Introduction

Nowadays, sedation has become an essential part of clinical practice. Despite this fact, we still lack data describing the exact impact of sedation on cardiac function.

Objective

The aim of this work is to compare the changes in cardiac function induced after sedation with midazolam or dexmedetomidine by magnetic resonance imaging (MRI).

A total of 30 volunteers were randomized into two groups: 15 patients in the midazolam (MID) group and 15 patients in the dexmedetomidine (DEX) group. Each participant underwent a one session MRI imaging of the heart during and after sedation administration. The following parameters were recorded: right ventricular (Pul-vol) and left ventricular (Ao-vol) stroke volume, maximal blood flow velocity through the aortic (Ao-flow) and through the pulmonary valve (Pul-flow) during systole, and maximal blood flow velocity through the mitral valve during early (E-diast) and late diastole (L-diast). The monitor recorded mean blood pressure (MAP), pulse (P), and blood oxygen saturation (SpO_2) at 5minute intervals.

Results

As for the parameters of ventricular systolic function dexmedetomidine decreased significantly Ao-vol ($p = 0.006$), Pul-vol ($p = 0.003$), Ao-flow ($p = 0.048$) and Pul-flow ($p = 0.007$). Midazolam also significantly decreased Ao-vol ($p = 0.001$), Pul-vol ($p = 0.01$), and Ao-flow ($p = 0.04$). Pul-flow value in the midazolam group was without statistically significant change ($p = 0.23$). Regarding the parameters of left ventricular diastolic function, midazolam sedation worsened E-diast ($p = 0.019$). E-diast value in the dexmedetomidine group was without statistically significant change. The value of L-diast was not affected by the sedation technique.

Conclusion

Both sedation modes lead to a decrease in stroke volume of both the left and right ventricles. In addition, dexmedetomidine worsened cardiac output of both ventricles because of the deterioration in ventricular contractility as assessed by blood flow velocity through the aortic and pulmonary valves. This negative inotropic effect was slightly observed in the midazolam group, which in our cohort negatively affected the contraction of the left but not the right ventricle. Midazolam and not dexmedetomidine adversely affected left ventricular relaxation. The systolic function of the left atrium was not affected by the sedation technique.