other Spanish hospitals. In addition, as the patients who died in the ICU were not included, the total number of hospitalizations has been slightly underestimated, although we believe that the differences between the two periods would not be significantly different.

Hospitalizations for STEACS have decreased significantly in Spain in the past 20 years. The age of these patients has increased, particularly that of women. Anterior infarctions and mean stay have decreased. Our data show a trend similar to that presented in other populations, although with lower absolute numbers.

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Electrocardiographic Changes Underlying Central Nervous System Damage

Cambios electrocardiográficos asociados a afección del sistema nervioso central

To the Editor,

We present the case of a 58 year-old woman with dyslipidemia, an ex-smoker, with no known history of heart problems, diagnosed with oat-cell lung carcinoma two years earlier. She experienced extreme fatigue and inability to make fine movements for 10 days and sought emergency care. A cerebral computed tomography (CT) revealed a space-occupying lesion in the right frontal region, indicative of metastasis. A CT of the chest, abdomen, and pelvis did not show any relevant findings. We started the patient on high doses of corticosteroids and presented the case to the brain tumor committee, which opted for excision of the lesion. The patient underwent a preoperative electrocardiogram (ECG) (Fig. 1) and scheduled surgery on a week later. During anesthetic induction, the patient had increased blood pressure with alterations in the ECG, suggesting subepicardial ischemia (Fig. 2). The procedure was halted and the cardiology department was consulted. An echocardiogram did not reveal any segmental changes in contractility or associated valvulopathies; and left ventricular ejection fraction was preserved. Markers of myocardial damage were not elevated. However, given the possibility of non ST-segment elevation acute coronary syndrome and the need for a definitive diagnosis, it was performed a coronary angiography. We did not observe any significant lesions in the angiography. The patient was discharged from cardiology with the same electrocardiographic alterations. Sixteen days after discharge, she was readmitted to neurosurgery, the preoperative ECG had normalized. A right frontoparietal craniotomy was performed with radical excision of the space-occupying lesion, using the same anesthetic induction as before, but this time without incident.

ECG is a useful diagnostic tool based on the recording of electrical activity in the heart. A differential diagnosis of acute T-wave changes can be time-consuming and complicated, and can

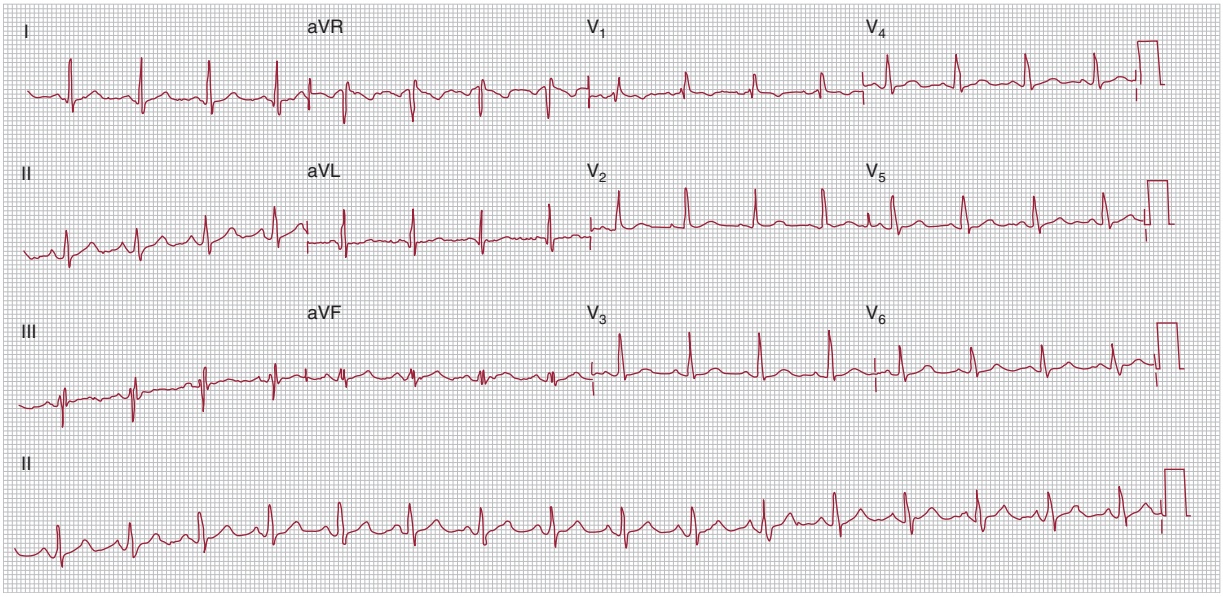

Figure 1. Preoperative electrocardiogram.

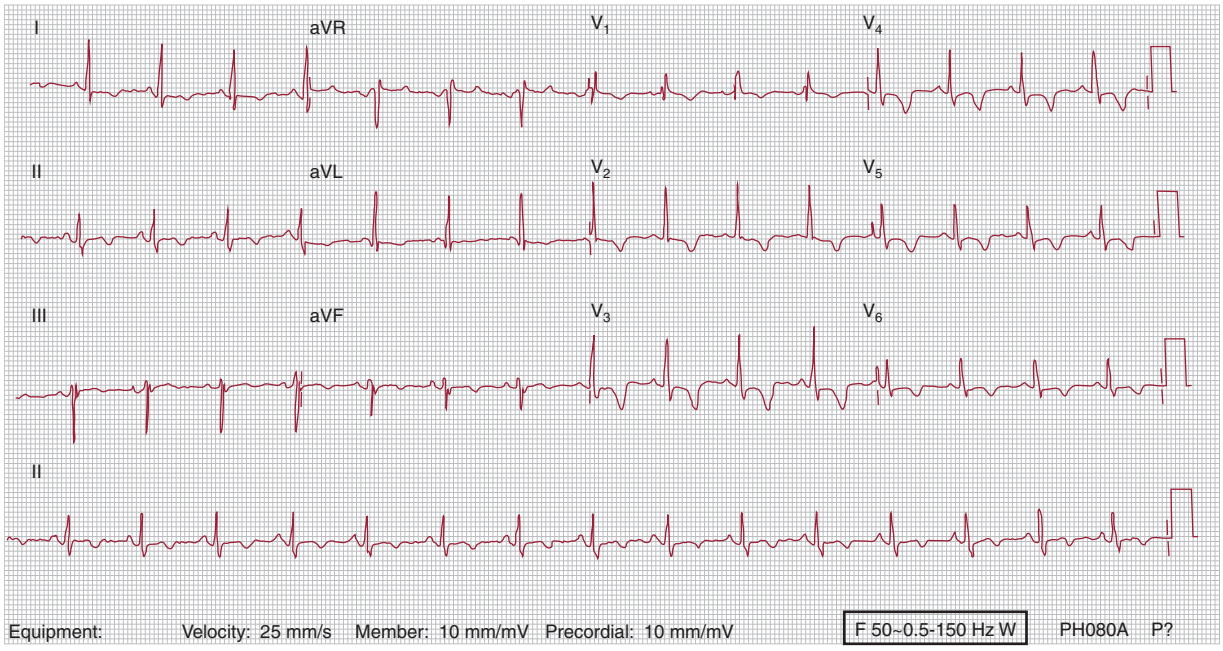

Figure 2. Electrocardiogram during anaesthetic induction, showing diffuse and deeply inverted T-waves.

include hydro-electrolytic alterations (especially hypocalcaemia and hyperkalemia), hypothermia, pulmonary embolism, cardiovascular disease, and central nervous system (CNS) disorders, among others.

The association of electrocardiographic abnormalities with intracranial damage has been known for decades. Burch et al.[1] were the first to described its association with acute cerebrovascular accidents (ACVA). These electrocardiographic changes can occur in patients with a previously normal electrocardiographic readout, and they can occur at any age, even adolescence. Although the majority appear in ACVA, can also occur in ischemic ACVA and non-vascular intracranial lesions.[2,3] Electrocardiographic anomalies can be found in 42% of patients with tumors of the brainstem and 56% with intracerebral tumours.[4]

Disorders of the CNS usually cause abnormalities in ventricular repolarisation. The most common findings are: elevated or depressed ST-segment, inverted T-waves, prolonged QT interval–the most common–and presence of U-waves.[3] These alterations can be transient or may remain for several weeks. The electrocardiographic changes observed in Figure 2 often occur in ACVA although they have also been described in Stokes-Adams attacks and intracranial tumours.[4]

The pathophysiology of this entity is still unclear. Several different mechanisms have been proposed, including elevated intracranial pressure, vagal tonicity, and excessive stimulation of the sympathetic nervous system with catecholamine production. The predominating theory involves the neurohormonal system. Damage to the CNS can result in increased catecholamine levels and sympathetic outflow, which would not only cause electrical problems, but also could lead to physical myocardial damage.[5] Catecholamines probably have a direct toxic effect on myocardial cells or mediate a vasoconstrictive effect on the coronaries.

Complications may arise during anaesthetic due to the cardiovascular depression caused by the anesthetic agents. In our case, we used: propofol, midazolam, and fentanyl, using the appropriate doses based on body weight. In this context, the potential side effect would be transient arterial hypotension. Benzodiazepines have mild hemodynamic effects, and as regards opioids, fentanyl produces no changes in myocardial contractility. With this in mind, the most probable effect of the anaesthetic in this context is transient arterial hypotension, which did not occur in our case; nor did we observe signs of hemodynamic instability. As such, we can rule out a relationship between the electrocardiographic changes and the anesthetic induction used; we consider that this finding was related to the neurological process occurred in the perioperative context. Abnormalities exclusive to T-waves are not diagnostic of any condition in particular. T-waves must be evaluated together with QRS complex, ST-segment, and clinical conditions of the patient. In this case, anamnesis was not possible, and in light of the suspicion of coronary disease and the need for a definitive diagnosis, we performed a coronary angiography.

Cardiac and neurological diseases frequently overlap.[6] The electrocardiographic changes that are associated with alterations in the CNS have both theoretical and practical importance, since in many occasions they may seem to be heart disease and can lead to an incorrect diagnosis, with therapeutic and prognostic implications.

As such, non-cardiac causes, including alterations of the CNS, should be considered in the differential diagnosis of diseases resulting in ECG changes, especially when the clinical history does not suggest acute coronary syndrome.

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Prehospital Thrombolysis: Two Years' Experience of the Community of Madrid Emergency Services (SUMMA 112)

Dos años de fibrinolisis extrahospitalaria: experiencia del SUMMA 112 en la Comunidad de Madrid

To the Editor,

In ST-segment elevation acute coronary syndrome (STEACS), maximum myocardial salvage is achieved within the first 2 h.[1] During this critical period, primary angioplasty is the reperfusion strategy of choice, and fibrinolysis is reserved for patients who are outside of this time limit.[2] Current delays in the transfer and management of these patients means that prehospital fibrinolysis (Fex) can be a complementary strategy in early treatment, but doubts remain about its efficacy and safety.

We designed a retrospective observational study that describes the clinical course of 102 patients with STEACS who received fibrinolysis from the Emergency Medical Service of the Community of Madrid (SUMMA 112) in 2007 and 2008 (5.13% of all patients with STEACS attended during the study period). Diagnostic criteria for STEACS were ischemic chest pain, ST-segment elevation and/or new-onset complete left branch bundle block. Following a strict protocol, Fex was administered if the clinical course of symptoms had lasted <3 h (or 3-6 h with time to arrival at the hospital >60 min). Two diagnostic errors occurred: one myoendocarditis and one intraparenchymal hemorrhage (patient with a low level of consciousness, severe high blood pressure and ST-segment elevation in the electrocardiogram).

Data were gathered from clinical records and SUMMA 112 Fex registers, supplemented by clinical case histories and in-hospital hemodynamic records. Survival was confirmed in June 2011 (mean follow-up, 43 [9.7] months) by consulting the Spanish national registry of deaths, the Cibeles registry, and by telephoning patients when necessary. In the descriptive analysis of the sample, quantitative variables are described as mean (standard deviation) or median [interquartile range] (asymmetric distributions) and qualitative variables as absolute and relative frequency. The Hospital Carlos III Ethics Committee approved this study.

Table 1 describes epidemiologic characteristics, risk factors, clinical data and attendance times for the series. Median values describe the typical patient as a 55 year-old man, smoker, with hyperlipidemia, high blood pressure, and chest pain; time from symptom onset to contacting the emergency services was 70 min; SUMMA 112 attended in 10 min and stabilized the patient, diagnosed STEACS, and administered fibrinolysis in 25 min; following fibrinolysis he took 38.5 min to reach the hospital.

Table 2 describes in-hospital clinical data, complications during the transfer and treatment at discharge. Six out of 10 patients presented reperfusion criteria and underwent deferred angioplasty during hospitalization. No cases of major bleeding or acute cerebrovascular accident hemorrhage due to the fibrinolytic agent were recorded. In-hospital mortality was 2% (1 patient recovered from cardiorespiratory failure but died 24 h after admission; 1 patient with multiple pathologies died of multiorgan failure on day 6). Except for a 92 year-old woman, all patients were alive at ≥2.5 years (1% mortality) after STEACS.

Our study reveals data relevant to making decisions on the optimal myocardial reperfusion strategy. Before calling for help,

Table 1
Demographic Characteristics, Risk Factors, Clinical Data and Attendance Times of the Patients Studied (n=100)

Variable	Results
Age, years	56.29±11.48
Age, years	55 [47-64.2]
Men	88 (88)
Risk factors	
Diabetes	18 (18)
Ex-smoker	14 (14)
Smoker	64 (64)
Hyperlipidemia	47 (47)
High blood pressure	39 (39)
Obesity	14 (14)
Previous case history	
AMI	5 (5)
Angina	8 (8)
Heart failure	2 (2)
Clinical data	
Anterior AMI (anterior, lateral)	54 (54)
Inferior AMI (inferior, posterior, right)	46 (46)
ST-segment depression	48 (48)
Killip I	93 (93)
Killip II	7 (7)
Killip III, IV	0
Deferred angioplasty	56 (57.7)
Rescue angioplasty	36 (37.1)
Aborted infarctions (peak troponin <1 ng/mL)	
Total	10 (10.3)
Symptoms to fibrinolysis time, min	75 [62.5-172.5]
Deferred angioplasty	9 (90)
Anterior AMI	7 (70)
Intervals, min	
Symptom onset-EMU activation	70 [45-110]
EMU activation-arrival at home	10 [6-15]
EMU activation-arrival at hospital	73 [60-89]
Symptom onset-arrival at hospital	150 [115-189]
Symptom onset-fibrinolysis	105 [73-140]
Fibrinolysis within first 2 h	
Symptom onset-fibrinolysis <60 min	15 (15)
Symptom onset-fibrinolysis <120 min	65 (65)

AMI, acute myocardial infarction; EMU, emergency medical unit.
The data express n (%), mean±standard deviation or median [interquartile range].