

Case Report

Massive Carbon Dioxide Gas Embolism: A Near Catastrophic Situation Averted by Use of Cardiopulmonary Bypass

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Abstract: Endovascular vein harvesting is used as a less invasive method when compared to the standard open surgical method of dissecting and procurement of the greater saphenous vein. The benefits include smaller incision, decreased blood loss, less pain, decreased transfusion, decreased risk of infection and enhanced epidermal cosmetics. However, endovascular vein harvesting does have a learning curve to master the technique and although endovascular vein harvesting is the preferred standard of practice over the open surgical technique, it does have inherent potential complications. During endovascular vein harvesting, there is a potential for the patient's circulatory system to collapse which can be identified initially through hemodynamic monitoring, blood gas results, and vigilance through clinical observation. The suspected source of the crisis was confirmed when the surgeon cannulated the right atrium, where a massive gas source that (appeared pressurized) escaped upon incision of the right atrial

appendage. Cardiopulmonary bypass (CPB) was utilized to support the patient and rectify the impending catastrophic event. Once full CPB was attained, we achieved hemodynamic stability and eventually all blood gases were normalized. Massive CO₂ embolism is a life threatening emergency which must be identified and corrected instantaneously. CPB was the modality used to salvage this situation. Attention to the set-up of the EVH equipment, use of the transesophageal echocardiography, cerebral oximetry, vigilance and cooperation of all disciplines in the OR are definite recommendations to prevent such an occurrence. Experience gained by the perfusion team with a previous case was applied and helped to solve the immediate problems presented in this case. **Keywords:** endovascular vein harvesting, cardiopulmonary bypass, transesophageal echocardiography, end tidal CO₂, vacuum assisted venous drainage, embolus, carbon dioxide. *JECT. 2009;41:110–113*

Carbon dioxide (CO₂) is used for many applications that include but are not limited to lasers, pH stat maintenance during deep hypothermic circulatory arrest in pediatric surgery, flushing of the surgical field during heart valve surgery, abdominal laparoscopy, flushing of the extracorporeal circuit, and endovascular vein harvesting (EVH). It is also known to be a potent respiratory stimulant and has quite a prominent role in human physiology. The Bohr/Haldane effect shows how the heme molecule of hemoglobin performs CO₂ uptake at the cellular level and O₂ uptake at the alveolar level (1). CO₂ readily dissolves into solution at a rate of ~20–30 times greater than that of oxygen (2).

Coronary artery bypass surgery routinely uses the left internal mammary artery (LIMA) and the greater saphenous vein as conduits. Other conduits may be used for this procedure. EVH is a modality used at our hospital, which uses the ACMI Turbo Flow 8000 CO₂-regulating device (ACMI, Southborough, MA) with the Guidant Vasoview Endo Harvesting System (Guidant, Santa Clara, CA). These devices use CO₂ gas as a means for creating insufflation to enhance visualization. CO₂ is introduced at a flow rate of 2–4 L/min, which is dependent on a servo-sensitive pressure loop system. A desired pressure between 10 and 12 mmHg is now used at our institution to regulate the CO₂ flow (previously between 15 and 20 mmHg). It is reported that 60% of systemic mishaps occur during initial CO₂ insufflation (3). These mishaps can include acute hypercarbia with its associated complications and/or cardiovascular collapse. An insufflation pressure increase from 12 to 15 mmHg has been shown to increase the systemic CO₂ incidences from 6.5% to 13.3%, respectively (4). CO₂ embolization must always be suspected in EVH whenever sudden hemodynamic deterioration occurs

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and/or is associated with a sudden increase and a subsequent decrease in end tidal CO₂ (ETCO₂ mmHg) (5).

We report a case of massive CO₂ embolism before cardiopulmonary bypass (CPB) and highlight the identification and management of this life-threatening situation. The CO₂ embolus was caused by EVH and identified by arterial blood gas before CPB, regional cerebral oximetry (rSO₂), ETCO₂, transesophageal echocardiography (TEE), and arterial blood gas pre- and post-initiation of CPB. Coronary artery bypass graft (CABG) was performed in the usual fashion and completed successfully. The patient had no immediate post-operative events related to the intraoperative situation and was discharged on post-operative day 10 from the hospital.

CASE REPORT

This 57-year-old woman presented with left lateral visual deficit associated with some left-hand numbness and cephalgia. Her height was 162.5 cm, weight was 115.7 kg, and body surface area (BSA) was 2.18 m². Ejection fraction was 55% with left ventricular wall thickness at the upper limits of normal. Final diagnosis was migraine cephalgia, sinusitis, systemic hypertension, hypercholesterolemia, type II diabetes mellitus, triple vessel coronary artery disease, gastro-esophageal reflux disease, congestive heart failure, myocardial infarction, and a transient ischemic attack in 1990. The patient had no significant stenosis (NSS) of the carotid arteries and no known drug allergies (NKDAs).

The patient was taken to the operating room (OR) for coronary artery bypass grafts × 3, with the intention of using the left internal thoracic artery (LITA) and the right leg greater saphenous vein. rSO₂ was measured through the right cerebral hemisphere (we monitored one side because there was no significant stenosis). A right brachial arterial line was inserted and transduced for arterial pressure monitoring. An intravenous catheter was introduced in the right arm and used for anesthetic drug administration: succinylcholine, 100 mg; pancuronium bromide, 10 mg; fentanyl, 1000 mg; versed, 10 mg. The patient was ventilated with 100% O₂ by mask before intubation.

Intubation was done with a #8 endotracheal tube, and a Swan Ganz catheter (Edwards Lifesciences LLC, Irvine, CA) was inserted through the right internal jugular vein. TEE showed no valvular disease. After preparation and draping, the surgical team proceeded to harvest the conduits according to standard protocols.

During EVH and LITA dissection, it was noticed that the blood pressure started to drift downward from 121/83 to 80/40 and then to 30, with a corresponding fall in the pulse oximeter saturation reading to a point where it did not read any longer. The rSO₂ fell from 66% to 38%, and bradycardia occurred, which progressed to a flat line on the EKG monitor. The surgeon aborted the left internal mammary artery

harvesting, and the anesthesiologist began administering epinephrine boluses and sodium bicarbonate. On the drop in mean arterial pressure, internal cardiac massage was performed with no visual changes in the arterial pressure waveform. The heart was enlarged, although the right heart was empty of blood as seen by TEE. Gross gas embolization of CO₂ had filled the right heart, causing it to be totally de-primed and hence leaving the left heart with no output, which resulted in a fall in blood pressure and ensuing cardiac arrest. The surgeon continued open chest cardiac massage, and 40,000 IU porcine heparin was given at this point. An arterial blood gas was obtained through the right brachial line, and the result was as depicted as no. 2 in Table 1. The Bayer 405 Rapid Point blood gas analyzer (Bayer, East Walpole, MA) has an upper CO₂ reading limit of ~185 mmHg. Before initiation of CPB, it was noticed that the capnograph read an ETCO₂ of 32 mmHg, and suddenly it increased to 35 mmHg but proceeded to fall to ~12–18 mmHg. During normal ventilation of the anesthetized patient, the capnograph will display ETCO₂ values, which reflect arterial blood PCO₂. Because of the total de-priming of the right heart, which resulted in no blood flow through the lungs, the ETCO₂ reading showed a dramatic decrease as was recorded. Capnographic recordings showing acute changes can be one tool used for diagnosing possible CO₂ embolus (6). This increased arterial CO₂ (Table 1, Lines 2, 3, and 4) was observed in a previously unpublished case in 1999 with the advent of EVH. During the initial use of EVH, long periods of times were spent in harvesting these veins because of the inherent learning curve of this procedure. This resulted in large amounts of CO₂ dissolving in the venous blood, which found its way back through the superior vena cava to the heart and then to all body tissues.

Based on the experience gained in that case, we were able to recognize and pinpoint the causative problem. At this point, the physician's assistant stopped the EVH and proceeded to the chest to help the surgeon; the EVH equipment was in operational mode and remained in situ with the insufflator on, causing massive amounts of CO₂ gas to bubble up through the side branch of the saphenous vein into the inferior vena cava (IVC). This added to the dosing of more CO₂, which was already introduced into the vascular system. A Terumo 8.0-mm soft flow aortic cannula (Terumo Cardiovascular Systems Corp., Ann Arbor, MI) was rapidly inserted into the aorta and connected to the arterial 3/8" line of the perfusion circuit. The right atrium was opened, and an excessive amount of pressurized bubbling was noticed through the IVC, with the venous blood coming to and out of the right atrium (RA).

When the source of the extraneous CO₂ was identified as coming from the EVH equipment, the CO₂ gas supply was immediately discontinued. After this, the RA was cannulated with a Medtronic 34 × 48-Fr dual stage

Table 1. Blood gas results.

No.	Time	pH	pCO ₂ (mmHg)	pO ₂ (mmHg)	HCO ₃ (mmol/L)	BE(B) (mmol/L)	Hct (%)	tHb (g/dL)	FO ₂ Hb (%)	Na (mmol/L)	K ⁺ (mmol/L)	Ca ²⁺ (mmol/L)	Cl ⁻ (mmol/L)	Glu (mg/dL)	ACT (seconds)	Events	rSO ₂ (%)	Action
1	8:59	7.432	40.3	81.8	26.3	1.9	41	13.8	95.5	139.0	4.73	1.16	103	169	115	Pre-induction	66	
2	10:33	6.755	↑	71.3	→	→	31	10.7	76.2	138.3	5.13	0.96	100	118	774	Pre-CPB	38↓	Emergent cannulation
3	10:45	7.338	74.7	478.6	39.2	11.7	24	8.1	98.2	140.9	4.44	0.81	100	216	800	ABG on CPB	54	Sweep gas 6 L/min
4	10:47	6.989	190.7	45.0	44.8	10.9	23	7.9	57.2	138.4	4.20	0.89	99	226	800	VBG* on CPB	53	Sweep gas ↑ to flush (~43 L/min)†
5	10:52	7.440	40.9	535.7	27.2	2.8	27	9.2	98.5	136.4	4.72	0.75	101	263	770		58	Sweep gas 9 L/min
6	10:59	7.383	45.8	399.5	27.3	1.7	32	11.0	99.0	137.9	4.34	0.88	101	276	763		62	
7	11:22	7.423	38.5	462.7	24.6	0.3	34	11.4	99.3	136.4	5.84	0.90	103	316	610		59	
8	11:40	7.431	36.1	459.0	23.5	-0.5	34	11.4	98.9	137.1	4.74	0.94	102	305	665		61	
9	12:03	7.481	30.1	318.8	22.0	-0.8	31	10.6	98.9	135.1	5.37	0.92	103	268	630		62	
10	12:24	7.412	34.9	306.1	21.7	-2.3	37	12.5	98.9	138.6	5.11	1.36	103	269	640		66	Sweep gas ↓ 3.5 L/min
11	12:37																	
11	13:05	7.433	37.1	293.5	24.2	0.1	30	10.2	99.2	141.7	3.83	0.88	102	235	103	Off CPB	66	
12	13:36	7.046	42.9	166.5	26.3	1.4	29	9.9	98.1	140.1	4.45	1.05	102	211		Post-protamine		
13	13:57	7.430	36.4	209.2	23.6	-0.4	33	11.3	98.3	139.5	4.32	1.21	104	191				

*Venous blood gas.

†Performed after results 3 and 4.

Table 2. Bayer blood gas analyzer measurement ranges.

Measured Value	Range
pH	6.676–7.700
PCO ₂	12.8–185 mmHg
PO ₂	40.6–569 mmHg
CL ⁻	70–129 mmol/L
Na ⁺	108.3–172.5 mmol/L
Glucose	23–715 mg/dL
K ⁺	1.36–11.74 mmol/L
Hgb	4.8–21.0 g/dL
Ca ²⁺	0.51–2.92 mmol/L

Reportable ranges for the Bayer Rapidpoint 405 blood gas analyzer.

Table 3. Unpublished blood gas result from December 8, 1999.

pH	pCO ₂	pO ₂	HCO ₃	TCO ₂	BE	THb	O ₂ Hb
7.080	102.9	416	30.9	34.0	-1.0	12.3	98.2

ABG pre-CPB during EVH.

Table 4. Event time line.

No.	Event	Time Line
1	Start of EVH	09:42
2	Patient's chest incision was performed	09:47
3	Amiodarone 300 mg × 25 minutes	09:52
4	Median sternotomy	10:07
5	Amiodarone 33 mL/h × 6 hours	10:18
6	Heart rate decreased from 64–53 beats/min	10:29
7	Systolic arterial blood pressure drops from 86–30 mmHg	10:29
8	40,000 units heparin sodium IV	10:29
9	Isoflurane off	10:29
10	Amiodarone off	10:29
11	Internal cardiac massage	10:30
12	Epinephrine 1 mg	10:30
13	Epinephrine 1 mg	10:31
14	SpO ₂ decreased from 100–97%, rSO ₂ ↓ 66–38%	10:31
15	Mean arterial pressure 30 mmHg	10:31
16	SpO ₂ did not register	10:32–10:35
17	Atropine sulfate .4 mg	10:34
18	Cerebral oximetry saturation (rSO ₂) down from 32–28%	10:29–10:35
19	50 mEq sodium bicarbonate	10:35
20	CPB initiated with 10,000 U heparin in prime	10:35
21	Removal of EVH	10:38

SpO₂, pulse oximetry saturation.

venous cannula (Medtronic Inc., Minneapolis, MN). At the onset of CPB, venous O₂ saturation was recorded at 32%. There was no venous return because of the large volume of gas (CO₂), and hence vacuum assisted venous drainage (VAVD) in the range of -10 to -30 mmHg was instituted. With a cardiac index of 2.0 L/min/m² and after 10 minutes of CPB, arterial and venous blood samples were drawn, and the results are documented in Table 1. The ventilating gas flow at this time was at 6 L/min. After these results, the gas flow was increased for 5 minutes to "FLUSH," which, in a laboratory setting, was measured later at ~43 L/min by using a Wright respirometer (Ferraris Respiratory Europe, Hertford, England) with an olympic style tachometer. The

operation proceeded successfully with one saphenous vein graft conduit anastomosed to the left anterior descending artery and the other vein graft anastomosed sequentially to the obtuse marginal and the diagonal arteries. Total pump time was 122 minutes, and cross-clamp time was 66 minutes. A total of 3100 mL microplegia (7) was delivered through a 4:1 ratio (blood:crystalloid). Two units of homologous blood were used, because there was some blood loss in the field when venous cannulation was attempted. The patient was rewarmed from a bladder temperature of 34°C to 37°C and was given 2 g magnesium sulfate (MgSO₄) and 1 g calcium chloride (CaCl₂) 5 minutes after the cross-clamp was removed. A total of 2200 mL of fluid was removed through the hemoconcentrator (Sorin Group Cardiovascular, Arvada, CO). With a reperfusion time of 20 minutes, separation from the CPB equipment was attained, and all the pump volume was given back to the patient through the retrograde cardioplegia cannula, which was repositioned in the RA. Protamine was administered, and a blood sample was taken to measure the arterial blood gas and activated clotting time (ACT). The results were documented as no. 11 in Table 1. TEE showed no hypokinesis, no gas embolus, and good ventricular function. Cardiac output was 4.1 L/min with a cardiac index of 2.1 L/min/m².

DISCUSSION

Massive CO₂ embolism during EVH is a life-threatening emergency that must be identified and corrected instantaneously (8). CPB was the modality used to salvage this situation. This patient was already prepared for CABG surgery and thus the appropriate equipment was immediately available. Indeed, this made the whole situation very optimized.

Attention to the set-up of the EVH equipment, use of the TEE, cerebral oximetry, vigilance, and cooperation of all disciplines in the OR are definite recommendations

to prevent such an occurrence (8). Experience gained by the perfusion team with a previous case in 1999 was an asset to identifying the immediate problems that this case presented.

We recommend that CO₂ insufflation pressures between 10 and 12 mmHg, TEE probe in situ at all times, continuous ETCO₂, and attention to hemodynamic parameters including arterial blood gases are all definite areas to monitor during EVH. The cardiovascular perfusionist must also be alert and prepared to intervene with CPB equipment when requested.

As a result of this experience, we also recommend that institutions that use this modality (EVH) have policies/protocols and procedures to prevent, recognize, and correct the occurrence of CO₂ embolism.

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