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Inferior myocardial infarction scars could be more arrhythmogenic than anterior ones: reply

We read the letter by Dr Čulić and we are grateful for his comments. We would like to reply to some of the issues raised regarding the hypothesized mechanisms underlying the increased susceptibility to substrate-related arrhythmias found in inferior myocardial infarction (MI). We suggested that protective vagal responses may be reduced after inferior MI given the preferential distribution of vagal sensory endings in the inferior wall. This hypothesis is questioned by Dr Čulić based on a study that demonstrated that cardiac parasympathetic activity, assessed by heart rate variability, was more reduced in anterior compared with inferior MI.[1] Some limitations however deserve emphasis. The difference in heart rate variability was apparent only during the first week after MI. Precisely because of the preferential distribution of vagal sensory endings, it is well known that the acute phase of inferior MI is often associated with transiently enhanced vasodepressor and cardioinhibitory reflexes, whereas anterior MI more frequently evokes sympathetic activation. Though the time after acute MI at which the depressed heart rate variability reaches the highest predictive value has not been studied, the general consensus is to assess heart rate variability at least 1 week after index MI. Of note, a study performed in the subacute and chronic phase of MI found heart rate variability to be more reduced in patients with inferior MI than in those with anterior MI.[2] Moreover, heart rate variability has been shown to be significantly related to left ventricular function, peak creatine kinase, and Killip class in the acute phase of MI.[3] The difference seen between anterior and inferior MI can therefore not be merely ascribed to the location of MI but may simply reflect the extent of MI. Finally, heart rate variability was used in the study to assess cardiac parasympathetic activity. This methodology reflects primarily tonic vagal activity, whereas baroreflex sensitivity, the other clinical autonomic marker of clinical interest, reflects predominantly reflex vagal activity. It has been shown experimentally that measures of vagal reflexes may be more useful than measures of vagal tone in identifying patients at higher arrhythmic risk in the absence of ischaemia or of a recent MI.[4] This was later supported in clinical studies.[5–6] Studies using this latter autonomic marker were therefore used to support our hypothesis. Baroreflex sensitivity has besides also been shown to be a more independent marker of arrhythmic risk since no correlation was found with left ventricular function.[7]

Dr Čulić also raises the interesting suggestion that the difference in the susceptibility of posterior and anterior papillary muscles (PMs) to infarction may potentially explain the propensity of inferior MI to suffer from substrate-related arrhythmia. Experimental and clinical evidence however challenges this hypothesis. Papillary muscles may contribute to post-infarction ventricular arrhythmias in two ways. First, as mentioned by Dr Čulić, surviving muscle bundles embedded within scarred PMs may give rise to circuitous conduction pathways that may lead to reentrant ventricular tachycardia. Secondly, the safety factor of impulse propagation depends on the relation between the source (amount of current available in the propagating wave front) and the sink (structure that determines current density when the wave front arrives). Papillary muscle creates a relative increase in muscle thickness and therefore increases the current sink for the incoming wave front. This may decrease the safety factor of impulse propagation resulting in conduction block and the formation of reenty.[8]

Both situations however require electrically active tissue and would not occur in complete PM infarction. In support of this, a recent clinical series of post-infarction patients with ventricular arrhythmia originating in PM reported that all arrhythmogenic PMs demonstrated heterogeneous uptake of gadolinium during magnetic resonance imaging, whereas none of the PMs with homogenous uptake were involved in the genesis of ventricular arrhythmias.[9]

The hypothesis raised by Dr Čulić is therefore questionable, since the posterior PM, owing to its frequently singular blood supply, has been shown to be more often involved in complete infarction and subsequent rupture rather than partial infarction. It is as yet, however, not known whether the latter occurs more often in posterior compared with anterior PM.

Conflict of interest: none declared.

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