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Birth weight and adult lung function in China

Y B Cheung; J P E Karlberg; L Low; M IP Thorax; Jan 2001; 56, 1; ProQuest Medical Library pg. 85

Birth weight and adult lung function in China

In a study published in Thorax in 1997 Stein and colleagues’ showed that birth weight was associated with adult lung function in an Indian population. We have carried out a similar analysis in a Chinese cohort of 59 men and 61 women born in Hong Kong in 1967 and followed up in 1997. This Hong Kong study has been described recently and has shown a significant inverse association between size at birth and adult blood pressure. Spirometric tests were performed according to the American Thoracic Society’s criteria to assure the quality. The data collected included forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and FEV1/FVC ratio at the age of 30 years, adjusted for sex, adult height, and smoking. However, there was no significant trend (each p>0.1). It can be seen from the findings presented by the Indian study that the previously reported associations between birth weight and FEV1 in men and between birth weight and FVC in both sexes were largely attributable to a lower lung function in subjects with a birth weight of less than 5 lb (2.27 kg) (Table 2 in Stein et al.).

In that study there was no obvious trend among the other subjects.

While we fully acknowledge the limitation of a relatively small sample, we believe it is important to report statistically significant findings and to compare them with the previous Asian study. Our study has shown a statistically significant relation between size at birth and adult blood pressure also suggests that the numbers were sufficiently large to reveal a clinically important association. Having considered findings from our study and the previous one we suggest that, while low birth weight may be associated with a reduced lung function in adults, variation in birth weight among subjects with a normal birth weight did not appear to be relevant.

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Hyperventilation syndrome

The editorial by Dr Gardner’ on controversial aspects of the hyperventilation syndrome (HVS) appears in the same issue of Thorax. This study showed that patients with HVS have an accentuated increase in ventilation as a response to change in body position from supine to standing. The editorial was a valuable addition to this difficult subject. We feel, however, that the interpretation of our paper in the editorial did not quite match the purpose or the message of the original study.

We agree with Dr Gardner that the definition of HVS in the literature are unfortunately variable. Dr Gardner suggests that the term HVS should be abandoned and that efforts should be made to find the initiating and sustaining causes of hyperventilation. This is also our strategy, so the subjects in our study underwent a comprehensive set of cardiological, respiratory, and other examinations. In clinical practice, however, the aetiology of hyperventilation often remains unknown and the only finding may be a disproportionate ventilatory pattern with resulting hypocapnia and alkalosis which may (at least partly) be the sustaining cause of the symptoms. Why would we not call the disorder HVSA or HVSB, thereby making the “unknown dyspnoea” which does not assure the patient of the benign nature of the disease? Dr Gardner suggests that our subjects “fit into a classification of dyspnoea and air hunger with secondary intermittent hyperventilation”. This classification would probably include a whole range of diagnostic categories with divergent diagnoses of dyspnoea and it is not justified, when several diagnostic procedures have been performed, to exclude cardiopulmonary diseases when the hyperventilatory component of the disorder has been objectively documented. In contrast to Dr Gardner, we also believe that the finding of hyperventilation may be of importance when the initiating cause is known, since not all patients with cardiopulmonary diseases have such a tendency. The assessment of evental panic disorder, symptom criteria described by the World Health Organization for research were used. Contrary to what Dr Gardner states in his editorial, the diagnosis of HVS in the study was based on episodic symptoms typical of HVS and documented respiratory alkalosis (with concomitant hypocapnia) in the arterial blood during such an episode. We consider this to be close to the original definition by Geisler et al. The approach to the definition of HVS was therefore physiological and unambiguous. As this was clearly described in the study, it is difficult to understand the confusion by Dr Gardner when he claims that the diagnosis was made in the presence of normal PaCO2. The measured orthostatic response which was the object of investigation in another manuscript and should not be confused with the process of diagnosis.

Finally, we would point out that the main purpose of our paper was to acknowledge the commonality of responses to orthostatic changes in patients with HVS. We hope that this finding will add to the knowledge of the causes and mechanisms of hyperventilation called for by Dr Gardner. Contrary to the repeated claim in his editorial, our intention was not to present the orthostatic test as a diagnostic tool for HVS but rather as a basis for its diagnosis—assessment of these patients

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