

but it is not clear that they are being implemented. The EuroHeart Failure Survey reported that only 24% of patients with HF recalled receiving advice to stop smoking.⁹ It is possible that a much higher proportion of patients received advice about smoking but, if so, the fact that they could not recall doing so indicates a failure in the strategy. It is also possible that HF is viewed as a near-terminal event and that it is too late to advice on smoking. This is a matter for debate, especially as smoking may adversely affect symptoms, exercise capacity, and quality of life as well as prognosis. Whether this calls for different smoking cessation strategies and what is the potential of such an intervention needs to be addressed in properly conducted trials.

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Smoking cessation in heart failure: easier said than done: reply

We are thankful to Dr Lainscak and colleagues for their interest in our article on the association between smoking and clinical outcomes in patients hospitalized with heart failure.¹ Several key issues are raised about the findings from OPTIMIZE-HF that smokers hospitalized with heart failure had lower risk adjusted in-hospital mortality, shorter length of stay, and similar early post discharge mortality compared with non-smokers. We concur that in addition to the variables included in our models, there are additional variables that, had they been adjusted for, may have strengthened or weakened the observed relationship between smoking status and clinical outcomes. A number of paradoxical associations between traditional cardiovascular risk factors (cholesterol, body mass index, and blood pressure) and clinical outcomes in patients with established heart failure have been previously reported by us and others.² The authors are in complete agreement that further studies are needed to confirm our findings before fully accepting that smoking represents another example of a paradoxical relationship in heart failure.

Effective smoking cessation methods should be vigorously employed in individuals with heart failure, including those hospitalized. In OPTIMIZE-HF, only 62.8% of smokers were documented to have received smoking

cessation counselling prior to discharge.¹ However, as previously reported, we did not find that smoking cessation counselling as documented by OPTIMIZE-HF participating hospitals was associated with improved post-discharge clinical outcomes.³ Smoking cessation counselling in the hospital setting may range in intensity from a generic statement in discharge instructions to detailed individualized counselling with outpatient program referral. Smoking cessation counselling as currently provided to hospitalized heart failure patients may not be sufficient to result in maintenance of actual abstinence from smoking post-discharge. More intensive hospital-based counselling, use of nicotine replacement/suppression therapies, and referral to outpatient cessation programs may be required to achieve clinical benefits.

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Tako-tsubo cardiomyopathy or aborted myocardial infarction?

We read with great interest the recent article by Eitel *et al.*¹ regarding the role of contrast-enhanced magnetic resonance imaging (MRI) in the differential diagnosis of suspected apical ballooning syndrome (ABS). The authors studied 59 consecutive patients with diagnosis of acute coronary syndrome, normal coronary arteries, and classical apical ballooning on left ventriculography; ABS was

diagnosed by MRI if patients had no signs of delayed enhancement typical for ischaemic heart disease or delayed enhancement patterns of myocarditis. Magnetic resonance imaging represents a powerful clinical tool for differentiating infarcted from normal (viable) myocardium and for characterizing the tissue pathology (necrosis, fibrosis, and oedema). Sub-endocardial or transmural myocardial delayed enhancement provides strong evidence of myocardial infarction but, when absent, it is not possible to distinguish tako-tsubo cardiomyopathy from a truly aborted myocardial infarction without myocardial necrosis. The features of ABS on cardiac MRI are myocardial oedema in the apical region in absence of necrosis, fibrosis, or scar formation. This 'pattern' of myocardial oedema is not typical for ABS: it also occurs in irreversibly injured myocardium, representing the marker of myocardium at risk of inflammation, or, in absence of late enhancement, it seems to be the magnetic resonance marker of myocardium 'at risk' of infarction that has been early reperfused and aborted.²

The early spontaneous reperfusion of acute thrombotic occlusion of the coronary artery could be the explanation of a self-limiting ischaemic episode. In this case, in fact, myocardial necrosis is either absent or very small and myocardial delayed enhancement may not be demonstrated. Moreover, the typical tako-tsubo wall motion abnormalities may be seen in patients with acute myocardial infarction and myocardial delayed enhancement on MRI in the presence of only mild atherosclerosis in the coronary artery. The involvement of the entire apical segments could be explained by the presence of a large portion of stunned myocardium around the infarcted myocardium.³ In a practical point of view, ABS may include a small number of patients with aborted myocardial infarction with typical apical ballooning pattern. Only a more accurate study of the left anterior descending coronary artery (i.e. with intravascular ultrasound or optical coherence tomography) is able to identify the presence of occult atherosclerotic plaques, not detected by conventional angiography.^{3,4} In conclusion, cardiovascular MRI may be useful in the assessment of ABS by demonstrating segmental dysfunction in the absence of myocardial irreversible damage (delayed enhancement) but, if myocardial delayed enhancement is absent, it may be difficult to distinguish ABS from a completely aborted myocardial infarction.

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Tako-tsubo cardiomyopathy or aborted myocardial infarction?: reply

We thank Dr Cattaneo and colleagues for their interest in our work.¹ Indeed, it appears that the typical 'apical ballooning shape' and the occurrence of oedema in tako-tsubo cardiomyopathy (TTC) is not

unique to this entity, and may also be seen in myocardial infarction (MI) and classical myocarditis. However, a lack of late gadolinium enhancement (LGE) in cardiovascular magnetic resonance (CMR) in the dysfunctional apical regions usually allows distinguishing between these different causative aetiologies including MI (subendocardial or transmural LGE corresponding to a vascular territory), infiltrative disease (typically diffuse or patchy LGE), and many cases of myocarditis (frequent but not universal presence of epicardial or patchy LGE).^{1,2}

We agree with Dr Cattaneo that a true aborted MI without myocardial necrosis (LGE) cannot be definitely distinguished from TTC using CMR. Nevertheless, in a recent study, most of the patients with fulfilled criteria of aborted MI showed LGE in the distribution of a coronary artery.³ The few patients with true aborted MI with the absence of myocardial scar had all a preserved left ventricular ejection fraction and no signs of wall motion abnormalities.³

The pathophysiological hypothesis of myocardial stunning resulting from a spontaneously aborted MI in patients with TTC was first described by Ibanez *et al.*⁴ In five consecutive patients, they demonstrated the presence of plaque rupture by intravascular ultrasound that was not detected by angiography. According to the authors, the involvement of the entire apical segments could be explained by the presence of a long recurrent distal left anterior descending (LAD) artery supplying a significant area of the inferior left ventricular segments.⁴

However, there are several reasons why this mechanism is unlikely to account for the pathophysiology of most cases in TTC. First, the regional wall motion abnormality is far greater than what can be accounted for by even a large wrap-around LAD. Second, plaque rupture has not been reported in any other large case series. Although no intravascular ultrasound was performed, in most cases the coronary arteries were completely normal making any plaque rupture extremely unlikely. Third, extensive regional wall motion abnormality of the right ventricle in one-third of patients cannot be explained on the basis of LAD territory ischaemia. Fourth, the female predominance would be unusual for a manifestation of aborted MI. Fifth, new patterns of TTC involving middle or basal and not apical segments have been recently described and it seems difficult to relate the transient midventricular or basal dysfunction to an ischaemic event.^{5,6}

In summary, CMR contributes to an understanding and differential diagnosis of this new entity by demonstrating the absence of