Heartbeat: treatment delays with telephone triage for acute myocardial infarction

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Prevention of myocardial damage and optimal outcomes in patients with acute myocardial infarction (MI) are achieved when primary percutaneous coronary intervention (PCI) is performed as soon as possible after symptom onset. Although some patients present directly to the emergency department or call an ambulance, others use telephone triage services which became the preferred route for receiving care in the UK during the COVID-19 pandemic. In order to identify issues that might lead to treatment delays with the telephone triage approach, Hodgins and colleagues[1] performed a retrospective study of all people admitted to Scottish hospitals with a diagnosis of MI between 1 January 2015 and 31 December 2017. In these 26 325 patients (63.1% men, 61.6% aged 65+ years), 47.0% called an ambulance, 23.3% presented directly to the emergency department and 18.7% called telephone triage. Patients who experienced multiple steps in the process between the initial contact and hospital admission had a higher mortality whether the initial contact was telephone triage (aOR 1.97, 95% CI 1.61 to 2.40) or another service (aOR 1.55, 95% CI 1.19 to 2.01).

In the accompanying editorial, Sze and colleagues[2] point out the challenges in early diagnosis of MI, especially by phone triage. 'Indeed, even when senior medical input is involved in the triage decision-making, myocardial infarction only accounts for one in nine of chest pain call-outs' (figure 1). They remind us that 'inclusion of high-sensitivity troponin measurement in algorithms which facilitate the early triage of chest pain has proven to be pivotal in safely ruling out myocardial infarction' and they suggest that prehospital point-of-care troponin measurements might be both safe and efficient in reducing delays in treatment in patients with MI, regardless of how the patient initially accesses the medical care system.

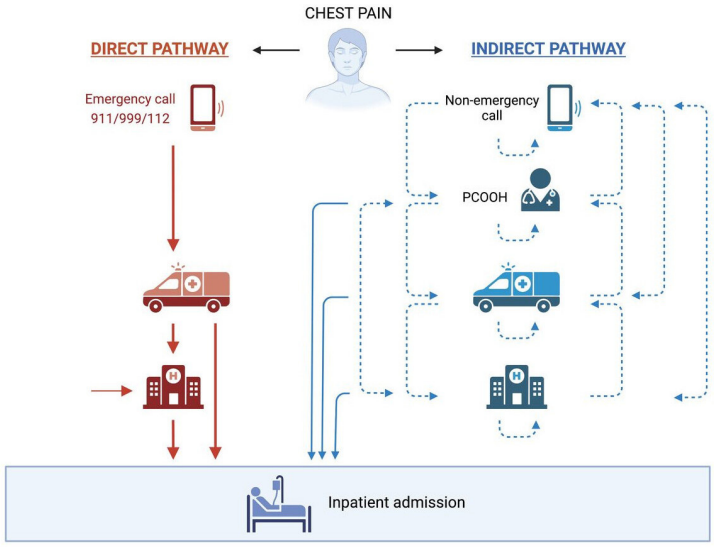

Figure 1 Direct and indirect pathways in the triage of suspected myocardial infarction. Direct pathways have fewer points of contact between the onset of symptoms and admission to a hospital bed. Direct pathways include early activation of emergency responders to make a rapid diagnosis and initiate optimal myocardial infarction management protocols. In contrast, indirect pathways for suspected myocardial infarction have multiple pre-admission points of contact following the onset of symptoms resulting in unnecessary system delays in diagnosing myocardial infarction. Indirect pathways occur more frequently when a non-emergency call handler or primary care out-of-hours (PCOOH) is the first point of contact.

A congenital bicuspid aortic valve (BAV) is present in about 1% of the population with familial inheritance in some patients. However, identification of a pathogenic gene variant in BAV patients has been elusive. Mutations in the NOTCH1 gene have been reported in some families but the relative importance of this gene variant across the population has been unclear. In this issue of *Heart*, after

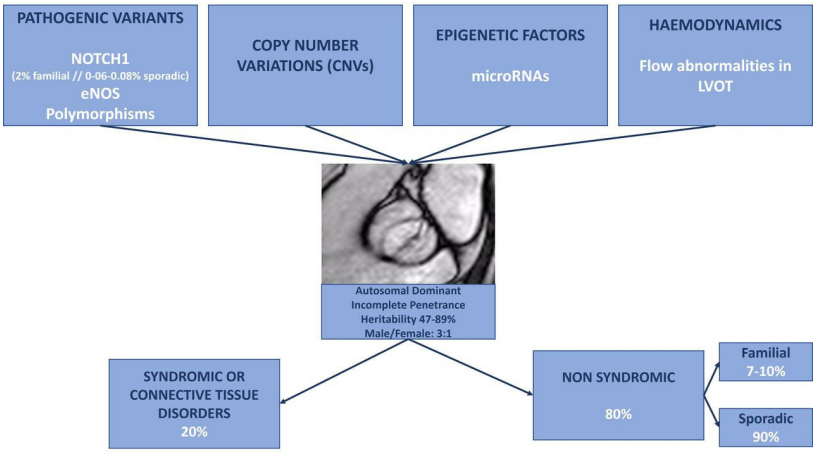

Figure 2 Schematic representation of the different aetiological mechanisms of the BAV, its inheritance pattern and its forms of expression in the different individuals. BAV, bicuspid aortic valve; eNOS, endothelial nitric oxide synthase; LVOT, left ventricular outflow tract.

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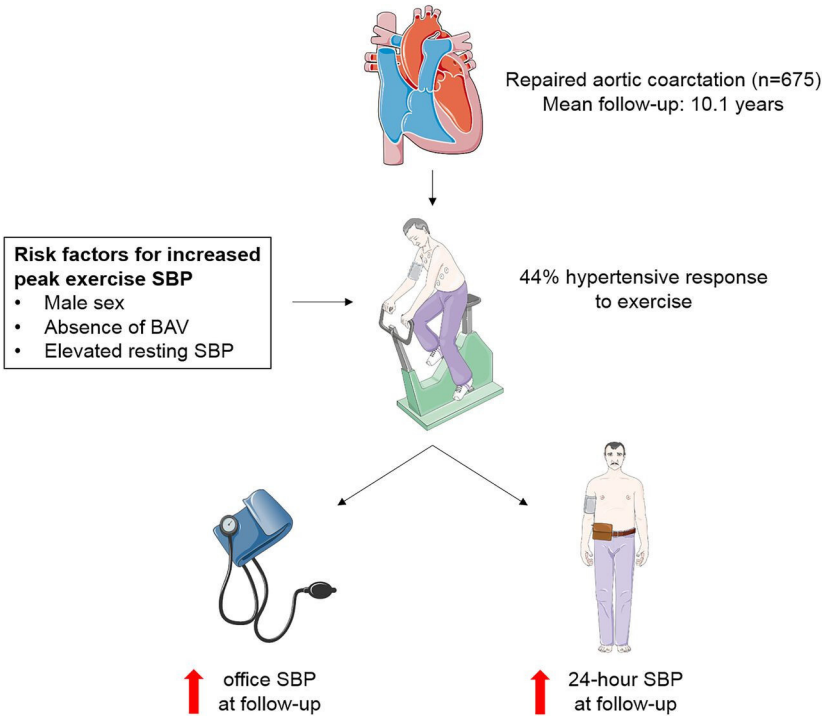

Figure 3 Graphical summary of the main findings in this study. Images from Servier Medical Art (smart.servier.com) were used to create this figure. BAV, bicuspid aortic valve; SBP, systolic blood pressure.

exclusion of syndromic patients, Debiec and colleagues[3] identified likely pathogenic or likely pathogenic NOTCH1 variants in 9/435 (2.1%; 95% CI 0.7% to 3.4%) patients with familial inheritance. In patients with a sporadic BAV, a pathogenic NOTCH1 variant was found in only 0.05% (95% CI 0.005% to 0.10%) and a likely pathogenic variant in 0.08% (95% CI 0.02% to 0.13%). Rodriguez-Palomares[4] discusses the complexities of genetic associations with BAV disease and provides the perspective that this data confirms 'that NOTCH1 variants explain only a small proportion of BAV disease and are associated with more complex congenital phenotypes such as tetralogy of Fallot or hypoplastic left heart syndrome.' Rodriguez-Palomares concludes that 'Collectively, the available evidence supports the notion that the clinical heterogeneity of BAV involves complex interactions between primary genetic defects, other genetic factors (gene modifiers), epigenetic factors (DNA methylation or histone modifications, miRNA) and haemodynamic abnormalities in the aortic mechanics and valve morphology (figure 2).

Another interesting paper in this issue by Meijs and colleagues[5] found that almost half of adults with a repaired aortic coarctation have a hypertensive response to exercise and that exercise systolic blood pressure independently predicted hypertension at follow-up (figure 3).

Commenting on this study, Lee and Grigg[6] conclude that 'Currently, exercise stress testing may be best used in identifying patients with repaired coarctation with normal resting blood pressure who may be at increased risk of developing hypertension in the future. However, it is currently unknown whether conventional antihypertensive treatment is effective for a hypertensive response to exercise (or other forms of hypertension for that matter) in adults with repaired coarctation.'

A review article[7] on the cardiovascular effects of scuba diving will provide clinicians with the information needed to advise patients with heart disease who wish to participate in this activity (figure 4). A detailed flow chart is presented for evaluation and risk stratification of patients, as well as recommendations for specific cardiac conditions. Given the lack of a robust evidence base, the authors recommend that 'A patient-centred approach facilitating shared decision-making between divers and experienced practitioners should be used in the management of prospective scuba divers.'

The *Education in Heart* article[8] in this issue provides an overview of diagnosis and management of adults with congenital left-sided obstructive lesions including both inflow and outflow obstruction. Examples of congenital inflow obstruction include cor triatriatum, congenital pulmonary vein stenosis and congenital mitral stenosis. Examples of outflow obstruction include left ventricular outflow obstruction (subvalvular, valvular and supravalvular aortic stenosis) and aortic coarctation.

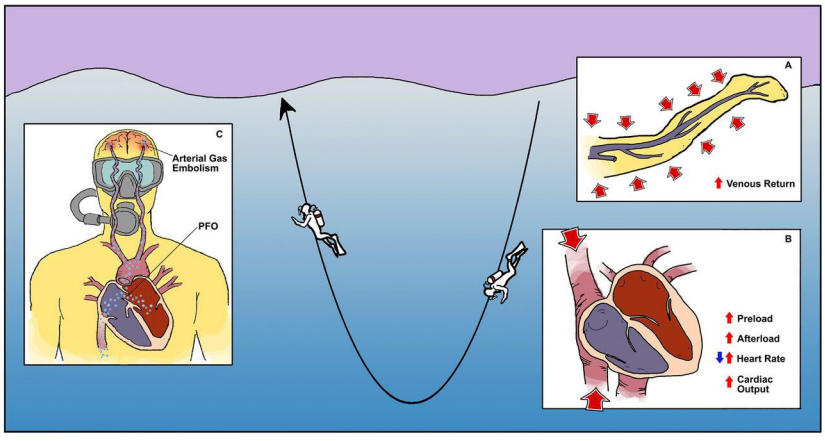

Figure 4 Physiological effects of scuba diving (A and B) and potential pathophysiological sequelae with rapid ascension (C). (A) Increased hydrostatic pressure leads to increased venous return from the extremities. (B) Haemodynamic shifts and mixed autonomic response to depth and temperature lead to increased preload and afterload and decreased, then increased heart rate, all yielding a net increase in cardiac output. (C) With rapid ascent, the formation of inert gas bubbles increases the risk of paradoxical arterial gas embolism, including in those with high-grade patent foramen ovale (PFO).

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