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Peri-anaesthetic cardiac arrest with administration of enalapril, spironolactone and β -blocker

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Although cardiac arrest during anaesthesia is rare, it may be of grave concern for the patient. The mechanism is a multifactor event, including personal treatment. Antihypertensive and diuretic drugs can induce dyskalaemia and heart rhythm problems *per se* while others, such as β -blockers, can controversially generate pulseless electrical activity. We report the occurrence of unanticipated peri-anaesthetic cardiac arrest where the use of such drugs need to be debated.

A 77-year-old male (ASA 3, NYHA 2) was scheduled for ambulatory laparoscopic hernia repair. He had a medical history of myocardial ischaemia, hypertension and stable aortic valvulopathy, as well as renal insufficiency (glomerular filtration rate: 33 mL min⁻¹). He was considered as stable and asymptomatic by his cardiologist. He had received daily acetylsalicylate acid 75 mg, bisoprolol 10 mg, lercandipine 10 mg, spironolactone 50 mg and enalapril 20 mg. The coagulation parameters and blood group were checked. Laparoscopic surgery was planned under general anaesthesia (remifentanyl and sevoflurane). Routine monitoring included heart rate, ECG, noninvasive blood pressure and pulse oximetry, baseline parameters were normal. The surgical procedure lasted around 45 minutes: gas insufflation and exsufflation were uneventful. Suddenly, his heart rate dropped from 65 beats per min to asystole while SpO₂ fell to 72% and capnometry to 20 mmHg while noninvasive blood pressure (BP) became unmeasurable and peripheral pulses could not be palpated. Cardiopulmonary resuscita-

tion was started immediately, including two repeated intravenous doses of 1 mg epinephrine and 100% oxygen while sevoflurane was suppressed. Within 2 minutes, the patient responded with a heart rate of 160 bpm, SpO₂ 92% and BP of 90/60 mmHg. The patient was admitted to PACU where the ECG became progressively sinusoidal at around 105 per min. Echocardiography (CB) eliminated a gaseous or pulmonary embolism by the absence of acute right ventricular heart failure. A moderate aortic stenosis was discovered with an aortic valve area of around 0.6 cm², a pressure gradient at 25 mm Hg and a left ventricular ejection fraction estimated at around 60%. No aspect of myocardial infarction or severe pericarditis was observed. BNP — was at 770 ng mL⁻¹, troponins around 30 μ g mL⁻¹, pH — 7.14, pCO₂ — 32 mm Hg, pO₂ — 153 mm Hg, while electrolytes were in a normal range, apart from potassium at 6.29 mEq L⁻¹. A chest X-ray showed no abnormality. The patient was admitted to the intensive care unit to undergo haemodialysis. He was extubated on day 1 with a pH of 7.44 and a potassium level of 4.39 mEq L⁻¹. The patient was discharged on day 4 and gave his informed consent to publish this case. Spironolactone was substituted with furosemide.

Obviously, the role of hyperkalaemia has to be discussed. Most hyperkalaemia-induced cardiac arrests may be observed in patients with neuromuscular dystrophy, blood transfusion or iatrogenic infusion [1, 2]. A combination of spironolactone with an ACE inhibitor may cause severe hyperkalaemia [3]. Abbas *et al.* [4], using health insurance claims data and performing a nested case-control study in a cohort of patients receiving ACE inhibitor/angiotensin receptor blocker therapy, showed that hyperkalaemia associated risk when combined with spironolactone is much greater in real-life practice than observed in clinical trials. The risk of hyperkalaemia in heart failure patients was strongly associated with spironolactone use (odds ratio

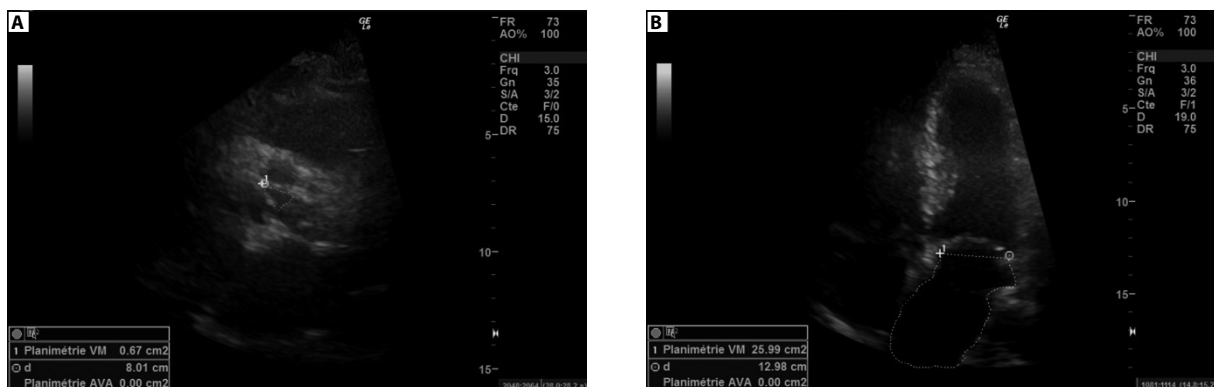


Figure 1. Cardioechography with absence of any gaseous or pulmonary embolism with absence of acute right ventricular heart failure

(OR 95% confidence interval (CI)) 13.59 (11.63–15.88). The association was stronger in those older than 70 years (OR (95%CI) 12.32 (9.35–16.23). Although the occurrence of hyperkalaemia with spironolactone is influenced by the dose, when used concomitantly with enalapril, losartan or candesartan, it may increase even if the dose of spironolactone is as low as 25 mg [5]. The EKG conduction parameters were normal in our patient and did not support the role of hyperkalaemia being associated with the cause of cardiac arrest. The role of some concomitant anomalies and/or drugs, such as sevoflurane slowing cardiac conduction, laparoscopic insufflation inducing acidosis, and trans-membrane potassium transfer should be discussed.

Though in our case, the initial cause of cardiac arrest was probably a conduction block, the β -blockers which can lead to such trouble should be discussed and the immediate efficacy of epinephrine to resolve the asystole supports this. β -blockers are the fourth most-commonly prescribed medication for hypertension, and about 60% of post-myocardial infarction patients are discharged on β -blockers. Both β -blocker use and the number of patients presenting pulseless electrical activity in cardiac arrest have increased over the past 20 years [6]. In contrast with patients having ventricular fibrillation-induced cardiac arrest who can often be shocked back into a normal rhythm, it may be very difficult to treat someone with pulseless electrical activity. Furthermore, β -blockade may thwart epinephrine while glucagon typically used to reverse β -blocker overdose may be useful [6]. During resuscitation, a speedy echocardiography is of serious importance to explore the causes of cardiac arrest and help the anaesthesiologist to eliminate other medical etiologies and manage emergency treatment [7] Knowledge of this case may prompt careful potassium level monitoring in concomitant users of spironolactone

and ACE inhibitor or angiotensin receptor blocker. According to French guidelines relating to routine preinterventional tests in a medium level procedure, electrolytes were not checked [8]. Considering our patient, this should have been considered in the preoperative period. In the same way, a combination of such comorbidities and treatments should be debated in an ambulatory process.

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