

Weak clinical effect of airway clearance on ADH and BNP concentration: mechanism or data collection BIAS

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To the editor,

Nocturnal enuresis is a disorder characterized by intermittent urinary incontinence that occurs during periods of sleep for at least one episode per month for at least 3 months (1). These episodes occur at a time when the child should, under normal circumstances, be able to control urination. In addition, although some causes are more common, such as lack of pelvic floor muscle control (2) and the presence of psychiatric disorders (3), there are also pathophysiological factors that can lead to the onset of nocturnal enuresis, such as obstruction of the upper airways (4).

This pathophysiological picture is captured by sympathetic receptors of the Autonomic Nervous System, which cause greater cardiomyocyte activity, secreting B-type natriuretic peptide (BNP) by the ventricles, which stimulates natriuresis and diuresis in order to compensate for the vasoconstrictor systems. that are activated in these situations. This increase in BNP, due to the pathophysiological condition of OSA, causes inhibition of the secretion of antidiuretic hormone (ADH), which regulates diuresis through the reabsorption of water in the collecting ducts (4).

The study by Ribeiro et al. (5), published in this journal in issue 48 (6), presents interesting results on enuresis. However, we bring an analysis based on clinical effect measurement to complement the probabilistic analysis presented by the authors. The measure of the clinical effect used is the Cohen's d, whose meaning is used to find out the usefulness of the probabilistic effect, which is influenced by the sample size and selection method (6).

The improvement shown for nocturnal enuresis in the study by Ribeiro et al. (5) was clinically very large (d=1.50) compared to the effects of surgery on the concentration of ADH (d=0.38) and BNP (d=0, 32), which are considered small clinical effects, even the comparison before and after BNP being significant from the probabilistic point of view.

From this, due to the paradoxical effect of ADH and BNP, some considerations of the mechanism of effect of upper airway obstruction on nocturnal enuresis in children arise: the small clinical effect of the increase in BNP is enough to strongly increase the proportion of nights dry? Can the delay time in data collection between 90 and 120 days affect hormonal indicators mainly in the heterogeneity of the sample? Is there another factor involved? Therefore, we can conclude that airway clearance modifications had little clinical impact on the hormonal actions of ADH and BNP as collected.

The Authors

CONFLICT OF INTEREST

None declared.

Table 1	- Measures of	f the clinical effect of	i upper airwa	/ clearance on enuresis and BNP	and ADH hormones.

	X_{before}	X_{after}	SD_{before}	SD_{after}	$X_{before} extsf{-}X_{after}$	SD _m	Ν	Square SD	Cohen's d
BNP (pg/mL)	116.5	156.2	126.5	112.3	-39.7	716291.05	49	120.90	-0.32
Dry nights %	32.3	75.4	24.7	33.4	-43.1	40003.81	49	28.57	-1.50
ADH (pg/mL)	5.8	14.6	3.2	35.4	-8.8	25360.16	49	22.74	-0.38

ADH = anti-diuretic hormone; BNP = brain natriuretic peptide; X = Mean; SD = Standard deviation; N = sample number.

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