Anxiety and hyperventilation in mild to moderate asthmatics

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Résumé

Les crises d'asthme s'accompagnent fréquemment d'hyperventilation. Nous avons comparé le pattern ventilatoire ($PetCO_2$, volume courant et fréquence respiratoire) de 80 asthmatiques légers à modérés à celui de 40 sujets contrôles sains appariés afin de déterminer si le groupe d'asthmatiques présente une hyperventilation clinique et/ou physiologique, et si c'est le cas, si les différences observées entre les deux groupes sont liées à des caractéristiques ventilatoires ou psychologiques. Les asthmatiques de notre étude ont une $PetCO_2$ considérablement plus basse (32.61±3.36 mmHg) que celle des sujets sains (34.8 ± 3.1 mmHg) à p=. 0009. L'analyse de régression montre que les variables qui expliquent le mieux la $PetCO_2$ basse chez les asthmatiques sont le volume courant (p=.0001), le sexe féminin (p=.0001) et l'affectivité négative (p=.04). Les femmes présentent un pattern ventilatoire et un profil psychologique calqués sur ceux des asthmatiques; comme eux, les femmes ont une $PetCO_2$ plus basse et un volume courant plus faibles, une plus grande sensibilité à l'anxiété, une affectivité négative, une conscience de soi et des symptômes d'hyperventilation plus importants, et moins de sens de la cohérence que les hommes. Par contre, elles ne décrivent pas significativement plus de symptômes d'asthme que les hommes. Ces résultats suggèrent qu'il est important d'identifier des catégories de patients qui requièrent une attention particulière en raison de leur profil psychologique. Il semble utile d'identifier l'hypocapnie chez les asthmatiques. Le traitement de l'hyperventilation physiologique est susceptible d'apporter une amélioration au niveau émotionnel et comportemental mais n'a pas été suffisamment étudié.

Mots clés : Asthme - Hyperventilation - anxiété

Abstract

Asthma attacks are frequently accompanied by hyperventilation. We have compared the breathing pattern ($PetCO_2$, tidal volume and respiration rate) in 80 mild to moderate asthmatics with that of 40 matched healthy control subjects in order to determine whether the group of asthmatics proved to have clinical and/or physiological hyperventilation, and if so, whether the differences observed in the two groups were related to ventilatory or psychological characteristics.

The asthmatics in our study had a considerably lower $PetCO_2$ (32.61$\pm$3.36 mmHg) than the healthy subjects (34.8 ± 3.1 mmHg) with *p*=. 0009. The regression analysis shows that the variables that best explain the low $PetCO_2$ in asthmatics are tidal volume (p=.0001), female gender (p=.0001), and negative affectivity (p=.04). Women seem to have a breathing pattern and psychological profile modelled on those of asthmatics; like asthmatics, women have a lower $PetCO_2$ and tidal volume, greater anxiety sensitivity, trait anxiety, negative affect, self-consciousness and symptoms of hyperventilation, and less sense of coherence than men. On the other hand, they do not describe significantly more asthma symptoms than men. These results suggest how important it is to identify the classes of asthmatic patients who require more attention because of their psychological profile. It seems to be useful to identify hypocapnia in asthmatics. The treatment of this physiological hyperventilation is likely to result in emotional and behavioural improvements, although this has not yet been sufficiently studied.

Keywords : Asthma – hyperventilation - anxiety

Introduction

Asthma attacks are frequently accompanied by hyperventilation (McFadden & Lyons, 1968; Hardonck & Beumer, 1979; Demeter & Cordasco, 1986; Gardner, Bass, & Moxham, 1992; Fried & Grimaldi, 1993; Peiffer, 1995; Gardner, 1996; Gardner, 2000; Osborne, O'Connor, Lewis, Kanabar, & Gardner, 2000; Thomas, McKinley, Freeman, & Foy, 2001). Hyperventilation in asthmatics may also be triggered by anxiety (Hibbert & Pilsbury, 1988a; Carr, Lehrer, & Hochron, 1992; Lehrer, Isenberg, & Hochron, 1993; Carr, Lehrer, Rausch, & Hochron, 1994; Carr, 1998). Similarly, hyperventilation, which chills the airways, is likely to provoke bronchoconstriction in vulnerable persons, which in turn exacerbates hyperventilation, leading to a vicious circle (Hibbert & Pilsbury, 1988b). However, the mechanisms underlying hyperventilation in asthmatics remain quite vague (Lavietes, 1984; Osborne *et coll.*, 2000). The etiological factors which contribute to its onset are largely undetermined. Many studies have shown that mild, often undiagnosed, asthma is a major etiological factor and may lead to a vicious circle of panic (Gardner *et coll.*, 1992; Gardner, 1996; Saisch, Wessely, & Gardner, 1996).

Osborne *et coll.* (2000) compared mild to moderate asthmatic patients with healthy controls but were unable to demonstrate the presence of clinical hyperventilation in the asthmatics. Nevertheless, they showed a statistically significant reduction in arterial and alveolar PCO_2 which is not related to pulmonary function, bronchial inflammation, anxiety or depression, but is associated with bronchial hyperreactivity. A low level of hypocapnia may therefore be related to the contractility of the bronchial smooth muscles in asthma.

A recent study using a mailing of the Nijmegen questionnaire attempted to assess the prevalence of a dysfunctional breathing pattern in adult asthmatics. It concluded that approximately one-third of women and one-fifth of men had positive scores (Thomas *et coll.*, 2001). However, the authors did not have objective data confirming the diagnosis of asthma or the severity of the disease. Moreover, the Nijmegen questionnaire has not been validated in asthmatic populations. Finally, we should note that even the existence of a 'hyperventilation syndrome' has been seriously questioned (Bass, 1997; Keeley & Osman, 2001).

Based on the literature, in this study we have compared the breathing pattern ($PetCO_2$, tidal volume and respiration rate) in mild to moderate asthmatics with that of matched healthy control subjects in order to determine (1) whether the group of asthmatics proved to have clinical and/or physiological hyperventilation, and (2) if so, whether the differences observed in the two groups were related to ventilatory or psychological characteristics. Clinical implications of the findings will be discussed.

Methods

Subjects

The experimental group was made up of 80 mild to moderate asthmatic subjects, selected in accordance with the American Thoracic Society's criteria (ATS, 1993). The subjects included in the study were between 16 and 60 years old and had to have asthma symptoms at the time of recruitment, and either a reversibility of 15% or 200 ml or a $PC_{20}M$ FEV_1 lower than or equal to 16 mg/ml. In addition, they could not suffer from any other pulmonary disorders, could not have contracted any recent respiratory infection, and had to have received a prescription for at least one anti-asthmatic medication.

The control group (N = 40), recruited in the personnel of the hospital and the university, was matched to the experimental group on the basis of sociodemographic characteristics (see Table I). Controls could not have any known respiratory diseases or allergies. In addition, a $PC_{20}M$ was carried out to be certain of excluding any bronchial hyperreactivity.

The patients were recruited in the Pulmonology Department of the Centre Hospitalier Universitaire de Liège (CHU) during consultations at the hospital clinic. The experiment was approved by the CHU's Ethics Committee. All subjects signed informed consent forms before taking part in the experiment.

Procedure

After recruitment and a bronchial challenge test, the patients went to the Functional Respiratory Test laboratory within eight days. They were told not to take bronchodilators for at least 24 hours before the test (48 hours in the case of long-acting beta-2 agonists) and not to take corticosteroids on the day of the test. After completing the questionnaires (cf. psychological measures, below), the subjects did spirometry and plethysmography tests. They were then seated in a quiet room and hooked up to the analyzer (MV Max 22, SensorMedics), which had a mouthpiece and nose-clip. Instructions concerning breathing were kept to a minimum. Recording only began after a five-minute stabilisation period; it lasted three minutes.

Bronchial reactivity

Bronchial hyperreactivity was measured by a methacholine bronchial challenge test modified according to the Cockcroft method (Cockcroft & Hargreave, 1991) and using quadrupled doses starting at 0.06 mg/ml. The maximum amount of methacholine used was 16 mg/ml. Only patients with a $PC_{20}M$ (concentration of methacholine necessary to achieve a 20% drop in FEV_1) lower than or equal to 16 mg/ml were included in the study. All patients were followed up in the outpatient clinic. The control subjects had to have a $PC_{20}M$ greater than 16 mg/ml.

Pulmonary function

The measurement of pulmonary function included the measurement of forced expired volume in one second (FEV_1), forced vital capacity (FVC) (MV Max 22 spirometer, SensorMedics), and airways resistance (Raw) and specific conductance (sGaw) (MV Max 22 plethysmograph, SensorMedics).

Ventilation and breathing pattern

Ventilation was measured in a seated position over three minutes after values had stabilised, using a mouthpiece and nose-clip. Tidal volume (V_t), respiration rate (R), respiratory minute volume (V_E), and partial end-tidal alveolar CO_2 pressure ($PetCO_2$) are recorded for each respiratory cycle. The values presented are the averages for the three minutes recorded.

Psychological measures

The *Anxiety Sensitivity Index – Revised* (ASI-R) (Taylor & Cox, 1998a) is an extension of the Anxiety Sensitivity Index (Reiss, Peterson, Gursky, & McNally, 1986), which measures fears of consequences that could result from anxiety symptoms. This is a scale measuring 'fear of fear', that is, the belief that anxiety symptoms have negative effects. The scale is made up of 36 items, the sum of which constitutes the total score. The subjects evaluate to what extent they agree with each item on a 5-point Likert scale (0 = very little to 4 = very much), resulting in a total score of 0 to 144. The scale contains six subscales that measure the following areas: fear of cardiovascular symptoms; fear of respiratory symptoms; fear of gastrointestinal symptoms; fear of publicly visible reactions; fear of dissociative and neurological symptoms; fear of loss of cognitive control. The six subscales have good internal consistency and a high alpha. Reiss *et coll.* (1986) consider anxiety sensitivity to be a predisposing factor for the development of anxiety disorders. It is supposed to be an individual difference associated with agoraphobia in particular and other anxiety disorders in general. Carr *et coll.* (1994) have shown that asthma alone (without panic disorders) is not associated with a high ASI, but they used the 16-item version. The ASI has excellent internal consistency in clinical and nonclinical populations (range of alpha coefficient 0.79 to 0.90) and good test-retest reliability.

The *Self Consciousness Scale – Revised* (SCSR) (Fenigstein, Scheier & Buss, 1975; revised by Scheier & Carver, 1985; French translation by Pelletier & Vallerand, 1990) is a scale intended to measure the individual differences related to the private and public aspects of self-consciousness. The term 'private self-consciousness' refers to a person's tendencies to think about and pay particular attention to the hidden and more intimate aspects of the self, that is, the aspects that are of a personal nature and not easily accessible to others. On the other hand, the term 'public self-consciousness' refers to the tendency to think about the aspects of the self that are more easily observable by the people around us or that can be used by another person to form an impression of us. Finally, the scale measuring social anxiety makes it possible to evaluate a specific type of reaction to the fact of being conscious of the public aspect of the self. This questionnaire includes 22 statements, assessed according to a 4-point scale (from 0 = not at all like me to 3 = very like me). The total score for the scale ranges from 0 to 66; the score for the private self-consciousness subscale ranges from 0 to 27; public self-consciousness, 0 to 21; social anxiety, 0 to 18. The scale has an acceptable level of internal coherence. The cronbach alpha measure of internal consistency of private self-consciousness is 0.73, the one of public self-conscioussness is 0.82; and the one of social anxiety is 0.74. Test retest reliability is respectively 0.82, 0.86 and 0.78.

The *State Trait Anxiety Inventory* (STAI) (Spielberger, Gorsuch, & Lushene, 1983) is very widely used in research and clinical practice. It includes two separate scales. The trait anxiety scale (YB form) has been widely used to measure clinical anxiety. It has also been used to identify anxious subjects in larger groups. It includes 20 items, evaluated by the subject from 1 (almost never) to 4 (almost always). The total score is transformed into a t-score based on the subject's gross score and sex. The psychometric qualities of the French-language version were established by Bruchon-Schweitzer and Paulhan (1993).

The *Nijmegen Questionnaire* (Van Doorn, Folgering, & Colla, 1982) evaluates the frequency of complaints of hyperventilation. This questionnaire is made up of 16 items assessed on a 5-point Likert scale (0 = never to 4 = very often). It draws on the symptoms most often found in hyperventilation syndrome. These include cardiovascular, neurological, respiratory, gastrointestinal, and psychological symptoms. A mark of higher than 23/64 indicates a probable hyperventilation syndrome. This is the questionnaire most often used to diagnose this syndrome. The sensitivity in relation to the clinical diagnosis is 91% and the specificity 95%.

The *Sense Of Coherence Scale* (SOC) (Antonovsky, 1987; 1993) presents a salutogenic theoretical model which focuses on the origins of well-being. It conceptualises health as a continuum between the opposite poles of well-being and illness. Antonovsky provides a model that makes it possible to assess the individual characteristics involved in this dynamic of adaptation and health: the sense of coherence model. The 13-item short-form version was used in this study. Subjects are invited to assess 13 statements on a 7-point scale (1 = very often to 7 = very rarely or never). Three items were reversed, with the result that a high score indicates a good sense of coherence. In 16 studies using SoC-13 the cronbach alpha measure of internal consistency has ranged from 0.74 to 0.91. The relatively few test-retest correlations show considerable stability.

The *Positive and Negative Affect Schedule* (PANAS) (Watson, Clark, & Tellegen, 1988) comprises two scales of 10 items each, the Positive Affect Scale and the Negative Affect Scale. These scales have a high internal consistency, are largely uncorrelated, and are stable at two months. They have converging and discriminating correlations with more long-term measurements of underlying mood factors. They show a degree of stability similar to that of a trait when used with long-term descriptors (e.g., 'in general, you feel…', as used in this study). According to Watson *et coll.* (1988), negative affect is a general dimension of subjective stress and disagreeable obligations which sums up a variety of moods, including anger, contempt, disgust, guilt, fear and anxiety; low negative affect is a state of calm or serenity. These two factors represent dimensions of the affective state, but Tellegen (1985) has shown that they are related to affective trait dimensions with a positive or negative emotion (individual differences in positive and negative emotional reactivity). The questionnaire has 10 items assessed on a 5-point scale (1 = very little/not at all to 5 = very much). A high value indicates a high degree of negative affect. The scale demonstrated appropriate stability over a 2-month time period. The scale is internally consistent. The coefficient alpha is 0.87.

The *Beck Depression Inventory* (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961 ; French translation Delay, Pichot, Lemperiere,& Mirouze, 1963) provides a quantitative estimate of the severity of depression. Each item comprises four sentences that correspond to four degrees of intensity of a symptom on a scale of 1 to 3. The subject must choose one or more sentences that apply to him or her for each item, but only the sentence that has the highest value is taken into consideration in the score. The global score (0 to 59) is obtained by adding up the scores for the 21 items. The higher the score, the more severe the depression. The test's internal consistency is satisfactory.

The *Asthma Symptoms Checklist* (ASC) (Kinsman, Luparello, O'Banion, & Spector, 1973, revised by Brooks *et coll.*, 1989) is a 5-point Likert scale (1 = very little/not at all to 5 = very much) on which patients report the frequency with which 36 symptoms specific to asthma are present at that exact time. The version used in this study is the one revised by Brooks *et coll.* (1989). These authors identified five factors: panic fear, bronchial obstruction, hyperventilation, fatigue, and irritability. The psychometric properties of the factor scores in the revised version are satisfactory. The reliability is strong—correlation analyses prove its validity, especially for the 'panic fear' factor. Psychometric properties of the factors are satisfactory. The reliability is high (from 0.84 to 0.94), and correlational analyses support the construct validity.

Data analysis

The difference between asthmatics and control subjects was analyzed using Student t-tests for independent samples; when the variables were nominal, they were compared with a Pearson's chi-square. The Bravais-Pearson correlation coefficient was calculated among all dependent variables for the asthmatic group only.

Multivariate analyses of variance (MANOVAs) were used to verify the general impact of sex and the presence of asthma on the dependent variables. Student t-tests made it possible to compare individual variables.

Logarithmic transformations were done on certain variables when the conditions of normality, homoscedasticity and symmetry were not respected, so that parametric statistics could be used.

A linear regression was done to evaluate the effect of certain dependent variables on $PetCO_2$. Due to the collinearity of certain variables and the need to retain approximately ten subjects per independent variable, the variables with an alpha threshold of greater than 1.5 in univariate mode were not introduced into the regression. Because of missing data, the degrees of freedom may vary somewhat.

All the analyses were done accepting the usual uncertainty level of 5%. Nevertheless, given that many comparisons were carried out, values of $p < .05$ may only suggest an association, whereas $p < .01$ indicates a clear association, and $p < .001$ indicates a strong association (Czajkowski *et coll.*, 1997).

Results

Characteristics of subjects

Insert table I

Table I indicates the mean and the standard deviation for the sociodemographic variables and pulmonary function of the two groups. The Student t-tests indicate that the means for the two groups are not statistically different with regard to age ($t_{(118)} = -0.14$, $p = .88$), education ($t_{(118)} = 0.79$, $p = .42$), height ($t_{(118)} = 0.83$, $p = .4$) and weight ($t_{(118)} = -0.84$, $p = .39$). Pearson's chi-square indicates the independence of group and sex ($\Pi^2_{(1)} = 0.16$, $p = .68$). The same is true of smoking ($\Pi^2_{(2)} = 2.94$, $p = .23$).

The pulmonary function of the mild to moderate asthmatic subjects in the sample was quite different from that of the control subjects with regard to FEV_1 ($t_{(118)} = 3.05$, $p = .002$), airways resistance ($t_{(118)} = -3.44$, $p = .0008$) and specific conductance ($t_{(118)} = 2.5$, $p = .01$), as expected. The means for the control subjects were normal mean values. On the other hand, the forced vital capacity ($t_{(118)} = 1.47$, $p = .14$) and Tiffeneau coefficient ($t_{(118)} = 2.54$, $p = .12$) did not differ for the two groups.

The average duration of the disease was 8.84 (12.68) years. The mean $PC_{20}M$ for asthmatics was 4.66 (5.38) mg/ml. Fifty-four asthmatics had an inherited predisposition to allergies, whereas 20 had no known allergies. The average dose of beta-2 agonists was 0.88 (1.59) inhalations per day, with a minimum of 0 and a maximum of 8.5.

Ventilation et psychological profile

A general 2 x 2 multivariate analysis of variance (MANOVA) (Status (asthmatic vs. control) x Sex (male vs. female)) was done to test the effect of asthma and sex on the four ventilatory variables, on the one hand, and the seven psychological variables, on the other hand.

With regard to breathing pattern, the effect of asthma is significant (p = .002), as is the effect of sex (p = .0000), but the effect of the interaction is not significant.

The psychological variables also indicate an effect for asthma (p = .0005) and one for sex (p = .04), but no effect for interaction. That is why the independent variables were tested separately with Student t's.

Influence of asthma

Table II shows that the $PetCO_2$ for asthmatics is in fact 2.21 mmHg lower than that for healthy subjects ($t_{(109)}$ = 3.4, p = .0009); on the other hand, respiration rate ($t_{(109)}$ = –1.6, p = 1.11), respiratory minute volume ($t_{(109)}$ = –1.33, p = .18), and tidal volume ($t_{(105)}$ = –0.65, p = .51) do not differ significantly. Both groups present normal values, albeit at the high end of the range.

With regard to psychological measures, the trait anxiety scale is equivalent for both groups ($t_{(117)}$ = –1.57, p = .12), but is remarkably high, for the control group as well. The asthmatics have a higher depression score than controls ($t_{(117)}$ = –3.2, p = .001), at the upper limit of the normal score (mild depression > 10). The sense of coherence differs ($t_{(116)}$ = 2.68; p = .008), with asthmatics revealing a significantly lower sense of coherence than healthy controls.

No difference was found between asthmatics and controls with regard to anxiety sensitivity ($t_{(117)}$ = –1.37, p = .17), negative affect (t(116) = –0.23, p = .81) and self-consciousness (t(117) = 0.02, p = .98).

Asthmatics have more symptoms of hyperventilation than healthy subjects (t(117) = –4.4, p = .00002); their high score on the Nijmegen questionnaire (25.4 ± 12.2) reflects probable hyperventilation (> 23).

Insert table II

Influence of gender

When one divides groups based on gender, one finds that women have a considerably lower $PetCO_2$ than men ($t_{(109)} = -3.52$, $p = .0006$). The t-scores also indicate a sex effect for V_t ($t_{(105)} = -4.17$; $p = .00006$) and V_E ($t_{(109)} = -2.81$, $p = .005$).

The same is true of the Nijmegen questionnaire, on which women score 9 points higher ($t_{(117)} = 4.19$, $p = .00005$).

The ASI-R shows a difference ($t_{(117)} = 2.66$, $p = .008$) on the subscales for fear of respiratory symptoms ($p = .0009$), fear of gastrointestinal symptoms ($p = .02$), and fear of dissociative and neurological symptoms ($p = .012$).

Negative affect is slightly higher in women ($t_{(116)} = 2.4$, $p = .02$), as is trait anxiety ($t_{(117)} = 1.92$, $p = .06$). Total SOC is lower among women ($t_{(116)} = -2.38$, $p = .02$).

Self-consciousness, both private and public, is no different ($t_{(117)} = 2.21$, $p = .3$); on the other hand, the social anxiety subscale indicates less anxiety in women (8.6 ± 4.3) than in men (6.9 ± 4.1), with $p = .037$.

Finally, results on the ASC are no different for men and women ($t_{(78)} = 0.65$; $p = .51$).

Insert table III

Relationship among variables

Table IV summarises the different correlations between physiological and psychological variables within the group of asthmatic subjects alone.

Insert table IV

Ventilation (V_E) is negatively correlated with $PetCO_2$. The latter is also negatively correlated with tidal volume, but not with respiration rate or the Nijmegen questionnaire, which is correlated with respiration rate.

One can see that the psychological variables are correlated with one another but none of them is correlated with the physiological variables, except the STAI and the Nijmegen questionnaire, which are inversely correlated with tidal volume.

Multiple regression

When one attempts to explain the $PetCO_2$ in the asthmatic group by a multiple regression analysis using the variables V_t, TAI, Neg, Sex, ASC, ASC PF, ASC irritability and ASC fatigue, one obtains an R^2 of .51 and $F_{(8,57)} = 7.36$, $p = .00001$.

Insert table V

One can see that the more important factors are tidal volume, sex, negative affectivity, and asthma symptoms, especially fatigue and irritation.

Discussion

We compared the breathing patterns of 80 mild to moderate asthmatic subjects and 40 healthy subjects, and we matched these physiological variables with psychological variables in order to try to explain any potential differences in the $PetCO_2$ of the two groups.

The sociodemographic data for the asthmatics were comparable to those for the subjects without asthma. Pulmonary function was different: the asthmatic group had a mean FEV_1 of 93%, a lower value than that of the control subjects (100.6%). Their mean $PC_{20}M$ was 4.66 ± 5.38 mg/ml.

The $PetCO_2$ in our healthy subjects (34.8 ± 3.1 mmHg) is lower than the generally accepted norm (40 ± 2 mmHg), but $PetCO_2$ measurements may vary from one device to another. Gardner (1996) recommends relaying on a control group rather than pre-established norms. However, Osborne, Gardner and Varley (1997) measured $PetCO_2$ using a mobile capgnograph in six healthy subjects; they obtained a mean of 34.4 ± 5.1 mmHg, and a range of 28.9 ± 2.2 to 40.9 ± 3.4 mmHg. These authors suggest that the range of the normal $PetCO_2$ should be extended downward.

The asthmatics in our study had a considerably lower $PetCO_2$ than the healthy subjects (2 mmHg). The other values are not significantly different, but the very large standard deviation is an indication of quite disparate breathing patterns.

Hormbrey, Jacobi, Patil and Saunders (1988) compared the breathing patterns of six patients with symptomatic hyperventilation, six mild asthmatics, and six healthy subjects. At rest, the hyperventilating subjects had normal mean ventilation and $PetCO_2$ numbers. The asthmatics had a higher ventilation rate (11.1 litres/minute and a respiration rate of 15.5) and a lower $PetCO_2$ (37 mmHg) than healthy (respiration rate of 13.9, ventilation of 9.7 and $PetCO_2$ of 40 mmHg) and hyperventilating subjects (respiration rate of 13, ventilation of 8.1 and $PetCO_2$ of 41 mmHg).

It is important to remember that it is very difficult to obtain breathing pattern measurements 'at rest' because of the impact of the mouthpiece on breathing. Han *et coll.* (1997) showed that the mere fact of breathing through a mouthpiece causes $PetCO_2$ to decline gradually in hyperventilating subjects and patients with anxiety disorders, but not in normal subjects. In subjects less than 28 years old, this decline in $PetCO_2$ results in an increase in tidal volume; in subjects over age 29, it creates an increase in respiration rate. Breathing through a mouthpiece makes the respiratory irregularities observed in everyday life disappear almost completely. This often results in respiratory discomfort and even anxiety, especially in women. Furthermore, use of the mouthpiece significantly increases tidal volume, again especially in women (Fried & Grimaldi, 1993). According to Gilbert, Auchincloss and Boden (1972), this triggers a slowdown in respiration rate and an increase in tidal volume, without any change in respiratory minute volume. Other researchers (Askanazi *et coll.*, 1980; Sackner, Boudy, Davis, Cohn, & Sackner, 1980) have reported similar increases in tidal volume, but changes in respiration rate are variable. Hirsch and Bishop (1982) also found a decrease in respiration rate and an increase in tidal volume when a mask is substituted for the mouthpiece, but without any change in minute volume (V_E). They do not attribute this change to blockage of the nostrils.

Awareness that the breathing pattern is being recorded also tends to prolong inhalation and exhalation time and decrease respiratory irregularities (Han, Stegen, Cauberghs, & Van de Woestijne, 1997). Thus, awareness of being recorded modifies the spontaneous breathing pattern: respiration rate decreases and tidal volume increases, whereas $PetCO_2$ does not appear to be affected.

The ventilation, tidal volume and respiration rate of the subjects in this study are normal, although at the high end in both groups; this is very surprising in the case of respiration rate, given that the mouthpiece, or at least the awareness of being recorded, generally decreases it (Han *et coll.*, 1997; Han, Stegen, Schepers, Van den Bergh, & Van de Woestijne, 1998; Han, Schepers, Stegen, Van den Bergh, & Van de Woestijne, 2000).

Physiological hyperventilation, and thus hypocapnia, are triggered by an increase in respiration rate and/or tidal volume. The correlation matrix shows that the low $PetCO_2$ is inversely correlated with the tidal volume in the sample of asthmatic patients. The increase in tidal volume is therefore responsible for the decline in $PetCO_2$, and thus for the physiological hyperventilation. It would appear that the clinical hyperventilation noted in the group of asthmatics (as measured by the Nijmegen questionnaire) is related to respiration rate. However, given the large number of comparisons, the *p* value must be accepted with caution.

This is corroborated by Han, Stegen, De Valck, Clément and Van de Woestijne (1996), who showed that, following breathing re-education, the improvement in the number of complaints by subjects suffering from hyperventilation syndrome (as measured by the Nijmegen questionnaire) was mainly correlated with the slowdown in respiration rate and, to a lesser extent, with the increase in tidal volume. Thus, the favourable influence of breathing re-education may be a consequence of its influence on respiration rate rather than on $PetCO_2$.

Finally, the regression analysis shows that the variables that best explain the low $PetCO_2$ in asthmatics are tidal volume, female gender, asthma symptoms, and negative affect. These two last are weakly correlated.

It appears that severe asthmatics are similar to mild asthmatics in terms of psychological characteristics (Ten Brinke, Ouwerkerk, Bel, & Spinhoven, 2001), more specifically anxiety sensitivity and the Nijmegen questionnaire. Nevertheless, our results only apply to persons with mild to moderate asthma.

The depression score for asthmatics, which is slightly above normal, is significantly higher than that for normal controls. Rimington, Davies, Lowe and Pearson (2001) measured depression in mild to moderate asthmatics using the Hospital Anxiety and Depression scale; in their sample, depression constituted the best predictor with regard to symptoms. Anxiety was a weaker predictor. The authors hypothesise that a certain proportion of asthmatics with mild depression in primary care may be over-medicated, since medication is increased in response to symptoms that are not caused only by their asthma. They suggest that the relationship between respiratory symptoms and the ‘objective’ severity of individual asthma is complex and involves many factors that are unrelated to asthma (GINA Guidelines, 1998).

The asthmatic subjects’ sense of coherence is surprisingly low, at 59.3 ± 11.3, which is below the norms established by Antonovsky (around 66).

With regard to anxiety sensitivity, the results are comparable to those of Carr *et coll.* (1994) and also Carr, Lehrer and Hochron (1995), who do not find any signs of anxiety sensitivity linked specifically to asthma.

The asthmatics’ score on the Nijmegen questionnaire is considerably higher than that of the control subjects; a high score suggests hyperventilation, but it must be interpreted with caution. In fact, this questionnaire has only been validated for a normal population; it contains 16 items, some of which present a clear overlap with asthma symptoms (Keeley & Osman, 2001). But since hyperventilation is an integral part of the asthmatic condition, the two diagnoses are not mutually exclusive; on the contrary, they co-exist, as we saw in the introduction. It is difficult to clearly distinguish between asthma and hyperventilation with a questionnaire alone

Women seem to have a breathing pattern and psychological profile modelled on those of asthmatics; like asthmatics, women have a lower $PetCO_2$ and tidal volume, greater anxiety sensitivity, trait anxiety, negative affect, self-consciousness and symptoms of hyperventilation, and less sense of coherence than men. On the other hand, they do not describe significantly more asthma symptoms than men. Centanni *et coll.* (2000) compared 80 asthmatics, 40 patients with chronic disease other than asthma, and 40 healthy control subjects. The levels of anxiety and depression were significantly higher among asthmatics than in the other two groups. In the population of asthmatics and healthy subjects, women had a higher incidence of anxiety and depression than men, whereas no statistically significant relationship appeared in the patients with chronic organic diseases. During the year before the study, cases of hospitalisation and emergency treatment due to asthma exacerbations were correlated with a high anxiety level in women. In addition, educational level in asthmatics is significantly related to the frequency of anxiety and depression: a better education is associated with a lower frequency of depression and anxiety.

Leidy and Coughlin (1998) state that sex-related differences do exist when it comes to experiences with asthma. Women receive more anti-asthmatic medications and have a higher mortality rate than men in the USA, according to Hindi-Alexander *et coll.*(1992), who also found that three times more central nervous system depressants were prescribed to women than men in a sample of 196 asthmatics; he interprets this as indicating that women have more anxiety and depression symptoms than men.

Hyland, Finnis and Irvine (1991) found that women have a lower quality of life score than men. Quirk and Jones (1990), Quirk, Baveystock, Wilson and Jones (1991) and also Marks, Dunn and Woolcock (1992) report differences between men and women with regard to asthma symptoms and its impact on certain aspects of everyday life, using a visual analogue scale. Marks *et coll.* (1992) showed that asthma has a greater impact on women's moods, feelings, and relationships, using a quality of life instrument specific to asthma.

All of these results are coherent with the sex-related effects that were highlighted in patients with chronic obstructive pulmonary disease and other populations where women tend to describe lower levels of well-being and quality of life.

These results suggest how important it is to identify the classes of patients who require more attention because of their psychological profile. It seems to be useful to identify hypocapnia in asthmatics. Conversely, Gardner *et coll.* (1992) presented the case of a patient in whom hyperventilation accompanied by tetany was actually provoked by undiagnosed asthma. This emphasises the importance of a systematic search for an organic disorder when hyperventilation is present.

Mild and moderate asthmatics are said to be more vulnerable to depression and hypocapnia than subjects without asthma. The treatment of this physiological hyperventilation is likely to result in emotional and behavioural improvements, although this has not yet been sufficiently studied.

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Tables

Table I

Sociodemographic characteristics of subjects (mean (SD))

	Asthmatics N=80	**Controls N=40**
Sociodemographic data		
Age (years)	36.7 (13.5)	36.32 (13)
Education (years)	13.16 (2.7)	13.57 (2.7)
Height (cm)	168.91 (9.3)	170.37 (8.6)
Weight (kg)	67.66 (13.4)	65.52 (12.2)
Sex : f/m	53/27	25/15
Smokers	42/12/18	25/10/5
Pulmonary function		
FEV_1 (% pred.)	93.33 (13.2)	100.62 (10.3)
CVF (% pred.)	100.43 (10.7)	103.42 (9.8)
Raw (kPa/l/s)	0.25 (0.2)	0.157 (0.1)
Sgaw (1/s/kPa)	1.59 (0.9)	2.05 (0.8)
Tiffeneau	78.92 (8.1)	82.82 (7.4)

Note. FEV_1: forced expired volume in one second; FVC: forced vital capacity; Raw: airways resistance; sGaw: airways conductance; Tiffeneau: FEV_1/VC

Table II

Ventilation and psychological measures in asthmatics and healthy subjects (mean (SD))

	Asthmatics N=80	Controls N=40
Ventilation		
$PetCO_2$ (mmHg)	32.61 (3.36)	34.82 (3.14)
F (bpm)	17.97 (3.88)	16.82 (3.13)
Vt (l)	0.65 (0.15)	0.638 (.12)
V_E (l/min)	11.25 (2.64)	10.59 (2.19)
Psychological measures		
ASI R	28.53 (19.7)	23.37 (18.4)
STAI trait	48.91 (10.74)	45.76 (8.96)
BDI	10.79 (8.5)	5.95 (4.69)
SOC	59.3 (11.33)	65.02 (10.08)
PANAS	18.15 (6.8)	17.45 (5.28)
Csce	32.02 (11.73)	32.07 (9.39)
Nijmegen	25.42 (12.2)	15.79 (8.7)

Note. f: respiration rate; V_t: tidal volume; V_E: respiratory minute volume

Table III

Ventilation and psychological measures in asthmatic and healthy subjects based on gender (mean (SD))

	Women **N=78**	**Men** **N=42**
Ventilation		
$PetCO_2$ (mmHg)	32.57 (3.28)	34.84 (3.26)
F (bpm)	17.77 (3.5)	17.2 (3.9)
Vt (l)	0.608 (0.13)	0.724 (0.15)
V_E (l/min)	10.52 (2.4)	11.86 (2.4)
Psychological measures		
ASI R	30.2 (20.1)	20.5 (16.3)
STAI trait	49.2 (11.2)	45.4 (7.7)
BDI	9.96 (8.6)	7.65 (5.6)
SOC	59.48 (11.8)	64.56 (9.8)
PANAS	18.85 (6.6)	16.14 (5.3)
SCSR	33.62 (11.5)	29.02 (9.2)
Nijmegen	25.46 (12.2)	16.4 (9.2)
ASC état	1.38 (0.49)	1.32 (0.28)

Table IV

Bravais-Pearson correlation matrix among physiological and psychological variables (in asthmatics only)

	Nij	STAI	ASI	Soc	PAN	Cs	BDI	PC2	VEM	PetC	f	Vt
ASC total	.68**	.23	.46*	-.16	.28*	.18	.36**	.06	-.28*	.196	.13	-
ASC PF	.523*	.19	.41*	-.13	.21	.18	.3*	.03	.37**	.15	.17	-.26*
ASC AO	.44**	.17	.4**	-.12	.17	.14	.32*	.13	-.21	.12	.12	-.04
ASC hyp	.73**	.13	.43*	-.09	.28*	.15	.3*	.001	.09	.07	.21	-
ASC fat	.53**	.28*	.44*	-.16	.3*	.16	.34**	.11	-	.19	-.01	-
ASC irri	.03**	.24*	.27*	-.18	.23	.15	.3*	.38	.07	.29*	.11	-.3*
VE	-.2	-.17	-.08	-.006	-.05	-	.001	-.1	.06	.37*	.32**	.63*
Vt	-	-.26*	-.15	.01	-.02	-	-.17	-.16	.4**	-	-	
F	.24*	-.02	.07	-.02	-.12	.07	.09	-.09	.14	.07		
PetCO2	.1	.18	.09	.078	.19	.09	.003	.09	-.08			
VEMS	-.14	-.04	-.04	-.007	-.19	-.1	-.12	.18				
PC20M	.09	-.005	.13	.1	-.05	-	.001					
BDI	.53**	.67**	.65*	-	.64**	.32						
Csce	.33**	.45**	.43*	.37*	.38**							
PANAS	.5***	.6***	.6**	-								
Soc	-	-	-									
ASI R	.65**	.41**										
STAI	.36**											

* p<0.05 ** p <0.01 *** p<0.005 **** p<0.001

Table V
Multiple regression analysis of $PetCO_2$ according to V_t, TAI, Neg, Sex, ASC, ASC PF, ASC irritability and ASC fatigue

	∃	P	partial r
	32.24	.00001	
Vt	-9.95	.0001	- .52
Sex	4	.0001	.6
TAI	-0.02	.48	.-.1
Neg	0.11	.04	.27
ASC	-7.14	.06	-.24
ASC pf	3.23	.18	.17
ASC fat	2.15	.04	.26
ASC irri	2.2	.03	.27

Note. Vt : tidal volume; TAI : trait anxiety; Neg : negative affectivity; ASC : Asthma Symptom Checklist.