intercalated disc surface area. In our study, we found an 8.5 times larger quasi-macroscopic currents in patches from the intercalated disc region, which, as set out by Petitprez and Abriel, would imply that cardiac and brain-type whole-cell Na\(^+\) currents have similar amplitude. We fully agree with Petitprez and Abriel that this amount of calculated brain-type Na\(^+\) current contrasts with findings in ventricular myocytes of mouse, dog, and rat, where brain-type Na\(^+\) currents account for <20% of the whole-cell Na\(^+\) current [see Verkerk et al.\(^1\), and primary references cited therein]. Since we used ventricular myocytes of rabbit, species differences may account for the contrasting findings. We have performed pilot experiments in rabbit ventricular myocytes in which we have tested the effects of 100 nM TTX on whole-cell Na\(^+\) current, using experimental conditions as we described previously.\(^4\) We found that 100 nM TTX never blocked more than 15% of whole-cell Na\(^+\) current, thereby excluding species differences as a potential explanation.

The simplified myocyte morphology as used by Petitprez and Abriel in their calculation, i.e. a cylinder with two intercalated discs (top and bottom of the cylinder) and a length-to-width ratio <5, may also contribute to the discrepancy. In reality, ventricular myocytes are not simple cylinders; intercalated discs can be found also along the lateral sides,\(^5\) the length-to-width ratio is 7–8,\(^6\) and the number of intercalated discs is ~10 [see Jongsma and Wilders\(^7\) and primary references cited therein]. Although an increase in length-to-width ratio will increase the calculated ratio of brain-type and cardiac Na\(^+\) current, the higher number of intercalated discs may result in a lower ratio. Unfortunately, despite an extensive literature search, we were unable to find quantitative data on the ratio between intercalated disc surface area and remaining surface area within one species and cell type, which makes a proper calculation impossible.

Finally, the discrepancy may also be related to the inevitable underestimation of the amount of cardiac Na\(^+\) current using macropatch recordings. First, it is easy to imagine that macropatches from the intercalated disc region may not contain intercalated disc surface only, but will be a mixture of ‘real’ intercalated disc (with cardiac Na\(^+\) channels) and ‘normal’ cell surface (without cardiac Na\(^+\) channels). Second, we have performed our measurements using close-to-physiological conditions and the activated quasi-macroscopic inward Na\(^+\) currents will depolarize the myocytes. Although the depolarization with the relatively small brain-type Na\(^+\) currents is limited,\(^1\) the larger intercalated disc Na\(^+\) currents may depolarize the cell substantially. Consequently, the driving force for Na\(^+\) will be reduced in the intercalated disc macropatch recordings, resulting in an underestimation of the intercalated disc Na\(^+\) current amplitude. A third possible explanation for the underestimation of the cardiac Na\(^+\) current may come from the total noise in our macropatches. When 50 nM TTX was present, quasi-macroscopic currents as well as single channel activity were virtually absent in macropatches from the middle of the cell. However, we cannot exclude the presence of remaining Na\(^+\) channels completely. Single Na\(^+\) channel measurements, especially at physiological temperature, require optimal signal-to-noise ratio,\(^8\) which was definitely not the case in our experiments. Thus, we may have overlooked residual cardiac Na\(^+\) channels in macropatches from the middle of the cell. This hypothesis seems supported by the presence of cardiac Na\(^+\) channels along the lateral membrane as observed in some immunohistochemistry studies [see Verkerk et al.\(^1\) and Abriel,\(^9\) and primary references cited therein].

In conclusion, the careful comments by Petitprez and Abriel clearly demonstrate that macropatch recordings are not suitable to give an accurate determination of the ratio of ‘cardiac’ and ‘brain-type’ Na\(^+\) currents in ventricular myocytes. However, these concerns do not affect the main conclusion of our study, i.e. density and gating properties of brain-type Na\(^+\) current are not altered in response to pressure and volume overload-induced heart failure in rabbit.

References

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**Ventricular oversensing in 518 patients with implanted cardiac defibrillators: incidence, complications, and solutions**

Rauwolf et al.\(^1\) report a 1.5% incidence of myopotential oversensing in their series of 518 ICD patients. The causes for myopotential oversensing were attributed either to contraction of respiratory muscles (by the diaphragm, which is in proximity to the ICD lead tip at the apex) or to contraction of the upper limb/trunk muscles (as was the case in the example shown in Fig. 2A). The latter finding is possible only when an integrated bipolar ICD lead is used with accidental inversion of the DF-1
connectors at implantation. In integrated bipolar leads, the distal (negative) DF-1 lead terminal supplies the anode for rate sensing. As the positive DF-1 connector is internally connected to the ICD casing, accidental inversion of the DF-1 lead terminals will result in the ICD casing becoming part of the sensing circuit. Therefore, myopotential activity may be sensed during contraction of the pectoral muscles, with a risk of inappropriate shocks. Furthermore, DF-1 lead inversion results in the presence of a shock vector directed between the SVC coil and the ICD casing that may shunt energy away from the ventricles, resulting in ineffective therapy. Owing to this, the DF-1 connections should be corrected, rather than simply modifying sensitivity settings to avoid inappropriate shocks.

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Myopotential oversensing due to inversion of the DF-1 connectors: reply

Thank you for the valid comment. Indeed, the problem of accidental inversion of the DF-1 connectors is resulting in quasi-unipolar configuration of the pace/sense part of the integrated bipolar ICD device. This is a generally accepted phenomenon.1,2 Clearly, we did not address this specifically in our paper. Therefore, careful connection of the device is essential for correct ICD function.

The described cause of myopotential oversensing, attributed to contraction of the upper limb, has a variety of causes. In our experience, the contraction of the upper limb musculature is associated with other muscle contractions, for instance diaphragmatic, abdominal, and intercostal muscles. An additional aspect is a sensing artefact based on a micro-ICD lead insulation damage with or without lead impedance changes.

In the case reported in Figure 2A,3 a high-energy device (Guidant Prizm HE with 41 J, Guidant/CPI, St Paul, USA) and a double-coil ICD lead system were implanted. The high amplitude of the myopotentials was the reason for an invasive revision of the ICD system. The intraoperative inspection of the leads, header, and connections was evidently not the cause of the malfunction in this specific patient. We decided to implant a new pace/sense lead in a high-septal position in the right ventricle.

With the new configuration, myopotential oversensing was absent during follow-up over the next 2 years.

References


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