We agree with Drs. La Regina and del Prato that problems in medicine can be approached from a variety of perspectives, although some basic directions should be shared by physicians working in different fields. There is no doubt that arterial hypertension is today a diagnosis that rarely leads to hospitalisation. There are however occasions characterised by a high risk for the patient, when this will be necessary, as in cases of pulmonary oedema or transient ischaemic cerebral attack. Concerning the patients without life-threatening hypertensive crises, the current Guidelines for the management of high blood pressure provide clear indications, suggesting the need for repeated visits to confirm the presence and the severity of an elevation of blood pressure, and to assess the global risk profile of the patient and the presence of target organ damage. In most cases there is no need to hasten the start of antihypertensive treatment until the diagnostic process has been completed. The time interval before starting treatment is obviously a function of the patient’s risk level. An appropriate diagnostic work-up should also lead to identification of cases with secondary hypertension. Primary Care Physicians (PCP) have the duty to start management of patients with hypertension, in cooperation with specialised centres that provide all the necessary support for definition of the patient’s risk profile and organ damage. This approach, accompanied by a good relationship between the patient and the physician, will also reduce patients’ utilisation of the emergency service for false hypertensive crisis, often due to anxiety and sympathetic hyper-reactivity.

The diagnostic work-up of the patient should not be carried out in the Emergency Department, but should be part of the routine interaction between PCP and specialists.
Sedation was performed with fentanyl (50 mcg) and propofol (60 mg). When the patient was sufficiently sedated, each shoulder was reduced with a two-physician technique. The first physician flexed the elbow to 90° and applied gentle axial traction, while the second physician applied gentle anterior pressure to the posterior aspect of the dislocated humeral head. Reduction was successful on each side on the first attempt. The neurovascular examination was normal on each side after the reduction.

Repeat radiographs showed successful reduction of both shoulders and no associated fractures. The patient was observed in the ED overnight for monitoring of his hypoglycaemia and had no further episodes. He was discharged the next morning to follow-up with orthopaedic surgery, but the patient did not attend this appointment and was subsequently lost to follow-up.

Posterior shoulder dislocations account for only 4% of all shoulder dislocations; anterior shoulder dislocations (95%) are far more common. Inferior shoulder dislocations (luxatio erecta), occurring in only 0.5% of cases, are extremely uncommon [2]. Posterior shoulder dislocations are frequently associated with seizure, trauma or electrocution, and almost all bilateral posterior dislocations are the result of a seizure [2]. Posterior shoulder dislocation or subluxation is also associated with neonatal brachial plexus injury, with incidence estimates of dislocation in 8% of infants and children with a brachial plexus injury [3]. Unilateral posterior dislocations are among the most commonly misdiagnosed joint injuries [4], with delays to diagnosis of over one year [5]. Diagnosis of bilateral posterior dislocations can also be delayed, with reports of erroneous initial work-ups for aortic dissection or myocardial infarction because of pain in the chest or shoulder area [4].

The mechanism of posterior shoulder dislocation is believed to be unbalanced muscle contraction. During seizure activity, the internal rotator muscles of the shoulder contract with greater force than the external rotators, which causes the humeral head to move superiorly and posteriorly [6]. Associated humeral head fracture can occur due to continuing pressure against the glenoid rim.

On physical exam, the arm is usually adducted and internally rotated. Anteriorly, the shoulder may appear flat and “squared off”, while posteriorly the humeral head may be palpable [2]. It is important to note that in cases of bilateral posterior dislocation the shoulders may be symmetric but still abnormal. Unlike in anterior shoulder dislocations, in which there may be damage to the axillary, musculocutaneous or radial nerves, neurologic or vascular injury is rare in posterior shoulder dislocations [2].

Proper joint imaging in cases of suspected posterior shoulder dislocation is crucial. AP radiographs can appear normal, as occurred in this patient (Fig. 1). In one series of 40 patients, only 50% of posterior dislocations were apparent when only AP and lateral radiographs were used [5]. Abnormalities commonly noted on the AP view include a humeral head that resembles a “light bulb” or “ice cream cone” rather than the usual “walking stick” [2]. Addition of axillary views raises the diagnosis rate to 100% [5].

If axillary views cannot be obtained because of patient pain (a common occurrence), computed tomography (CT) can show both the dislocation and any associated fractures [4]. For infants and children, ultrasound may be preferable to conventional imaging, as it does not expose the child to radiation or necessitate sedation, and it also allows for real-time assessment of reduction attempts [3].

In adults, reduction should be attempted with procedural sedation or general anaesthesia. The most commonly described technique is a two-operator method in which the first operator applies constant, gentle longitudinal traction on the adducted arm while a second operator uses both thumbs to press on the humeral head from the back to push it forward and rotate it internally. Special care should be taken in cases...
with concomitant fracture; although some authors recommend one attempt at closed reduction, it is likely that such patients will require open reduction with fixation of the fracture, so early consultation with an orthopaedic surgeon is advisable [7, 8]. If closed reduction appears successful, the arm should be immobilised and post-reduction radiographs should be obtained to verify placement and identify any new fractures.

Patients with posterior shoulder dislocations should be seen by an orthopaedic surgeon, either in the ED or within 5–7 days after discharge, and the patient should remain in a shoulder immobiliser until this evaluation. Some patients may require early surgical intervention, while others may be treated with immobilisation [9]. Rotator cuff exercises or physical therapy can be useful in preventing recurrence of dislocation, especially in those with seizure disorders who are at risk of future dislocations during seizures [10].

We report a case of bilateral posterior shoulder dislocations that were identified and successfully reduced in the ED. Posterior shoulder dislocations occur rarely but are often missed on initial presentation, resulting in ongoing patient discomfort, long-term morbidity and elevated health care costs. Posterior shoulder dislocations should be considered in post-ictal patients with shoulder pain or an abnormally appearing shoulder. ED physicians may attempt to reduce the dislocation if there is no concomitant fracture, but early consultation with orthopaedic surgery is often advisable.

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Amino acid sequence homologies between HCV polyprotein and thyroid antigens

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Recent evidence in the literature suggests that molecular mimicry between viral and self antigens may be involved in the pathogenesis of autoimmune thyroid diseases in patients with chronic hepatitis C virus (HCV) infections [1–3]. Chronic HCV infection has been reported to be associated with thyroid autoimmunity and thyroid function disorders with a mean incidence of 10% and 3%, respectively [4, 5]. Alfa-IFN therapy may exacerbate or induce underlying latent thyroid disorders, increasing the incidence of thyroid autoimmunity and thyroid function disorders to 20% and 11%, respectively [4, 5].

In keeping with the tenets of the clonal selection theory of acquired immunity, an infectious agent may circumvent the deletion of anti-self lymphocytes activating clones with receptors sufficiently degenerated to respond to mimicking epitopes and host antigens [6]. A minimum of five to six amino acids are necessary to induce an immune response, and the probability of 20 amino acids occurring in six identical residues between two proteins is 20\(^4\) (for each peptide, irrespective of the sequence) or 1 in 128 000 000 [7].

We performed the comparison between the amino acid sequence of the HCV polyprotein and five tissue-specific antigens of human thyroid, available in the database on www.ncbi.nlm.nih.gov/pubmed.

In particular, we examined the following HCV genotypes (with the respective NCBI sequence identification number): HCV1a (GI:130455), HCV1b (GI:130469), HCV1c (GI:385131), HCV2a (GI:130466), HCV2b (GI:130468),