Xenon based anesthesia does not alter cardiac QT-interval

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Prolongation of the corrected QT-interval (QTc) may cause potentially critical ventricular arrhythmias. Most volatile anesthetics as well as sympathetic activation during endotracheal intubation or surgical interventions can lead to an increase of QTc, due to blockade of rapidly acting potassium rectifier channels [1]. Xenon (Xe) may alter QTc by both direct effects and indirectly through its sympathomimetic properties [2]. Accordingly, we test the hypothesis that xenon based anesthesia alters QTc.

Material and Methods: Following local IRB approval (Ref. No.: MO-LKP-394 + 3386) Xe was evaluated in eight healthy volunteers (Eudra CT No.: 2009-012449-48; ClinicalTrials.gov Identifier: NCT01043419) and in a clinical observational study including 35 patients (Reg. No.: AL-PMS-01/07GER). In volunteers (age: 25 years ±2), surface ECG, heart rate and radial arterial pressure were recorded in the awake state, following denitrogenation (F_iO_2>0.9) and during Xe mono-anesthesia while breathing spontaneously via a face mask. In the observational study, Xe was administered in patients (age: 44 years ±11) scheduled for general surgery or traumatology. Following oral premedication with midazolam (75-150 µg kg^{-1}), general anesthesia was induced by intravenous propofol (2.5 mg kg^{-1} + 6 mg kg^{-1} min^{-1}), remifentanil (0.2 µg kg^{-1} min^{-1}) and rocuronium (0.6 mg kg^{-1}). Following denitrogenation (F_iO_2>0.9) Xe administration was started (endtidal Xe: 0.65) when steady state conditions were achieved propofol was discontinued. Surface ECGs and non-invasive blood pressure were recorded at 3 different stages: In the awake state, following anesthesia induction and during steady-state of xenon anesthesia before initiation of surgery. QT intervals were always determined of three consecutive cardiac intervals from ecg print outs (feed 50 mm/sec) in a double blinded fashion and corrected by using Bazzet's formula [3].

Statistics: Means±SD, one-way ANOVA for repeated measurements, Newman-Keuls post hoc test, $p<0.05$

Results: In healthy volunteers, xenon slightly increased mean arterial pressure (from 93 mmHg ±5 to 107±6) but did not alter heart rate (awake: 64 min^{-1}±10; xenon: 70±10) and QTc (awake: 398 ms ±21; F_iO_2>0.9: 409±42; xenon: 409±28; p=0.37). In patients, induction of anesthesia with propofol and remifentanil decreased arterial pressure (systolic/diastolic: from 129 mmHg ±13 / 70 mmHg ±8 to 97±8 / 51±6, $p<0.001$) as well as heart rate (from 69 min^{-1} ±11 to 61±12, $p<0.001$) and was associated with the tendency to decrease QTc from 414 ms ±25 to 405±24 (p=0.06). Discontinuation of propofol and administration of xenon increased arterial pressure (to 113 mmHg ±13 / 62±8, $p<0.001$), reduced heart rate (to: 58 min^{-1} ±10, p=0.04) and normalized QTc to preanesthetic values (417 ms ± 32, p=0.3 vs. awake). The calculated power of the study is greater 0.99 in volunteers and patients, respectively.

Conclusion: Xenon mono-anesthesia in healthy volunteers and general anesthesia maintained with xenon/remifentanil did not reveal increased QTc. Thus, we could not identify any adverse event of xenon on QTc.

References:

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