

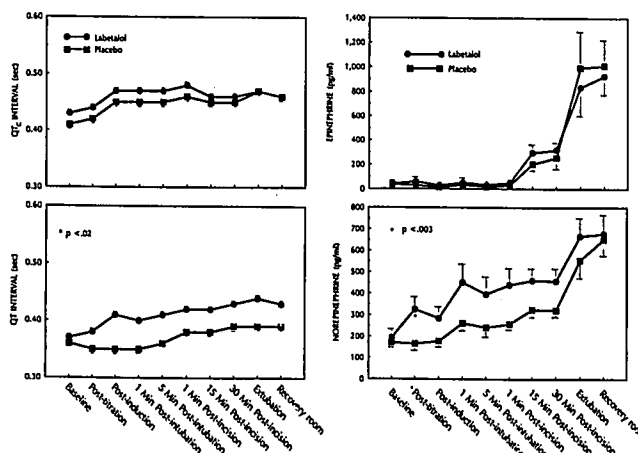
TITLE: PERIOPERATIVE CHANGES IN THE QT INTERVAL IN RESPONSE TO ANESTHETICS AND PLASMA CATECHOLAMINES
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The QT interval may be influenced by heart rate, catecholamine state and anesthetic agents. The effects of these factors were studied during an IRB approved study of 16 consenting ASA I females undergoing elective abdominal surgery. Eight patients were randomized to receive labetalol, titrated prior to induction to reduce mean arterial pressure by 15%. Anesthesia consisted of thiopental, vecuronium and maintenance with N₂O, isoflurane and vecuronium. The QT and QTc intervals and plasma catecholamines were evaluated. Labetalol titration was associated with significantly higher QT and plasma norepinephrine levels not seen in control. Following induction with thiopental the QT increased further while plasma norepinephrine decreased in the labetalol group, however, neither changed significantly in control. Surgical stimulation was associated with small increases in QT and plasma catecholamines in both groups. Peak elevations of plasma catecholamines occurred at extubation and early recovery without significant changes in QT or QTc. The QTc interval varied within a narrow range in both groups.

Conclusions. a) In contrast to a previous report (1), there was no increase in QT in the control

group following thiopental administration. b) The perioperative changes in the QT observed in both groups did not predictably correspond to plasma catecholamine concentrations. c) The changes in the QTc interval seen throughout the perioperative period were limited in both groups and are unlikely to be of clinical significance.

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TITLE : RIGHT VENTRICULAR END-SYSTOLIC PRESSURE VOLUME RELATIONSHIP (ESPVR) DURING NOREPINEPHRINE (NE) INFUSION IN SEPTIC SHOCK PATIENTS.
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In patients with hyperdynamic septic shock syndrome, NE has been proposed to treat hemodynamic perturbances. A major effect of this drug is to increase ventricular afterload (increase in Vascular Resistances - VR) and thus potential effects on ventricular contractility are difficult to evaluate using the classical Frank-Starling relationship. In animals and humans, the ESPVR has been introduced as an index of ventricular contractility, independent of the loading conditions of the ventricles. The present study was designed to determine whether NE could improve ESPVR in septic shock patients.

After institutional approval and informed consent from the family was obtained, 8 patients were studied. They all presented with hyperdynamic septic shock (Systolic Blood Pressure (SBP) < 90 mmHg, Systemic VR (SVR) < 600 dynes.sec.cm⁻⁵, cardiac index > 4 l.min⁻¹.m⁻², oliguria) and required controlled mechanical ventilation. Their pulmonary artery was catheterized with a modified Swan-Ganz catheter equipped with a fast response thermistor (93A 431 H 7.5 F Edwards laboratories). Right ventricular ESV (RVESV) was calculated using the REF 1 computer (Edwards Laboratories) and RVESP was recorded from the proximal injectate lumen of the catheter

positioned in the RV. The ESPVR was calculated by best fit linear regression using the formula: $RVESP = E \times RVESV + \text{constant}$. The slope E is the elastance and is proportional to RV contractility. Regression lines were computed before and after fluid challenge (7 ml.kg⁻¹ of colloid solution within 30 min), before and during NE infusion (1.1 ± 0.2 mcg.kg⁻¹.min⁻¹) used to restore SBP within the normal range.

During NE infusion, SBP increased from 73 ± 7 to 122 ± 8 mmHg (p < 0.0001), SVR from 440 ± 45 to 940 ± 60 dynes.sec.cm⁻⁵ (p < 0.001) and CI did not change (4.8 ± 0.5 to 5 ± 0.5 l.min⁻¹.m⁻²).

The baseline elastance slope derived in the 8 patients before NE infusion was 0.16 mmHg.ml⁻¹. ESPVR was: $RVESP = 0.16 RVESV + 7.4$, r = 0.72, p < 0.005. During NE infusion the elastance slope was 0.18 mmHg.ml⁻¹ (NS from baseline) and the ESPVR was: $RVESP = 0.18 RVESV + 9.6$, R = 0.76, p < 0.0001.

The ESPVR obtained in this study showed very good linearity. No change was observed in elastance, reflecting the absence of an NE inotropic effect on the RV. This study does not provide information regarding the left ventricle.

REFERENCES

1. Crit. Care Med. 15 : 134-137, 1987
2. Circulation 63 : 1223-1227, 1981