LETTERS TO THE EDITOR

Stokes-Adams syndrome caused by a faulty secondary pacemaker

Sir,

Syncope of cardiac origin is usually of sudden onset and short duration. Recovery is generally rapid. Syncope in Stokes-Adams syndrome Adams is sufficiently long to display signs of cerebral anoxia (seizures, sphincter incontinence, cyanosis etc.)[1]. Among its causes are AV block (AVB) in 50-60% of cases, sinoatrial block (30-40%) and tachyarrhythmia (0-5%)[2].

We report the case of an 84-year-old man with a pacemaker since 1980, attended at our emergency department for seizure. According to family report, he had experienced several convulsive episodes, without tongue biting or sphincter incontinence, and had shown complete recovery after each episode. Physical examination showed jugular vein distension, with cardiac arrhythmia and lower lung rales on auscultation, and slight perimaleolar edema in both legs. Blood test was normal. Plain chest X-ray showed discrete cardiomegaly and a single-chamber pacemaker. While being monitored, the patient experienced sudden dizziness followed by tonic-clonic seizure of short duration, with spontaneous recovery. We obtained an electrocardiogram recording (ECG) of more than 15 seconds duration which showed a pacemaker spike without subsequent QRS complex. With the diagnosis of complete AVB and pacemaker malfunction, tracheal intubation and connection to an external pacemaker was performed after sedation and relaxation. He was referred to a reference hospital for definitive treatment.

Rapid-onset short-duration syncope in a patient with heart disease or pathologic EGG should suggest arrhythmia a a cause[1]. Correct medical history is of great importance in differentiating convulsive syncope from epilepsy[3]. The ECG only allows causal detection of syncope in 2-10% of cases[1], but in patients with pacemakers we consider that device malfunction must be ruled out. In this case, partial disconnection of the stimulating electrode was found to be responsible for the clinical picture. In our review of the literature we found case reports of ECG-confirmed Stokes-Adams syndrome secondary to AVB, but none related with pacemaker malfunction.

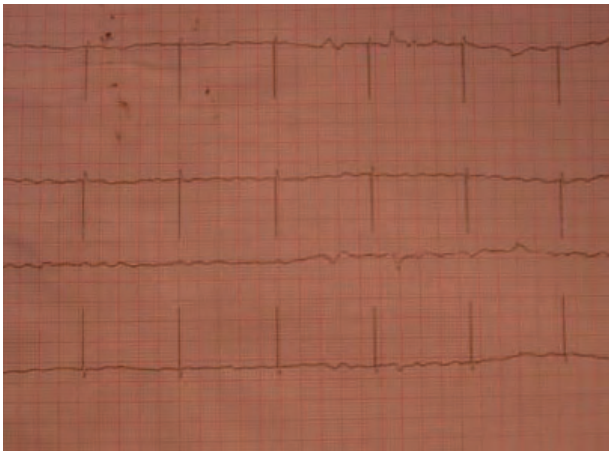

Figure 1. ECG showing pacemaker spike with no subsequent QRS complex.

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Acute confusional state secondary to tumor-associated hypercalcemia

Sir,

The main metabolic complications observed in cancer patients are hypercalcemia, hyponatremia (inappropriate secretion of ADH), tumor lysis syndrome, lactic acidosis, hyperuricemia, renal failure, hyperammonemia, hypoglycemia and potassium alterations[1]. Hypercalcemia is the most common metabolic disorder[2], found in 10-30% of patients with cancer[3]. Despite its high prevalence, it is often undiagnosed because of the non-specific clinical presentation. Encephalopathy of different degrees is the most common and severe symptom[4], thus preventing adequate treatment that would lead to clinical improvement[5,6].

We report the case of a 62-year-old man presenting general deterioration of one week's evolution with fever up to 39ºC, malaise, psychomotor retardation, disorientation and sphincter incontinence. Medical history included paroxysmal atrial fibrillation, chronic ischemic heart disease, residual pulmonary tuberculosis, a possible hepatic hemangioma and chronic moderate alcohol abuse. Physical examination showed blood pressure 156/91 mmHg, heart rates of 85 bpm, 95% oxygen saturation and body temperature 37.5ºC. General condition was poor, with dry skin and mucous membranes, acceptable level of consciousness with a tendency to somnolence, bradypsychia with coherent speech and normal cranial nerves. The remainder of the neurological examination was normal. Cardiopulmonary auscultation and abdominal examination showed no abnormalities. Laboratory findings were: 14,100 WBC with 78% neutrophils, urea 77 mg/dl, creatinine 2.2 mg/dL, normal sodium and potassium, lactate 1.4 mmol/L, pH 7.46 and normal urine sediment. Toxins in urine were negative. Chest X-ray: pachypleuritis in upper right lobe. The electrocardiogram showed a sinus rhythm without atrioventricular conduction disturbances or repolarization. Computed tomography (CT) scan was normal. A subsequently performed lumbar puncture showed clear fluid without cell biochemistry. Abdominal ultrasound showed images suggestive of liver metastasis. With the diagnosis of acute confusional state and suspected liver metastasis, he was admitted to the Department of Internal Medicine. Once there, biochemical analysis showed: calcium 17.3, phosphorus 4.5 mg/dL, AST 35 U/L, ALT 73 U/L, FA 86 U/L, GGT 82 U/L, LDH 550 U/L, total protein 6.4 g/L and CRP 122 mg/dl. Chest-abdominal CT scan (Figure 1) showed a tumor lesion in the upper right lobe with hilar lymphadenopathy, and liver images compatible with metastasis. Hypercalcemia was diagnosed and severe tumoral therapy was started with intravenous fluids, furosemide, corticosteroids and bisphosphonates, which steadily improved the patient's level of consciousness. Finally, bronchoscopy and cytology confirmed the diagnosis of poorly differentiated squamous cell lung cancer.

The differential diagnosis of hypercalcemia must be performed considering primary hyperparathyroidism (54%), tumors [lung (35%), breast (25%), myeloma and lymphoma (14%), kidney (3%), prostate gland (3%)], and others such as hypervitaminosis D and A, immobilization, osteoporosis, Paget's disease, hyperthyroidism, tuberculosis, sarcoidosis, pheochromocytoma, Addison's disease, HIV, milk-alkali syndrome, lithium, thiazides, chronic renal failure and the diuretic phase of acute renal failure, and familial hypocalciuric hypercalcemia (20%)[3].

Tumor hypercalcemia is often of rapid onset and can cause lethargy, mental confusion, anorexia, nausea, vomiting, constipation, polyuria and polydipsia. Some complications may be added as a result of persistence of the hypercalcemia, such as dehydration, nephrolithiasis, nephrocalcinosis, renal failure, hypertension, cardiac arrhythmias, peptic ulcer, pancreatitis and coma[7].

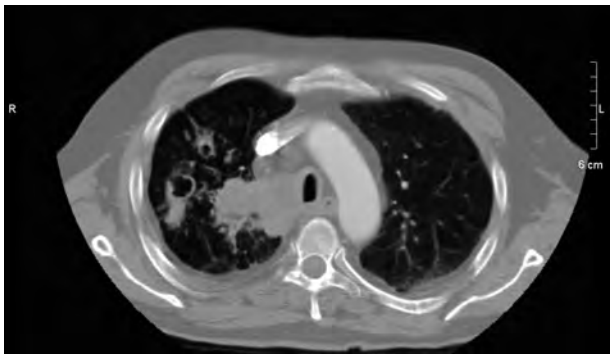

Figure 1. Chest computed tomography showing a cavitary lesion in the right upper lobe and right parahiliar adenopathies.

Treatment of acute hypercalcaemia involves rehydration with isotonic saline, depending on the patient's cardiovascular status and renal function; loop diuretics (furosemide), apart from controlling excess volume after rehydration, increase the renal elimination of calcium, bisphosphonates (etidronate, alendronate, clodronate,pamidronate or zoledronate) adhering to the surface of hydroxyapatite and inhibit the re-absorption of calcium by osteoclasts, and they have some analgesic effect and may prevent vertebral fractures; calcitonin inhibits osteoclast resorption and promotes renal excretion of calcium; corticosteroids (hydrocortisone, prednisone) to inhibit bone resorption and decrease gastrointestinal re-absorption. General measures include: removal of nutritional calcium and any drugs that can cause hypercalcemia, increased mobility of the patient and, if possible, withdrawal of sedatives and analgesics in order to improve the mental confusion. In any case, the most effective treatment is tumor treatment with surgery or radio/chemotherapy, although in some cases the hypercalcemia may persist despite removal of the tumor[1,5,8,9]. Prognosis is usually poor, and 50% of patients die within a month and 75% within three months after starting treatment. The prognosis is slightly better in those patients that respond to specific cancer therapy[3].

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Cardiorespiratory arrest due to hyperkalemia

Sir,

Hyperpotasemia[1], defined as serum potassium concentration greater than 5.5 mEq/l, obliges us to consider the possibility of oliguric renal failure, severe haemolysis or excessive tissue catabolism. Cardiac manifestations are the most serious. When levels exceed 7.5 mEq/l[2], wide QRS ECG patterns may appear, with flattening or disappearance of the P wave, which may trigger ventricular fibrillation, atrioventricular block (AVB) or asystole.

We report the case of a 66-year-old man attended by the Emergencies Health Sector 112 ambulance team for syncope with spontaneous recovery. Our medicalized Emergency Unit registered a clinical picture compatible with acute pulmonary edema with signs of poor peripheral perfusion. The patient received oxygen therapy, monitoring, peripheral venous canalization and 60 mg of furosemide iv. During the transfer, wide QRS complexes without P waves were recorded (Figure 1), followed by asystole coinciding with two episodes of cardiopulmonary arrest (CPA) which resolved after about 20 seconds with basic life support. Once in the hospital ED, the patient presented complete AVB, and two new episodes of PCA lasting a few seconds which responded to basic life support. He then received an external pacemaker before referral to the intensive care unit where he subsequently required endotraqueal intubation and mechanical ventilation for respiratory distress. The medical history included chronic obstructive pulmonary disease, diabetes, hypertension, recurrent low-grade papillary urothelial bladder cancer under palliative treatment and infiltrating left hydronephrosis. Additional tests showed: glucose 662 mg/dL, creatinine 2.4 mg/dL, sodium 124.5 mEq/L, potassium 7.75 mEq/L and chloride 88.5 mEq/L. The pH was 7.09. The temporary pacemaker was placed via the femoral artery and haemodialysis was performed for persistent anuria. After this, potassium values normalized and the patient recovered sinus rhythm at 90 bpm. The patient evolved favourably.

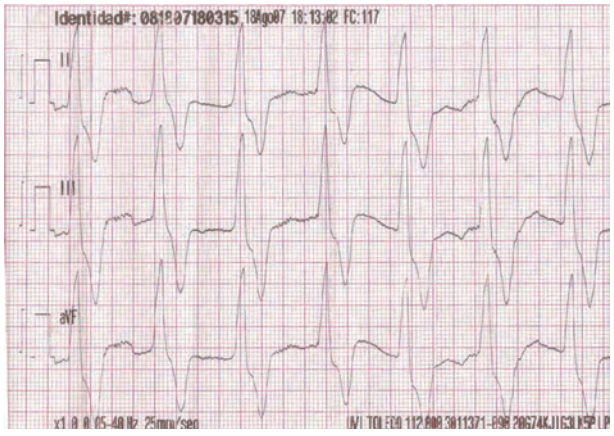

Figure 1. ECG showing disappearance of sinusal rhythm and widened QRS complex, coinciding with a plasma K value of 7.7 mEg/t.

In conclusion, therefore, the CPA suffered by this patient was secondary to hyperkalemia in the context of obstructive acute renal failure and the underlying pathology.

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Hemorrhagic shock secondary to aortoenteric fistula

Sir,

We present the case of a 75-year-old German man with a history of hypertension and an aortoaortic bypass for abdominal aortic aneurysm in 1989. He was previously admitted to another centre for melena of 24 hours evolution. He reported a 15-day history of fever with chills and continuous low back pain treated with nonsteroidal anti-inflammatory drugs without clinical improvement. On examination he presented mean blood pressure of 70 mmHg and heart rate of 95 bpm. Laboratory tests showed: haemoglobin (Hb) 7.1 g/L, haematocrit (HCT) 22.3 L/L, platelets 120 x 109/L, prothrombin (PT) 96.6% and activated partial thrombo-

plastin time (APTT) 32 sec. After transfusion with three packs of red blood cells, he was transferred to our centre for urgent upper fibrogastroscopy (FGS) for suspected upper gastrointestinal bleeding (UGIB). In the emergency department, he was haemodynamically stable but low back pain persisted despite the administration of morphine. Post-transfusion laboratory tests showed: Hb 8.2 g/L, Hct 23.5 L/L, leukocytes 2.0 x 10^9/L, TP 89%, APTT 29 sec and platelets 101 x 10^9/L. He was admitted to the intensive care unit. FGS showed some remnants of blood clots in the stomach and duodenum. Abdominal CT scan (CT) showed aortoenteric fistula in the duodenum. The patient suddenly entered a state of haemodynamic shock with abundant bleeding through the nasogastric tube and required intensive volume resuscitation, haemoderivatives and vasoactive drugs. Surgery was rejected by the attending team on duty due to the precarious clinical condition of the patient.

Aortoenteric fistula is an uncommon cause of UGIB, less than 0.6-2.3% of cases[1]. Its fundamental aetiology has not been described but different possibilities have been postulated: infection of the prosthesis (primarily S. epidermidis[2]) or of mechanical origin by pulsatility due to the lack of interposing retroperitoneal tissue. Clinic symptoms are usually unremarkable, characterized by occasional previous episodes of GI bleeding without clinical impact until the final event and/or signs of sepsis without a focus of infection. A high degree of clinical suspicion is therefore required for prompt intervention given the high mortality rates[1,2]. The diagnostic technique of choice is abdominal CT with high sensitivity (85.7%). The only treatment is surgical, with low survival in patients with hemodynamic instability, although the use of stents before surgery is gaining ground[3,4].

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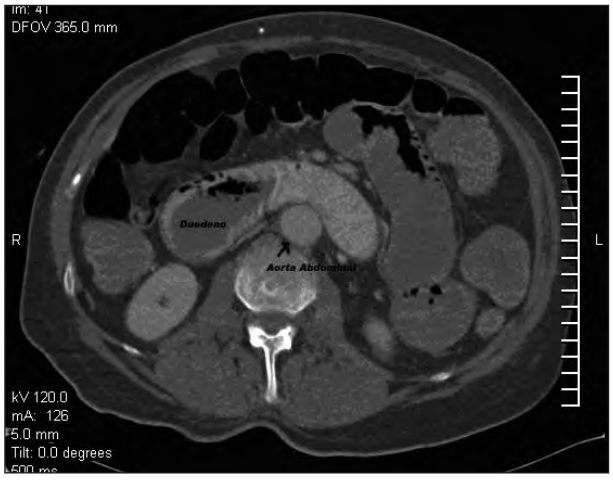

Figure 1. Abdominal CT scan performed 10 minutes after intravenous contrast administration.

Headache and convulsions during delivery an postpartum recovery

Sir,

Iatrogenic pneumocephalus is a relatively rare complication that may occur after inadvertent dural puncture during epidural or combined (spinal - epidural) anaesthesia[1-3].

We report the case of a 19-year-old woman with a history of congenital brachial palsy, smoking (20 packs/year) and bronchial asthma, with 39 weeks of pregnancy. She was attended at a private maternity centre for prodromal labour, receiving epidural anaesthetic with loss of resistance (LOR) technique]. During insertion of the spinal catheter, the patient reported intense holocraneal headache of sudden onset. The baby was born by vaginal eutocic delivery. After two hours, the woman presented generalized seizures, three times in a period of 90 min, with recovery of consciousness between seizures. She was treated with diazepam and phenytoin, and then referred to the emergency department for evaluation. Physical examination showed: blood pressure (BP) 157/94 mmHg, heart rate of 120 bpm, bradypsychia and disorientation without neurologic focality. Laboratory tests including blood count, biochemistry, urine sedimentation, toxin and coagulation studies, were normal. Computed tomography (CT) scan (Figure 1) showed an air density image in the right frontal region and other smaller ones, all intraparenchymal, located posterior to the splenium of the corpus callosum. We also observed a small air bubble in the occipital midline, superimposed on the sagittal sinus. The condition was therefore diagnosed as pneumocephalus secondary to iatrogenic spinal anaesthesia. Evolution was favourable, with no new convulsive episodes, and the headache had disappeared after 72 hours. The electroencephalogram showed no specific

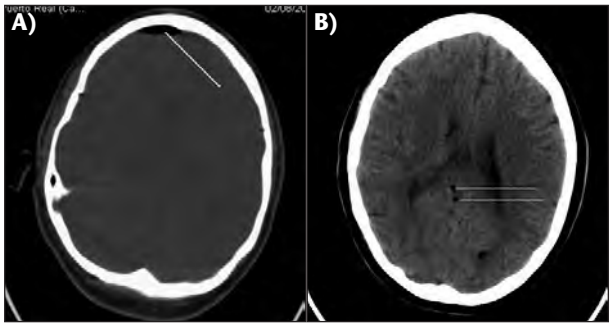

Figure 1. CT scan showing an air density image in the right frontal region (A, arrow) and other smaller ones, all intraparenchymal, located posterior to the splenium of the corpus callosum (B, arrows).

abnormalities. Follow up brain CT scan one month later found complete resolution of the pneumocephalus.

In epidural anaesthesia, the LOR technique is used to confirm catheter location in the epidural space by administering air, saline solution or both[4]. When the needle passes through the yellow ligament, pressure is reduced and the content of the syringe, air or saline solution, flows freely. There is no consensus on the best LOR technique[4]. The incidence of pneumocephalus and other complications is increased when more than 3 ml of air is injected or repeated attempts are made in complicated blockade[4]. The clinical manifestations are non-specific and correspond to a syndrome of variable degrees of intracranial hypertension[5], which may include altered consciousness, headache, vomiting, diplopia, and neurologic focality and haemodynamic instability. In the differential diagnosis, the following should be considered eclampsia, venous sinus thrombosis, reversible posterior leukoencephalopathy, vasculitis, infectious disease and demyelinising diseases. Intense fronto-occipital headache is the most frequent symptom, characteristically of sudden onset during or immediately after the procedure. Exceptionally, as in this case, seizures represent the main manifestation, although more often deferred in the other reported cases[6]. The prognosis is favourable, with spontaneous re-absorption of the air and symptom remission within 48-72 h. Supine rest, hydration and analgesics are recommended. In refractory cases, the use of high-concentration oxygen seems useful[3]. Decompression treatment is only recommended in exceptional cases, reserved for tension pneumocephalus6. In conclusion, the possibility of iatrogenic pneumocephalus should be considered in the presence of sudden headache and other neurological manifestations in direct relation with epidural anaesthesia.

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Unusual anomalous positioning of a central venous catheter

Sir,

Central venous catheters can be inserted using a central or brachial approach (antecubital crease). Incorrect positioning may be due to anatomic vascular variables. We describe the case of a patient with abnormal positioning after left basilic vein canalization.

This was a 64-year-old male patient diagnosed with septic shock of probable pulmonary origin, where anteroposterior chest X-ray showed a catheter entering the chest through the left subclavian vein and abruptly changing direction caudally along the left sternal edge (Figure 1). To discard possible arterial placement, central venous and arterial blood gases pressure were monitored through the catheter, which proved to be compatible with vein positioning. Echocardiogram findings showed no significant alteration. Lateral chest X-ray did not show catheter positioning in the posterior mediastinum. No further complementary tests could be performed before the patient died.

Incorrect catheter placement depends on the site of insertion, technique, patient positioning and anatomical peculiarities of the veins used[1]. The recommended site for catheter tip insertion is in the lower third of the cava superior vein. Incorrect locations described are the caval, liver,axillary, azygos, cervical plexus, hemiazygos, pericardiacophrenic, left internal thoracic, intercostal veins and the superior vena cava, with a frequency of 0.3 to 5.7%[2]. Chest X-ray is one of the most commonly used methods to detect catheter positioning. Transesophageal echocardiography allows visualization of the right atrial and the superior vena cava.

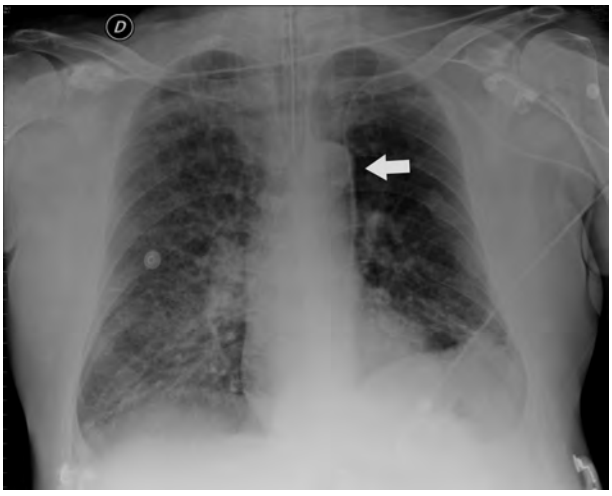

Figure 1. Anteroposterior X-ray. Catheter positioned at the left paravertebral level, reaching the retrocardiac area (arrow).

In our case, the anatomic variants could have been access through the left superior vena cava (it crosses to the right because it follows the anomalous cava vein to where it opens into the coronary sinus)[3], advancing the catheter along the left subclavian vein to the superior intercostal and left hemiazygos veins or through the left internal mammary vein. Here, the latter two are possible. With the mammary vein, a plain lateral X-ray would show the catheter situated in front, while for the intercostal vein, it would be situated behind. In the event of duplication of the vena cava, trans-thoracic echography can allow diagnosis of up to 70% of these defects[3,4]. The use of contrast in the left superior vena cava allows direct viewing, entering the right atrium through the coronary sinus, which may also be viewed on computed tomography or magnetic resonance imaging[5].

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ERRATA

On page 275 of the journal EMERGENCIAS, In volume 21, Issue Nº 4, August 2009, in the article "Advanced life support units in Spain 2008: current situation", the authors listed in the abstract are incorrect The correct authors are: E.E. Pesqueira Alonso, P. Juliani Izquierdo.