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

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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 she 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 she started to have pathological breathing suggestive of CSR. A second TCD was performed with continuous measurement of BP (Finometer) and end-tidal CO₂ (EtCO₂, 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 ABP, following the increase in EtCO₂ (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, reinforcing 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 CO₂ concentrations; and 3) RAP
represents myogenic control, following changes in BP, more than those in CO₂;
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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References


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$_2$). Figure 1B. 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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