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Atrial fibrillation ablation: beyond electro-mechanical matters: reply

We thank Dr Martignani et al. for their interest in our work.1 We totally agree that the drop of ANP level at Day 1 after ablation is probably the consequence of acute atrial damage caused by radiofrequency (RF). An acute atrial endocrine stunning was previously reported with surgical AF ablation.2 However, in our study, it reverses within 3 days with concomitant elimination of fluid overload. But the most interesting point is that at 3 months, despite extensive RF application, the ANP level was normal in the vast majority of patients being in sinus rhythm, meaning that there is no longer evidence of atrial endocrine injury at 3 months.

We also agree that LV function is very difficult to assess in AF and that is the reason why LV dimensions were measured in sinus rhythm just after ablation (Table 2).1

We also agree that rhythm control strategy has not been proved to be more effective than rate control, but all studies cited by Dr Martignani et al. involved anti-arrhythmic drugs and not catheter ablation. Moreover, the percentage of patients with effective sinus rhythm in the rhythm control group was not so different from the rate control group in these studies (63% vs. 35% in AFFIRM3), and the maintenance of sinus rhythm was associated with a decreased risk of death (HR = 0.53).4

Concerning the indication of persistent and permanent AF ablation, the aim of our study was not to promote AF ablation in all patients with persistent/permanent AF. Ablation in this population is a long procedure with a potential risk of complications and requires experienced centres. Those relatively young patients (53 ± 12 years) included in our study were referred for catheter ablation because they were highly symptomatic (asthenia, dyspnoea, and 12% had left ventricular ejection fraction (LVEF) < 50%). At the present time, there is no doubt that catheter ablation of persistent/permanent AF ablation should not be performed in symptomatic patients without LVEF alteration.

Concerning the complex interaction between AF and impaired LVEF, the patient’s history is crucial. In cases where AF occurs following the onset of heart failure (HF) without a change in the LVEF, it is unlikely that AF is contributing to the HF. Conversely, some patients with established HF demonstrate further impairment of LVEF following the occurrence of AF and these patients may well benefit from catheter ablation.5 Rate is not the only factor in these patients as an improvement in LVEF may be seen even in cases of good rate control.5,6

In most cases, we agree that it is difficult to assess the impact of AF. However, recent studies have consistently showed a very significant improvement in LV function following AF ablation, suggesting that the deleterious impact of AF has been largely underestimated in the past, when drugs or ‘ablate and pace’ were the only available strategies.

To conclude, the strategy to treat patients with persistent/permanent AF has to be tailored to every single patient. The patients must be extremely well informed in this decision process.

References

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Suggestion to list acute aortic dissection as a possible cause of type 2 myocardial infarction (according to the universal definition)

The most feared cause of acute myocardial infarction (AMI) has been aortic dissection (AD), because the antithrombotic and thrombolytics treatments double hemorrhagic complications and mortality. Cannesson et al.1 found 25 patients with AMI induced by AD, erroneously treated by fibrinolysis. There have been more recent reports on this probably underreported problem with mortality 69–100%.

There is a long-standing debate about imaging testing for AD in ACS. Argument pro is to omit dangerous antithrombotics and thrombolitics or high doses of antithrombotics (if primary PCI is prepared) in AD. Argument contra is not to lose time needed for imaging study for AD, because ‘time is myocardium’ in AMI and a great majority of AMI patients do not have AD. The difference in incidence of AMI vs. AD is high: the ratio is 750:1 or nearly 800:1 and only a minority (roughly 2–5%) of AD patients has AMI, making this ratio much higher.

Thus, it may not be clever to delay the antithrombotic therapy in overwhelming majority of AMI patients for the sake of rare patients with both AD and AMI. According to the current balanced guidelines, patients with ACS and features suggestive for AD ought to be checked for the presence of AD.1,2,4

AD as a cause of AMI can be overlooked, because many usual risk factors described...
Clinical signs of ischaemia (among others) in cell necrosis might occur in the absence of definition of MI, it is stated that myocardial demand/supply imbalance (and type 2 MI, etc. Thus, AD may mimic AMI. To the contrary, AD may cause AMI. AD can masquerade as or actually produce AMI, accordingly.

In the cornerstone document ‘Universal definition of MI’, it is stated that myocardial cell necrosis might occur in the absence of clinical signs of ischaemia (among others) in AD. Also in the ESC Guidelines for NSTE-ACS, AD was mentioned as a non-coronary condition with troponin elevation.

On the other hand, there have been numerous descriptions of direct influence of AD upon coronary artery, producing myocardial ischaemia/infarction by coronary artery: (i) compression (by false lumen); (ii) ostium obstruction (by intimal flap); (iii) dissection; and (iv) avulsion.

Neri’s classification has been useful, too. All aforementioned direct mechanisms suggest the decrease/cease of the coronary artery blood flow, with the consequent diminished myocardial supply (recognized as type 2 MI). Indirect mechanisms may be operative in AD too, leading to myocardial oxygen demand/supply imbalance (and type 2 MI, consequently): hypertension, arrhythmia/tachycardia and spasm (due to sympathetic/ catecholamine surge) or hypotension and anaemia (due to aortic rupture/blood leaking).

There have been extremely rare reports (we found only two) suggesting even thrombus in a coronary artery in patients with both AD and AMI. Accordingly, chances for real type 1 AMI produced by AD are minimal. To conclude, there is little doubt (if at all) that AD can cause AMI. If so, we suggest that aortic dissection should be listed as a possible cause of type 2 AMI.

References

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Comment on ‘Comparison of operator radiation exposure with optimized radiation protection devices during coronary angiograms and ad hoc percutaneous coronary interventions by radial and femoral routes’

We read with extreme interest the paper by Brasselet et al.1 showing an increased radiation exposure for operators performing percutaneous coronary angiographies and interventions through the radial approach in comparison to the femoral access. We believe the prospective, operator-blinded design and the careful acquisition of the radiation exposure data are major strengths of this study. However, there are limitations that in our opinion seriously undermine the conclusions of the manuscript, where the authors suggest reconsidering promptly the radial approach in light of their findings.

First, the non-randomized nature of this study is a major drawback and the authors did not do any effort to adjust statistically their results at least for the available variables collected. For example, body weight has been found to be significantly higher among patients in the radial group.1 Body weight can influence the total amount of X-rays needed to visualize properly the coronary arteries as the X-ray apparatus automatically increases or reduces the emission according to the impedance that finds from the body of the patient. An adjustment of the results according to the baseline data is needed in a study designed as a registry, and a regression analysis or a propensity matching analysis would be extremely welcome.2,3

Secondly, no mention of the possible rates of crossover from femoral to radial access or vice versa has been made or considered, and this would be also important, mainly in an intention to treat analysis.

Thirdly, it is not clear whether the procedures were performed all in one room or in different rooms. The authors mention only that the cine-angiography units were the same and were all 9 years old; however, if located in different rooms, it is possible that a different rate of use of these machines could have had an impact on the results.

Finally, safety is a comprehensive issue, and radiation safety is only a part of the whole picture. Periprocedural bleedings after percutaneous coronary procedures threaten safety.4,5 Yet, no mention of the minimization of bleeding risks by the radial access and of its potential impact on overall patient safety is made in the analysis or discussion.6

In conclusion, the radial approach has been repeatedly shown to have several benefits over the femoral access for coronary angiographies and interventions in thousands of patients.6 We believe that a single registry of 420 patients without a randomized design cannot prompt to reconsider the radial approach for these indications. On the other hand, seen the provocative results of this study and the expertise of the authors in radiation exposure measurement, we urge them to plan a prospective randomized comparison, setting radiation exposure as primary endpoint and using the results of the current pilot study to define a proper power analysis and an adequate sample size.