A reply

The authors appreciate the important comment made by Kamath and Lip that a hypercoagulable state may be yet another mechanism increasing the risk of thromboembolism in the setting of cardioversion of atrial fibrillation. In our Editorial on the study by Roijer et al.[1], we tried to focus on the mere ‘mechanical’ aspects of thrombus formation in the atria after cardioversion. Yet, as Kamath and Lip point out, there is evidence that patients may be in a hypercoagu-

able state during atrial fibrillation. In fact, pharmacological cardioversion of recent-onset atrial fibrillation may also lead to some increase in markers of thrombin activity underscoring the impor-
tance of a hypercoagulable state in this setting[2]. However, the fact that most thrombi originate in the left atrial appendage indicates that the mechanical dysfunction is the primary insult and that increased coagulability may be a contributing factor, i.e. the reduced contractility during atrial fibrillation or post-cardioversion probably leads to blood stasis which then leads to a hypercoagulable state.

As again pointed out by Kamath and Lip, unfractionated heparin is widely used in patients with de novo atrial fibrillation although this is largely based on clinical judgement rather than evidence-based medicine. The advantages of using low-
molecular-weight heparin in these patients seem obvious, although there are again no randomized studies available. In addition to the ACUTE II study mentioned by Kamath and Lip a similar study comparing enoxaparin to a conventional anticoagulation scheme (either with or without guid-
ance by transoesophageal echocardiography) is currently being performed in Germany, the ACE study (Antico-
agulation for Cardi oversion With Enoxaparin). Hopefully, these two studies will answer many of the still open questions.

We also agree that the data of the ACUTE-I study—even though not yet published as a full paper—were somewhat disappointing because the study was simply underpowered. Unfortunately, our Editorial was sub-
mitted to the European Heart Journal before these data were presented at the meeting of the American College of Cardiology last year. Clearly, more questions were raised than answers provided by the ACUTE data. More studies are needed in order to clarify whether an approach guided by trans-

esophageal echocardiography is as safe as the conventional approach, whether it is necessary in all patients undergoing cardioversion and what the optimal anticoagulation scheme should be, especially in ‘short-lasting’ atrial fibrillation.

Finally, the question, whether aggressive restoration and mainten-
ance of sinus rhythm is beneficial compared to rate control and anti-

ticoagulation, is currently being investigated in two trials: the PI AF-

study (Pharmacological Intervention in Atrial Fibrillation), performed in Germany[3], and the AFFIRM trial in the U.S.[4]. Both studies, despite many differences in study design and size, principally compare two treatment strategies: restoration and mainte-
nance of sinus rhythm vs rate control during atrial fibrillation. The results of both studies are eagerly awaited.

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Cardiac nomenclature

The disposition and anatomy of the atrioventricular valves are not adequately or accurately described in the Consensus statement from the Cardiac Nomenclature Study Group of Arrhythmias, European Society of Cardiology and the Task Force on Cardiac Nomenclature from the North American Society of Pacing and Electrophysiology by Cosio et al.[1].

The coordinates and disposition of the atrioventricular valves and coronary sinus are misleading because the structures are depicted inverted in the left anterior oblique schematics in Figs 1, 2 and 8. In Fig. 1, the inverted valves and coronary sinus are depicted in the upright human model so that the aorta and pulmonary artery appear to be positioned posteriorly. Recognizing distortions in anatomical positions due to optical illusions in radiographic views is an important first step for accurately interpreting fluoroscopic images. The inversion can be appreciated by direct comparison of photographs of the transected hu-

man heart in Fig. 4[1] to the same region in the inverted breadloaf slice of the dog heart (Fig. 4B)[2]. The righted opposite (flip) side of the slice in this case (Fig. 4A)[3] shows the course of coronary sinus adjacent to the left atrium and its entry into the inferoposterior medial atrial wall. Moreover, the atrial septum does not come in contact with the valves but instead the septum is limited to a superior region of the fossa ovalis[3] (Fig. 1C)[4]. The atrioventricular junction region contains no atrial septal component, but rather only in-

ferior regions of the medial atrial wall[3]. Terms such as septal and paraseptal are therefore inaccurate as applied.

The anatomy of the atrioventricular junction is also not accurate in their schematics in Figs 1, 2 and 8[1]. As seen in the human heart (Fig. 4) and in the dog heart[2] at this same level, the aorta shares a common wall with the right ventricle in the form of the ventriculoaortic septum (Fig. 4B)[2] (Figs 4A–D, 5A–D)[3] (and also more
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superiorly with the right atrium via the atrioaortic septum) and is continuous with the crista supraventricularis (Figs 2A, 4B, 7B)[2]. It is anatomically inaccurate to exclude these structures from their schematics because the septum is an integral component of the tricuspid valve and the crista supraventricularis occupies the anterior region shown as ventricular septum. Histological sections confirmed these important relationships and contacts with the conduction tissues in perpendicular, transverse (Figs 4A-D, 5A-D)[3] and parallel orthogonal planes (Fig. 7A, B)[3].

These crucial anatomical relationships deserve serious consideration by all who are working in the living heart, as rupture of the crista supraventricularis could cause tamponade with leakage of blood into the pericardial sac, whereas rupture of the septa as with aortic aneurysms would provide patent high–low pressure communications, and the clinical consequence of these events have been reported[4].

Thus, if the intent is to provide accurate designations as teaching aids, the proposed nomenclature and anatomic depictions must be accurate.

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References

Atrioventricular junctional structures

We welcome the opportunity to reply to the letter of Dr Racker. Our purpose in producing the review, however, was not to discuss the detailed anatomy of the atrioventricular valves[5]. Rather, we wished to show that, at present, there was a discrepancy between the way the atrioventricular junctional structures are described relative to their true locations within the body. And, of course, all structures within the body are described relative to the anatomic position, which is why we depicted them in the upright human model. Dr Racker, however, dislikes our depiction of Figs 1, 2 and 8 because they ‘are depicted in the upright human model so that the aorta and pulmonary artery appear to be positioned posteriorly’. As explained, the images are shown in this position because that is how the interventionist sees them on the fluoroscopic screen in the electrophysiological laboratory. In this respect, it is inadvisable to make comparisons with the canine heart, as suggested by Dr Racker, because the dog never uses the upright posture. Furthermore, we have purposely not shown the aorta and the pulmonary trunk in any of these figures.

We are further criticised for apparently stating that the atrial septum comes into contact with the valves. Nowhere in our review do we make this statement. We certainly illustrate a ‘septal’ component of the right atrioventricular junction, but we do not argue that this area is occupied by the atrial septum. In fact, this is the area where the septal leaflet of the tricuspid valve takes origin from the ventricular septum, and hence is accurately depicted as ‘septal’. The aorta does not share a common wall with the right ventricle and the right atrium. The areas which Dr Racker describes as the ‘ventriculo–aortic’ and ‘atrioaothic’ septums are better known as the interventricular and atrioventricular components of the membranous septum, respectively. These are an integral part of the aortic root, or in other words the subaortic outlet component of the left ventricle, but they are not part of the aorta. The aorta, of course, is limited proximally by the semilunar lines of attachment of its valvar leaflets. The areas in dispute are on the ventricular aspect of these lines of attachment. They are, therefore, an integral part of the left ventricle, but not the aorta. In this respect, the membranous septum is, indeed, an important component of the right atrioventricular junction. This is why part of the junction is correctly labelled as ‘midseptal’ in Fig. 1, and ‘septal’ in Fig. 8, albeit that the area occupied by the membranous septum is perhaps smaller than implied in these figures.

We do not imply that the supraventricular crest is part of the ventricular septum. It is common knowledge that the supraventricular crest is part of the free-standing sleeve of the muscular subpulmonary infundibulum which lifts the leaflets of the pulmonary valve away from the body of the right ventricle, and makes possible the Ross procedure. But this fact has nothing to do with the structure of the atrioventricular junctions, and again was purposely not included in our review.

In summary, we agree totally with Dr Racker that both nomenclatures and anatomical description must be accurate. We see nothing in her letter which shows that ours are incorrect.

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Reference