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Editorial medical reflection

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Conceptual semantics, ethnicities, demonyms, scientific language, and political correctness

Semántica conceptual, etnias, demónimos, lenguaje científico y corrección política

Eduardo Meaney,* Alejandra Meaney‡

Science and scientific medicine must be expressed in the most pristine, precise, and straightforward language to describe even complex phenomena in the uttermost transparently and unmistakably conceivable way. Coloring and analogies confer formal richness, poetic licenses, and elaborate and elegant style to speech or literary writings in literature and daily language. However, not in science, where each word must coincide, without ambiguities, with the material and objective phenomenon that it discovers, describes, modifies, or interprets.

It is surprising that still in this new Century of Lights, the United States Census Bureau recognizes five «races» in the United States (US) population: White or Caucasian, Black or African American, American Indian or Alaska Native, Asian, and Native Hawaiian or other Pacific Islanders (although accepting that this classification «reflects a social definition of race recognized in this country and not an attempt to define race biologically, anthropologically, or genetically»)[1] not only equally surprising but also preposterous is the fact that the term «race» continues to be present in modern medical literature to indicate and differentiate human populations into distinct ethnic groups.[2] This «racial» medical differentiation is understandable to a certain extent, as it is known that genetic inheritance and ethnic belonging often determine the risk or resistance to contracting a particular disease or pathological condition and the severity or modality of a clinical entity. Among ethnicities are also contrasting biological behaviors of some systems, functions, or molecules. For example, in the «biracial» cohort study REGARDS,[3] low concentrations of high-density lipoproteins cholesterol (HDL-c) increase coronary heart disease risk only in White people but not in Blacks.

Nevertheless, «race» is based on a handful of phenotypic visible physical features such as skin tone, eye color and shape, hair color and texture, height, etcetera, which depend on variations of 0.1% of our genome. Genetic studies (mainly the monumental effort known as the Human Genome Project) have proven that there is only one race, humans.[4,5] The visible phenotype differences among ethnic groups depend on climate, exposure to sunlight, isolation or interbreeding with other human groups, nutrition, epigenetics, and other social and environmental factors.[6] As an example of what phenotypic variability signifies, dogs (descendants of primitive wolves) and modern gray wolves share 99.96% of their genome structure. Both species have changed through time due to hybridizations with jackals and coyotes, and among themselves, climate modifications, and in the case of dogs, the effects of domestication. However, despite their genetic homology, the physical differences between a wolf, a Great Dane dog, or a Chihuahua pet are remarkable.

Although «race» is not scientifically based, it is indeed a social and political construct.[2,5] It is deeply enrooted in some racist-prone societies like the US, Germany, England,

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Spain, Mexico, and Brazil. It has been used to explain and justify or normalize all kinds of injustices, dispossessions, discrimination, and barbaric acts.

Medicine and science must be apolitical but never anti-ethical. In these times of the apparent fading of globalism and the rebirth of nationalism everywhere, amid the universal struggle for the rights of all, especially minorities, we must be cautious in our medical texts to use words that can offend any national, ethnic, or minority group sensitivities. At the same time, scientific language obliges us to employ terms and concepts based on knowledge and common sense (although, as the classics say, it is the least common of all senses).

The division of the genus Homo sapiens into «subspecies» or «races» was the product of a disparate group of valuable scientists, limited by their time and Eurocentrism prejudices (Carl Linnaeus, Georges Cuvier, and Charles Darwin) and others that were, even if unintentionally, ideological precursors of White supremacism.[5] For example, the word Caucasian (as a synonym of White or Europoid) is an old-fashioned, unscientific, and racist myth invented by Germans pseudoscientists Christoph Meiners and Johann Friedrich Blumenbach in the late 1700s following the absurd Linnaeus ideas to ascribe North and Western European individuals the more elevated intellectual and moral virtues «Europaeus albus», and at the same time, to describe Yellows (Asians, «Asiaticus fuscus»), Blacks (African Negroes, «Africanus niger») and Reds (Malaysians and Amerindians, «Americanus rubescens») as intellectual and moral inferior «races». Calling White individuals of European origin (because there are ethnically Arabs, Turks, and Iranians, among others, with white skin) Caucasians is an enormous error from genetic, anthropological, geographical, and historical points of view. The Caucasus is an ethnically kaleidoscopic region where Russians, Georgians, Armenians, Azerbaijanis, Turkish, Chechens, Iranians, Mongols, and other ethnic groups have mixed genetically, socially, and culturally for centuries. Modern Europeans are the result of a gigantic mishmash consequence of migratory flows from Africa in the first place and from the Middle East and Siberia lately.[7-9] In conclusion, «Caucasian» must be excluded from the medical and scientific language as unscientific, insulting, and racist.

Another matter that goes against logic, history, physical geography, and anthropology is the word used to name the people from the nation known as the United States of America (US or USA). US people called themselves «Americans», and beyond the demonym, the word is also used as an adjective to name things, institutions, customs, qualities, and virtues from that nation: «the American way of life», the «American Heart Association», the «American Government», the «American literature», the «American democracy», and the like. Nonetheless, America is a vast continent stretching from the easternmost part of the Aleutian archipelago to Patagonia. Indeed, Americans are all the inhabitants of this continent, its surrounding islands, and archipelagos of the Caribbean, the Pacific, and the South Atlantic Oceans, comprising 35 sovereign states and 14 territories still under colonial dominion. The noun North American, sometimes applied to the US people, is also incorrect because North America, geopolitically speaking, is the northernmost part of the continent. It encompasses the Aleutian Islands, Greenland, Alaska, Canada, the continental US, and most of the Mexican territory until the strait of Tehuantepec. However, some also add the Caribbean islands and signal the Panama strait as the limit of the northern part of the continent. The Spanish term «estadounidense» (literally United Statesman, «états-unien» in French, and «statunitense» in Italian) is almost proper but less utilized. The correct demonym for the US's inhabitants (that comprises all the elements of that nation's official name) must be US Americans (as it is in German, for example, *US-Amerikaner*). Other mistakes emerge from the initial error of appropriating the name of an entire continent. Calling persons of Mexican origin living in the United States «Mexican Americans», apart from being redundant, is tantamount to inferring that Mexicans do not belong to the American continent. Correct denominations must refer to them as *US inhabitants or citizens of Mexican origin or ancestry*. In the same order of ideas, to name Black persons, Afro-Americans (as if Black or Negro, were derogatory words, while White

and Brown are not) is another anti-geographical and anti-anthropological term. Africa, a big continent, houses a considerable number of ethnic groups. There are Arabs in the north and Whites mainly in the Republic of South Africa (but with sizable White communities in Kenya, Zimbabwe, Namibia, Angola, Mozambique, and Congo, among others). Also exists a complex mixture of ethnic groups that compose Ethiopians, Somalis, and Eritreans (mainly Afro-Asiatic and Nilo-Saharan ethnic groups), and of course, a significant number of countries with a majority Negro population. With the current denomination, an Egyptian or a White South African residing in the US could be appropriately named «Afro-American». The same mistake is made in Mexico when the population of Black origin is called «Afro-Mexicans». They are indeed Mexicans of Black descent.

The term United States has been used by other nations as a shameful copycat. For example, in Mexico, the term «Estados Unidos Mexicanos» was adopted as the country's official name after the promulgation of the 1917 Constitution currently in force. After a long dispute, the constituents' legislators gave our country the unfortunate name of the United Mexican States as an unimaginative imitation of the one they had taken for themselves, our northern neighbors, since the beginning of their independent life. There is currently an initiative in the Chamber of Deputies to restore the ancestral name of Mexico, which is how our country is known abroad and the one that all Mexicans use.

What does it mean, ethnically, to be Mexican? There is no Mexican «race» or a specific ethnic group that could characterize us. Referring to Mexicans as Aztecs, as a second or nickname demonym, is improper because it leaves out other great native cultures, the Olmecs, the Teotihuacans, the Mayans, the Mixtec-Zapotecans, and the like, and denies our entire genetic lineage. In effect, Mexico is a clear-cut example of ethnical diversity and admixture, being mestizo, the more significant proportion of our population.[10] The three main genetic trunks admixed in the composition of the current Mexican population are European (64.9%), Amerindian (30.8%), and Black (4.2%), according to a study that examined the paternal lineage through the analysis of the non recombining region of the Y-chromosome.[10] Nevertheless, there is significant heterogeneity in the proportion of these three ancestral origins, with a north-south gradient, according to which European ancestry predominates in the northern and western States of the Republic.[10] In contrast, Amerindian ancestry is more important (37-50%) in Central and Southeastern Mexico. The Black genetic contribution was low and more homogeneous in all the territorial zones (0-8.8%).[10]

However, studying the maternal inherited mitochondrial DNA, the Amerindian contribution to the genetic composition of the current Mexican population was almost omnipresent (more than 90% of the mitochondrial DNA, related exclusively to maternal lineage, pertains to one of the main Pan-American indigenous haplogroups).[11] That means that the contribution of female Europeans to our genetic ancestry was relatively small, contrasting with the significant genetic ancestry of European males. This phenomenon indirectly indicates the sexual domination Mexican women suffered during the conquest and colonial consolidation. So, as the more substantial proportion of the Mexican population is of mixed origin, the term Hispanic is only partially accurate, genetically speaking.

However, racist orientation often yields blatant senselessness. The US Census authorities use the term «Hispanic» to designate any «person of Cuban, Mexican, Puerto Rican, South or Central American, or other Spanish culture or origin regardless of race».[12] Curiously, people from Spain do not receive that appellative but are named Spaniards. The people from The Philippines, a former Spain colony where Spanish is not the predominant language, are not considered Hispanics. Neither the Portuguese-speaking people from Brazil, whose territory was colonized by another Iberian country, Portugal. The Roman conquerors named Hispania the entire Iberian Peninsula and Hispanicus to their original inhabitants. Spain (España, in Spanish) derives from the Latin noun Hispania. Although this term enclosed all the peninsular territory, in less ancient times, it was awarded exclusively

to Spain, which as a country, was born at the end of the 14th century. The Spanish term Hispanoamérica (or obsolete Spanish America) leaves again out Brazil, the biggest non-English-speaking country on our continent, and other Caribbean or South American countries or colonized enclaves, where French or Dutch are the dominant languages.

Although the terms *Iberoamerica* and *Iberoamericans* include all the countries derived from the Spanish-Portuguese conquest, they leave out the other Caribbean or continental national entities colonized by France and the Netherlands. On the other hand, the noun Latino is used by the US Census Bureau as a synonym for Hispanic. It is another kind of nonsense! Latin was the language of ancient Rome, and relevant modern occidental idioms (Italian, French. Portuguese, Spanish, and Rumanian) derive from the vulgar original Roman tongue. Furthermore, a lot less extended idioms (unfairly called «regional») like Catalan, Galician, Corso, Ladino, Calabrese, and Napolitano, among many others, are also Latin-derived romances tongues. Amazingly, neither Italians nor other European national groups with Latin-derived languages are named Latinos, just those from the Americas! Instead of all this nonsense, the correct terms Latin America (and Latin Americans) encompass most of our countries and populations from Spain, Portugal, and France's ancestry and cultural heritage, combined with our original Amerindian settlers' splendorous native cultures and the significant African Black contribution. We share the same geographic space, the Americas, with Caribbean nations and peoples with mixed ancestry and idioms (as other continental enclaves or countries such as the Guyanas and Belize), some Spanish-speaking, and others who speak English, French, or Dutch. Except for the latter, all of them fall into the definition of Latin Americans, the name that the components of the second most numerous ethnic block in the US (more than 62 million people representing 19% of the total population of that country) should receive. To identify the different Latin Americans residing in the US it is necessary to indicate which country they come from: US inhabitants of Mexican, Bolivian, Haitian, Argentinian, or whatever descent.

Political correctness must be based on wisdom and rationality, not prejudices or convenient false politeness. Finally, each ethnic group, nation, and population, without exception, must hold a healthy pride in its values, traditions, culture, and language, as all belong to that diverse, multifaceted, and great fraternity called humanity.

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Original research

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Echocardiographic and electrocardiographic factors associated with non-response to cardiac resynchronization therapy

Factores ecocardiográficos y electrocardiográficos asociados con la no respuesta a la terapia de resincronización cardiaca

Araceli Villanueva-Balandrán,* Oswaldo Aldana-Varela,* Marco Antonio Hernández-Mercado‡

Keywords:
cardiac resynchronization therapy, heart failure, fragmented QRS, dilated left ventricle.

Palabras clave:
terapia de resincronización cardiaca, insuficiencia cardiaca, QRS fragmentado, ventrículo izquierdo dilatado.

ABSTRACT

Introduction: Heart Failure (HF) is a public health problem in Mexico and worldwide. It is one of the most frequent causes of hospitalization and death in the cardiology and internal medicine departments. Cardiac Resynchronization Therapy (CRT) has been an effective treatment for selected patients with HF. However, even in patients with class I recommendations, the rate of non-response is up to 30%, which makes it necessary to identify predictors of non-response. **Objective:** to find predictors of non-response to CRT. **Material and methods:** the electrocardiographic and echocardiographic features of patients who received CRT at ISSEMyM Medical Center of Toluca (IMCT) were analyzed between June 1st 2003, and June 1st 2019. We looked for an association between these features and the response or non-response to CRT one year after the implantation. A sample of 24 patients with a class I recommendation for CRT were studied; a Multivariate logistic regression analysis was performed to identify predictors of non-response. **Results:** 62.5% of the patients with a Left Ventricle End-Diastolic Diameter (LVEDD) ≥ 77 mm were non-responders (p = 0.003); 75% of the patients with fragmented QRS in the electrocardiogram previous to the CRT were non-responders, although this last result with a non-statistically significant p (0.083). **Conclusions:** the LVEDD ≥ 77 strongly predicts non-response to CRT. As for the fragmented QRS pattern, even when 75% of patients had it were non-responders, a bigger sample might be required to find statistical significance.

RESUMEN

Introducción: *la Insuficiencia Cardiaca (IC) es un problema de salud pública en México y a nivel mundial, es una de las causas más frecuentes de hospitalización y de muerte en los servicios de cardiología y medicina interna. La Terapia de Resincronización Cardiaca (TRC) ha sido un tratamiento efectivo en pacientes seleccionados con IC. Sin embargo, aun en los pacientes con recomendación clase I, la tasa de no respondedores es de hasta 30%, por lo que es necesario identificar predictores de no respuesta.* ***Objetivos:*** *encontrar predictores de no respuesta a la terapia de resincronización cardiaca.* ***Material y métodos:*** *se revisaron las características electrocardiográficas y ecocardiográficas en pacientes que recibieron TRC en el Centro Médico ISSEMyM Toluca entre el 1º de junio de 2003 y el 1º de junio de 2019. Se buscó la asociación entre estos factores y la respuesta o no respuesta a un año del implante. Se analizó una muestra de 24 pacientes con recomendación clase I para TRC. Se utilizó análisis de regresión logística multivariable para identificar predictores de no respuesta.* ***Resultados:*** *el 62.5% de los pacientes que tenían un Diámetro Telediastólico del Ventrículo Izquierdo (DTVI) ≥ 77 mm fueron no respondedores (p = 0.003); 75% de los pacientes con patrón fragmentado del QRS en el electrocardiograma fueron no respondedores, aunque este último resultado tuvo una p estadísticamente no significativa (0.083).* ***Conclusiones:*** *el DTVI ≥ a 77 mm es un fuerte predictor de no respuesta a la TRC. En cuanto al patrón fragmentado del QRS, a pesar de que 75% no respondió, podría ser necesaria una muestra de pacientes más grande para encontrar significancia estadística.*

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INTRODUCTION

Heart Failure (HF) is a public health problem with a high mortality rate. A recent United States of America (USA) study found that the deaths caused by HF had increased from 275,000 in 2009 to 310,000 in 2014.[1] In Europe, the incidence of HF is 3/1,000 people per year (all age groups) or 5/1,000 people per year in adults.[2,3] The treatment for HF is costly for any country. In the USA, the annual cost of HF in 2010 was estimated at $39.2 billion, corresponding to 2% of the total US healthcare budget.[4] A revision from different European countries found similar HF-related costs for overall health care expenditure.[5,6] Not many studies have evaluated the incidence of HF in Latin America. Ciapponi et al.[7] report an incidence of 199 cases per 100,000 person-years, a prevalence of 1%, and a 1-year mortality rate of 24.5%; for these reasons, it is imperative to optimize HF management. Cardiac Resynchronization Therapy (CRT) reduces morbidity and mortality in selected patients[8] and improves cardiac function and quality of life.[9,10] The currently approved class IA indication for CRT is in patients with optimal drug therapy who are still symptomatic and have Left Ventricle Ejection Fraction (LVEF) < 35%, QRS width > 150 ms and QRS morphology of Left Bundle Branch Block (LBBB).[11] Despite implementing CRT in patients with class I recommendation, the rate of non-response is up to 30%.[12-14] Although, studies have shown that response rates range from 32 to 91%, depending on the criteria used to define response.[15] The definitions adopted in randomized essays versus clinical practice remain discrepant. Clinical essays typically measure variables based on events, while less defined criteria are used in practice. Various definitions of CRT response have been proposed, and the response rate is different in every case.[15,16] Response rates are higher when clinical parameters such as symptoms are used but much lower when using outcome measures or ventricular remodeling.[12] For this study, the definition by Hu YR et al.[17] was used, in which after one year of follow-up, patients were defined as non-responders if the LVEF increased 24.5 ± 3.7% vs 26.2 ± 4.0% or less with a reduction of the Left Ventricle End-Diastolic Diameter (LVEDD) of 76.8 ± 6.3% vs 75.3 ± 7.3%. Patients were classified as responders if their LVEF increased by 27.4 ± 5.2% vs 42.5 ± 10.4% or more and had a reduction of the LVEDD of 70.3 ± 9.1 vs 61.8 ± 10.3 mm or greater. Many parameters have been used to predict non-response to CRT, such as electrocardiographic, echocardiographic and clinical parameters. Hu YR et al,[17] after much logistic regression analysis, found that two variables were strongly associated with non-response to CRT after one year of follow-up: fragmented QRS and LVEDD ≥ 77 mm. If any of the two was present, the probability of non-response was 14-17%; if both variables were present, the probability of non-response was close to 50%. For this study, electrocardiographic and echocardiographic features pre-CRT and their association with non-response to the therapy were analyzed.

MATERIAL AND METHODS

A retrospective and observational study was performed with patients from the cardiology department of the ISSEMyM Medical Center of Toluca (IMCT), who were treated with CRT with or without an Implantable Cardioverter Defibrillator (ICD) from June 1st, 2003, to June 1st, 2019. A sample of 24 patients was used. The inclusion criteria were 18 years old or older, both genders and having received CRT. Exclusion criteria were a basal QRS length of less than 120 ms and patients with LVEF of 40% or greater before the procedure. The only elimination criterion was death before the control echocardiogram post-CRT. The electrocardiographic features analyzed were the length of the QRS, the presence of LBBB and the presence of a fragmented QRS complex previous to the CRT. The echocardiographic feature was the end-diastolic diameter of the left ventricle previous to the CRT. A descriptive statistics analysis was performed using normality and symmetry tests for the quantitative variables. The minimum, maximum, median and standard deviation were calculated, with a 95% confidence interval for the median. For the qualitative variables, we obtained absolute and relative frequencies; cross-tabulation

for variables association. Inferential statistics consisted of applying Pearson's χ^2 test, the one-factor ANOVA test and Wilcoxon's test, all with a p-value < 0.05. The database was organized in excel, and we used the SPSS program 22 version for the analysis.

RESULTS

A total of 24 patients were included, two were excluded, and two were eliminated. The average age was 65.8 ± 12.0 years, with a minimum of 38 years and a maximum of 88 years. Sixteen patients were classified as responders, and eight patients as non-responders. When comparing age among groups, no statistically significant differences were found. The distribution by gender mainly corresponded to males, with 79% (19 cases). When comparing gender among groups, no statistically significant association was found. However, it is worth noting that 100% of the females were responders, vs 57.9% of males, with an Odds Ratio (OR) of 1.7 times for the female gender. Previous LBBB was associated with response to CRT with statistical significance, with a protection odds ratio of 0.722 times. We found that 75% of the patients with a fragmented QRS complex were non-responders to CRT *(Table 1)*.

Table 1: Association between a fragmented QRS complex and response to Cardiac Resynchronization Therapy.

	Responder		
Fragmented QRS complex	Yes n (%)	No n (%)	Total
Yes	6 (37.5)	6 (75.0)	12 (50.0)
No	10 (62.5)	2 (25.0)	12 (50.0)
Total	16 (100.0)	8 (100.0)	24 (100.0)

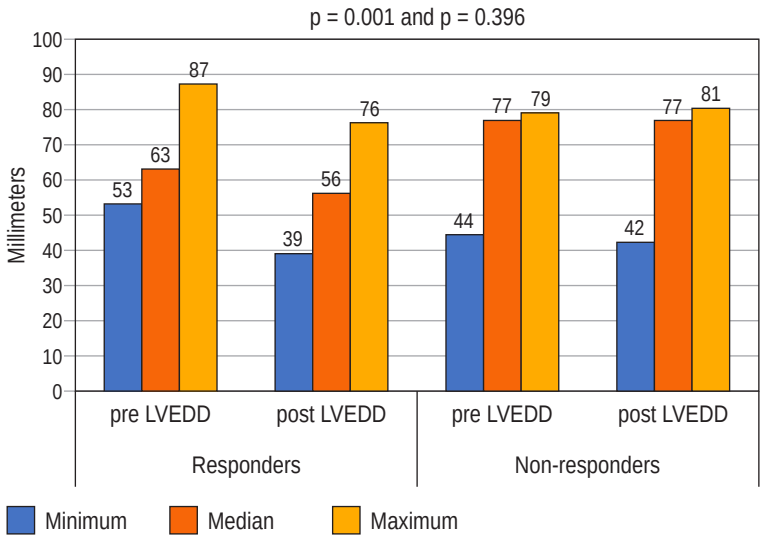

Figure 1: Comparison of pre and post-LVEDD between groups.
LVEDD = Left Ventricle End-Diastolic Diameter.

Figure 1 shows a comparison between pre-LVEDD and post-LVEDD. The investigators found that patients with the highest values of LVEDD pre-CRT, with a mean of 77 mm, were classified as non-responders, vs a mean value of 63 mm for patients classified as responders.

Figure 2 shows a comparison of the frequency of the different variables and their association with the response or non-response to CRT. It is noticeable that the percentage of responders was greater in patients without a fragmented QRS pattern in their electrocardiogram, with a previous LVEDD smaller than 77 mm and a QRS length equal to or greater than 150 ms, though only the LVEDD < 77 mm previous to CRT was statistically significant.

In counterpart, *Figure 3* shows the association between the variables and non-responders. It is evident that the variable that had a greater association with non-response to CRT was the fragmented QRS pattern, with 75%, though without statistical significance. 62.5% of patients with an LVEDD of 77 mm or greater were classified as non-responders, with statistical significance. The length of the QRS < 150 ms was present in 37.5% of non-responders without statistical significance.

DISCUSSION

Many trials have found electrocardiographic features associated with response to CRT, such as the length of the QRS > 150 ms and the presence of LBBB.[18,19] Moreover, they have also used echocardiographic parameters like the LVEF, LVEDD and the Left Ventricular End-

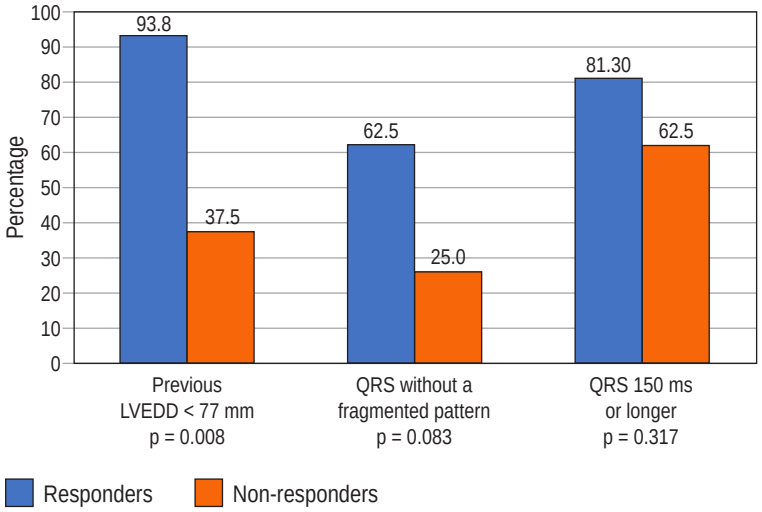

Figure 2: Frequency of responders vs non-responders and variables of the study.
LVEDD = Left Ventricle End-Diastolic Diameter.

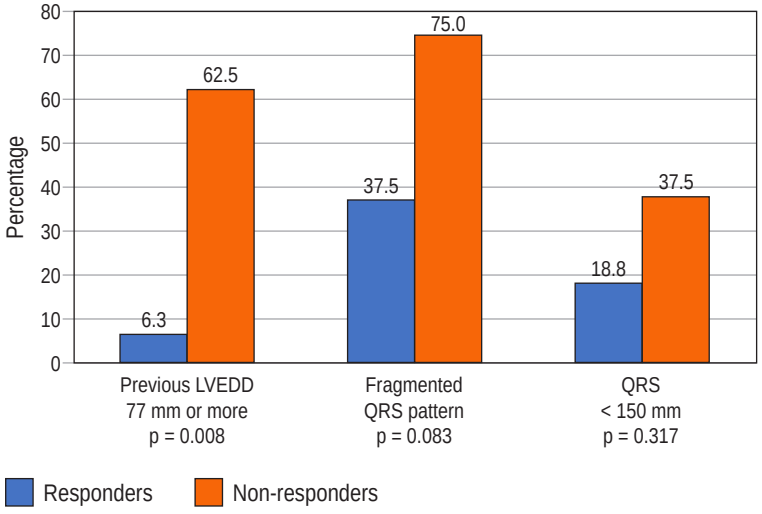

Figure 3: Variables association with non-responders.
LVEDD = Left Ventricle End-Diastolic Diameter.

Systolic Volume Index (LVESVI) to determine the positive response to CRT.[18,19] However, very few studies have been designed to find predictors of non-response. After a comprehensive revision of the literature, we found only two trials designed that way.[17,20] Hu YR et al.[17] found that the presence of a fragmented QRS and a dilated LVEDD prior to CRT are strong predictors of non-response. In patients with both features, the rate of non-response was 46.2%. Shanks M et al.[20] concluded that patients with a shorter QRS duration (150.6 ± 29.9 milliseconds vs 156.0 ± 32.5 milliseconds, p = 0.041) and larger left atrial volumes (44.9 ± 16.9 mL/m^2 vs 40.9 ± 17.6 mL/m^2, p = 0.006) were more frequently non-responders. In the present study, we found that most patients with QRS length < 150 ms were non-responders, with a p-value of 0.317. The atrial volumes were also determined, but there was no association with response to CRT. Similar to the results obtained by Hu YR et al.,[17] we found that two parameters were associated with non-response to CRT, an LVEDD > 77 mm and a fragmented QRS pattern. Nevertheless, only the first one had a significant p-value. The main limitation of this study was the small sample size, it is likely that if the number of patients is increased, we might obtain more statistically significant results.

CONCLUSIONS

The LVEDD greater than 77 mm prior to the cardiac resynchronization therapy is a strong predictor of non-response. The fragmented QRS pattern previous to CRT was associated with non-response, although with a p-value > 0.05. It is necessary to do studies with much bigger sample sizes to increase these findings' statistical significance.

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Brugada phenocopy induced by aluminum phosphide intoxication: a case report

Fenocopia de Brugada inducida por intoxicación con fosfuro de aluminio: reporte de un caso

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Keywords:
Brugada, phenocopy, aluminum, phosphide, intoxication.

Palabras clave:
Brugada, fenocopia, aluminio, fosfuro, intoxicación.

ABSTRACT

Introduction: Brugada phenocopy is an electrocardiographic phenomenon that presents a type 1 or 2 Brugada pattern triggered by various underlying clinical conditions. **Case report:** a 23-year-old male patient was admitted due to the ingestion of aluminum phosphide. At admission, an electrocardiogram showed a Brugada type 1 pattern with a transthoracic echocardiogram showing a left ventricular ejection fraction of 34% and apical dyskinesia. A second electrocardiogram was performed 24 hours later with sinus tachycardia and no evidence of the Brugada pattern. After 72 hours of clinical improvement, the apical contractility was reported as normal. **Conclusion:** this acute stress-induced cardiomyopathy by Aluminum phosphide intoxication met the criteria to be regarded as a type 1 class B Brugada phenocopy.

RESUMEN

Introducción: *la fenocopia de Brugada es un fenómeno electrocardiográfico que presenta un patrón de Brugada tipo 1 o tipo 2 desencadenado por diversas condiciones clínicas subyacentes.* ***Caso clínico:*** *paciente masculino de 23 años que ingresó por ingesta de fosfuro de aluminio. Al ingreso, el electrocardiograma mostró patrón de Brugada tipo 1 con un ecocardiograma transtorácico que mostró una fracción de eyección del ventrículo izquierdo del 34% y discinesia apical. Se realizó un segundo electrocardiograma 24 horas después con taquicardia sinusal y sin evidencia del patrón de Brugada. Luego de 72 horas de mejoría clínica se reportó la contractilidad apical como normal.* ***Conclusión:*** *esta miocardiopatía aguda inducida por estrés por intoxicación con fosfuro de aluminio cumplió con los criterios para ser considerada como fenocopia de Brugada tipo 1 clase B.*

INTRODUCTION

Brugada phenocopy is an electrocardiographic phenomenon characterized by presenting a type 1 or 2 Brugada pattern triggered by various underlying clinical conditions that resemble the Brugada phenotype in an individual not carrying the genetic mutation.[1] This case highlights the fact that the search for Brugada phenocopies triggering factors remains open, and the list continues to grow, confirming the importance of timely recognition of Brugada phenocopies and their clinical approach.

CASE PRESENTATION

A 23-year-old male patient was admitted due to the intentional ingestion of a tablet with 1.7 grams of aluminum phosphide and 1.3 grams of excipients. An electrocardiogram (ECG) was performed *(Figure 1)*. The patient was admitted with neurologic deficit and shock; cardiovascular support, mechanical ventilation, and vasopressors were initiated.

Cardiac biomarkers (troponin I, brain natriuretic peptide) were elevated beyond the 99th percentile values at admission; no

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electrolyte imbalance was present when the ECG was taken; a transthoracic echocardiogram was performed, documenting a 34% biplane left ventricular ejection fraction (LVEF) with apical dyskinesia. A second ECG was performed 24 hours later, showing sinus tachycardia without evidence of the Brugada pattern *(Figure 2)*. After 72 hours of admission, vasopressors were discontinued, and the patient was weaned from mechanical ventilation. A second transthoracic echocardiogram showed improvement in systolic function (LVEF 48% by biplane) without alterations in apical contractility. In a 24-hour Holter, the Brugada pattern was not documented again. Due to the patient's age and contractility improvement, the patient was not considered in need of coronary angiography. Acute stress-induced *(tako-tsubo)* cardiomyopathy was presumed. The patient continued to show clinical improvement until discharged.

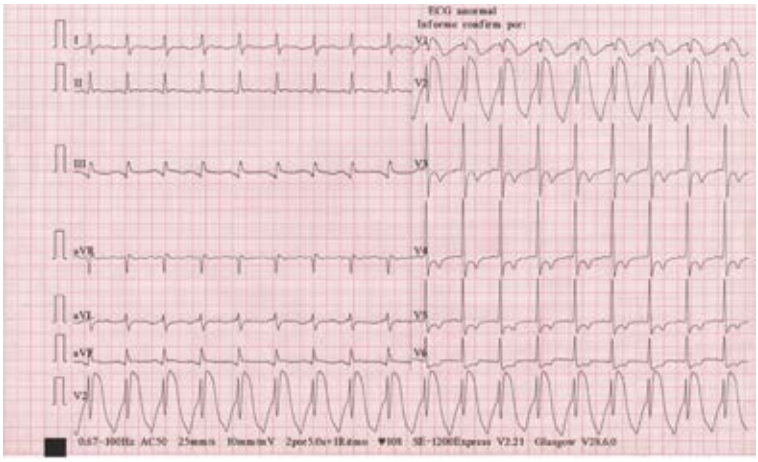

Figure 1: Electrocardiogram was taken at the presentation in the emergency room.

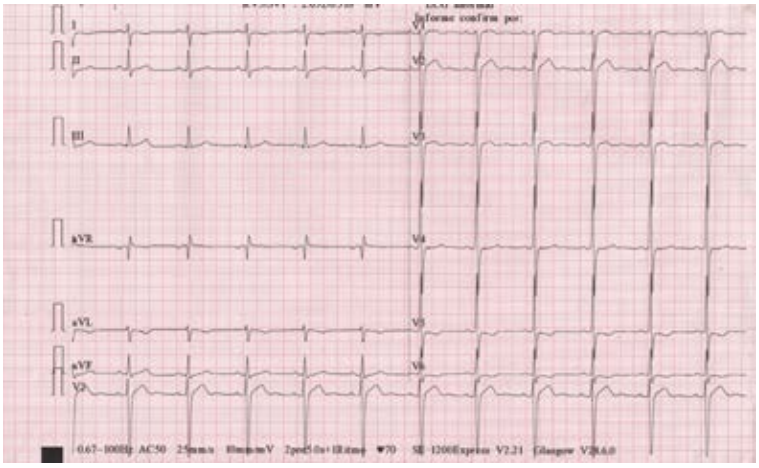

Figure 2: Electrocardiogram taken after patient's recovery.

DISCUSSION

Aluminum phosphide (Alp) is used to preserve grains worldwide. It is also one of the most dreaded poisons. The poisoning mechanism after ingestion of Alp is thought to be secondary to the release of phosphine gas by a chemical reaction of the phosphide with water and hydrochloric acid in the stomach.[2] Phosphine is a metabolic poison that noncompetitively blocks cytochrome C oxidase in rat liver preparation.[3] It also boosts the extra-mitochondrial release of oxygen free radicals, resulting in lipid peroxidation and protein denaturation of the cell membrane.[4]

A cardinal feature of Alp poisoning is early hypotension (within six hours) with a clear mental state. Features of shock include a thready pulse, cold extremities, sweating, and oliguria present in 70% of patients.[5] Myocardial injury is common with global hypokinesia of the left ventricle and interventricular septum with decreased LVEF on echocardiography, which occurs during the first 1 to 4 days in up to 50% cases.[6] Apical dyskinesia was not found in Alp poisoning in previous reports. In this case, it was attributed to *tako-tsubo* cardiomyopathy.

In Alp intoxication nonsurvivors, examination of myocardial tissue has been reported with focal areas of congestion, necrosis, and edema along with inflammatory cell infiltration; histopathological changes related to left ventricular dysfunction have been reported.

The ECG may show the following:

1. Widening of QRS complex.[4]
2. ST depression.
3. ST elevation.
4. T inversion in V2 and V3.
5. Arrhythmias and conduction disturbances include atrial fibrillation, ventricular tachycardia, wandering atrial pacemaker, complete heart block, bundle branch block, and sinus arrest.[7]

Asymmetrical T-wave inversion in precordial leads was attributed to the acute stress-induced cardiomyopathy found in the patient.

Previous cases of Brugada phenocopy induced by intoxication by Alp have been reported, none of them in conjunction with acute stress-induced cardiomyopathy.[8,9]

The diagnostic criteria for Brugada phenocopy have been established (The first four criteria are mandatory):

1. ECG pattern with type 1 or type 2 Brugada morphologic criteria.
2. Presence of an underlying condition that is identifiable and reversible.
3. Resolution of the ECG pattern upon elimination of the underlying condition.
4. Low pretest probability for Brugada syndrome determined by the lack of symptoms, clinical history, and family history.
5. A provocative negative test with a sodium channel blocker drug (e.g., ajmaline, flecainide, or procainamide).
6. A negative genetic test.[1]

CONCLUSIONS

This acute stress-induced cardiomyopathy by Aluminum phosphide intoxication met the criteria to be considered a type 1 class B Brugada phenocopy according to the Morphologic classification system from the international registry of Brugada phenocopies.

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Congenital absence of the right coronary artery with chronic total occlusion of the left coronary artery: a rare clinical situation

Ausencia congénita de la arteria coronaria derecha con oclusión total crónica de la arteria coronaria izquierda: un escenario clínico raro

José Martín Alanís-Naranjo,* Óscar Manuel Granados-Casas‡

Keywords:
absence of the right coronary artery, chronic total occlusion, coronary angiography.

Palabras clave:
ausencia de arteria coronaria derecha, oclusión total crónica, angiografía coronaria.

ABSTRACT

In the literature, few cases of absence of the coronary artery have been reported. In addition, the absence of the right coronary artery (RCA) with chronic total occlusion (CTO) of the left coronary artery is uncommon. We present the case of a Mexican patient with a history of exercise-induced chest pain in which a congenital absence of RCA with CTO was confirmed by coronary angiography. The patient was discharged symptom-free and remained so after conservative treatment. The absence of coronary arteries results from abnormal embryonic development, with various clinical manifestations; most patients show symptoms of myocardial ischemia. This case illustrates the importance of coronary angiography for diagnosis, which is considered the gold standard. However, there are no standardized treatment guidelines; conservative or revascularization therapy may be used to treat the condition. Due to its extremely low incidence, it is challenging to determine the prognosis of this coronary anomaly.

RESUMEN

En la literatura se han reportado pocos casos de ausencia de la arteria coronaria. Además, es infrecuente la ausencia de la arteria coronaria derecha (CD) con oclusión total crónica (OTC) de la arteria coronaria izquierda. Presentamos el caso de un paciente mexicano con antecedentes de dolor torácico inducido por el ejercicio, en quien se confirmó, mediante angiografía coronaria, una ausencia congénita de CD con OTC. El paciente fue dado de alta asintomático y permaneció así tras el tratamiento conservador. La ausencia de arterias coronarias resulta del desarrollo embrionario anormal, con diversas manifestaciones clínicas; la mayoría de los pacientes muestran síntomas de isquemia miocárdica. Este caso ilustra la importancia de la angiografía coronaria para el diagnóstico, que se considera el estándar de oro. Sin embargo, no existen pautas de tratamiento estandarizadas; se puede utilizar una terapia conservadora o de revascularización para tratar la afección. Debido a su incidencia extremadamente baja, es difícil determinar el pronóstico de esta anomalía coronaria.

INTRODUCTION

Right coronary artery (RCA) absence is a rare congenital cardiovascular malformation with few cases reported in the literature.[1-3] Congenital absence may result from agenesis or congenital occlusion of the RCA during embryonic development. It has also been shown to be associated with congenital heart disease.[1] In most cases, the congenital absence of the RCA is asymptomatic. However, a small proportion of these patients develop life-threatening clinical complications, including acute myocardial infarction, stroke, or sudden death.[3] The absence of RCA with chronic total occlusion (CTO) of the left coronary artery is extremely rare.

We present the case of a Mexican patient with a history of exercise-induced chest pain in which a congenital absence of RCA with CTO was confirmed by selective coronary angiography (CAG).

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CASE PRESENTATION

A Mexican man of 80 years old was admitted to our hospital following six months of exercise-induced chest pain. The chest pain was retrosternal, oppressive, lasting two minutes, and waned with rest. Over the past two months, he experienced an increase in the intensity and frequency of chest pain episodes. The patient denied any history of chronic diseases, including cardiovascular disease or a family history of coronary heart disease. A physical examination revealed a body temperature of 36.5 ºC, a pulse rate of 86 beats per minute, a breathing rate of 20 breaths per minute, a blood pressure of 104/69 mmHg, and a body mass index of 28.5 kg/m^2. There were no abnormal findings during the cardiac examination. It was decided to admit the patient for further investigation and treatment.

The electrocardiogram showed sinus rhythm, inversion of T wave in leads aVL, V2, and V3 with Q waves in lead aVR *(Figure 1)*. In laboratory tests, a creatine kinase-MB (CK-MB) level of 7 IU/L was found (reference range, 0-10 IU/L) *(Table 1)*.

The coronary angiography revealed a CTO in the middle of the left anterior descending artery with a congenital absence of the right coronary artery and ectasia in the left circumflex artery, resulting in three posterolateral arteries and the posterior descending artery *(Figures 2 and 3)*.

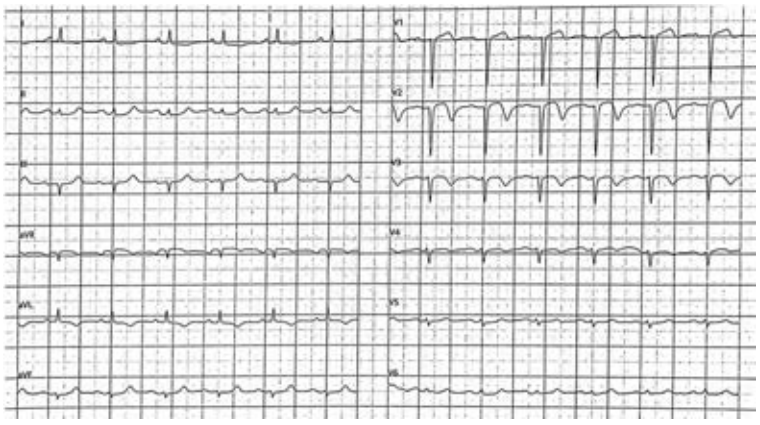

Figure 1: 12-lead electrocardiogram: sinus rhythm and inversion of T wave in leads aVL, V2, and V3 with Q waves in lead aVR.

The patient was then offered only symptomatic treatment. The patient was prescribed aspirin, atorvastatin, and metoprolol during his hospitalization. Hospitalization was uneventful. On the day of discharge, he was symptom-free and has continued to be symptom-free for the duration of his follow-up treatment.

DISCUSSION

Coronary arteries develop in two stages during embryonic development. First, endothelial cells derived from venous sinuses form immature primary coronary vascular networks in the subepicardial and myocardial layers. Second, epicardial cells undergo an epithelial-mesenchymal transition to produce smooth muscle cells and fibroblasts, which are then remodeled to create a mature cardiac artery system. Coronary artery anomalies are the result of abnormal embryonic development.[1,4,5]

In 1882, Hyrutle published the first report of a single coronary artery in an autopsy study.[6] According to Smith, it is an isolated coronary artery that arises from the aortic trunk and supplies the entire heart regardless of location.[7] White and Edwards first described the congenital absence of the RCA in 1948.[8] A prevalence of 0.014 to 0.066% has been reported in the literature for the congenital absence of RCA.[1] At the time of diagnosis, the mean age of patients with congenital absence of RCA is 53 years, with 54 percent being females and 87% without any associated congenital heart defects.[3]

The angiographic classification of a single coronary artery was first proposed by Lipton in 1979 and is now widely used in clinical practice. It is first separated into types R and L based on the location of the coronary artery's origin (right or left coronary sinus). Secondly, it is classified into groups I, II, and III according to its anatomical distribution of branches. In group I, one coronary artery extends to supply the heart that the contralateral coronary artery should have supplied; in group II, a single coronary artery that has transverse branches that extend to supply the heart that the contralateral coronary artery should have supplied; in group III, an LCX and LAD branch

Table 1: Laboratories on admission to hospital.

	Value	Reference range
Leukocytes, [mm^3]	8,390	5,000-10,000
Platelets, [mm^3]	220,000	130,000-400,000
Hemoglobin, [g/dL]	14.3	14-18
Sodium, [mEq/L]	138	136-145
Potassium, [mEq/L]	4.45	3.5-5.1
Chlorine, [mEq/L]	106	98-117
Phosphorus, [mg/dL]	3.2	3.7-7.2
Calcium, [mg/dL]	9.3	8.6-10.6
Magnesium, [mg/dL]	2.1	1.9-2.7
Glucose, [mg/dL]	94	74-106
Creatinine, [mg/dL]	0.94	0.7-1.3
Urea, [mg/dL]	48.79	18-50
Blood urea nitrogen, [mg/dL]	22.8	9-23
Total creatine kinase, [IU/L]	59	21-232
Creatine kinase-MB, [IU/L]	7	0-10
Triglycerides, [mg/dL]	160	0-150
Total cholesterol, [mg/dL]	99	100-200

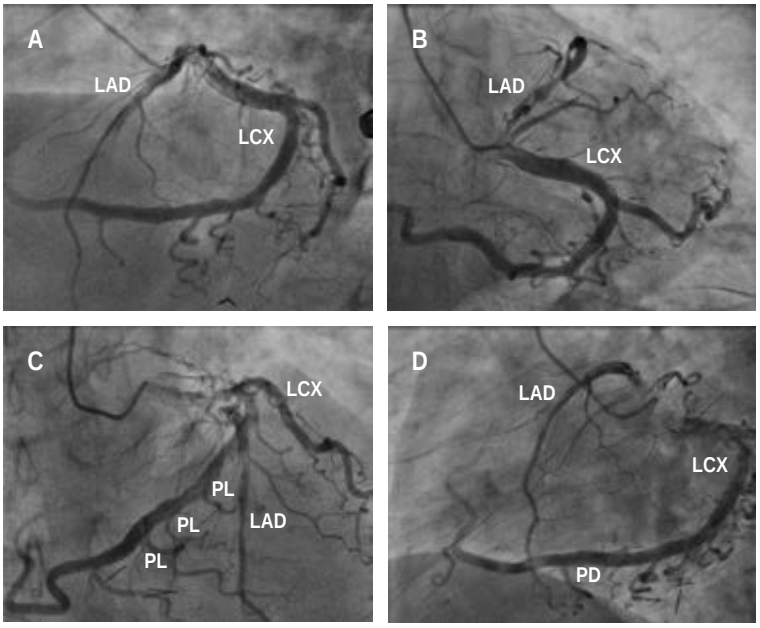

Figure 2: Coronary angiography: chronic total occlusion of the middle segment of the left anterior descending artery, the ectatic left circumflex artery in the proximal portion giving rise to three posterolateral arteries, subsequently originating posterior descending artery. **A)** Left anterior oblique view. **B)** Right anterior oblique caudal view. **C)** Left anterior oblique cranial view. **D)** Left anterior oblique caudal view. LAD = left anterior descending artery. LCX = left circumflex artery. PL = posterolateral artery. PD = posterior descending artery.

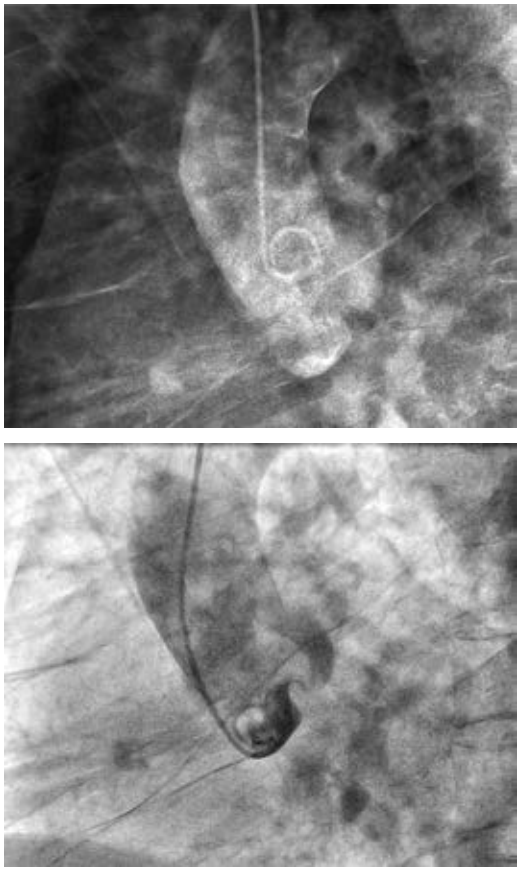

Figure 3: Aortogram in which the absence of the right coronary artery is observed.

arise separately from the trunk of a coronary artery. Lastly, according to the route taken by the large transverse trunk as it crosses the base of the heart to reach the normal contralateral coronary artery, it is classified into three groups regarding the aorta and the pulmonary artery: A (anterior), B (between), and P (posterior).[9] It has been reported that the L-I pattern is more likely to occur than the L-II pattern.[3] In accordance with Lipton's classification, this patient is classified as a type L-I.

Clinical manifestations vary; most patients present with symptoms of myocardial ischemia, such as angina pectoris, chest tightness, palpitations, and atypical chest pain. Symptoms such as these are caused by arterial stenosis and obstruction. It is important to note that the electrocardiogram manifestations vary and are non-specific; approximately half of

these patients will have left coronary artery branch lesions, and approximately 60% will have ST-T changes; other findings include sinus bradycardia, third-degree atrioventricular block, or atrial fibrillation.[10]

The CAG remains the gold standard for diagnosing congenital absences of the RCA and is widely used in clinical practice. Nevertheless, it often confuses the operator during angiography, resulting in repeated non-standard angiographic projections, rapid consumption of contrast medium, a prolonged procedure time, and an increased risk of complications.[3] CT angiography is a noninvasive examination commonly used for diagnosis, treatment, and follow-up; it can also detect anatomical variations in the cardiovascular structure and discover other cardiac conditions.[10]

As few cases have been reported, there are no standardized treatment guidelines for patients with single coronary arteries. However, medical therapy with anti-platelets, anti-coagulation, lipid-lowering agents, and revascularization therapy, including interventional or surgical procedures, are available as treatment options.[1,2,3,10] The percutaneous coronary intervention (PCI) of CTO lesions has drawn considerable interest recently. The procedure, however, is technically challenging, and the complication rate is higher than when PCIs do not involve CTOs. Moreover, there are minimal data from randomized controlled trials comparing CTO-PCI with medical therapy. Most studies examining PCI outcomes for CTO are either observational single-center or multicenter registries. Clinical trials have not demonstrated an improvement in ventricular function, a difference in symptoms, or clinical outcomes with CTO PCI compared with optimal medical care. Therefore, all patients with CTO should receive optimal guideline-directed medical therapy, and revascularization should be considered only for those with refractory angina despite medical therapy and for whom benefits exceed the potential risk of the procedure.[11,12]

CONCLUSIONS

In the literature, there are a limited number of reports of congenital absence of RCA, which can sometimes lead to life-threatening complications. The absence of RCA with CTO of the left coronary artery is a rare condition. This case illustrates the importance of coronary angiography for diagnosis, which is considered the gold standard. Treatment options include conservative or revascularization therapy; in this case, we used antiplatelet and lipid-lowering agents without any symptoms during follow-up. Due to its extremely low incidence, it is difficult to determine the prognosis of this coronary anomaly. Further studies are needed to determine this condition's most appropriate treatment and prognosis.

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Review

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Clinical application rules

Reglas de aplicación clínica

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Keywords:
epidemiology, clinical, application.

Palabras clave:
epidemiología, clínica, aplicación.

ABSTRACT

Decision-making in the daily practice of medicine requires amalgamating the experience of the physician, the data derived from scientific studies as well as the available resources and preferences of patients. Evidence-based medicine helps in these tasks and clinical guidelines have served as support, but the doctor must reinforce his knowledge of epidemiology, statistics and scientific method to be better prepared when deciding which data can be incorporated into the practice of medicine, which should be discarded and identify those that require confirmation. This article presents the most important elements to consider when deciding on the applicability of data from research studies and clinical epidemiology.

RESUMEN

La toma de decisiones en la práctica diaria de la medicina requiere amalgamar la experiencia del médico, los datos derivados de estudios científicos, así como los recursos disponibles y las preferencias de los pacientes. La medicina basada en evidencias ayuda en estas tareas y las guías clínicas han servido de apoyo, pero el médico debe reforzar sus conocimientos de epidemiología, estadística y método científico para estar mejor preparado a la hora de decidir qué datos se pueden incorporar a la práctica de la medicina, cuáles deben descartarse e identificar aquellos que requieren confirmación. Este artículo presenta los elementos más importantes a considerar al decidir sobre la aplicabilidad de los datos de los estudios de investigación y la epidemiología clínica.

INTRODUCTION

Medicine had a fast evolution since science allowed a better understanding of health and disease, and technology paved the way for prevention and control. However, the art of medicine, which includes variability in all its expressions and human preferences and attitudes, puts medicine suddenly inside the complexity that rules the function of the universe including biology, and human behavior. Decision-making in the daily settings of medical practice incorporates data derived from investigations that are proven effective under some rules that are not universal and leave much room for improvement.[1]

Stephen Jay Gould, an anthropologist from Harvard University, wrote many years ago that nature is not conformed by clearly defined entities but works in many different levels that interact diffusely in their borders. Now the concept is named «complexity», and in the words of the German scientist Hans-Peter Dürr, «the whole is greater than the sum of its parts and the new paradigm is complexity instead of reductionism».

The large number of medical papers published negates the possibility of reading all of them, even if the selection is limited only to one topic.[2] Some groups help by classifying the papers according to their quality, but even so, they are too many, and the selection methods have been subject to criticism. Another method for applying clinical investigation to the daily practice of medicine is to follow the recommendations of clinical guides. However, currently, there are too many clinical guidelines of very different quality, and some of them are signaled as having different types of bias, like lack of preparation of the authors and conflicts of interest by obeying not the scientific evidence but the industry's interests.[3,4]

Paradigms are the backbone of medical recommendations and give the false security

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of interpreting them as rock-solid concepts. Nevertheless, history tells us that the opposite is true; as an example, Mayo Clinical Proceedings published an analysis in which, after ten years of publication, only 40% of the concepts remained state of the art.[5]

Among the published papers, there will be some that are relevant to the care of patients. According to J Ioannidis, most investigation in science is of low quality, so it is necessary to identify those with good quality (internal validity) and which can be applied to a particular setting (external validity or applicability in the real world).[6]

The Cochrane foundation lists those whose qualify was evaluated by the GRADE method. However, to properly evaluate which of them must be chosen to read, many published methods aim to rate their quality and the possible impact on medical care rather than accepting the authors' conclusions and recommendations.[7]

The Cochrane foundation mentioned above published its ladder of evidence *(Figure 1 and Table 1)*, where they place the backup of any medical recommendation in one of three steps. The first one is named «efficacy» and includes those concepts derived from medical papers but without confirmation in various settings. At best, they have been shown to function in near-to-ideal settings. Karl Popper states that there should be falsification studies before a concept is confirmed. Even so, currently, they are never done because of a lack of financial backup and the interest to continue promoting findings that will not be reproduced in new investigations. The second step is effectiveness which can be reached when the «epidemiological arrow» described by Jeremiah Stamler points in the right direction by the added value of several studies. Confirmation studies and their application in clinical settings must corroborate the data obtained in the initial clinical trials. The third step relates to the cost/benefit ratio. While medical associations give their advice, this step falls within the realm of governments and official agencies, where decisions take place concerning priorities in money expenditure.

Notwithstanding all the limitations that medical knowledge confronts, clinicians must decide which recommendations apply to a particular patient and circumstance in every consultation. Here is a list of some scientific characteristics that back up the concept of clinical applicability and will help in the decision.

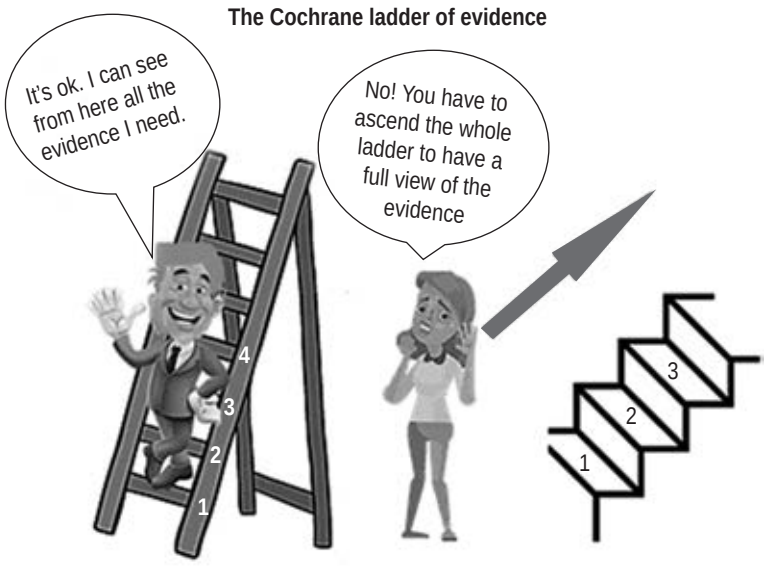

Figure 1: It should be clear for everybody, that for a new and just identified concept you have to ascend the whole staircase before it is established as a solid paradigm and then observe the direction of the epidemiological arrow as described by Jeremiah Stamler.[11,12]

Table 1: Interpretation of the Cochrane ladder of evidence.[13,14]

Can it work?	Under ideal circumstances, the degree of the intervention producing more benefit than harm	Efficacy	Step 1
Does it work?	The same concept but in the clinical setting	Effectiveness	Step 2
Is it worthwhile?	Measures the intervention effect vs its cost	Cost/benefit ratio	Step 3

RULES FOR CLINICAL APPLICABILITY

1. Look for the causality rules (Koch, Bradford Hill, Evans). Although subject to some criticism, they give a good view to distinguish etiological and risk factors, and those which are merely accompanying and not causal ones
2. Identify the absolute or attributable risk reduction and do not accept only the relative risk reduction or the risk ratios. Their confidence limits are used to calculate the statistical significance but do not measure the size of the effect. Furthermore, sometimes when the effect is small, the absolute risk reduction is hidden, so it is necessary to dig further to find it.
3. The number needed to treat (NNT): how many patients are to be treated to obtain a reduction of the endpoint selected (i.e., mortality of morbidity). The number needed to harm (NNH): how many patients are treated for every harm or complication detected. Although there is no precise number, the ratio of NNT/NNH must be in favor of the benefits. For example, Sacket published that an NNT greater than ten is unacceptable. However, others currently considered beneficial interventions in medicine stray far from that point, with some numbers reaching the hundreds.
4. Internal and external validity. Those investigations with a proper design, valid protocol, and representative sample, adequately executed, evaluated, and presented, excel over those poorly done. Besides, we must consider at the top of quality evaluation those investigations based on meta-analysis and prospective randomized medical trials, followed by non-randomized trials, retrospective, cohort, and case/control studies. At the bottom are the observational studies and original case reports, which with some exceptions usually indicate the need for more advanced trials. Again, there are exceptions, but as one goes up in the ladder of the quality class of investigations, there is evidence of better scientific merit and a lower bias risk.
5. A good prognosis of greater survival in the first five or ten years must be confirmed in different settings. Clinical trials are the best scenario that an intervention will have. Usually, the actual level of its benefit must be observed in subsequent trials or observational studies during its clinical application (i.e., databases or real-world studies).
6. Clinical setting varies widely from controlled clinical trials (CCT). Patients will differ in their characteristics and their preferences. Other points include economic issues and acceptance to perform a study or to follow a treatment. The physicians' points of view will also influence the application of the procedure or treatment.
7. Be aware of distraction and even of deceit and fraud. The general under-preparation in epidemiology and biostatistics makes physicians feel overwhelmed by numerical information, so before it is too late, practitioners must start their instruction in scientific methodology.[8]
8. Do not consider statistical significance as the main factor in judging the quality of a study. In recent decades, based on the publications of Sir Ronald Fischer and the statisticians and clinical researchers who refined and adopted them, the statistical significance of research findings has become the main factor to consider. Researchers put it as their primary factor «p fishing», and readers of publications rely on that number to decide on the «success or failure» of the study. However, it must be emphasized that the more important thing is the size of the effect (i.e., correlation, comparison), and these facts are not measured by the «p» values or the confidence limits of the central tendency.[9] In contrast, there are many more points that can guide decision-making. The pillars that support decisions in medicine are its proportions of art and science that comes, in the words of Sir William Osler, from the pursuit of excellence, the practice of abstraction, and learning the method. We could add here the practice of common sense.[10]

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REVIEW

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Influenza vaccination for primary and secondary prevention of cardiovascular risk: Call to action of Cardiology Societies of Mexico

Vacunación contra la influenza para la prevención primaria y secundaria del riesgo cardiovascular: una llamada para la acción de las Sociedades Cardiológicas de México

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ABSTRACT

Influenza substantially increases the risk of triggering acute coronary syndromes and heart failure. Vaccination against influenza decreases these risks and diminishes the occurrence of complications. Vaccination of the entire population is recommended, especially for people at risk or with established cardiovascular disease.

RESUMEN

La influenza aumenta sustancialmente el riesgo de desencadenar síndromes coronarios agudos e insuficiencia cardiaca. La vacunación contra la influenza disminuye estos riesgos y disminuye la aparición de complicaciones. Se recomienda la vacunación de toda la población, especialmente las personas con riesgo o con enfermedad cardiovascular establecida.

INTRODUCTION

Influenza is associated with increased cardiovascular risk, especially in coronary syndromes and heart failure patients.

Considerations: 1. Influenza is an acute respiratory infection caused by the influenza virus that circulates in all parts of the world and is predominantly seasonal. It represents a high disease burden and is estimated to cause 3 to 5 million cases of severe illness and 290,000 to 650,000 deaths yearly.[1] 2. Several studies have confirmed that people with cardiovascular disease (CVD) are at increased risk of severe influenza illness. In addition, influenza-type respiratory infections are associated with cardiovascular events at follow-up, including acute myocardial infarction, stroke, hospitalizations for heart failure, atrial fibrillation, and deaths from cardiovascular causes.[2] 3. About 11.7% of patients with confirmed influenza develop an acute cardiovascular event. (6.2% heart failure, 5.7% acute coronary syndrome).[3] 4. Influenza vaccination has been associated with a reduction in cardiovascular events.[4] 5. Several studies have reported that influenza vaccination in patients with coronary heart disease is associated with a reduction in cardiovascular events and low-risk people over 65 years of age, i.e., absence of comorbidities, no established CV disease, or diabetes.[5] 6. Patients with *Acute Heart Failure* had a 45% lower risk of complications when they were vaccinated during their hospital stay.[6] 7. Vaccination in high-risk people reduces this risk by 45%. It is

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required to vaccinate 56 patients to prevent one case of myocardial infarction (NNT).[7] 8. After an acute coronary syndrome episode, vaccination during the first 24 h reduces major cardiovascular events by 28% and CV mortality by 41%.[8] 9. Recently, the Inter-American Society of Cardiology and the World Heart Federation (WHF) issued a consensus document entitled «Influenza Vaccination for the Prevention of Cardiovascular Disease in the Americas».[9]

RECOMMENDATIONS

Given the overwhelming evidence of the benefit of influenza vaccination and aligned to the Inter-American Society of Cardiology and the World Heart Federation, the cardiological societies of Mexico recommend vaccination against influenza under the following conditions: 1. Preferably the entire population over six months of age and older will be vaccinated for influenza annually 2. If vaccines are not available for the entire population: Vaccinate patients over 65 years of age, even without CVD or risk factors 3. Vaccinate patients with chronic coronary heart disease annually with or without a history of revascularization, regardless of the patient's age. 4. Vaccinate all patients with heart failure annually against influenza. 5. Vaccinate annually against influenza people living with diabetes or high blood pressure, even without underlying CVD and regardless of their age. 6. Vaccinate patients with acute coronary syndromes or revascularization procedures before their hospital discharge, whatever the epidemiological season.

CONCLUSION

The lack of vaccination against influenza in adults can be considered a modifiable cardiovascular risk factor

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Review

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Mexican College of Interventional Cardiology and Endovascular Therapy (COMECITE) international multidisciplinary consensus statement regarding catheter-based pulmonary artery monitoring

Declaración de consenso internacional y multidisciplinario del Colegio Mexicano de Cardiología Intervencionista y Terapia Endovascular (COMECITE) sobre la monitorización invasiva de la arteria pulmonar

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Keywords:
Swan-Ganz catheter, invasive pulmonary monitoring, cardiogenic shock, cardiac intensive care.

Palabras clave:
catéter de Swan-Ganz, monitoreo invasivo de la arteria pulmonar, choque cardiogénico, cuidado intensivo cardiovascular.

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ABSTRACT

The Swan-Ganz (SG) catheter is an indispensable tool for invasive hemodynamic monitoring but is underused due to controversy for misunderstandings after several confounding studies. The Mexican College of Interventional Cardiology and Endovascular Therapy (COMECITE) invited a select group of international specialists in interventional cardiology, critical cardiology care, and general intensive care for a consensus statement on SG catheter use, endorsed by COMECITE and the Mexican College of Critical Care (COMMEC). The consensus recommends the SG as a diagnostic tool in cardiogenic shock from any etiology and at any class and level, involving one ventricle or both; during worsening heart failure/hemodynamic instability, despite adequate treatment; for differential diagnosis during failed treatment for respiratory distress, hypotension, and or progressive renal failure; for simultaneous monitoring of the pulmonary artery and right atrial pressures during severe right heart-related shock. The consensus encourages centers with low SG utilization to include and master its hemodynamic monitoring benefits.

RESUMEN

El catéter de Swan-Ganz (SG) es una herramienta indispensable para la monitorización hemodinámica invasiva, pero está subutilizado debido a la controversia después de varios estudios con resultados que llevaron a interpretaciones erróneas. El Colegio Mexicano de Cardiología Intervencionista y Terapia Endovascular (COMECITE) invitó a un grupo selecto de especialistas internacionales en cardiología intervencionista, cuidados cardiológicos críticos y cuidados intensivos generales para una declaración de consenso sobre el uso del catéter SG, avalada por COMECITE y el Colegio Mexicano de Cuidados Críticos (COMMEC). El consenso recomienda el SG como herramienta diagnóstica en el choque cardiogénico de cualquier etiología y de cualquier clase y nivel, con compromiso de un ventrículo o de ambos; durante el empeoramiento de la insuficiencia cardiaca e inestabilidad hemodinámica, a pesar del tratamiento adecuado; para el diagnóstico diferencial durante el tratamiento fallido de dificultad respiratoria, hipotensión o insuficiencia renal progresiva y para la monitorización simultánea de las presiones de la arteria pulmonar y la aurícula derecha durante un choque grave relacionado con el corazón derecho. El consenso alienta a los centros con baja utilización de SG a incluir y dominar sus beneficios en el monitoreo hemodinámico.

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INTRODUCTION

It has been more than fifty years since the Swan-Ganz (SG) catheter was first used for invasive hemodynamic monitoring and there has been ongoing controversy regarding benefits and risks of its use.[1-4]

The Mexican College of Interventional Cardiology and Endovascular Therapy (COMECITE: *Colegio Mexicano de Cardiología Intervencionista y Terapia Endovascular*) invited a select group of international specialists in interventional cardiology, critical cardiology care, and general intensive care, to discuss the current use of invasive pulmonary artery monitoring, its benefit/risk and to publish a consensus statement on SG catheter use, endorsed by COMECITE, the Mexican College of Critical Care (COMMEC: *Colegio Mexicano de Medicina Crítica*) through its cardiovascular care working group, plus other invited medical organizations.

MATERIAL AND METHODS

The consensus group emerged from members of COMECITE, COMMEC and SCAI plus international experts on cardiogenic shock (CS), further electing chair, co-chair, and the rest's specific functions.

The meetings took a nominal group technique format, which consisted of the face-to-face discussion on video conference, in which each member presents their proposal and their reasons, without a time limit. Delphi rounds finally solved disagreements.[5-8]

The consensus group defined the authors' nomination from the beginning of the consensus work and modified it during its process. According to the International Committee of Medical Journal Editors (ICMJE), were authors all the people who contributed and who strictly complied with every one of the following aspects:

1. Contributed substantially to the conception or design of the work; or the acquisition, analysis, or interpretation of data.
2. Wrote the work or critically reviewed it.
3. Approved the final version for publication.
4. Confirmed the accuracy and completeness concerning every part of the work.

The acknowledgments section mentions the contributors who have not complied with every one of the four points outlined above, but worth mentioning for relevant participation.

The magnitude of consensus' contribution ordered the authorship and the corresponding author designation, with a preponderance of the person who originated the idea and who presides and coordinates. In case of disagreement and dispute over the order, an anonymous vote in a ranking format of importance decides, and, in extreme cases, the consensus might call an internal or external judge.[9]

CURRENT KNOWLEDGE

The Society for Cardiac Angiography and Interventions (SCAI) stated on 2019, a classification of the CS (document endorsed by the American College of Cardiology [ACC], the American Heart Association [AHA], the Society of Critical Care Medicine [SCCM], and the Society of Thoracic Surgeons [STS]).[10]

This statement stresses the relevant accurate invasive hemodynamic information obtained by the utilization of the pulmonary artery catheterization during the monitorization for CS, measuring directly right atrial pressure (RA), pulmonary artery pressure (PA), pulmonary capillary wedge pressure (PCWP), mixed venous oxygen saturation and cardiac output (CO), which derives cardiac index (CI), systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), pulmonary artery pulsatility index (PAPi), and cardiac power output (CPO).

This tool is essential for early recognition, differential diagnosis, phenotyping, therapeutic titration, escalation to mechanical circulatory support (MCS), weaning of therapies, prognosis, and identification of univentricular versus biventricular failure. This expert panel recommends invasive pulmonary artery monitoring in CS and recognizes the reluctance for its utilization based on currently unjustified controversy.

Unfortunately, the controversy about the invasive right heart monitoring currently provokes its underuse, surely with a significantly negative impact on CS patients, because the old studies did not include a significant volume of

patients with CS or those treated with MCS, while there is indeed a significantly lower mortality in CS under SG monitoring (29.7% versus 38.1%). This kind of monitoring, when properly managed and interpreted, may help to identify worsening heart failure and CS and will help to guide treatment in clinically conflicting and mixed shock conditions.[4]

Finally, severe right ventricle dysfunction may require continuous right heart monitoring, particularly during intense bi-ventricular failures, such as right coronary-related myocardial infarction with significant right ventricle involvement, in which the simultaneous monitoring of the pulmonary artery and right atrial pressures, is valuable to determine the diastolic relationships between both.[11]

Several medical organizations wrote current guidelines for invasive right heart monitoring (American College of Cardiology Foundation, American Heart Association, European Society of Cardiology, Heart Failure Society of America, International Society of Heart and Lung Transplantation), as follows:[12]

1. On anesthesia induction on CS patients for coronary bypass graft surgery (class I; level of evidence C).
2. To estimate intracardiac filling pressures on respiratory distress or impaired perfusion with clinical discrepancy (class I; level of evidence C).
3. On heart failure persistence despite therapeutic adjust and any of the following (class IIa; level of evidence C):
   a. Uncertain systemic or pulmonary vascular resistance, fluid or perfusion status.
   b. Unresponsive hypotension.
   c. Worsening renal function.
   d. Need for vasopressors.
   e. On candidates for mechanical circulatory support or heart transplantation.
4. On patients with mechanical circulatory support (class I; level of evidence B).
5. On hemodynamic instability due to unknown worsening mechanism or refractory heart failure (class IIb; level of evidence C).
6. To withdraw mechanical circulatory or pharmacologic support.

RECOMMENDATIONS

Regarding the utilization of the Swan-Ganz catheter for continuous right heart monitoring, this consensus recommends:

1. The SG catheter is a hemodynamic diagnostic tool; it is not a device for treatment.
2. Do not utilize the SG catheter to monitor respiratory insufficiency without heart failure.
3. Indicate the SG catheter on any cardiogenic shock from any etiology and at any class and level, involving one ventricle or both.
4. Consider the SG catheter:
   a. During worsening heart failure/ hemodynamic instability, despite adequate treatment.
   b. For differential diagnosis during failed treatment for respiratory distress, hypotension, and or progressive renal failure.
5. Consider simultaneous monitoring of the pulmonary artery and right atrial pressures during severe right heart-related shock.
6. Encourage centers with low SG utilization to include and master its hemodynamic monitoring benefits.

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