

Body fat, body fat distribution and the respiratory system: a "fat neck" syndrome?

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The clinical syndrome of obesity, somnolence, sleep apnoea and hypoventilation was described many years ago. The occurrence of dyspnoea and a "cardiac respiratory Pickwickian" syndrome in obese patients was noticed as early as 1936, as quoted by BARRERA *et al.* [1]. From the early fifties, extreme obesity was repeatedly reported to be associated with respiratory abnormalities, alveolar hypoventilation, polycythaemia and somnolence [2, 3]. The sleep apnoea syndrome has been correlated with increasing age and obesity [4], leading to the hypothesis that several symptoms in patients with the so-called Pickwick syndrome can be attributed to sleep apnoea [5].

Variation in body shape and proportion, especially with regard to the distribution of subcutaneous fat, is visually obvious and is exaggerated in obesity. The degree of overweight and/or obesity, usually derived from height/weight indices or anthropometry, can be simply regarded as an excess of adipose tissue or as the regional distribution of fat. It has long been recognized that the heterogeneity of adipose tissue is not only confined to morphology but rather to function. It is also known that not all obese subjects show the same metabolic profile regarding risk and complications. Regardless of the degree of overweight, obese men show a higher frequency of arterial hypertension, lipid disturbances and cardiac abnormalities. In addition, in obstructive sleep apnoea a strong male predominance was found [4].

At about the same period, the interest for research on obesity-induced respiratory abnormalities became apparent. VAGUE [6] reported the relationship between upper trunk (male) android obesity and the prevalence of a series of metabolic alterations. Over the following years, fat accumulation in the abdominal region has attracted considerable interest as an important and possibly independent factor in the development of endocrine and metabolic disturbances such as non-insulin dependent diabetes [7], lipid and lipoprotein abnormalities [8, 9], hypertension and an increased risk of atherosclerosis in both sexes.

The more recent background on morbidity and mortality includes the prospective work of Swedish investigators who found that abdominal adipose tissue

distribution is the best anthropometric predictor of cardiovascular disease and death. Using the waist-to-hip circumference ratio (WHR), LARSSON *et al.* [10] prospectively studied almost 800 middle-aged men and found that abdominal adipose tissue increased the risk for myocardial infarction or death by a factor of about 2 for a given body mass index. When total fat mass was accounted for, the authors indicated that for myocardial infarction and death the highest risk was found in men who had abdominal accumulation of fat and a (relative) low body mass index.

Upper body, android or central fat predominance can be measured by skinfold measurements and the WHR. Whereas skinfold thickness is related to subcutaneous fat, WHR is correlated more with the proportion of intra-abdominal fat [11]. Intra-abdominal fat, however, can be measured exactly by computed tomographic (CT) scan or magnetic resonance imaging (MRI) methods; it reflects the exact amount of intraperitoneal or visceral body fat, which is closely related to intrathoracic (mediastinal) fat accumulation [12].

Even in non-obese subjects with visceral fat accumulation, topographic and metabolic markers are closely related and visceral fat also correlates with circulatory disorders, such as left ventricular dilatation and hypertension.

It was recently shown that respiratory disturbances occur mostly in obese subjects with visceral fat predominance [13]. Expiratory reserve volume, functional residual capacity and arterial oxygen tension were reported to be reduced in visceral obesities of both sexes. Also, the frequency of sleep apnoeas is significantly higher in male obese patients with visceral fat predominance [13]. The CT observation of a significantly reduced pharyngeal area during normal breathing in supine position suggests that the peripharyngeal fat accumulation, rather than the severity of overweight, may explain the higher prevalence of nocturnal obstructive sleep apnoea. Similar findings have also been reported in women.

In the present issue of the Journal, the article by HOFFSTEIN and MATEIKA [14] provides an interesting addition to this emerging field of study. The authors report upon the use of abdominal and neck circumferences in the prediction of obstructive sleep apnoea and snoring. Among a large number of patients, apnoeic patients were characterized by a high body mass index (BMI) and larger neck and abdominal

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circumferences. When matched for age and BMI, the neck circumference remained the most important determinant of snoring. Even in a subgroup of non-obese subjects, an important degree of the variance of sleep apnoea and snoring was explained by the neck circumference alone.

The present study is interesting, not only in showing a series of factors involved in the sleep apnoea phenomenon, but also for its findings in the "metabolically obese" non-obese subject.

Although there is no direct evidence from these results that the neck circumference reflects upper body or pharyngeal fat accumulation in an objective way, they are promising or provocative enough to stimulate further study in this field. If the neck circumference could be validated by CT measurements of the fat content of the neck region, as was recently done for the waist circumference and WHR, such a new, easy to perform and inexpensive parameter might be a valuable screening test for obstructive sleep apnoea and snoring. In addition to helping clinicians to identify patients at risk, this could provide new insight for future research on the possible involvement of body fat topography on respiratory physiology, metabolism and disease. Such further research could help us to explain whether respiration abnormalities - in particular nocturnal desaturation and respiratory arrest - might contribute to the sudden death observed in obese and non-obese subjects with upper body fat predominance.

Whether neuroendocrine dysfunctions, a common finding in sleep apnoea and upper body fat, can be considered as primary or secondary common underlying mechanisms, has to be established.

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