

The role of hyperventilation in dissociative disorders.

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Abstract

Hyperventilation is well documented in panic disorder. In dissociative and somatization disorders, hyperventilation is suspected to be important, but it has not been studied. The authors studied the arterial pressure of Oxygen and Carbon Dioxide, and other gasometric measures in the arterial blood of 5 dissociative, 5 somatization and 7 control female inpatients in a psychiatric ward. In addition to the clinical diagnosis, the patients were submitted to a diagnostic interview schedule (DDIS), and scales to detect dissociative (DES), and somatoform dissociative experiences (SDQ20), which confirmed the diagnoses. The main result was a lowering of *Bases* (*Bicarbonate*, *Total CO₂*, and *Actual Base Excess*) in dissociative patients, which was interpreted as a consequence of chronic overbreathing (frequent sighs), and as a factor of sensitivity to circumstantial variations of breathing, including acute hyperventilation. Anaerobic neuronal metabolism is suspected to be part of dissociative phenomena.

Introduction

The relationship between neurotic disorders and irregular breathing has been recognized for several years (Lopez Ibor, 1950; Salkovsky, 1988). Hyperventilation has been reported as a symptom of “hysteria” (Mai & Merskey, 1980) and “hysterical epidemics” (Alexander & Fedoruk, 1986; Brodsky, 1988; Olkinuora, 1984), but its causative role has not been sufficiently emphasized. However, it is known from clinical practice that voluntary hyperventilation can provoke a dissociative or conversive attack (Lopez Ibor, 1950: 79).

Hyperventilation is also related to panic attacks, and there is some evidence for its panic-precipitation effect (e.g. Hibbert, 1984; Ley, 1987; Salkovsky, Jones & Clark, 1986;

Wilhelm, Trabert, & Roth, 2001). Since there is some heterogeneity in the phenomenology of panic (Pio-Abreu, Ramalheira & Valente, 1998), a main role for hyperventilation may be assumed in a percentage of the attacks (Moynihan & Gevirtz 2001; Nardi et al., 2001). Re-breathing in a bag or breathing control can be therapeutic tools (Parker & Curtis, 1987), and clinical improvement is related to normalization of arterial pCO₂ (Salkovsky et al., 1986). There is already a lot of research and controversies in this field.

In contrast, the evidence for a causative and therapeutic role of breathing in dissociative and conversive attacks is only anecdotal (Goldman, 1992; Lopez Ibor, 1950; Margarian & Olney, 1984), but it can indeed be postulated. These attacks consist of varied phenomena, which frequently mislead the diagnosis, and correspond to atypical dissociative attacks, including possession, and to some of the pseudo-neurological DSM-IV symptoms of somatization disorder. Since they are different from one person to another, it is difficult to define them. However, they share the following features: (1) there is some motor activity and physical manifestations taking place in a standardized sequence, also present in past attacks; (2) they occur under crepuscular consciousness and generally leave no memory trace or personality integration; and (3) they imply secondary benefit and seem to be purposeful, in spite of the patient's absence of awareness.

When these attacks are a main clinical problem, we usually ask the patient to hyperventilate and produce his or her attack; we also teach the patient or relatives to use a bag or other strategies in order to control or stop overbreathing and the attack. We sometimes record the attacks on videotape, which is afterwards shown to the patient. These procedures have a dramatic impact on the patient and his/her family, contribute to improvement, and permit the patient to be aware of the attacks. Therefore, we can add a fourth feature to define this type of attack: (4) they can begin with hyperventilation and be stopped by re-breathing into a bag.

Thus, hyperventilation may be a touchstone in acute manifestations of neurotic disorders. However, a number of questions remain unanswered. For instance, we may ask why certain patients are more sensitive to hyperventilation, and why it provokes either panic or dissociative attacks in different people. We can invoke conditioning and habits, but this is not enough.

Hyperventilation provokes some well known physiological effects felt as similar to panic and somatization symptoms (Fried, 1987; Pincus, 1978). These effects may depend on intensity, pattern and timing of hyperventilation, previous physiological state of the organism, and concomitance of motor activity. However, in the literature of dissociative disorders, no systematic research has been devoted to this issue. Our goal is to study the physiological state of the organism as defined by arterial gasometry at rest, in female inpatients diagnosed with dissociative disorders.

Material and Methods

In the Women's Psychiatric Ward of Coimbra University Hospital (H.U.C.), five patients were diagnosed as having dissociative disorders in 2001. These patients were selected for the study, as well as eight women clinically diagnosed as belonging to “histrionic spectrum”, including somatization disorder, and nine with other diagnoses. The patients were interviewed with the Dissociative Disorders Interview Schedule (DDIS; Ross, 1989) and were asked to fill in the Dissociative Experience Scale (DES; Carlson, 1997) and the Somatoform Dissociation Questionnaire (SDQ20; Nijenhuis, 2000).

After the scheduled interview, two patients were diagnosed as having dissociative amnesia, one dissociative fugue, one depersonalisation, and one identity disorder. This was the study group. Five patients who fit somatization disorder criteria formed a second group. Finally, seven patients whose diagnoses did not belong to “histrionic spectrum”, borderline personality or panic disorder, but whose demographic data were close to the study group, were taken as a control sample. It was represented by two schizophrenics, one manic, two depressed, one obsessive-compulsive, and one drug-abusing patient. Table 1 shows data related to the samples.

Table 1: mean age of each sample and results of self-administration scales

	N	Age	DES	SDQ20
Dissociation	5	38,4	39,6	48
Somatization	5	39,4	26,7	43
Controls	7	39,4	12,6	26

The patients were of similar ages. The figures of DES distinguish dissociative disorder, and SDQ20 is higher in both groups diagnosed by DDIS. These findings were expected and they confirmed the diagnoses.

Three to 8 days after the admission and informed consent, these patients were submitted, at rest, to an arterial puncture in the radial artery. The blood was sent to the laboratory in order to measure the following data: *Acidity/Alkalinity* (pH), *Arterial Pressure of Carbon Dioxide* (pCO₂), *Arterial Pressure of Oxygen* (pO₂), *Bicarbonate* (HCO₃), *Total Carbon Dioxide* (TCO₂), *Actual Base Excess* (ABE), *Haemoglobin Saturation* (SAT), and arterial *Lactates*.

Means and standard deviations were computed for every measure in each group. These means were compared using the t-Test.

Results

Table 2 shows the overall results. The main results are a significant lowering of *Bases* (*Bicarbonate*, *Actual Base Excess*, *Total CO₂*) in the dissociative group. These patients also tend significantly to have *acidosis* at rest, in spite of no significant lowering of pCO₂. They also have a higher concentration of arterial *lactates* (p<0,05).

Table 2. Gasometric measures in arterial blood, comparing the 3 groups of patients. The significant differences are highlighted

		pH	pCO ₂	pO ₂	HCO ₃	TCO ₂	ABE	SAT	Lactate
Dissociation (N=5)	<i>Mean</i>	7,38	36,5	99,9	22,4	23,6	-1,72	97,20	1,92
	<i>SD</i>	0,05	3,35	17,9	1,2	1,3	0,95	1,79	0,68
Somatization (N=5)	<i>Mean</i>	7,42	37,3	94,5	23,6	24,8	-0,48	97,14	1,28
	<i>SD</i>	0,02	3,94	12,9	3,2	3,3	2,99	1,01	0,44
Control (N=7)	<i>Mean</i>	7,44	38,9	104,3	25,6	26,8	1,49	97,93	1,19
	<i>SD</i>	0,03	4,76	22,3	1,92	2,1	1,32	0,96	0,35
p (T test)	<i>Dissociation/Control</i>	0,036	0,360	0,723	0,009	0,012	0,001	0,457	0,038
	<i>Somatization/Control</i>	0,299	0,536	0,404	0,214	0,228	0,150	0,459	0,688

The somatization patients show means between the other samples (except the lower

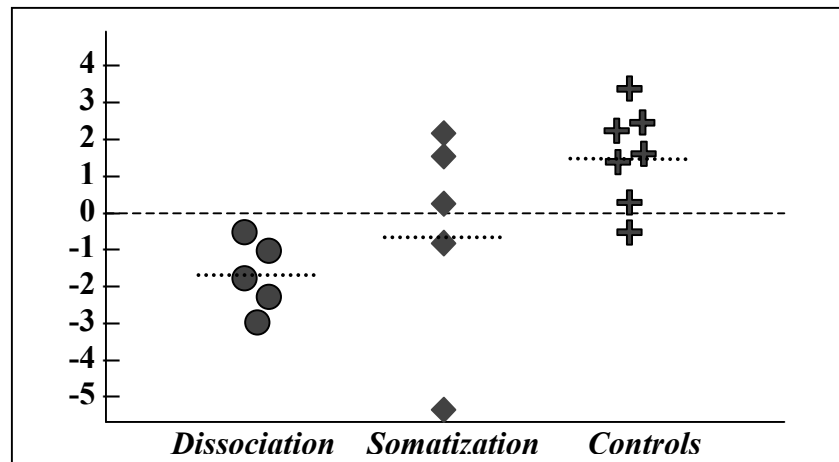


Fig. 1— Individual results of Actual Base Excess in the 3 groups of patients.

pO₂), but without statistical significance. With respect to the *Bases* (Bicarbonate, Actual Base Excess, Total CO₂), this group shows a higher Standard Deviation, revealing that the data are more widespread. This can be seen in fig. 1, where individual measures of *Actual Base Excess* are displayed.

Discussion

The results of the present study must be treated with caution, since they are based on a small number of patients. However, they are impressive: every dissociative patient had negative *Base Excess* (see fig.1), mainly as a result of low *Bicarbonate*. As far as we know, this is the first study where such a measure was performed, and therefore it must be replicated in subsequent research. The present finding of the lowering *Bases* may be a consequence of frequent overbreathing, which leads to chronic respiratory alkalosis and compensatory *bicarbonate* loss via the kidneys (Compensatory Metabolic Acidosis). In fact, the dissociative patients tend to have acidosis at rest (when the arterial puncture is performed), but they can compensate for it by overbreathing during normal life and, as is frequently observed, they indeed hyperventilate during the dissociative attacks.

Margarian & Olney (1984) described a 66-year-old man suffering “absence spells”, which fit DSMIV criteria for dissociative fugue, for 17 years. After several negative treatments and laboratory studies, arterial blood revealed a pH of 7.44; pO₂ 85 and pCO₂

33. Deliberate hyperventilation was found to provoke the “absence” symptoms, which ceased after rebreathing into a bag. After an explanation of the symptoms, and breathing education, the patient improved. The authors also report a previous gasometry (pH 7.50; pO₂ 99 and pCO₂ 25), but they did not report any measure of *Bicarbonate* or *Base Excess*.

In fact, measures of pH, pO₂ and pCO₂, are evanescent and dependent on the ventilation at the time of measuring. Since the arterial puncture is performed at rest, these measures may be normal (asking the patient to hyperventilate in order to provoke an attack would give obvious results). However, concentration of *Bicarbonate* is longer lasting and independent of circumstantial ventilation. Furthermore, *Bicarbonate* is an important buffer solution. Without it (or with a *Base* deficit), breathing variations of CO₂ provoke easier alkalosis or acidosis (Rose, 1997). Thus, the lowering of *Bases* can explain the hypersensitivity to the effects of hyperventilation (and also, perhaps, alternate hypoventilation).

The point of departure of the present study was a group of psychiatric patients with a well-documented diagnosis of dissociative disorder. Gasometry was carried out as part of the study, but it was not a clinical requirement. According to Margarian & Olney (1984), the presence of a pathophysiological mechanism should preclude dissociative diagnosis. However, in view of our results, we could ask how many diagnosed cases would remain without, at least, a slight deficit of *Bases*. Perhaps, gasometry should be a routine laboratory test in psychiatric wards.

Some somatization patients and controls with other diagnoses have a similar *Base* deficit (see fig. 1), and anxious or panic patients may share ventilation mechanisms (Salkovsky, 1988; Nardi, Valença, Mezzasalma & Zin, 2001; Wilhelm, Trabert & Roth 2001). Thus, chronic hyperventilation and lower Bicarbonate may be a concurrent but not sufficient cause of dissociation. Defence against unbearable experiences, previous conditioning and habits, personal and cultural beliefs, and social support, must be concomitant causes (Cardena, 1997; Kirmayer 1997). One main difference between panic and dissociative patients is the fact that immobilization is the rule in fearful panic patients, while there is always some motor activity in dissociative/conversive patients. Since hyperventilation causes cerebral vasoconstriction, and hyperventilation-induced alkalosis

makes haemoglobin retain oxygen, both provoking paradoxical hypoxia, we can consider the possibility of anaerobic metabolism during the dissociated activity of patients.

The metabolism of the Central Nervous System is strongly aerobic, but there are some anaerobic means of neuronal transmission. At present, these pathways are scarcely known. However, they can play an important role in emergency mechanisms, in the same way that anaerobic muscle fibres do (MacKey, 1997: 172). Provoking a dissociative attack by hyperventilation can be a way of putting into action such mechanisms. Anaerobic metabolism could be verified by an excess of *Lactate*, the arterial concentration of which is higher in our dissociative patients. However, this question deserves more research.

Somatization patients, in spite of belonging to the histrionic spectrum, have gasometrical figures closer to the control sample. However, their *Bases* and *Bicarbonate* tend to be slightly lower but without statistical significance, since they show a wider dispersion (see fig. 1). Perhaps the present diagnostic criteria for somatization disorder may detect a heterogeneous population, where some patients are close to dissociative disorder.

In short, the dissociative patients we have studied were in a physiological state of metabolic acidosis at rest, during the time that they could display memory and consciousness symptoms. The presence of this state can be explained by chronic hyperventilation (frequent sighs), and it makes the patient more sensitive to the effects of irregular breathing, which includes acute overbreathing. These effects putatively facilitate anaerobic metabolism which mediates automatic behaviour.

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