

# Correspondence

## Progressive lung disease in a malt-worker

Sir,—The case report described by Ellis and Friend in your July issue (*Thorax* 1981;**36**:552-4) is indeed unusual, particularly the granulomas found in the liver. I see in the article no note of tests of the leucocyte function. Since odd cases of the so-called chronic granulomatous disease of childhood can become manifest well into adult life without any remarkable history of earlier infections (Chusid *et al.* *JAMA* 1975;**233**:1295-6; Dilwontu, Mandell. *Am J Med* 1977;**63**:233-4). I would suggest the NBT test (Nitroblue Tertrazolium Reduction Test, Good *et al.* *Semin Hematol* 1968;**5**:215-54, should be performed on the leucocytes of the patient.

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We were most grateful to Dr Hillerdal for his helpful suggestion that our patient might have chronic granulomatous disease presenting in adult life. The NBT test has now been performed on the patient's leucocytes and the result was positive, a normal result. We feel that this must make chronic granulomatous disease most unlikely, bearing in mind also that the patient is now aged 47 years. There has been no material change in the patient's condition since we first reported the details.

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## Valid evaluation of plasma catecholamines

Sir,—We agree with the opinion of Barnes *et al* (*Thorax* 1981;**36**:435), that for valid evaluation of plasma cate-

cholamines a radioenzymatic assay is necessary.

In previous studies we had analysed the catecholamine-cAMP- and cGMP-plasma levels in 51 patients with extrinsic allergic bronchial asthma before and several times after bronchoconstriction with acetylcholine-inhalation. For comparison we studied 51 healthy volunteers (*Prax Pneumol* in press; *Prax Pneumol* 1980; **34**:585; *Med Welt* 1981;**32**:309). The catecholamines and cGMP were measured by a specific and sensitive radioenzymatic method (*J Neural Transmission* 1979;**45**:219; Amersham kit) and cAMP by a competitive protein binding assay (*Clin Chem Acta* 1974;**56**:221).

The adrenaline levels of patients (median 83.5, range 27.0-1085.4 pg/ml) and normal controls (median 84.5, range 10.8-356.2), the noradrenaline levels (median 258.4, range 88.0-702.0 pg/ml; median 289.2, range 179.0-760.6 pg/ml respectively), the cAMP levels (median 23.6, range 12.6-44.5 pmol/ml; median 25.1, range 13.3-51.0 pmol/l respectively) and the cGMP levels (median 7.00, range 1.90-15.70 pmol/ml; median 6.35, range 1.50-14.40 pmol/ml respectively) did not differ significantly. These results are, in part, in accordance with those found by Barnes *et al*.

After the acetylcholine-provocation in the patients there was a 2.5 fold rise in the adrenaline and a 1.0 fold rise in the noradrenaline concentrations maximum five to 15 minutes after the inhalation. For the healthy individuals only a 0.5 fold rise in the noradrenaline levels could be demonstrated. The cAMP and cGMP concentration showed no difference between the groups before and after the inhalation. In conclusion, there is higher sympatho-adrenergic activation in patients with allergic bronchial asthma after bronchial provocation with a cholinergic substance.

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Course organisers: Dr JMB Hughes, Dr NB Pride, Dr M Rudolf. Application forms may be obtained from: The School Office, Royal Postgraduate Medical School, Hammersmith Hospital, Ducane Road, London W12 0HS. Tel: 01-743 2030, ext 351.