

having a first face-to-face consultations rate higher than the national average really “add value”?). The dashboard for the CU management team should be fed with outcome indicators as well as cost indicators, but the latter are absent from the proposal.

- “Process management.” The proposal includes several elements related to an approach to health care management processes that should be debated. Establishing outcome indicators by functional unit (cardiac catheterization, levels of care, etc) within the CU gives rise to overlapping indicators, probably unnecessary, that should be measured at the end of the process rather than in each functional unit (at discharge or at 30 days). The metrics by functional unit should probably not be monitored by management, but by the head of the CU. Another debatable aspect is that, if integrated health care process indicators are really included, then most of them should refer to the hospitals as a whole and others to their geographic-population catchment area.³ It probably makes more sense to measure in-hospital or 30-day mortality due to heart failure in hospitals as a whole rather than just in CUs, given that most patients with this disease are treated in hospitals by internal medicine departments; likewise, should not “readmissions after 30 days for heart failure” be an indicator for the whole area, including primary care? If internal medicine or primary care (such as emergencies and, in many hospitals, level 2 and 3 care) are outside the scope of CU management, then they would not be “integrated” health care processes. That is, the CU would not be providing a care service that aligns in which all the health care departments involved in the process are aligned with the best scientific evidence available and in which health care managers promote collaboration between all units in the preparation, implementation, management, and assessment of health care process outcomes.
- “To compare, adjust.” The proposed indicators, such as those of INCARDIO, lack adjustment systems.⁴ This approach to monitoring the performance of a given CU over time may make sense, assuming that patient profiles remain stable (which is a lot to assume). However, because patients’ characteristics affect outcomes regardless of the quality of care, the indicators must be adjusted to the independent variables (age, sex, presence of comorbidities, etc) of the patients treated in each CU, if they are to be compared with each other.^{2,5} The need to “adjust” is applicable to the comparison of other indicators between different units, such as those relating to the frequency of unit use. These indicators should be weighted by the age and sex of the reference population.

There are many more elements in the proposal of González-Juanatey et al.¹ that should be debated. These include the number of indicators, hospital mortality vs 30-day mortality, the absence of health level indicators, and other elements proposed by Porter,² such as Patient Reported Experience (PREM) and Patient-Reported Outcome Measure (PROM), the information and data recording system itself, and so on. In fact, the list of such elements exceeds the scope of this letter, whose aim is to warmly acknowledge the editorial by González-Juanatey et al.¹ and encourage the SEC to promote its debate.

CONFLICTS OF INTEREST

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Clinical management indicators for the cardiovascular area. A note for the debate. Response



Indicadores de gestión clínica en el área cardiovascular. Un apunte para el debate. Respuesta

To the Editor,

We read with interest the letter by Dr Elola Somoza regarding our editorial published in *Revista Española de Cardiología* (REC)¹ in which we reflect on components of clinical management, focusing on cardiovascular medicine. We included a proposal for organiza-

tion, as well as indicators that would allow us both to determine the efficiency of our activity and to make comparisons with certain benchmarks and outcomes from centers of excellence, and, essentially, identify opportunities for improvement. In his letter, Dr Elola Somoza makes some statements that we would like to clarify, although we believe that a careful reading of the editorial would clarify most of his questions.

In his letter, Dr Elola Somoza suggests that only a third of our indicators refer to health outcomes and that in many cases they are “overlapping”. This is not the case. Two thirds are outcome indicators. It all depends on what Dr Elola Somoza understands as an outcome indicator. Is low frequency of hospitalization not, perhaps, a good outcome indicator of quality of outpatient care? And, if by overlapping he means redundant, we did not believe it necessary to clarify that in the design of key outcome indicators, such as

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mortality or health care-associated complications, it was essential to identify which area or areas of care show deviation in order to effectively apply corrective measures. The letter also refers to the existence of limitations with the measurement of indicators related to cardiology units, health care area management, epidemiology, etc, when our proposal for indicators was concerned exclusively with the organizational structure of heart disease care. In addition, although already mentioned in our editorial, we have a specific program for costs and professionals' and patients' experience.

We do not share his opinion on the lack of adjustment in the indicators, which extends to those proposed in INCARDIO.² In both cases, the indicators are similar to those suggested by top-ranking scientific societies and agencies and which should be used as reference standards for health care areas dealing with large volumes of patients, and they are good markers of quality. As indicated in both documents, certain analyses require adjustment techniques that go far beyond the age- and sex-adjustment mentioned by Dr Elola Somoza.

We reiterate our appreciation for the letter received and we suggest a re-read of the editorial. We agree that the Spanish Society of Cardiology should encourage this type of debate, as it can help clarify areas of uncertainty and identify opportunities for improvement for all of us.

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Elevated baseline renin levels as a possible cause of worse prognosis of COVID-19 in patients with heart disease



Altas concentraciones basales de renina como posible causa del peor pronóstico de la COVID-19 en pacientes con cardiopatía

To the Editor,

We read with interest the article by San Román et al.,¹ recently published in *Revista Española de Cardiología*. The results of their registry confirm that COVID-19 is associated with a worse prognosis in heart disease patients, who show a higher incidence of respiratory failure and high mortality rates. The study reports very elevated in-hospital mortality in patients with heart disease (43%), and an even higher rate among those diagnosed with cardiomyopathy (64%).

The reasons for the worse prognosis of COVID-19 in patients with previous heart disease has not yet been fully explained. One hypothesis is that it could be related to the mechanisms described by Garvin et al.² in a study analyzing gene expression in cells from bronchoalveolar lavage material in COVID-19 patients. These authors report a decrease in angiotensin converting enzyme (ACE) expression and an increase in expression of ACE2, an enzyme homologous to ACE that is the entry point of the virus into cells and a mediator of angiotensin I conversion^(1–9) and angiotensin II

conversion^(1–7) into angiotensin. ACE2 overexpression during SARS-CoV-2 infection would lead to increased angiotensin production,^(1–9) which has a sensitizing effect on receptors of bradykinin, a vasodilatory peptide that is degraded by ACE under normal conditions (figure 1). Thus, in SARS-CoV-2 infection, bradykinin would have a more potent and persistent action, a situation known as “bradykinin storm”, in which vascular dilation and permeability would be increased (among other effects), thereby triggering many of the symptoms related to a poor clinical course during COVID-19.

Patients with previous heart disease could be more vulnerable to this pathophysiological mechanism due to higher baseline production and release of renin. Among other events associated with elevated renin, which is the limiting factor of angiotensinogen conversion to angiotensin I, it has been related to a higher prevalence of heart failure and sympathetic hyperactivation.³ Durante SARS-CoV-2 infection, ACE2 overexpression would convert excess angiotensin I into angiotensin-(1–9) and this would lead to a more marked effect of bradykinin, with consequent clinical worsening. The effect of ACE inhibitors is controversial in this situation. Although they cause a renin increase by decreasing angiotensin II production, it is unknown whether this effect is added to effect produced by SARS-CoV-2 by this same mechanism. It is possible that chronic administration of these agents could induce mechanisms alternative to ACE to inactivate bradykinin,⁴ which might be beneficial during COVID-19.

The promising results of the recent pilot clinical trial by Trainas Castillo et al.,⁵ in which calcifediol (25-hydroxyvitamin D) administration was associated with a significant reduction in

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