definition "pulmonary tuberculosis with negative sputum smear and positive or negative culture". I would argue that improved case finding as a result of the evident increase in the number of patients with respiratory symptoms studied (from 62 370 in 1993 to 179 493 in 1997) and in the number of contacts per case of tuberculosis studied (from 4.8 in 1993 to 12.5 in 1997) led to the sudden decrease in the rate of smear negative patients. This decrease is more appropriate and credible for a country in which the distribution of tuberculosis cases by age is similar to that found in industrialised nations,[2 3] a fact that Dr Marrero *et al* point out in their paper.

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AUTHORS' REPLY We appreciate the comments by Dr Garcia-Zamalloa on our paper on the control of tuberculosis in Cuba. We stated in the discussion that "better case finding and the change in the case definition may explain the increase in the period 1992–1994". The improvement in case finding is related to the comments by Dr Garcia-Zamalloa concerning the better detection of patients with respiratory symptoms and contacts per case studied. However, in 1994 there was a change in the case definition in Cuba because, before this year, patients with clinical symptoms and radiological features suggestive of tuberculosis who were smear and culture negative were not included in the register of the programme. This change and the better case finding also influenced the decrease in the percentage of smear positive cases.

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Birth weight and adult lung function in China

In a study published in *Thorax* in 1997 Stein and colleagues[1] 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.[2] Spirometric tests were performed according to the American Thoracic Society's criteria to assure the quality.[3] The same equipment (Sensor Medics 2200) was used in all subjects. Data were analysed by multiple linear regressions; adult height, smoking status, and sex were included as covariates.

The mean birth weight was 3.1 kg (range 2.3–4.1). Table 1 shows that babies of low birth weight (≤2.5 kg) had lower mean values of forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), and FEV_1/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 FEV_1 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*[1]). In that study there was no obvious trend among the other subjects.

Table 1 Mean (SD) FEV_1, FVC, and FEV_1/FVC ratio at 30 years of age, adjusted for sex, height at 30 years, and smoking (vs non-smoking) according to birth weight (n=120)

Birth weight (kg)	n	FEV_1 (l)	FVC (l)	FEV_1/FVC (%)
≤2.50	4	3.09 (0.29)	3.64 (0.33)	84.7 (6.2)
2.50–2.74	18	3.23 (0.46)	3.69 (0.47)	87.9 (8.9)
2.75–2.99	26	3.38 (0.32)	3.85 (0.50)	89.1 (5.6)
3.00–3.24	36	3.34 (0.44)	3.79 (0.53)	88.8 (5.4)
3.25–3.49	19	3.34 (0.41)	3.81 (0.54)	88.4 (6.0)
3.50–3.74	11	3.32 (0.28)	3.76 (0.36)	88.5 (4.7)
≥3.75	6	3.36 (0.37)	3.68 (0.47)	91.0 (4.1)
p for trend *		0.36	0.87	0.30

* Using birth weight as a continuous variable.

While we fully acknowledge the limitation of a relatively small sample, we believe that it is important to report statistically negative findings and to compare them with the previous Asian study. That 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[1] on controversial aspects of the hyperventilation syndrome (HVS) refers to our study[2] 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 definitions 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 cardiopulmonary 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 HVS? An alternative label would be "unknown dyspnoea" which does not assure the patient of the benign nature of the disorder. Dr Gardner suggests that our subjects "fit into a classification of dyspnoea and air hunger with secondary intermittent hyperventilation". This classification would probably include the whole spectrum of differential 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 disproportionate compensatory mechanisms of ventilation and the resulting hypocapnia may therefore be a sign of inherent susceptibility to hyperventilation and may be responsible for part of the patients' symptoms.

In our study[2] the organic causes of dyspnoea were excluded as far as possible by specialist care in a university hospital. For the assessment of eventual 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.*[3] 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 $Paco_2$. The measured orthostatic response which was the object of investigation is another matter and should not be confused with the process of diagnosis.

Finally, we would point out that the main purpose of our paper was to describe the accentuated breathing response 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,[1] our intention was not to present the orthostatic test as a "diagnostic criterion for HVS" nor as a basis for its diagnosis—assessment of these patients