Professor Harrison suggests that cilia remaining at the posterior part of the nose are unable to transport the relatively large particles of snuff back to the nasopharynx, and this, he thinks, accounts for the incidence of nasopharyngeal carcinoma being much the same in the Bantu as in the European. In fact, flecks of snuff can be seen at the back of the throat even after many years of snuff-taking. I remember the well-controlled start of surprise when my family doctor looked at my throat and saw what he thought at first glance were petechial haemorrhages.—I am, etc.,

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Expiratory Airways Collapse

SIR,—The phenomenon of expiratory collapse of the airways¹ is not uncommon, but recognition usually depends on the characteristic pattern being seen on a spiogram. There is, however, a diagnostic physical sign which does not seem to be widely known. This is a characteristic movement of the upper abdomen immediately after the commencement of a sharp expiration.

If the lower chest and upper abdomen are closely observed the expiratory movement will be seen to start and then immediately check, giving the appearance of a quiver or bounce. The expiratory movement then continues smoothly. This "bounce" occurs at the moment of shut-down—i.e., when the airflow from the chest is cut off and the upward movement of the diaphragm is abruptly checked, the checkwave being reflected back to the abdominal wall. This sequence is the exact counterpart of the familiar spiographic trace. "Abdominal bounce" seems a reasonably descriptive term for the phenomenon. The sign may be obvious or may have to be sought at different breathing rates. When found it is diagnostic.

All patients with expiratory collapse adopt the "pursed lips" method of expiration, the object of which is to raise intrabronchial pressure and thus keep airways patent. This should be treated as an indication to look for "abdominal bounce." Another suggestive sign is supraclavicular bulging on expiration, which is a further indication of positive intrathoracic pressure. It is important to diagnose airways collapse. These patients are considerably disabled. Twenty-six of these cases have been investigated in this hospital over the past two years. The Table summarizes arterial blood-gas findings.

Pco ₂ under 50 mm. Hg	
Oxygen saturation 95% or over	Nil
Pco ₂ under 50 mm. Hg	
Oxygen saturation 90-95% ...	3
Pco ₂ over 50 mm. Hg ...	17
Oxygen saturation under 90%	19

Thus only three patients had arterial-gas levels approaching normal values.

Therapeutically, also, it is important to diagnose the condition. As pointed out by Gandevia, these patients can be helped, often dramatically, by instruction in relaxed respiration. They have often been taught the opposite—i.e., to try to empty the chest by forced expiration, and this, of course, only serves to increase obstruction.

I am dubious about the operation of tracheal and bronchial splinting proposed by Herzog. Pressure-volume studies show the shut-down to occur during normal breathing at oesophageal pressures of +3 to +6 cm. water pressure, the majority at pressures of +4 to +5 cm. water pressure. This is indicated by an abrupt interruption of expiratory flow with a concomitant sharp rise of oesophageal pressure as soon as pressures in this range are reached. Maximum pressures reached in normal breathing range from 8 to 16 cm. w.p., with an average of 11 cm. w.p.; the majority being in the 10 to 12 cm. w.p. range. Inspection of Herzog's pressure/calibre graphs shows that at these pressures all but one of his cases retain more than 50% of their lumen—the majority above 60%. Our pressure-volume studies show a resistance in the region of 30 cm. w.p./l./sec. This is of the same order as that of a single small tube 3 mm. in diameter. Herzog's graphs show clearly that narrowing of this order is not produced by intra-thoracic pressures in the range which causes shut-down. In other words, shut-down occurs before pressures sufficient to produce bronchial or tracheal collapse are reached. This would indicate that the site of collapse is more distal, in the smaller airways. The collapse of trachea and major bronchi is an artifact produced by the forced expiratory manoeuvre.—I am, etc.,

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- ¹ Gandevia, B., *Quart. J. Med.*, 1963, **32**, 23.
- ² Herzog, H., *Triangle*, 1963, **6**, 85.

Thyrotoxicosis in Childhood

SIR,—Dr. F. J. Pick (21 November, p. 1329) has correctly emphasized the importance of the protein-bound iodine as a test in the diagnosis of thyroid disorders and in following up the progress of therapy as we had indicated in our paper. We would, however, like to point out some conditions where P.B.I. gives an erroneous idea of the thyroid status.

In most laboratories the normal range of P.B.I. in children as well as adults is 3.5 to 8 $\mu\text{g.}/100\text{ ml.}$ Higher levels are found in the newborn and persist for a few weeks. Similarly high levels are seen in pregnancy and during oestrogen therapy due to increased thyroxine-binding protein. Inappropriately high P.B.I.s may be met with in children with lymphocytic thyroiditis or goitrous cretinism. In thyrotoxic children under propylthiouracil therapy we have seen persistent high levels of P.B.I. for the first four to six weeks while the child became euthyroid or even hypothyroid, as judged clinically or by other thyroid-function tests. In some children when hypothyroidism set in there was a rebound increase in the size of the thyroid gland which subsided on thyroid administration. Later in the course of therapy P.B.I. more closely correlated with the thyroid status.

From the point of practical importance it should be remembered that treatment of hypothyroidism with thyroxine (T₄) causes P.B.I. levels to range between 8 and 12 $\mu\text{g.}/100\text{ ml.}$ when euthyroidism is achieved. It should be recalled that substitution therapy with tri-

iodothyronine (T₃) will result in a P.B.I. of only 2-3 $\mu\text{g.}/100\text{ ml.}$ The P.B.I. remains in the normal range of 3.5 to 7.5 $\mu\text{g.}/100\text{ ml.}$ when desiccated thyroid is administered. In the presence of normal thyroid function a low P.B.I. may be seen in nephrosis, hypoproteinaemia, and occasionally in patients on mercurial diuretics, androgen, or prolonged adrenocortical steroid therapy.

In most of the conditions pointed out above, except thyroiditis and goitrous cretinism, the butanol-extractable iodine level parallels the P.B.I. A simultaneous determination of the red-cell-T₃-¹²⁵I-uptake test will, however, be of great help in indicating the true thyroid status. We have found this test to be very reliable in following up the course of children undergoing treatment for either hyper- or hypo-thyroidism.—I am, etc.,

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Leprosy and Gynaecomastia

SIR,—You recently discussed gynaecomastia in your columns (19 December, p. 1548) and mentioned "generalized skin diseases" and "testicular atrophy" as causes. You do not, however, mention lepromatous leprosy. Your leading article the previous week (12 December, p. 1478) claims that there are some 12 to 15 million cases of leprosy in the world. The proportion of cases which are lepromatous (multibacillary) varies tremendously in reported series, but an average of 23 series reported by Skinsnes¹ would give a reasonable basis for calculation—about 30%. Roughly 10% of cases of lepromatous leprosy show gynaecomastia.² Therefore there must be at least 360,000 cases in the world.

Gynaecomastia occurs in leprosy because the mycobacterium attacks the testis as a very superficial organ; breast growth is stimulated by the pituitary in consequence of the hormonal imbalance produced. It is rather amusing, however, to note that several such affected patients claim fatherhood. Perhaps hormonal imbalance occurs before total azoospermia. The pathogenesis of this condition was recently reviewed by Davey and Schenck.³—I am, etc.,

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- ¹ Skinsnes, O. K., in *Leprosy in Theory and Practice*, ed. R. G. Cochrane and T. F. Davey, 1964, 2nd ed., p. 164. Wright, Bristol.
- ² Davey, T. F., and Schenck, R. R., *ibid.*, p. 194.
- ³ ——— *ibid.*, p. 195.

Red-cell Aplasia

SIR,—I was interested to read the article by Dr. C. C. Entwistle, Dr. P. H. Fentem, and Dr. A. Jacobs (12 December, p. 1504) reporting the association of red-cell aplasia with carcinoma of the bronchus. Having had some interest in the congenital type (Diamond-Blackfan anaemia) and read the adult cases by way of comparison, I was puzzled by some seemingly apparent omissions when the authors had obviously undertaken such a comprehensive review of the literature.