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Evaluation of Intracranial (Cerebral) Compliance in Myocardial Revascularization: Prospective Pilot Study

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Abstract

Background: Coronary Artery Bypass Grafting (CABG) is a critical treatment for coronary artery disease, but it carries a risk of neurological complications. The relationship between cardiac surgery and Intracranial Compliance (ICC) is not fully established, particularly whether improved coronary perfusion leads to improved ICC. This study utilized the non-invasive B4C device to monitor ICC, aiming to identify perioperative changes.

Methods: This was an observational, prospective pilot study of repeated measures, including 16 adult patients undergoing elective on-pump CABG at a single center in Porto Alegre, Brazil. Patients with pre-existing structural neurological disease were excluded. Non-invasive ICC was monitored using the B4C device, which analyzes the waveform of the intracranial pressure (ICP) pulse. The P2/P1 ratio (the ratio of the second to the first peak of the ICP waveform) was used as the primary indirect marker of compliance, with a P2/P1 ratio >1.2-1.4 indicating reduced compliance. Monitoring was performed at three time points:

- T0: Preoperatively (baseline)
- T1: Immediately post-surgery, with continuous sedation and initial stabilization in the Intensive Care Unit (ICU)
- T2: Immediately post-extubation, without sedation, in the ICU

Repeated measures ANOVA with Bonferroni post hoc correction was used for statistical analysis, with $p< 0.05$ considered significant.

Results: The sample was predominantly male (75%), with common comorbidities including systemic arterial hypertension (62.5%) and diabetes mellitus type II (31.3%). A statistically significant variation over time was observed for both the P2/P1 ratio ($p=0.003$) and Time to Peak (TTP) ($p=0.019$). The analysis revealed a biphasic pattern: T0 (Baseline): The mean P2/P1 ratio was 1.28 \ TTP 0.37, suggesting a state of reduced or abnormal ICC. T1 (Post-Surgery/Sedation): A significant improvement in compliance was noted, with the mean P2/P1 ratio dropping to 0.85 \ TTP 0.28 (significantly different from T0 and T2, $p < 0.05$), moving into the normal range. All patients were under continuous sedation at this point. T2 (Post-Extubation/No Sedation): The ventilatory and hemodynamic variables, though recorded, did not show significant differences that would interfere with the ICC assessment.

Conclusion: Non-invasive evaluation of cerebral compliance using the B4C device proved to be a viable and safe method in the perioperative period of complex cardiac procedures. The study demonstrates that ICC is compromised preoperatively (T0) and returns to a normal state post CAGB

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(T1,T2). The most robust finding was the significant, temporary improvement in ICC immediately post-surgery under continuous sedation (T1), which may be due to the surgery itself, post-aggression self-regulation, or a protective effect of the continuous sedation on cerebral autoregulation. Further studies with larger samples are recommended to clarify the causes of the observed dysfunction and the physiological mechanism responsible for the transient improvement.

Keywords: Intracranial compliance; B4C; Coronary Artery Bypass Grafting (CABG); Cardiac surgery; Heart; Neurological risks

Introduction

Coronary Artery Bypass Graft (CABG) surgery is an essential treatment for coronary artery disease, aiming to restore blood flow to the heart muscle. However, the procedure is not without potential neurological risks, which raises crucial questions about its impact on cerebrovascular compliance - the ability of cerebral vessels to adjust to variations in pressure and volume. The interaction between cardiac surgery and the cerebrovascular system is an area marked by both certainties and important questions, especially when considering intracranial compliance (ICC) [1]. The B4C device was created to assess the neurovascular health of patients in bed, without the need for invasive interventions that carry risks of infection and bleeding. It can stratify patient risk based on brain waves. These assessments aim to minimize the risk of complications in cases where damage is still in its early stages [2].

Myocardial revascularization (CABG) improves cardiac perfusion but poses a challenge in managing ICC, requiring rigorous identification of risk factors and the adoption of preventive measures to protect brain function. Medical literature demonstrates that CABG can impact cerebral hemodynamics, but there is no robust evidence that it directly improves ICC. Studies show that patients with ischemic heart disease present changes in ICC, but cardiac rehabilitation does not significantly modify this parameter [3]. Furthermore, cardiac interventions can temporarily increase cerebral perfusion and improve specific cognitive functions, such as psychomotor speed, but these effects do not necessarily translate into improved ICC [4]. Although there is strong evidence, the cause-and-effect relationship between myocardial revascularization surgery and cognitive impairment (often temporary mental sequelae) is not yet fully proven. The challenge lies in determining the exact cause of these neurological risks; and whether improved coronary perfusion can lead to improved ICC. There is no evidence in the literature that myocardial revascularization improves cerebrovascular compliance. The beneficial effects on cerebral hemodynamics after cardiac procedures are limited and do not include proven improvements in ICC.

Methods

An observational, prospective, repeated-measures study was conducted in the Surgical Center and Intensive Care Unit (ICU) of Hospital São Francisco / Santa Casa de Porto Alegre (RS, Brazil) to evaluate CC in the perioperative period of on pump CABG. Seventeen adults undergoing elective CABG were included. The following criteria were included: age ≥ 18 years, clinical indication for CABG, and technical feasibility of noninvasive CC monitoring. Individuals with known prior structural neurological disease, severe active systemic infection preoperatively, decompensated hepatic or renal dysfunction, impediments to adequate cranial sensor placement, and lack of consent were excluded. The protocol was approved by the Institutional Research Ethics Committee, with free and informed consent obtained, and follows the STROBE guidelines for reporting observational studies.

ICC was measured with the B4C device, morphologically analyzing the noninvasive ICP waveform, in which the peak ratio (P2/P1) is used as an indirect marker of intracranial compliance. Monitoring occurred at three standardized time points: preoperative (T0), immediately after surgery with sedation and initial stabilization (T1), and immediately post-extubation without sedation(T2), up to 30 minutes after withdrawal of mechanical ventilation, in the ICU. In each assessment, the B4C sensor was positioned over the skull according to the manufacturer's manual, ensuring standardized fixation and contact pressure, with the head in a neutral position, and the headrest between 0° and 15°. At least 10 minutes of continuous tracing were acquired per time point, with real-time signal quality checks and marking of movement artifacts, aspiration, or loss of contact, which were excluded from the analysis.

Concomitantly with the acquisition of the tracing, physiological variables were recorded and potentially confounding contextual factors: mean arterial pressure (MAP), heart rate, peripheral oxygen saturation, core temperature, capnography and arterial blood gas analysis. In patients on mechanical ventilation, the ventilation mode, volume current, PEEP, FiO2 and EtCO2. Sedation and analgesia were also documented in use and employment of vasoactive agents. Intraoperative data included technique surgical, extracorporeal circulation times, minimum temperature, water balance, transfusions, minimum hemoglobin and lactate. Baseline information included age, sex, BMI, relevant comorbidities and ejection fraction. This set aimed to allow operational standardization between collection times and subsequent analytical adjustment of confounders recognized in the cardiac perioperative literature, according to practices

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methodological and physiological findings reported in indexed articles.

The B4C signals were processed by an automated algorithm to detect the percussive (P1) and compliance (P2) components of each beat, with review visual when necessary, and calculation of the aggregated P2/P1 ratio in time windows, reporting by time. The operational definition of reduced CC was P2/P1 ≥ 1.2-1.4, according to technical descriptions of the method in previous study [2]. The primary outcome was the variation of P2/P1. Secondary outcomes included: proportion of patients with P2/P1 ≥ 1.2-1.4 at each time point, absolute and relative differences in P2/P1 between the moments, correlations between P2/P1 at T0, T1, T2, and perioperative factors (e.g., CPB times, minimum hemoglobin, temperature), time on mechanical ventilation, ICU and hospital stay. To reduce biases, the team was trained in sensor positioning and checking quality of the routes, checklists were used to standardize the conditions of collection (position, head of bed, MAP targets and normocapnia/normothermia), and a minimum of 8 minutes of "clean" tracing was established per time for inclusion.

Statistical Analysis

For statistical analyses, SPSS version 23.0 was used. Descriptive analyses were performed for all variables, presenting means and standard deviations (SD) for continuous variables, and relative and absolute frequencies for categorical variables. Were performed repeated measures ANOVA and Bonferroni post hoc correction for multiple comparisons. We considered significant differences for the confidence level less than 5% ($p \leq 0.05$).

Results

A total of 16 patients comprised the sample. Of these, the majority were male (75 %). The most prevalent morbidities were systemic arterial hypertension (62.5 %), diabetes mellitus type II (31.3 %) and atrial fibrillation (18.8 %), presented in Table 1.

Table 1: Characterization of the sample regarding sociodemographic and clinical variables.

Features (n= 16)	n (%)
Sociodemographic	
Male gender	42705
Age (years)*	63 (13.1)
Morbidities	
SAH	10 (62.5)
DM	5 (31.3)
Atrial Fibrillation	3 (18.8)
Intervention with ECC	
CABG	10 (62.5)
CABG + mitral prosthesis	3 (18.8)
CAGB + aortic prosthesis	3 (18.8)
ECC	
Time (minutes)*	93 (25)
Ischemia (minutes)*	77 (16)
Post-operative	
Analgesia	9 (56.3)
Norepinephrine	14 (87.5)
Vasopressin	1 (6.3)
Nitroprusside	3 (18.8)
Intravenous insulin	4 (25)

SAH: Systemic Arterial Hypertension; DM: Diabetes Mellitus type II; ECC: Extracorporeal Circulation; CABG: Coronary Artery Bypass Grafting; *data as mean ± standard deviation.

Table 2 shows the behavior of clinical and hemodynamic variables during the assessment of ICC, in the evaluated moments.

Table 2: Behavior of clinical and hemodynamic variables during the assessment of ICC.

Variable (n= 16)	T0	T1	T2
MAP (mmHg)	-	75.3±10.2	77.9±10.6
SBP (mmHg)	-	123±20.9	121.4±15.6
HR (bpm)	-	87.6±14.5	86.1±16.7
SpO2 (%)	-	91±25.2	98.1±1.3
PH	-	7.3±0.1	7.4±0.1
PaO2 (mmHg)	-	141.3±37.4	102.9±24.8
FiO2 (%)	-	35.9±15	-
Hematocrit (%)	35.5±9.9	30.6±5.2	28.8±4.2
Hemoglobin (g/dL)	12.9±1.6	10.5±1.8	9.9±1.5
Glucose (mg/dL)	117.8±19.6	163.6±52.2	168.4±49.2
Creatinine (mg/dL)	1.1±0.4	1.1±0.3	1.2±0.4
Sodium (mmol/L)	-	140±2.0	138.6±3.5
HBA1C (%)	-	5.8±0.8	-
Lactate (mmol/L)	-	18.5±15.6	-

ICU: Intensive Unit Care; MAP: Mean Arterial Pressure; SBP: Systolic Blood Pressure; HR: Heart Rate; SpO2: Peripheral Oxygen Saturation in Hemoglobin; PaO2: Partial Pressure of Oxygen in Arterial Blood; FiO2: Fraction of Inspired Oxygen; HBA1C: Glycated Hemoglobin. Data as mean ± standard deviation.

Table 3 provides an analysis of ICC across three time points in patients, using the P1/P2 ratio and Time to Peak (TTP). The pattern of variation over time for both parameters is statistically significant (p=0.003 for P1/P2 and p=0.019 for TTP), suggesting a real physiological change between the evaluated time points.

Comparative analysis with established studies suggests a biphasic response pattern for brain compliance in the group:

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1. T0 (baseline): The group starts with parameters {P1/P2} = 1.28; {TP} = 0.20, indicating reduced/abnormal ICC. 2. T1 (significant improvement): There is a significant improvement in compliance at T1 (P1/P2 drops to 0.85, TTP to 0.14), with values moving into the normal range. This change is the most significant and statistically robust finding in the Table 3. T2 (return to borderline): The parameters return to borderline values at T2 {P1/P2} = 1.04; {TTP} = 0.20, which, although numerically better than T0, are statistically similar to the initial state and indicate that compliance has returned to the "gray zone" or abnormal state.

The findings at T1 represent the state of best ICC in the group, while T0 and T2 represent states of compromised compliance according to the cut-off points in the literature. The ventilatory and hemodynamic parameters did not interfere and showed significant differences in the group. All patients in T1 were under continuous sedation.

Table 3: Comparison of variables obtained in the assessment of ICC over time.

Variable (n=16)	T0	T1	T2	p value
P1/P2 ratio	1.28^{A} ±0.37	0.85^{B} ±0.28	1.04^{A} ±0.34	0.003
TTP	0.20^{A} ±0.07	0.14^{A} ±0.06	0.20^{A} ±0.07	0.019

ICU: Intensive Unit Care; TTP: Time to Peak. Data as mean ± standard deviation. Means with different letters differ significantly ($p<0.05$) by repeated measures ANOVA and Bonferroni post hoc correction for multiple comparisons.

Discussion

Neurological complications after CABG remain relevant despite advances in perfusion and cerebral protection [1]. Standardization of early physiological markers that signal postoperative (PO) risks is urgently needed, as ventilatory and hemodynamic interventions potentially alter the clinical course. In the context of CABG and cardiopulmonary bypass (CPB), associated with CC, events such as hemodilution, changes in PaCO2, PEEP, time to extubation, temperature, and venous congestion can reduce CC and predispose to increases in ICP and decreased cerebral perfusion [5,6]. In CABG, Mills (1993) describes that the combination of macro/ microembolization and inadequate cerebral perfusion during CPB disrupts autoregulation and increases the permeability of the blood-brain barrier, culminating in vasogenic edema [7]. Increased intracranial volume increases ICP and decreases compliance, making the parenchyma vulnerable to perioperative hemodynamic fluctuations. Thus, the pathophysiological mechanisms outlined by Mills link CABG to increased ICP and worsening ICC via embolism and cerebral edema [7].

Two centuries ago, Monro-Kellie introduced a new approach to intracranial hemodynamics. In a rigid, intact skull, the sum of intracranial volumes is approximately constant. In 2012, Mascarenhas and his group proposed a practical extension of the Monro-Kellie doctrine [8]. Elastic microdeformations exist in the cranial vault that synchronize with the arterial pulse. By measuring these microdeformations on the surface of the head, it is possible to infer the characteristics of intracranial waves noninvasively, safely, and with the patient in bed [8]. This becomes possible when we introduce new technologies for such monitoring. ICC is a promising physiological marker for monitoring and risk stratification, using pulse wave morphology to identify its morphology and thus understand the pathophysiology of brain diseases [9].

In the context of CABG and the immediate postoperative period, pCO2 and Mean Arterial Pressure (MAP) are the main determinants of ICC. However, in this pilot study, we cannot accept the hypothetical condition that these variables did not influence this initial result. For the purposes of critique, the focus shifts to the analysis of the intrinsic physiology and the methodological/statistical rigor of the study. By invalidating the external variables (hemodynamics and ventilation) due to the small number of patients, the critique focuses on the interpretation of intrinsic damage and the robustness of the analysis. If ventilation and hemodynamics are not the cause, the analysis should focus on why compliance is altered at the evaluated times. The study fails to present the underlying cause of this pre-existing dysfunction, not relating it to the etiology and duration of the pathology.

In the postoperative period following CABG, continuous sedation can contribute to hemodynamic stability and reduce the stress response by decreasing catecholamine and cortisol levels [10]. Modulation of the neuroendocrine response protects the brain against sudden changes in blood pressure and episodes of cerebral hyperperfusion or hypoperfusion, factors that affect cerebral autoregulation. Adequate sedation facilitates ventilatory management and prevents psychomotor agitation, keeping the patient responsive and comfortable, thus facilitating neurological monitoring [11]. Continuous sedation, in our pilot study, may have improved the stability of cerebral autoregulation, and justify the findings in ICC.

If sedation did not interfere, the improvement may be an effect attributed to the surgery itself or to an intrinsic factor of post-aggression self-regulation/adaptation (the surgery). It is unusual for cardiac surgery, which involves systemic inflammatory stress and cardiopulmonary bypass (which can affect cerebral microcirculation), to result in an immediate and significant improvement in compliance. Is the improvement in myocardial perfusion sufficient to explain the improvement in cerebral perfusion and compliance? The high variability of the P2/P1 ratio and TTP remains a weakness in this small number of patients. Even without external interference, such a high standard deviation indicates that the group response is highly heterogeneous. The analysis of means (T0, T1, T2) may be hiding subgroups with different responses. An

analysis by subgroups or by individual rate of change would be necessary to draw more precise conclusions. Despite the post hoc analysis of variance (ANOVA) and Bonferroni correction, the failure to detect a significant difference between T0 and T2 raises the question of whether the sample size is insufficient to detect a clinically relevant difference.

Further studies should be conducted with a larger number of patients to justify the compliance dysfunction observed preoperatively (T0); explain the unusual physiological mechanism that would lead to improved compliance at T1 without the interference of critical therapies (sedation/ventilation); and better address heterogeneity, as the high variability at T0 and T2 suggests that the exclusive use of the mean is insufficient and that the population is very heterogeneous in its response.

Conclusions

Noninvasive cerebral compliance evaluation showed to be a viable and safe method that may add data related to ischemia / reperfusion in highly complex cardiac procedures.

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