Journal of Electrocardiology xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

Journal of Electrocardiology

journal homepage: www.jecgonline.com

Changes in spatial QRS-T angle and QTc interval in patients with traumatic brain injury with or without intra-abdominal hypertension

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ARTICLE INFO

Available online xxxx
Keywords:
Vectorcardiography
Intra-abdominal pressure
Brain-heart interaction
ST segment

ABSTRACT

Introduction: Traumatic brain injury (TBI) affects cardiac electrical function, and several extra-cerebral factors, including intra-abdominal pressure (IAP), might further modulate this brain-heart interaction. The purpose of this study was to investigate the impact of TBI, and of increased IAP during TBI, on cardiac electrical function as measured by vectorcardiographic (VCG) variables.
Methods: Survival, IAP and changes in VCG variables including spatial QRS-T angle and QTc interval were measured in consecutive adult patients with either isolated TBI (iTBI), or with TBI accompanied by polytrauma to the abdomen and/or limbs (pTBI). For all patients, observations were performed just after the admission to the ICU (baseline) and at 24, 48, 72 and 96 h after admission.
Results: 74 patients aged 45 ± 18 were studied. 44 were treated for iTBI and 30 for pTBI. In all patients, spatial QRS-T angle and QTc interval increased after TBI ($p < 0.001$), relatively more so in patients with pTBI. Compared to survivors, non-survivors also ultimately had greater widening of the spatial QRS-T angle ($p < 0.001$), most notably just before foraminal herniation. Wider spatial QRS-T angle and longer QTc interval were also noted in patients with IAP > 12 mmHg ($p < 0.001$), and with right compared to left hemispheric injury ($p < 0.001$). ST segment level at the J point decreased 24 and 48 h after TBI in leads I, II, III, aVR, aVF, V_1, V_2, V_3 and V_6, and increased in lead V_1, especially in non-survivors.
Conclusions: Spatial QRS-T angle and QTc interval increase after TBI. If foraminal herniation complicates TBI, further widening of the spatial QRS-T angle typically precedes it, followed by notable narrowing thereafter. Increased IAP also intensifies TBI-associated increases in spatial QRS-T angle and QTc interval.

Introduction

Traumatic brain injury (TBI) is a common cause of traumatic death worldwide. The overall, at-discharge and the 3-month mortality rates are high and depend on the extent and type of treatment [1]. Mortality after TBI relates to severity of brain injury and brain-related complications, the presence of second insult, intracranial bleeding or malignant cerebral edema, as well as extra-cranial pathologies, especially cardiac electrical dysfunction [2]. Cerebrogenic cardiovascular damage may result in sudden cardiac death (SCD) via disturbed central autonomic function with raised catecholamine concentrations, shift in potassium-sodium ions following activation of renin-angiotensin system, and cerebral injury-related inflammatory responses [2–5]. Different kinds of electrocardiographic disturbances have been reported in 60%–70% of

Abbreviations: APACHE, the Acute Physiology and Chronic Health Evaluation; ARIC, the Atherosclerosis Risk Communities Study; CI, cardiac index; CO, cardiac output; CVP, central venous pressure; DAI, diffuse axonal injury; ECG, electrocardiography; GCS, Glasgow Coma Score; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; ICP, intracranial pressure; ICU, intensive care unit; iTBI, isolated traumatic brain injury; pTBI, polytrauma with traumatic brain injury; PVI, pleth variability index; ROC, receiver-operator characteristic; QTc, corrected QT interval; SCD, sudden cardiac death; SOFA, Sepsis-Related Organ Failure; SpO_2, peripheral oxygen saturation; SrO_2, regional cerebral saturation; STJ, ST segment with J point level; SVRI, systemic vascular resistance index; SVV, stroke volume variation; TBI, traumatic brain injury; VCG, vectorcardiography.

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https://doi.org/10.1016/j.jelectrocard.2017.12.038
0022-0736/

patients with intracerebral haemorrhage, in 40%–70% of patients with subarachnoid haemorrhage and in 15%–40% of patients with ischemic stroke [6–8]. Several clinical observations have documented an association between cerebrogenic cardiac arrhythmias and electrocardiographic disorders such as prolonged QT, inverted T or depressed ST [5, 8–11]. These changes may also affect vectorcardiographic variables, particularly spatial QRS-T angle.

Vectorcardiography (VCG) is a spatial method of electrocardiography that enables visualizing, through the cardiac cycle, the continuous moments of the cardiac vector as loops. Vectorcardiograms and associated parameters such as the spatial QRS-T angle, i.e., the spatial angle between cardiac depolarization and repolarization, can now be easily derived from standard 12-lead electrocardiograms [12]. While normal ranges for spatial QRS-T angle vary by method and by study, most studies have suggested that normal values lie below 100°-110° for men and below 90° for women [13–15]. Of note, a widened spatial QRS-T has been reported as an independent predictor of cardiac arrhythmias and SCD [14–16]. Several pathologies widen spatial QRS-T angle. In the Rotterdam Study, spatial QRS-T angle >105° was noted in 20% of diabetic patients [17]. In patients with chronic kidney diseases, both disease duration and poor outcome of dialysis is associated with increased spatial QRS-T angle [18,19]. Severe coronary atherosclerosis is also an independent predictor of widened QRS-T both in patients with chronic kidney diseases and in patients with isolated coronary artery disease [18,20]. Additionally, some anaesthetics and sedatives can affect VCG and increase risk of SCD [21–24]. Interestingly, rapidly induced intra-abdominal hypertension (IAH) to 15 mmHg also acutely widens spatial QRS-T angle to above 100° in some healthy young women undergoing elective gynaecological laparoscopy [25].

Intra-abdominal pressure (IAP) is the steady-state pressure within the abdominal cavity [26]. Raised IAP above 12 mmHg, defined as IAH, has been associated with increased morbidity and mortality in critically ill patients [27,28]. Additionally, IAH strongly affects intracranial pressure (ICP) and blood venous outflow, often leading to venous congestion in the cerebral circulation and brain ischemia [29–32]. Raised IAP positively correlates with ICP in critically ill patients with traumatic and non-traumatic brain injury [30,33]. IAH can also depress cardiac relaxation and impair coronary perfusion pressure leading to cardiac and circulatory failure and changes in VCG variables [25,34,35]. In patients treated for TBI, therefore, investigation of changes in spatial QRS-T angle should ideally also take IAP into consideration.

The aim of this study was to observe the effect of TBI, either without or with concomitant IAH (generally caused by polytrauma), on VCG variables. We hypothesized that worsening TBI, or TBI associated with IAH, would be associated with relatively greater increases in spatial QRS-T angle.

Patients and methods

This prospective observational study was conducted in accordance with the intensive care unit (ICU) protocol, the Declaration of Helsinki and applicable regulatory requirements as approved by the Institutional Review Board and the Bioethics Committee of Medical University at Lublin, Poland (KE-0254/308/2016). Informed consent was obtained from the legal representatives of patients because all patients were sedated and on mechanical ventilation.

Patient selection

Adult patients who required artificial ventilation due to severe or moderate TBI assessed by the Glasgow Coma Score (GCS) were enrolled. Pregnant women, patients below the age of 18 years, polytrauma patients with thoracic or/and cardiac contusion, and patients with known cardiac diseases including previous cardiac surgery or pacemaker implantation were excluded. Moreover, transplant recipients, patients with drug overdose, and those treated for neoplastic or severe chronic pulmonary, hepatic, endocrine or metabolic disorders were also excluded.

For the entire duration of the ICU stay, relevant demographic, clinical and laboratory data were registered in an electronic database, along with daily sepsis-related organ failure assessment (SOFA) score, fluid balance, IAP and advanced hemodynamic monitoring variables (see below), supplemented by mortality on day 28.

On admission day, severity of trauma was described by the Revised Trauma Score (RTS: GCS + systolic blood pressure + respiratory rate). For GCS the value assessed by the rescue team during first aid was used and confirmed immediately after admission into the Emergency Department. Severity of TBI was described by Rotterdam CT score (R-CT) [36].

Monitoring and treatment techniques

Systemic arterial blood pressures and heart rate were measured continuously. Additionally, hemodynamic variables such as cardiac output/index (CO/CI), stroke volume variation (SVV), systemic vascular resistance index (SVRI) and central venous pressure (CVP) were monitored using an EV 1000 platform (Edwards Lifesciences, Irvine, USA). Continuous measurement of regional cerebral oxygen saturation (SrO_2), fronto-temporal electroencephalography, peripheral oxygen saturation (SpO_2) with haemoglobin level and Pleth Variability Index (PVI) were obtained via Masimo Root monitor (Irvive, CA) with SEDLine. Immediately after the admission into the ICU, all patients received hyperosmotic therapy with 15% mannitol at the dose 1.5 g/kg body weight per day connected with loop diuretics. This treatment was discontinued in patients with osmolality higher than 310 mOsm/kg H_2O. Blood potassium concentration was measured 5 times per day and decrease in its concentration was corrected using continuous infusion of potassium and magnesium mixture. Fluid administration and vasopressor (norepinephrine) were titrated to obtain adequate SrO_2 higher than 50% and mean arterial pressure higher than 70 mmHg. Antibiotic therapy was based on bacteriological results. Patients with gastrointestinal dysfunction grade III or IV [37] received total parenteral nutrition using NuTRIflex Lipid Special® (B Braun, Melsungen, Germany). In patients with detectable bowel sounds, enteral feeding was supplemented with Nutrison Multi Fibre (Nutricia, Schiphol, Netherlands) to reach the target caloric energy supply. In patients with acute kidney injury continuous renal replacement therapy with citrate anticoagulation was used.

ECG, derived vectorcardiogram (VCG), IAP measurement and study protocol

Surface 12-lead resting ECG was recorded using a Cardiax device (IMED Co Ltd. Budapest. Hungary). The recordings at each time period were automatically converted to a single median beat, and transformed into three orthogonal leads X, Y and Z according to the inverse Dower method [13,38]. The projections of the maximum vectors of QRS and T-waves in the frontal, transverse, and left sagittal planes and on the x, y, and z axes were then obtained. Next, the value for the spatial QRS-T angle as automatically calculated from the maximum spatial QRS and T vectors, as well as for the QTc interval and ST segment J level (STJ), were obtained directly from the Cardiax commercial software.

IAP was measured indirectly via the urinary bladder using a modified Kron's technique: After clamping the Foley's catheter, 25 ml of a sterile saline solution was administered as priming. All residual urine from the urinary bladder was drained immediately before each IAP measurement. Measurements were performed in the supine position and the midaxillary line was established as a zero reference point where it crossed the iliac crest.

The observations were made at five time points: just after admission into the ICU (Baseline) and at 24, 48, 72 and 96 h after admission. Selected data were also analysed in relation to patient outcome.

Please cite this article as: Dabrowski W, et al, Changes in spatial QRS-T angle and QTc interval in patients with traumatic brain injury with or without intra-abdominal hypertensi..., Journal of Electrocardiology (2017), https://doi.org/10.1016/j.jelectrocard.2017.12.038