Cerebral hemodynamic effects of Cheyne-Stokes respiration in a patient with stroke.

Nogueira RC[1], Panerai RB[2,3], Teixeira MJ[4], Robinson TG[2,3], Bor-Seng-Shu E[4]

[1]Department of Neurology, Hospital das Clinicas, University of São Paulo School of Medicine, São Paulo, Brazil

[2] Department of Cardiovascular Sciences, University of Leicester, Leicester, LE2 7LX, UK

[3] Biomedical Research Unit in Cardiovascular Science, Glenfield Hospital, Leicester, LE3 9QP, UK

[4]Department of Neurosurgery, Hospital das Clinicas, University of São Paulo School of Medicine, São Paulo, Brazil

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Corresponding author: R.C. Nogueira
Department of Neurology
University of São Paulo School of Medicine
São Paulo, Brazil, 05410020
TEL: +55 (11) 26617207
rcnogueira28@gmail.com

Running title: Cerebrovascular effects of Cheyne-Stokes respiration

Abstract

Introduction: Cheyne-Stoke respiration (CSR) and Central Sleep Apnea (CSA) are common in patients with heart failure (HF) and/or stroke. We aim to describe the cerebrovascular effects of CSR during the acute phase of stroke in a heart failure patient. *Case Report:* A 74 year-old male with previous dilated cardiomyopathy had sudden onset of right hemiparesis and aphasia. A transcranial Doppler was performed with continuous measurement of BP (Finometer) and end-tidal CO_2 ($EtCO_2$, nasal capnography). Offline analysis of hemodynamic data disclosed relatively large periodic oscillations of both CBFV and BP related to the CSR breathing pattern. Derivate variables from the cerebrovascular resistance were calculated (critical closing pressure, CrCP and resistance-area product, RAP) demonstrating that there may be a myogenic impairment of CBF control in the affected hemisphere of this subgroup of patient. *Conclusion:* There is an impairment of CBF regulation in the affected hemisphere of the patient with ischemic stroke and CSR, highlighting the role of cerebral hemodynamic monitoring in this scenario.

Key words: ischemic stroke, Cheyne-Stokes, cerebral autoregulation, cerebral blood flow control, transcranial Doppler and ultrasound.

Introduction

Cheyne-Stoke respiration (CSR) and Central Sleep Apnea (CSA) are common in patients with heart failure (HF) and/or stroke (1, 2); the metabolic and cardiovascular changes observed in these conditions can have impact on the cerebral circulation (3, 4).

The control of cerebral blood flow (CBF) comprises a number of complex mechanisms to maintain cerebral perfusion despite changes in arterial blood pressure (BP) which is known as cerebral autoregulation (CA)(5). We aim to describe the cerebrovascular effects of CSR during the acute phase of stroke in a heart failure patient.

Case Report

A 74 year-old male with previous dilated cardiomyopathy had sudden onset of right hemiparesis and aphasia. Initial National Institute of Health Stroke Scale (NIHSS) was 20 and ~~s~~he received ~~was submitted to~~ thrombolytic therapy 3.5 hours after ictus. The patient had a dramatic neurologic response to therapy with final NIHSS of 1. Transcranial Doppler ultrasound (TCD) performed after thrombolytic therapy excluded any intracranial arterial occlusion or stenosis. Echocardiography revealed systolic ventricular dysfunction (ejection fraction of 24%). One day after admission ~~s~~he started to have pathological breathing suggestive of CSR. A second TCD was performed with continuous measurement of BP (Finometer) and end-tidal CO_2 ($EtCO_2$, nasal capnography).

Offline analysis of hemodynamic data disclosed relatively large periodic oscillations of both CBFV and BP related to the CSR breathing pattern (Fig. 1 A). For this analysis, a two-parameter model (critical closing pressure, CrCP and

resistance-area product, RAP) was proposed to replace the classical concept of cerebrovascular resistance (CVR). Previous studies demonstrated that CrCP reflects the metabolic control of CA while RAP reflects myogenic control(6). Curiously the oscillation of CrCP had the same pattern of oscillations as found in CBFV and ~~A~~BP, following the increase in $EtCO_2$ (Figure 1 A). However, RAP oscillations were less prominent and the pattern of oscillation for the affected and non-affected hemispheres was different in the coherent average (Fig. 1 B).

Discussion

Previous studies with TCD in patients with CSR disclosed marked changes in CBF velocity from the apnea to the hyperpnoea phases (3, 4). Moreover, it is hypothesized that these hemodynamic changes may influence clinical outcome in both situations (HF and stroke). Despite this concern, the contributions of regulatory mechanisms to explain the observed changes in CBF have not been reported previously.

This case report is the first to highlight the changes of CBF regulatory mechanisms, reinforcinges some physiological concepts of CA studies: 1) CBF regulation mechanisms are not impaired in the non affected side of this patient subgroup ~~of patients~~; 2) CrCP more likely represents the metabolic control of CA with higher oscillations mainly driven by CO_2 concentrations~~; and~~ 3) RAP represents myogenic control, following changes in BP, more than those in CO_2; and~~.~~ 4) The different pattern of RAP in both cerebral hemispheres suggests an impaired myogenic control of CA in the affected side (5). These findings highlight the role of cerebral hemodynamic monitoring in this scenario.

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Figure Legends

Figure 1A. Systemic and cerebral hemodynamic parameters (affected hemisphere, continuous line; unaffected hemisphere, dotted line), during Cheyne-Stokes breathing. CBFV (cerebral blood flow velocity), BP (blood pressure), CrCP (critical closing pressure), RAP (resistance-area product), $EtCO_2$ (end-tidal CO_2). Figure1B. Coherent average of multiple respiratory cycles of systemic and cerebral circulation parameters (affected hemisphere, continuous line; non-affected hemisphere, dotted line). Signals were normalized in percent. CBFV (cerebral blood flow velocity), BP (blood pressure), CrCP (critical closing pressure), RAP (resistance-area product), $EtCO_2$ (end-tidal CO_2).

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Figure(s)

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