leak), only 35% showed improvement over 24 h, which was significantly different ($P < 0.01$, Wilcoxon rank sum test) from the treatment group.

Cancer (22 patients), primary sepsis (15 patients), multiple trauma (11 patients), intracranial bleeding (11 patients), subarachnoid haemorrhage (11 patients) and aortic aneurysm rupture/repair (11 patients) were among the more common admission diagnosis in patients with adrenal and relative adrenal insufficiency. Patients with a history of hypertension, coronary artery disease, diabetes mellitus, chronic obstructive pulmonary disease and/or vascular disease seem to be at higher risk for these conditions although our study size was too small for logistic regression analysis. Etomidate use for intubation could be identified in only one patient.

Overall, our findings support the notion that adrenal insufficiency diagnosed with a 1-μg cosyntropin test is common in surgical patients with volume refractory shock and responsive to hydrocortisone substitution independent of confirmed sepsis.

Larger scale epidemiologic and prospective studies are warranted to determine whether patients should be routinely tested with low-dose or high-dose cosyntropin tests or empirically treated with hydrocortisone and classified as clinical responders or non-responders after 24 h.

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Fatal outcome during anaesthesia induction in a patient with amiodarone-induced thyrotoxicosis

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EDITOR:
Amiodarone is a class III anti-arrhythmic drug which has been used in Europe since 1967 to treat angina and refractory arrhythmias of supraventricular, junctional and ventricular origin [1]. It is an iodinated benzofuran derivative with a molecular structure that is similar to thyroxine and tri-iodothyronine [2]. It has a high iodine content (37% per weight). A significant amount of iodine is liberated during drug metabolism [3]. The large iodine load has been implicated as the main factor in the pathogenesis of amiodarone-induced thyroid

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dysfunction, such as amiodarone-induced thyrotoxicosis [2,3]. We report a case of amiodarone-induced thyrotoxicosis after a single dose of amiodarone for the treatment of atrial fibrillation and the impact on induction of anaesthesia for thyroidectomy.

A 53-yr-old female with a medical history of a dilatated cardiomyopathy was admitted to the medical ICU with congestive heart failure and first time occurrence of atrial fibrillation. The patient had hyperthyroidism (free thyroxine: $4.1\,ng\,dL^{-1}$, thyroid-stimulating hormone $<0.01\,mU\,L^{-1}$). She had been treated with potassium perchlorate and thiamazol for 5 days before the actual hospital admission. After thrombus exclusion by echocardiography, the patient was electrically cardioverted but sinus rhythm could not be established. A single dose of 600 mg amiodarone intravenous (i.v.) was administered, again followed by electrical cardioversion. Sinus rhythm was achieved and the patient rapidly recovered from congestive heart failure. However, 1 day later thyrotoxicosis with tachyarrhythmia, haemodynamic instability, sweating and severe neuromuscular weakness developed. Despite additional treatment with carbimazole and corticosteroids, the symptoms of thyrotoxicosis worsened. Finally, the patient was scheduled for thyroidectomy 13 days after the development of amiodarone-induced thyrotoxicosis. On arrival in the operating room, the patient had a heart rate of about 140 beats per minute with atrial fibrillation and a bifascicular block. An arterial line was placed in the left radial artery. Mean arterial pressure was 80 mmHg. Oxygen was applied by a mask until oxygen saturation reached 100%. Induction of anaesthesia was with sufentanil $0.3\,\mu g\,kg^{-1}$, etomidate $0.25\,mg\,kg^{-1}$ and rocuronium bromide $0.6\,mg\,kg^{-1}$. Immediately after induction, mean arterial pressure decreased to 40 mmHg and norepinephrine infusion was started $(0.1\,\mu g\,kg^{-1}\,min^{-1})$. The patient was intubated and ventilated with 100% oxygen. At this time, ventricular tachycardia developed and this rapidly progressed to ventricular fibrillation. Resuscitation in accordance with The International Liaison Committee on Resuscitation (ILCOR) guidelines was initiated. Transoesophageal echocardiography did not reveal any treatable cause, e.g. thrombus formation or pericardial fluid. The unsuccessful resuscitation attempts were ceased after 90 min.

Amiodarone-induced thyrotoxicosis is a life-threatening disease with a mortality of up to 50% in patients who are refractory to medical treatment [4]. However, mortality is reduced to 0–13% when operative thyroidectomy is performed [1,4]. Here, we report the fatal outcome of a patient with amiodarone-induced thyrotoxicosis refractory to medical, mechanical and electrical treatment during induction of anaesthesia for thyroidectomy.

In previous reports, amiodarone-induced thyrotoxicosis developed in patients with long-term oral amiodarone treatment [2,3,5,6]. In our case, a single dose of amiodarone 600 mg i.v. was administered. The patient had a goitre and manifest hyperthyroidism at the time of amiodarone administration. The high iodine load of the amiodarone molecule was responsible for excessive thyroid hormone synthesis. Therefore, excessive hormonal synthesis was blocked by thionamides. Additionally, potassium chlorate $(KClO_4)$ was administered for the depletion of intrathyroidal iodine stores. Finally, i.v. glucocorticoid therapy was initiated to treat a possible amiodarone- or iodine-induced destructive thyroiditis [5]. The pharmacological properties of amiodarone make the medical management of this situation difficult. Amiodarone accumulates in the thyroid gland and produces a large increase in iodine stores. It also has high lipid solubility with an elimination half-life of up to 107 days. [2]. When medical treatment is not successful, plasmapheresis and peritoneal dialysis have been applied for transient improvement [1]. These patients have been successfully treated with thyroidectomy [1–4], but patients with manifest hyperthyroidism have a high anaesthesia-related risk for morbidity and mortality due to haemodynamic instability and cardiac arrhythmias.

There is no specific recommendation for the management of anaesthesia induction in these patients. In one report, drugs similar to those in our case were used with success [1]. Thyroidectomy could have alternatively been performed after the infiltration of local anaesthetic. However, surgical management is more complex under these circumstances and endogenous catecholamine release could have contributed to a further deterioration of haemodynamic dysfunction and tachyarrhythmia. Our patient had refused surgery with local infiltration. The use of dantrolene has been reported to stabilize the haemodynamics in a patient with thyroid crisis. Dantrolene might inhibit the effect of circulating thyroid hormones on calcium flux across the sarcoplasmatic reticulum [5]. This treatment is experimental and therefore dantrolene was not administered in our case.

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Thoracic epidural anaesthesia in valvular cardiac surgery

EDITOR:

The use of thoracic epidural anaesthesia (TEA) in patients undergoing cardiac surgery, although increasing in popularity, remains controversial [1,2]. Moreover, while relatively large series of patients undergoing coronary revascularization under TEA have been studied, a small number of cases has been reported in which TEA has been used in valvular cardiac surgery [3]. We report the successful use of TEA in a challenging case of mitral surgery, in a patient with severe co-morbidities and allergies contra-indicating the administration of non-steroidal anti-inflammatory drugs (NSAIDs) and opioids. The patient was previously rejected by other cardiosurgical centres.

A 49-yr-old (157 cm, 59 kg) female with severe mitral regurgitation was scheduled for valve replacement. Co-morbidities included hypertension, a history of congestive heart failure and transient acute renal failure, pectus excavatum with severe respiratory deficit, multiple allergies (including various antibiotics, paracetamol and NSAIDs), previous transfusion reaction, glucose-6-phosphate dehydrogenase (G-6-PD) deficiency, post-thyroidectomy hypothyroidism and hypoparathyroidism. The patient was receiving amlodipine, clonidine, spironolactone, budesonide, calcium fumarate, salbutamol, beclomethasone, oxygen, l-thyroxine and calcium carbonate. She complained of fatigue and dyspnoea on minimum exertion and was orthopnoeic, with reduced chest expansion, reduced dipharagmatic mobility and abnormal sounds in the inferior pulmonary fields. Electrocardiography showed sinus

rhythm and signs of left ventricular hypertrophy. Chest X-ray was consistent with the patient's cardiac and pulmonary diagnosis. Transthoracic echocardiography revealed moderate-to-severe mitral regurgitation caused by functional tethering and fibrosis of the posterior leaflet, moderate concentric left ventricular hypertrophy, hypokinetic cardiopathy with no geometric remodelling and left ventricular ejection fraction of 40%. It also showed increased pulmonary artery pressure (50 mmHg systolic), with moderate tricuspid regurgitation and mild right ventricular failure. No preoperative transoesophageal examination was performed because the patient did not tolerate the procedure. Spirometry confirmed the severe obstructive–restrictive pattern with forced expiratory volume in 1 s (FEV_1) 31% and forced vital capacity (FVC) 43% of predicted. Partial pressure of O_2 (PaO_2) and CO_2 ($PaCO_2$) were 69 and 40 mmHg, respectively, with saturation of O_2 (SaO_2) 92% at arterial blood gases analysis on room air. Other biochemical and haematological parameters were within normal limits.

In view of her preoperative status, it was believed that the patient would benefit from TEA. The risks and benefits were explained to her and she gave written informed consent. Epidural catheterization was performed on the day prior to surgery. Her pre-operative platelet count (390×10^9 mL^{-1}) and coagulation (international normalized ratio (INR) 0.87, partial thromboplastin time (PTT) ratio 0.87) were within the normal range. Catheter placement was accomplished by an experienced anaesthesiologist in a high-dependency area. Monitoring included electrocardiogram, non-invasive blood pressure and pulse oximetry. The patient was positioned sitting upright and an 18-G epidural catheter was inserted at T5–T6, using the loss-of-resistance technique and a midline approach, in a single attempt. The catheter was advanced 6 cm cephalic

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