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Case Report

Cardiac arrest: should we consider norepinephrine instead of epinephrine?

Abstract

A patient scheduled for a laparoscopic cholecystectomy had an anaphylactic shock during induction of anesthesia. After the injection of vecuronium, an unusual fall of arterial pressure occurred, with bradycardia, enlargement of the QRS complex, then a circulatory arrest. Chest compressions were initiated, while intravenous epinephrine 1 mg was administered. The cardiac rhythm turned into a ventricular fibrillation (VF). Despite continuous chest compressions with repeated boluses of epinephrine and several external electric shocks, the patient remained in VF. Because of obviously β-adrenergic adverse effects, epinephrine was replaced with norepinephrine. Return of spontaneous circulation was observed, with the recovering of sinusal activity. After staying for several weeks in intensive care unit because of multiorgan failure, the patient recovered without sequelae. Blood samples and cutaneous testing confirmed an allergy to vecuronium. This case report of a cardiac anaphylaxis with prolonged cardiac arrest illustrates the dual activity and adverse effects of epinephrine. Although vasoconstriction is mandated during cardiopulmonary resuscitation to provide an acceptable perfusion pressure to organs, β-adrenergic stimulation seems deleterious to the heart. Experimental studies have shown that blocking the β-adrenergic effects of epinephrine attenuates postresuscitation myocardial dysfunction or helps the return of spontaneous circulation after VF. Norepinephrine, a potent α-adrenergic drug nearly devoid of β-adrenergic properties, could be an interesting alternative to epinephrine. It can improve organ perfusion during cardiopulmonary resuscitation and could be more efficient than epinephrine in case of VF.

Epinephrine for cardiac arrest could do more harm than good [1]. A case of prolonged cardiac arrest acutely illustrates this point of view.

A 59-year-old man was scheduled for a cholecystectomy. Clinical and echocardiographic examinations were normal. On the morning of the intervention, blood pressure (BP) was 120/80 mm Hg, with a regular pulse of 60 beats per minute. After administration of sufentanil, 30 μg and propofol, 200 mg, BP was 110/65 mm Hg (8:06 AM). After the injection of 8 mg vecuronium, BP fell to 59/34 mm Hg (8:10 AM). The trachea was intubated. Blood pressure continued to fall and was at 44/24 mm Hg despite the injection of titrated ephedrine (24 mg). A bradycardia occurred with enlargement of the QRS complex (8:15 AM). Because of a cyanosis testifying to a circulatory arrest, chest compressions were initiated with intravenous administration of epinephrine, 1 mg. An anaphylactic shock was suspected. Despite the cardiopulmonary resuscitation, the capnia collapsed (Figure), and the cardiac rhythm turned into a ventricular fibrillation (VF). Continuous chest compressions were maintained with the administration of incremented doses of 1, then 3 mg intravenous epinephrine. Despite several external electric shocks, repeated boluses of epinephrine, and uninterrupted cardiopulmonary resuscitation (CPR), the patient remained in VF.

At 8:45 AM, cardiac activity recovered with a supraventricular tachycardia (180 beats per minute) but with a rapid return to VF. Epinephrine (total dose, 45 mg) was replaced with norepinephrine. Return of spontaneous circulation was observed, with the recovering of sinusal activity. The patient stayed for 6 weeks in intensive care unit because of multiple organ failure. He eventually recovered without sequelae. Blood samples (specific immunoglobulin E) confirmed an allergy to vecuronium.

This case of an anaphylaxis with prolonged cardiac arrest illustrates the adverse effects of epinephrine. Although vasoconstriction is mandated during CPR to provide an acceptable perfusion pressure [2], β-adrenergic stimulation seems deleterious and probably responsible for the VF refractory to multiple electric shocks. Indeed, the use of vasopressin as a systemic vasopressor instead of epinephrine carried better results in a piglet model of cardiac arrest [3]: cardiac function and survival were greater in the vasopressin group. However, this was not confirmed in humans [4].

Moreover, an experimental study showed that blocking the β-adrenergic effects of epinephrine attenuates postresuscitation myocardial dysfunction [5]. Accordingly, esmolol helps the return of spontaneous circulation after VF [6]. Although epinephrine use for cardiac arrest is the subject of controversial results [7-9], norepinephrine, a potent α-adrenergic drug nearly devoid of β-adrenergic properties, could be an alternative. It has been experimentally proven that it can improve organ perfusion during CPR [10] and that it could be more efficient than epinephrine in case of VF [11]. Yet, human data are too scarce to identify a clinical benefit of norepinephrine in cardiac arrest [12].

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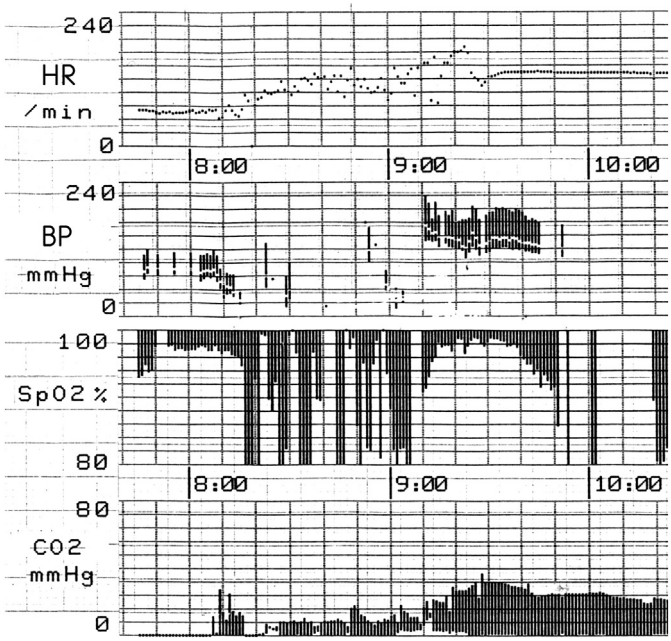

Figure. Record of heart rate, arterial pressure, pulse oxymetry, and capnometry of the patient during CPR in operating room. Cardiac arrest lasted from 8:15 to 9:10 AM.

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