory hypoxemia and paradoxical embolism during the course of pulmonary embolism. Transesophageal echocardiography showed an atrial septal aneurysm and a patent foramen ovale in all patients. The latter was detected by an early and massive passage of contrast from the right to the left atrium. We suggest that the presence of an atrial septal aneurysm plus a patent foramen ovale greatly enhances both magnitude of shunting and the risk of systemic embolism. The presence of an atrial septal aneurysm plus a patent foramen ovale should be considered and checked using transesophageal echocardiography in every patient with significant pulmonary embolism.

(CHEST 1996; 110:846-48)

Key words: atrial septal aneurysm; paradoxical embolism; patent foramen ovale; pulmonary embolism; refractory hypoxemia

A patent foramen ovale may be responsible for severe hypoxemia due to shunting when the right atrial pressure exceeds the left atrial pressure1 and for paradoxical embolism,2 especially during pulmonary embolism.3 The relative rarity of these complications, despite the very frequent occurrence of both pulmonary embolism4 and patent foramen ovale,5 is intriguing. This may suggest that there is a predisposing factor in patients experiencing these complications. An atrial septal aneurysm, described as a bulge in the fossa ovalis, is also a known cause of cardiac embolism.2 This malformation could greatly enhance the risk of complication during pulmonary embolism. Nevertheless, there have been no reports, to our knowledge, of the association of a patent foramen ovale and an atrial septal aneurysm being a risk factor of both severe hypoxemia and paradoxical embolism during pulmonary embolism.4 This may be because transesophageal echocardiography was not routinely practiced in previous studies. Transesophageal echocardiography is the best technique available today for detecting an atrial septal aneurysm.6,7 Nevertheless, even recent publications on the state of the art of transesophageal echocardiography do not mention the documentation of an atrial septal aneurysm during pulmonary embolism.8 Our report describes three patients with pulmonary embolism who suffered from refractory hypoxemia and stroke. Trans-

*From Service de Réanimation Médicale, Hôpital Louis Mourier, Colombes, France.
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Reprint requests: Dr. Dreyfuss, Service de Réanimation Médicale, Hôpital Louis Mourier, 92700 Colombes, France