HIGH-QUALITY cardiopulmonary resuscitation (CPR) during cardiac arrest is critical to maintaining perfusion of vital organs. The 2010 American Heart Association Guidelines define high-quality CPR in adults as: (1) at least 100 compressions per minute, (2) compressions of at least 2 inches in depth, (3) complete chest recoil between compressions, and (4) minimization of interruptions to compressions. Conventional CPR is performed in the supine position on a firm surface. Alternative compression techniques, such as open-chest cardiac massage, interposed abdominal compression CPR, “cough” CPR, precordial thump, and percussion pacing have been described, but none have been shown to be superior to traditional supine chest compressions.

Intraoperative cardiac arrest can present a challenge when patients are in the lateral or prone position, as a quick transition to the supine position can be difficult. There are four reported cases of successful intraoperative CPR in the prone position, but to our knowledge, CPR in the lateral position has not been reported. Here, we describe a case of successful intraoperative CPR with chest compressions performed on a patient in the lateral position, and calculate the ratio of cardiac output (CO) during chest compressions to CO prearrest.

CASE REPORT

A 67-yr-old man (183 cm tall, 84 kg) with a left renal tumor extending into the renal vein underwent an open nephrectomy. Past medical history was notable for nonobstructive coronary artery disease, and Wolff–Parkinson–White syndrome refractory to ablation treatment. The patient was asymptomatic with excellent exercise tolerance. Baseline electrocardiogram displayed sinus rhythm with a short PR interval and Δ wave consistent with a Wolff–Parkinson–White pattern, a right bundle branch block, and nonspecific T-wave abnormalities (fig. 1). Aspirin was held preoperatively, given the bleeding risk.

After placement of a large-bore intravenous catheter, radial arterial line, and mid-thoracic epidural, induction of general anesthesia and tracheal intubation were completed uneventfully. The patient was positioned in the right lateral decubitus position with the bed flexed. Surgical exposure was achieved through an incision from the tip of the eleventh rib to the lateral border of the rectus abdominus, and maintained with an Omni Retractor System (Integra LifeSciences, Plainsboro, NJ), similar to that shown in figure 2. Anesthesia was maintained with sevoflurane and a continuous epidural infusion of bupivacaine 0.1% and hydromorphone 20 μg/ml. Volume-controlled ventilation parameters were: tidal volume = 540 ml/breath, rate = 12 beats/min, and FIO₂ of 0.5.

The patient remained hemodynamically stable with no vasopressor requirement and minimal blood loss until the 2-h mark, when he developed a wide-complex tachycardia at 140 beats/min, with a mean arterial blood pressure around 20 mmHg. The surgical team was instructed to start chest compressions immediately, and defibrillation equipment was immediately requested. While one surgeon initiated chest compressions with the patient in lateral position, the other surgeon worked to remove the Omni Retractor system to allow for supination. Visibly forceful chest compressions were performed using a two-handed technique at approximately 100 compressions per minute, with the palm of the right (dominant) hand against the patient’s mid-thoracic spine, and the palm of the left (nondominant) hand directly opposite, against the sternum, as demonstrated in figure 3.

During resuscitation, FIO₂ was increased to 1.0, but all other ventilation settings remained unchanged. A carotid
pulse was palpable with compressions, and the arterial line waveform appeared sinusoidal with systolic values near 70 mmHg, end-tidal CO₂ above 20 mmHg, and oxygen saturation greater than 95%. We did not administer a beta-blocker, calcium channel blocker, or adenosine during the arrest, for fear of accentuating a possible Wolff–Parkinson–White rhythm, such as a deterioration of antedromic atrioventricular tachycardia to ventricular tachycardia or fibrillation. We did not have procainamide or amiodarone immediately available, which would have been the two best medical agents to use at the time. Furthermore, we did not administer epinephrine or vasopressin in the setting of a known

Fig. 1. Preoperative electrocardiogram. Short PR interval and δ wave consistent with a Wolff–Parkinson–White pattern, right bundle branch block, and nonspecific T-wave abnormalities.

Fig. 2. Omni Retractor System (Integra LifeSciences, Plainsboro, NJ) with a patient in lateral decubitus position.

Fig. 3. Surgeon reenactment of chest compressions on a patient in the lateral decubitus position with an Omni Retractor System (Integra LifeSciences, Plainsboro, NJ) in place.
Wolff–Parkinson–White wide-complex rhythm as these would not have terminated the arrhythmia. Ultimately, after 4.5 min of CPR, the patient's rhythm converted to sinus with systolic blood pressure near prearrest values, before pharmacologic or electrical intervention.

The remainder of the case was uneventful, and the patient was hemodynamically stable. Immediate postresuscitation arterial blood gas (7.40/47/174; FIO₂, 1.0; Hb, 13 g/dl) did not display signs of metabolic acidosis, and pH was relatively unchanged compared with an arterial blood gas before the arrest (7.45/40/270; FIO₂, 0.5; Hb, 13.6 g/dl) when accounting for PaCO₂ correction. The patient had received his normal Nadolol dosage the morning of surgery, and an intraoperative cardiology consultation advised no other antiarrhythmics or medication were needed. The patient was emerged from anesthesia and extubated. Postoperatively, he had no new electrocardiogram findings that caused concern, had normal cardiac markers, normal renal and liver function, and no neurologic deficits. After an uneventful postoperative course, he was discharged home on postoperative day 3, in good condition, with no sequelae of the arrest at 6-month follow-up.

**Discussion**

A complete recovery, without any postoperative neurologic deficits, combined with lack of clinical or laboratory evidence of tissue ischemia or infarction, suggests that adequate tissue perfusion and oxygenation were achieved during resuscitation performed in the lateral position. We mathematically validate this claim by calculating the ratio of CO during resuscitation to the prearrest value using physiologic concepts of oxygen supply and demand.

1. **Determine basal oxygen consumption under general anesthesia**

Before cardiac arrest, the patient was hemodynamically stable, with a constant anesthetic depth, temperature, FIO₂, and a normal arterial blood gas. Given these steady-state conditions, the patient’s basal oxygen consumption must be equivalent to the amount of oxygen extracted from alveoli.

- Basal oxygen consumption = oxygen extracted from alveoli
- Oxygen extracted from alveoli = minute alveolar ventilation × (FIO₂–ETO₂)
- Oxygen extracted from alveoli = 255 ml/min

The patient consumed 255 ml O₂/min under general anesthesia. Because there was no evidence of anaerobic metabolism on pre- or postarrest arterial blood gas samples, we can assume that this basal oxygen consumption remained constant before, during, and after CPR.

2. **Define the relationship between CO and “oxygen deficit”**

Imagine if the CO generated by chest compressions had been exactly the same as the prearrest CO, then the amount of oxygen extracted from alveoli would have matched the patient’s basal oxygen consumption of 255 ml O₂/min, and the “oxygen deficit”, as we define it, would be zero.

To the other extreme, if CO had been zero during chest compressions and resuscitation efforts were completely ineffective, then no oxygen would have been extracted from alveoli, and the oxygen deficit would have been 255 ml/min (equal to the basal metabolic rate). In reality, the chest compressions generated CO that was between these two extremes.

Oxygen deficit arises when there is insufficient delivery, either due to reduction in arterial oxygen content, or CO.

![Cardiac Output as a Function of Oxygen Deficit](image)

**Fig. 4.** Cardiac output generated during cardiopulmonary resuscitation (CPR) is inversely proportional to the oxygen deficit (ml O₂/min). Recall that oxygen deficit is defined as: (basal metabolic oxygen consumption – oxygen extracted from alveoli). In this case, the oxygen deficit was 155 ml O₂/min, which extrapolates to a cardiac output during CPR of 39% of baseline.
Oxygen content of arterial blood mainly arises from the product of hemoglobin and oxygen saturation, which remained nearly constant throughout the resuscitation. Therefore, CO is the only variable that is inversely proportional to the acquired oxygen deficit (fig. 4), and is the rate-limiting factor determining oxygen extraction from alveoli.

3. Determine the “oxygen deficit” during resuscitation.

Oxygen deficit (ml/min) = Basal metabolic oxygen (ml/min) − Oxygen extracted from alveoli (ml/min)

To determine oxygen deficit, we need to know the amount of oxygen extracted from alveoli during resuscitation. If $FIO_2$ were constant during the arrest, we could calculate oxygen deficit as we did in Step 1. However, because $FIO_2$ was rapidly increased to 1.0 at CPR initiation with a subsequent delayed and lagging rise in $ETO_2$, which indicates accumulation of oxygen in alveoli, this calculation would result in overestimating oxygen deficit. Therefore, we need a different method to calculate oxygen deficit.

After 4.5 min of CPR, the patient had a return of spontaneous circulation and compressions were halted. For the next 3.5 min, the patient extracted more oxygen per minute from the alveoli than his prearrest oxygen consumption rate of 255 ml/min. The patient extracted an additional 698 ml of oxygen, for a total oxygen extraction of 1,591 ml (698 ml + 255 ml/min × 3.5 min). The additional amount (698 ml) of oxygen extraction must be the “oxygen deficit” accumulated during the arrest due to the low CO generated by chest compressions. Because cardiac arrest and CPR lasted for 4.5 min, the patient’s oxygen deficit was 155 ml O₂/min (698 ml O₂/4.5 min).

4. Estimate CO generated by chest compressions.

Using figure 4, an oxygen deficit of 155 ml O₂/min corresponds to a CO ratio of 39%. Therefore, the CO generated by chest compressions in the lateral position is estimated to be 39% of the prearrest value.

Conclusion

Cardiac arrest with a patient in the lateral position leads to unique resuscitation challenges. The primary objective remains as commencement of high-quality chest compressions as soon as possible in the supine position. However, as demonstrated, moving to the supine position may not be possible in a timely manner due to bulky surgical retractor systems, surgical clamps on delicate structures, and various other reasons. Chest compressions on a patient in the lateral position can provide CO that is adequate for tissue perfusion and gain time for more advanced treatments.

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Competing Interests

The authors declare no competing interests.

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