Atrial septal defect closure in the adult remains controversial

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‘Treatment of atrial septal defect in the adult is still controversial.’ is the opening sentence of the paper by Nyboe et al. [1] in this issue of the journal. Whether this paper has been able to enlighten us from the darkness of controversiality, is the question at stake. As with most surgical procedures, there has been no prospectively randomized trial into the merits of closing atrial septal defect (ASD), and the Nyboe paper is no exception in this aspect. The consequence being that we have no other option than to make inferences from cohort studies. In this paper, a cohort of 220 patients is described having had their ASD’s closed in a single institution over an 18-year period. The selection criteria for ASD closure are, however, not specified, and scrutiny of Table 3 detailing pre- and postoperative symptoms brings us no further. Whether or not these patients came to medical attention through some sort of screening or through symptoms or a combination thereof is entirely unclear. The
selection process being unclear, this leaves us in the dark about all the patients with an ASD that were not accepted for closure, the reasons for which we can only speculate about. As the indications for ASD closure have undergone a noticeable evolution in the past decades, this is an essential element in the appreciation of the Nyboe paper.

That the indications for ASD closure have undergone a rapid evolution is clearly seen in the way the guidelines changed in a mere 7 years. The 2010 European Guidelines state: ‘Patients with significant shunt [signs of right ventricle (RV) overload] and pulmonary vascular resistance (PVR) <5 wood unit should undergo ASD closure regardless of symptoms’ [2]. However, the 2003 European Guidelines stated as indications for intervention: ‘large defects (>10 mm) unless pulmonary vascular disease (PVR >8 wood unit × m², L–R shunt <1.5, no response to pulmonary vasodilators). Paradoxic embolism’ [3]. Interestingly, the exact size of the defect has been abandoned as a criterion, while the unquantified ‘RV overload’ is currently en vogue. It is also surprising that the size of the defect in the 2003 guideline is not normalized for body size, while it is known that the Danes are among the tallest people in the world. A 10-mm defect has far less significance in Denmark than, for example, in the generally much smaller Asians. Also, the haemodynamic shunt size (quantified by \( Q_p/Q_s \)) is now abandoned, while it was a criterion in 2003! Let us presume that the indications for operation in Aarhus changed with the guidelines over the years, then it is unclear what their indications were prior to 2003. In any case, let us presume the size of the defects was >10 mm since 2003, in the absence of other criteria.

Whatever the indications, this cohort of 220 patients was submitted to invasive treatment and one—apparently untypical—patient died at a later pacemaker implantation, the interval between the operations being unclear. We can then safely conclude that mortality is not a major issue in this cohort. The mixed lot of complications, however, listed in Table 2 are arbitrarily lumped together to form some sort of composite end point to compare the two age groups on one side and catheter intervention vs surgery on the other. Subsequently, a dazzling \( P \)-value is produced in favour of catheter intervention. Nevertheless, everyone will agree that pericardial effusion and minor bleeding are of a totally different magnitude than emergency surgery for an apparently failed catheter intervention and pneumonia. Lumping these diverse complications together does not improve insight into the fate of these patients. Furthermore, it is unclear how dichotomizing the cohort into two age groups helps the analysis. Why the age of 50 years was chosen is not specified. In general, by dichotomising the age variable, much of its information is unnecessarily discarded statistically.

The benefits for this cohort of patients are detailed in Table 3 and 5, but again these tables do not bring us any further in answering the questions at stake. On the level of individual symptoms, these tables have a go at quantifying improvement, but we are dealing with patients in their integral entirety that often have more than one symptom. In this paper, it remains unclear what happens on the level of the patient. How many patients improved in how many symptoms and how this correlated with objectively measured variables remain totally unclear.

My conclusion is that this paper brings no enlightenment to the controversy of closing ASD in the adult. On the contrary, because selection criteria are unclear, complications and patient benefits are not worked out, this paper adds to the existing confusion. In all likelihood, much more could have been made of analysis of this data set, particularly, if data had been added on the size of the defect and of the shunt and on patients that had not been accepted for ASD closure.

REFERENCES