Nutritional Status, Heart Failure and Minimum Basic Data Set. Response

Situación nutricional, insuficiencia cardiaca y Conjunto Mínimo Básico de Datos. Respuesta

To the Editor,

We wish to thank Dr. Sánchez-Muñoz for his letter. First, we would like to express our agreement with him about the limited capacity of the Minimum Basic Data Set (MBDS) to reflect nutritional status, a view that we mention in the discussion section of our article.

Our group has recently published a review concerning reports of malnutrition in Spanish internal medicine departments[1] in which we found that this condition was recorded in only 1.4% of patients, whereas in prospective studies the incidence is around 50%.[2] In our review, we pointed out that the presence of malnutrition was accompanied by twice the mortality rate and a longer mean hospital stay than patients who were not undernourished, and we commented with surprise on the failure to report such an important finding.

The MBDS is "fed" mainly by the data provided in discharge records, and we consider it remarkable that information like this, which may reinforce important indicators of functional capacity, is not reflected in our records.[3] In fact, for the article published in your journal, we reviewed codes V85.0 to V85.5 as suggested by Dr. Sánchez-Muñoz in his letter, and found that these existed in a very small proportion of the cases, fewer than 1/10 000 of all the records. This appears to reflect the fact that, despite its importance, body mass index is not provided in the vast majority of discharge records or, if reported, the encoders do not systematically include it.

On the other hand, as the MBDS and other administrative databases are mandatory under the law they contain a very large amount of information that combines demographic, epidemiological patient care data. This not only provides information for administrators on the efficiency and quality of care, but can also be useful for clinicians involved in research projects, and would be even more so if compliance with the discharge record on the part of the physician preparing it were more complete.[4]

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P-Wave Terminal Force and Atrial Fibrillation: A Lesson Learned From Old Masters

Fuerza terminal de la onda P y fibrilación auricular: una enseñanza de los viejos maestros

To the Editor,

We read with interest the Martín García et al.[1] paper. The authors analyzed negative deflection of the P wave in precordial lead V1 (NTDV1) which is, in fact, one of the possible patterns of "P-wave terminal force V1" introduced in 1964 by Morris et al.[2] He did not study patients with atrial fibrillation (AF) but since his cohort had rheumatic heart disease one can assume that many of them experienced AF. His results showed that P-wave terminal force in V1, ie, NTDV1, "may be abnormal even in the face of normal mean left atrial pressure and normal left atrial size by x-ray examination." He called it "left atrial involvement" to stress that this electrocardiographic sign is independent of structural or pressure changes of left atrium. Those observations were confirmed later by Robitaille et al.,[3] who studied patients with lone AF and found significant differences in NDTV1 between patients with lone AF and the control group.

The report by Martín García et al.[1] gives more evidence that NTDV1 is independent of the size of left atrium, a predictor of AF recurrence. Interestingly, NTDV1 can be "positively" modified with pulmonary vein antrum isolation.[4] Striated myocardial sleeves of left atrium extend a variable distance into the pulmonary veins. They constitute the arrhythmogenic substrate and triggers implicated in AF.[5,6] Additionally, patients with AF have significantly larger diameters of pulmonary vein.[7,8] In that way NTDV1 may reflect not only retrograde activation of left atrium but enlarged pulmonary veins, most of which are posteriorly activated areas.[4]

According to Coumel's triangle of arrhythmogenesis,[9] three cornerstones are required for the onset of clinical arrhythmia: the arrhythmogenic substrate, the trigger factor, and the modulation factors such as autonomic nervous system or inflammation. When analyzing NTDV1 we analyze the arrhythmogenic substrate, which could be electrical or structure remodeling of left atrium or both. Electrical remodeling (completely reversible after restoration of sinus rhythm), the result of alterations in ionic changes, eg, L-type Ca2+ current down-regulation, leads to decreases in action potential duration and in conduction velocity. Structural remodeling (a far less reversible process of myocyte loss, diffuse and patchy fibrosis, scarring) leads to nonhomogeneity,

slowed conduction and electrical uncoupling in electrical tissue, facilitating AF.[10]

Restoration of sinus rhythm and continuation with antiarrhythmic drugs allows us to fight electrical remodeling. In patients with structurally changed atria we need inhibition of angiotensin converting enzyme and angiotensin I receptors (as angiotensin II has a central role in the development of atrial fibrosis) as well as statins and antioxidants. Other substances, including antagonists of the TGF-ß1 pathway and corticosteroids, are under evaluation.[10] Pulmonary vein antrum isolation, by eliminating focal triggers, may reverse electrical remodeling but cannot be expected to stop or reverse structural remodeling.

Two groups of patients described by Garcia,[1] without and with NTDV1 after cardioversion, most probably had electrical and structural remodeling of left atria, respectively. Modification of NTDV1 with pulmonary vein antrum isolation may be the result of cutting off depolarization of only the most posterior left atrial area.[4] In these settings the standard 12-lead ECG tracing with additional evaluation of NTDV1 could appear to be the easiest everyday clinical tool for primary evaluation and further follow-up of patients with AF.

CONFLICTS OF INTEREST

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P-Wave Terminal Force and Atrial Fibrillation: A Lesson Learned From Old Masters. Response

Fuerza terminal de la onda P y fibrilación auricular: una enseñanza de los viejos maestros. Respuesta

To the Editor,

It was with great interest that we read the comments by Wojcik et al. on our recent publication.

It is true that in 1964, Morris et al.[1] were the first to discover the presence of P terminal force in lead V1 in patients who had left-sided valvular disease. At a later date, Robitaille et al.[2] demonstrated the presence of greater terminal P negativity in lead V1 in a group of patients with a history of atrial fibrillation (AF) and no structural heart disease. Recently, Ogawa et al.[3] followed by Janin et al.[4] showed that terminal negative deflection of the P-wave in lead V1 (NTDV1) often disappears after isolation of pulmonary veins in ablation procedures.

Our objective was not to analyze the mechanisms involved in cases of NTDV1 appearing after cardioversion for AF. Beyond any doubt, they are the result of changes in the electrical activation pattern of the left atrium. In any case, none of these studies refer to prognostic implications of NTDV1, as Janin et al.[4] have stated. In this context, our group demonstrated that NTDV1 (very likely to be a manifestation of a more advanced form of left atrial disease) is an independent marker of AF recurrence.[5]

We completely agree with Wojcik et al. that it is necessary to revisit lessons taught by the "Old Masters" so they will not be forgotten. If additional original contributions continue to be made, so much the better. After all, that is the purpose of scientific research: to rely on the support of existing knowledge and to contribute new findings, even modest ones like our own.

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