Data on coil occlusion is more relevant to the paediatric than to the adolescent and adult patient. Coil occlusion of PDAs in the adult is usually possible only in the smallest of adult PDAs. The vast majority of adult ducts require closure with a plug or umbrella device.

What do we learn from voluntary, non-standardized registries? Usually very little. In this instance, the literature is replete with studies on coil implantation, noting it to be both safe and effective. Since the year this study was begun, 1994, there have been 126 publications on duct occlusion with one form or another of spring coils. These studies have promoted improved understanding of technical issues in implantation, and have described both complications and outcomes. In general, the authors have concluded that implantation of these devices worked and that their use should be encouraged. Thus, the object of the present study, to define efficacy and safety of such devices and techniques, has already been well established in the literature. The difficulty with such voluntary, uncontrolled registries is the inability to address specific in-depth technical or outcome questions due to the lack of a uniform protocol.

Nonetheless, there is strength in numbers, and the present study further confirms and encourages the application of this technology to the paediatric management of the persistently patent arterial duct.

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Recurrence of atrial fibrillation and the need for new definitions

See page 1822 for the article to which this Editorial refers

Electrical cardioversion is the method of choice in persistent, i.e. non-self terminating atrial fibrillation. However, the Achilles’ heel of cardioversion is the frequent recurrence of atrial fibrillation. It becomes increasingly important to categorize outcomes of cardioversion and to distinguish between (a) complete shock failure, (b) immediate recurrence (IRAF), (c) subacute recurrence and (d) late recurrence[1]. First, the atria may resist the transthoracic electrical current completely: no electrical silence in the atria, i.e. complete shock failure. Next, if one single sinus beat emerges, the following 1 to 2 min are crucial since in this short time window the immediate recurrences (IRAF) happen[2]. After the ultra short IRAF period and up to 1 to 2 weeks, the so-called subacute recurrences present, and 1 and 2 weeks after the shock the phase of the late recurrences starts[3]. This

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pattern of recurrences suggests distinct arrhythmogenic mechanisms. Probably IRAF is due to instantaneous hypervulnerability of the atria, whereas subacute recurrences may relate to reversed electrical remodelling producing temporary heterogeneity. Up till now research into the separate mechanisms has been sparse.

In the present issue, Fynn et al. studied electrophysiological factors associated with recurrence of atrial fibrillation in patients with persistent atrial fibrillation undergoing internal electrical cardioversion[4]. Considering the difficulties of the authors’ human model, they have to be commended for their achievements. As a measure of local refractoriness they recorded atrial fibrillation cycle lengths at four different sites. The fifth percentile of all atrial fibrillation cycle lengths (AFCL$P_5$) was considered to reflect the refractory period. Also the refractory period using the extrastimulus method (AERP) was recorded. Dispersion was calculated as the difference between the longest and shortest AFCL$P_5$ or AERP. Out of 37 patients, 28 had a recurrence, i.e. seven probably had IRAF (they left the lab in atrial fibrillation) and the other 21 a subacute recurrence (after 122.5 h in sinus rhythm). The investigators found that dispersion of AFCL$P_5$ (but not AERP) was the only predictor whereas the refractory period itself did not matter. Other electrophysiological parameters such as conduction velocity or dispersion of conduction were not recorded. Although the technique used to determine dispersion is not easily applied in routine clinical practice, it may, however, play a role in the future in the management of patients undergoing internal cardioversion, in particular with the development of automatic determination of dispersion. The study however, does, not report whether dispersion of atrial fibrillation cycle length was predictive of IRAF rather than subacute recurrences or vice versa. The small numbers may have precluded such conclusions. Obviously, when trying to elucidate recurrence mechanisms it may not be helpful to lump the different types of recurrences together. It is tempting to speculate whether in the study of Fynn et al. dispersion predicted IRAF rather than subacute recurrences because at the time of the subacute recurrence, dispersion appeared to have decreased.

In the same paper the authors showed that in patients attending for a repeat internal cardioversion, dispersion of AFCL$P_5$ was ameliorated. Of note, these repeat cardioversions and electrophysiological measurements were done in patients with a subacute recurrence. Thus, the subacute recurrences occurred at a time when the dispersion had diminished. This finding casts doubt on the effectiveness of medical interventions that are aimed at prolonging the shortest refractory periods to decrease dispersion, in reducing the recurrence rate. Therefore, one cannot support the authors’ conclusions that AFCL dispersion may provide a mechanistic link between atrial remodelling and atrial fibrillation recurrence, and hence be a target for treatment in patients with persistent atrial fibrillation. Rather, one would favour the notion that a high dispersion indeed relates to recurrence but only because it marks atrial heterogeneity associated with a high probability of atrial fibrillation recurrence.

What does AFCL$P_5$ stand for? The authors used the fifth percentile of all atrial fibrillation cycle lengths (AFCL$P_5$) as a measure of the refractory period. However, the AERP did not correlate well with the AFCL$P_5$. In addition, whilst AFCL$P_5$ dispersion had decreased after cardioversion after an average of 122.5 h of sinus rhythm (a sign of reversed atrial electrical remodelling as the authors suggest), the AERP dispersion remained unchanged. Although the lack of an association may be related to chance (low numbers), it was probably due to AFCL$P_5$ representing the overall electrophysiological behaviour, including not only AERP but also conduction and excitability properties of the atria.[5]

Traditional factors predicting atrial fibrillation recurrence include long atrial fibrillation duration, atrial enlargement, high age, heart failure and hypertension[6]. Fynn et al. found AFCL$P_5$ predictive but traditional factors not[4]. Is AFCL$P_5$ more sensitive than these factors or does it represent something else? The answer to these questions is probably no. The electrophysiology of the above clinical factors ageing, hypertension, heart failure and long atrial fibrillation duration all translate into atrial enlargement and pathophysiological adaptations including loss of atrial myocytes, decreased coupling of myocytes[7] and fibrosis[8]. These adaptations form the basis for dispersion of refractoriness and conduction. Thus atrial patho-anatomical remodelling and electrical remodelling as described in the report by Fynn et al.[4] are clearly related. The above is far from saying that the findings of Fynn et al. are not important, since they provide important insights into atrial electrophysiology after cardioversion. In addition, it underscores the notion that we need a proper definition of the various forms of post-cardioversion recurrences of atrial fibrillation. Nevertheless it remains to be determined whether we need to measure dispersion of AFCL$P_5$ to help us predict atrial fibrillation recurrence.

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