University of Ottawa, Ottawa, Canada
  Division of Emergency Medicine
  (JVQ, IGS)
  Department of Medicine (PCH)

Need for Sedation in a Patient Undergoing Active Compression–Decompression Cardiopulmonary Resuscitation

James V. Quinn, MD, Paul C. Hebert, MD, Ian G. Stiell, MD

The authors report the case of a 57-year-old man with a history of ischemic heart disease who presented to the emergency department with an acute myocardial infarction and hypotension. Despite aggressive pharmacotherapy, the patient's heart rate decreased, and he developed pulseless electrical activity within 15 minutes of his arrival. Cardiopulmonary resuscitation (CPR) was begun with an active compression-decompression (ACD) device, and the patient became agitated, making purposeful movements. When ACD-CPR was discontinued for a rhythm check, the patient had no pulse and became motionless. Agitation and purposeful movements occurred on two subsequent occasions with the initiation of ACD-CPR. The patient required physical restraints, sedation, and paralysis for personnel to perform endotracheal intubation and facilitate treatment. The implications of this case are discussed.

Survival rates for cardiac arrest patients remain poor even with the early initiation of cardiopulmonary resuscitation (CPR).[1] Traditional CPR often fails to provide significant blood flow to vital organs. Specifically, cardiac output and cerebral blood flow are only 5–15% of those in the prearrest state with standard chest compressions.[2,3] Recently the concept of active compression–decompression (ACD) CPR has been proposed as a means to improve hemodynamic variables in patients undergoing CPR.[4,5] Prospective field trials are under way to assess the impact that this device may have on survival of cardiac arrest patients.[6]

The University of Ottawa is currently part of a multicenter trial in Canada comparing ACD-CPR with standard CPR. We report a case during this ongoing trial in which a patient in cardiac arrest showed clinical signs of cerebral perfusion during ACD-CPR that were absent when ACD-CPR was discontinued. We discuss the clinical and investigational implications of this phenomenon.

A 57-year-old man who had a past history of stable exertional angina presented to the emergency department after awakening with retrosternal chest pain. The pain had started 45 minutes prior to his arrival. He described an increasing frequency of angina with exertion over the preceding week. His medications were diltiazem, aspirin, and nitroglycerin. Cardiac risk factors included smoking, non–insulin-dependent diabetes, and elevated cholesterol.
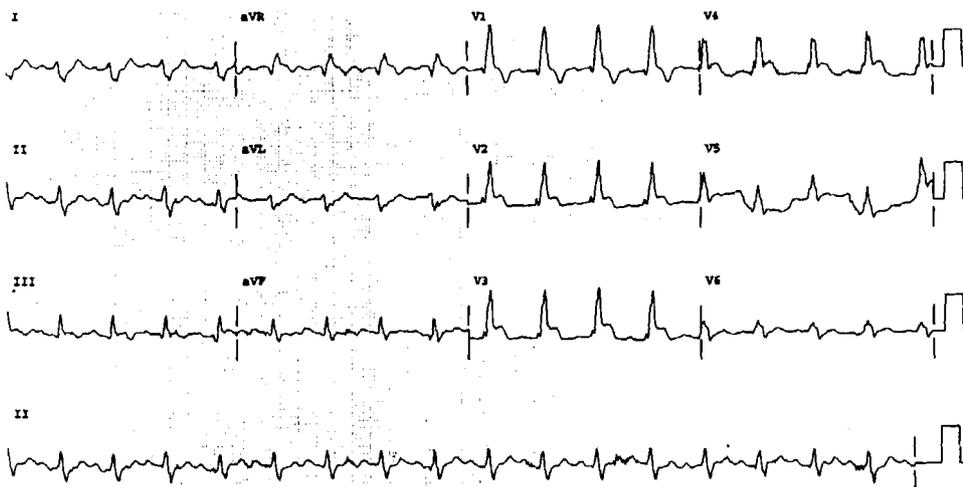
FIGURE 1. Electrocardiogram on arrival in the emergency department.
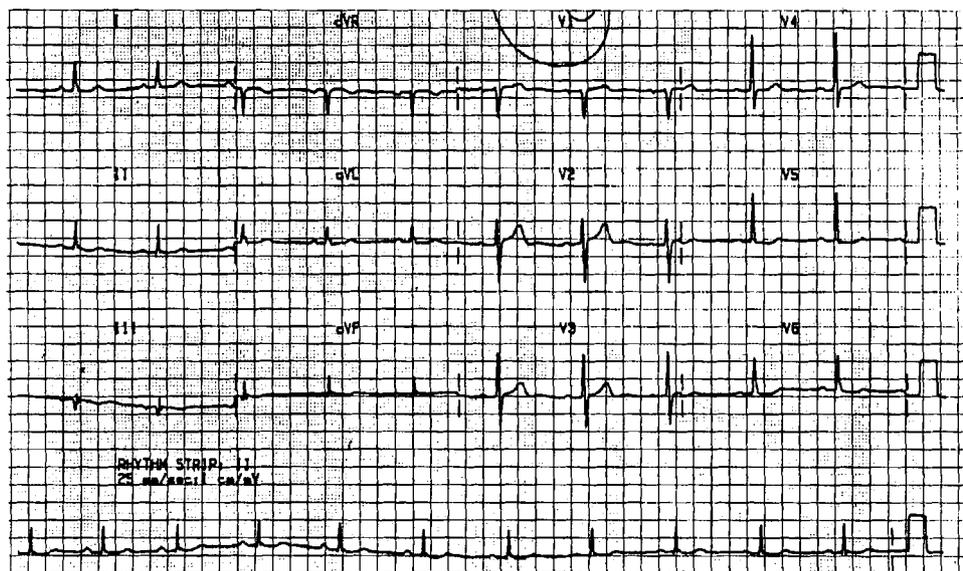
FIGURE 2. Previous electrocardiogram three months earlier (most recent available electrocardiogram).

distress, appearing pale, diaphoretic, and dyspneic. Blood pressure was 80 torr by palpation; heart rate was 100 beats/min; and respiratory rate was 30 breaths/min. The jugular veins were evident 6 cm above the sternal angle; crepitations were auscultated in both lung fields; heart sounds were distant; and there were no audible heart murmurs. The patient weighed approximately 110 kg.

Electrocardiogram showed a new bifascicular block with anterior ST-segment elevation (Figs. 1 and 2). Significant laboratory results included: glucose 21.9 mmol/L (normal range 4.2–6.4); white blood cell count 17.9 10^9/L (normal range 4.0–10.0); total creatine kinase 71 U/L (normal range 45–220). Serum electrolytes, blood urea nitrogen, and creatinine were within normal limits.

Within 15 minutes of arrival in the emergency department, the patient became unresponsive, with a progressive bradycardia, and subsequently developed pulseless electrical activity. This progression occurred despite high flow oxygen, atropine, and dopamine. At the initiation of CPR, he was randomized to the ACD arm of the multicenter trial.

CPR Events

4:25 AM The patient became unconscious. Femoral and carotid pulses were not palpable. Blood pressure could not be determined by Doppler signal. The patient was found to be in pulseless electrical activity, with a heart rate of 30 beats/min. Chest compressions were started with an ACD device, and the patient received bag–valve–mask ventilation. After a few compressions with the device, the patient progressed from being motionless to an agitated state, with purposeful movement of hands (i.e., pulling at the ACD device and ventilation mask).

4:26 AM CPR was stopped. The patient again was found to be in pulseless electrical activity and to be motionless without ACD CPR.

4:27 AM Epinephrine (1 mg IV) was given with a 250-mL fluid bolus of 0.9% normal saline, and ACD-CPR

was restarted. The patient again became agitated, violently moving his arms and legs. Limb restraints were applied. CPR was stopped to check his rhythm, and the patient became motionless. The patient was in pulseless electrical activity, showing complete heart block at a rate of 30 beats/min. Atropine (1 mg IV) was given, and intubation was attempted unsuccessfully.

4:30 AM The patient was found to have a slow agonal rhythm without pulses. ACD-CPR was restarted, and the patient again began to move purposefully. ACD-CPR was continued.

4:30 AM The patient underwent a rapid-sequence induction with midazolam (0.1 mg/kg IV) and succinylcholine (1.5 mg/kg IV) to allow tracheal intubation and facilitate treatment.

4:38 AM The patient experienced muscle fasciculations (1 min, 15 sec after the succinylcholine was administered) and the trachea was intubated successfully.

4:40 AM Epinephrine (1 mg IV) was given.

4:41 AM Femoral pulse was good during ACD-CPR. When CPR was stopped, the patient was found to have pulseless electrical activity with a slow agonal rhythm. ACD-CPR was restarted.

4:42 AM Atropine (1 mg IV) was given.

4:45 AM A central line was inserted into the right internal jugular vein.

4:47 AM ACD-CPR was stopped. The patient was in asystole. Epinephrine (1 mg) and atropine (1 mg) doses were repeated and ACD-CPR continued.

4:52 AM An attempt at transcutaneous pacing was unsuccessful.

4:55 AM The patient remained in asystole. Epinephrine (1 mg) and atropine (1 mg) doses were repeated with a 250-mL normal saline bolus. ACD-CPR was continued.

4:57 AM The patient remained asystolic. Magnesium sulfate (2 g IV) was given. ACD-CPR was continued.

5:01 AM In light of continued asystole, the patient was pronounced dead.

∎ DISCUSSION

This unfortunate patient presented with massive, acute myocardial infarction. He arrived with hypotension and a new bifascicular block that progressed to complete heart block and eventually asystole. He probably had a lesion in the left anterior descending coronary artery that caused infarction to a large portion of the myocardium and conduction system with associated heart block and cardiogenic

shock. Unfortunately, we were unable to confirm our clinical suspicion because of our inability to obtain family permission for an autopsy.

We had not previously witnessed purposeful movements by a patient undergoing CPR while in full cardiac arrest. Prior reports do indicate that an alert patient in ventricular fibrillation can maintain consciousness while coughing.[7] Given the reproducibility of our patient's witnessed agitation and purposeful movements, significant improvement in cerebral perfusion occurred during ACD CPR.

When performed optimally, traditional CPR has been shown to provide, at best, cerebral perfusion pressures only 30–60% of normal and coronary perfusion pressures only 20–25% of normal.[2,8] Various devices and techniques, such as interposed abdominal compression (IAC), CPR, vest CPR, and ACD-CPR, have been proposed to improve these perfusion pressures.[9] The ACD device used in our case was developed in response to a case of successful home resuscitation that used a toilet plunger to compress and decompress the victim's chest.[10]

The ACD device consists of a rubber suction cup connected with a bellows to a plastic handle with grips that allow active decompression. The suction cup is placed on the midsternum at the level of the nipples. The compression phase and rate are maintained in accordance with American Heart Association guidelines.[11] The decompression phase consists of active lifting of the device by its handle without dislodgement of the device from the chest. The speculated mechanism of action is that the ACD device produces a negative intrathoracic pressure during decompression, thereby improving venous return, transmitral ventricular filling, and cardiac output.[4,5]

Use of the ACD device in humans has commonly occurred late, after traditional CPR.[4] Our use of the device early in a witnessed cardiac arrest scenario may explain the dramatic neurologic response of our patient to ACD-CPR. Unfortunately, our patient suffered massive myocardial infarction with heart block and cardiogenic shock; this insult was not reversible with standard pharmacologic therapy.

This case raises several issues:

1. Would our patient have shown similar clinical evidence of cerebral perfusion during standard CPR had it been used rather than ACD-CPR? Our ongoing trial design did not permit a crossover assessment of standard and ACD-CPR techniques.

2. If ACD-CPR does improve coronary and cerebral perfusion pressures over standard CPR, will the improvement increase survival rates or instead produce more prolonged resuscitations and increased intensive care unit admissions without restoration of prearrest level of function? Large-scale clinical trials must examine the

ultimate effect of this intervention on hospital dis-
charge rates and subsequent patient function.

3. Is it inhumane to do CPR without sedation or analgesia?
These agents were used in our case because of the need
to restrain the patient and to facilitate intubation. Are
there other patients who are aware of the resuscitation,
but who cannot express their fears or discomfort?

■ REFERENCES

1. Cummins RO, Ornato JP, Thies W, et al. Improving survival from
cardiac arrest: the "chain of survival" concept. Circulation.
1991;83:1832–47.
2. Del Guercio LRM, Coomaraswamy RP, State D. Cardiac output and
other hemodynamic variables during external cardiac massage in
man. N Engl J Med. 1963;269:1389–404.
3. Koehler RC, Chandra N, Guerci AD, et al. Augmentation of cerebral
perfusion by simultaneous chest compression and lung inflation with
abdominal binding following cardiac arrest in dogs. Circulation.
1983;67:266–75.
4. Cohen TJ, Tucker KJ, Redberg RF, et al. Active compression-
decompression resuscitation: a novel method of cardiopulmonary
resuscitation. Am Heart J. 1992;124:1145–50.
5. Cohen TJ, Tucker KJ, Lurie KG, et al. Active compression-decom-
pression: a new method of cardiopulmonary resuscitation. JAMA
1992;267:2916–23.
6. Callaham M, Schwab T, Schultz JJ, et al. A randomized prospective
trial of active compression-decompression CPR versus manual CPR
in pre-hospital cardiac arrest [abstract]. Ann Emerg Med. 1993;
22:885.
7. Criley JM, Blaufuss AN, Kissel GL. Cough induced cardiac com-
pression. JAMA 1976;236:1246–50.
8. Paradis N, Martin G, Rivers E, et al. Coronary perfusion pressure
and the return of spontaneous circulation in human cardiopulmonary
resuscitation. JAMA. 1990;263:1106–13.
9. Halperin HR, Weisfeldt ML. New approaches to CPR: four hands, a
plunger, or a vest. JAMA 1992;267:2940–1.
10. Lurie KG, Lindo C, Chin J. CPR: the P stands for plumber's helper
[letter]. JAMA. 1990;264:1661.
11. Emergency Cardiac Care Committee and Subcommittees, American
Heart Association. Guidelines for cardiopulmonary resuscitation and
emergency cardiac care. JAMA. 1992;268:2171–302.

Further Thoughts from the Reviewers

■ As noted by the authors, this case report has implica-
tions beyond the issue of whether active compression–
decompression (ACD) cardiopulmonary resuscitation
(CPR) is more effective than standard CPR. The return of
spontaneous purposeful movement during CPR is indeed
uncommon, but several of the reviewers have anecdotally
seen this phenomenon with standard CPR. We suspect that
the paucity of reports in the literature may not reflect its
rarity, but instead may denote its limited novelty. It is likely
that a short interval from cessation of measurable perfusion
to initiation of an effective CPR technique is required for
this phenomenon. We cannot determine from this case

report whether the degree of neurologic response reported
indicates an improvement of cerebral blood flow that was
possible only with ACD CPR or an improvement that
would have been found with standard CPR also.

The details of the patient's resuscitation reflect several
interventions that should be questioned. First, the large
dose of midazolam administered may have reduced vaso-
motor tone and reduced coronary perfusion pressure. Fur-
ther, the administration of magnesium sulfate for asystole
is unsupported by current resuscitation paradigms and may
have had a similar negative vasomotor effect. Third, the
delay in application of transcutaneous cardiac pacing
would have limited the effectiveness of this modality.
However, it is unlikely that these interventions affected the
outcome of this resuscitation, and the case is worthy of our
attention for other reasons.

The cardiac arrest patient who regains neurologic re-
sponse during CPR is uncommon. However, when this
phenomenon does occur, the final outcome can be emo-
tionally disturbing to the rescuer, who expects that such a
neurologic response always will be associated with surviv-
al. In addition to the humane use of sedation and analgesia
during resuscitation of these patients, there may be a role
for alternative therapeutic approaches in select cases.

Was the authors' patient a candidate for immediate
revascularization using thrombolytic therapy? While CPR
is commonly considered a contraindication to thrombolysis
in acute myocardial infarction patients, Merriman and
Kalbfleisch have argued that thrombolysis after CPR can
be beneficial.[1] However, data on the use of thrombolysis
during CPR are sparse. If such therapy is to have a role in
resuscitation, the patient who 1) demonstrates neurologic
function during CPR, 2) is in pulseless electrical activity,
and 3) has preceding electrocardiographic evidence of a
massive infarction appears to be the ideal candidate.

The results of the ongoing multicenter trial will help us
determine whether survival can be improved with ACD
CPR. We also must be attuned to the emotional and
financial costs of such an improvement. Should marginally
improved hemodynamic variables lead more commonly to
prolonged resuscitations without improvements in final
clinical function, we will have done a disservice to our
patients. We eagerly await the results of the authors'
ongoing clinical trial and further reports that address the
frequency of neurologic responses during CPR and wheth-
er these patients might benefit from alternative therapy.

The Authors Respond

■ We appreciate the opportunity to respond to the re-
viewers' questions regarding the resuscitation.

To paralyze and not sedate our patient would have been
inhumane. Midazolam was used for sedation because it has
less myocardial depressant effects than thiopental. Ket-

amine would have been the best choice in hindsight. The dose used was 0.1 mg/kg, the minimum recommended anesthetizing dose for rapid-sequence induction.[2-4] Given the uniqueness of this situation, the proper dosage is uncertain. While the dose used in this setting of hypotension may still have been excessive, the patient's degree of agitation suggested the need for a full induction dose.

The magnesium sulfate used in this case, while not recommended for asystole, was used for the underlying acute myocardial infarction.[5] The hemodynamic effects of magnesium are minimal, and despite its potential to decrease general vasomotor tone, magnesium has been suggested to improve coronary perfusion.[6] Earlier pacing in this patient would have been ideal.[7] We considered early pacing in this patient, but difficulty controlling the arrest scene delayed its use.

We believe this case demonstrates some of the challenging dilemmas faced when resuscitation efforts produce effective cerebral perfusion that cannot be sustained when CPR is halted.

■ REFERENCES

1. Merriman CS, Kalbfleisch ND. Thrombolysis in acute myocardial infarction following prolonged cardiopulmonary resuscitation. Acad Emerg Med. 1994; 1:61–6.
2. Dronen SC. Pharmacologic adjuncts to intubation. In: Roberts JR, Hedges JR, eds. Clinical Procedures in Emergency Medicine. 2nd ed. Philadelphia: W.B. Saunders, 1991: 37.
3. Danzl DF. Advanced airway support. In: Tintinalli JE, Krome RL, Ruiz E, eds. Emergency Medicine. A Comprehensive Study Guide. 3rd ed. New York: McGraw-Hill, 1992: 18–9.
4. Walls RM. Rapid sequence intubation: a primer for physicians in the emergency department. In: Emergency Update 92: Airway Management and Pain Control. Ottawa, Ontario: Canadian Association of Emergency Physicians, 1992: 10–5.
5. Finkelstein JA, O'Keefe KP, Butzin CA. Magnesium in acute myocardial infarction. Ann Emerg Med. 1993; 22:754–6.
6. Yusuf S, Teo K, Woods K. Intravenous magnesium in acute myocardial infarction: an effective, safe, simple and inexpensive intervention. Circulation. 1993; 87:2043–6.
7. Guidelines for cardiopulmonary resuscitation and emergency cardiac care: recommendations of the 1992 conference. JAMA. 1992; 268: 2171–302.

■

Reflections

■ INAPPROPRIATE CONSULTATION

■ Inappropriate consultations have become an increasing problem over the last two decades. Although this pervasive and insidious disorder is common, there has been little discussion of it. Therefore, little is known and less has been published. I hope that this essay will stimulate discussion and dispel myths.

Epidemiology and Pathophysiology

Because this disorder has a seasonal incidence, occurring predominately in the late summer with a sharp peak in July, it has been postulated that an infectious agent may be the cause. Despite an intensive search for various organisms, no evidence for an infectious link has been found. In particular, there is no evidence of a link to retroviruses. Nevertheless, inappropriate consultation does seem to occur in small epidemics among housestaff and fellows on various subspecialty services. There is no explanation for the fact that there is a significant negative correlation between the prevalence of inappropriate consultation and the incomes of both the consulter and the consultant.

Attention has recently shifted away from the realm of infectious diseases toward an endocrine cause. There are several reasons for this. First, there is a definite circadian variation in the incidence of inappropriate consultation, which occurs mainly at night, especially after 10 PM. Clearly the pituitary–adrenal axis is involved in some way. In addition, it has been noted that there is a strong correlation between inappropriate consultation and both the heart rate and the blood pressure of the consultant at the time of initial consultation. Therefore, abnormalities of the sympathetic nervous system have been postulated. It is important to note that there is no evidence to date that this is another presentation of pheochromocytoma. Others have suggested thyroid abnormalities or excessive interleukins as a cause of the syndrome, but no study of these hormones has been reported.

A genetic link has been postulated. This may be one of the few Y-linked chromosomal disorders. Some investigators have noted excessively large Y-chromosomes in afflicted male consultants. The disorder is uncommon but not unheard of in women. Whether or not abnormal adrenal steroid metabolism is involved is not known.

There is a positive correlation between the number of consultations with a particular patient and the prevalence