the fetus, which could explain a low fetal/maternal ratio. Conversely, at low doses a greater proportion of the amalgam Hg could sequester in the fetus, thus explaining the higher fetal/maternal ratio. We know of no study comparing fetal Hg distributions under these exposure conditions.

Perhaps the most compelling refutation of Larsson and Sagulin's belief that the fetal liver can render Hg unavailable for uptake by other fetal/neonatal organs is Yoshida and colleagues' experiment in guineapigs.[4] They found that Hg initially stored in the fetal liver is not excreted by the kidney post partum but is redistributed to other tissues, especially kidney and brain, in amounts much higher than levels seen in utero.

Amalgam Hg exposure is not limited to vapour inhalation. Other absorption routes[1-3] that may alter the dynamics of tissue uptake and distribution were not factors in the investigations cited by Larsson and Sagulin, which were directed solely at inhalation of vapour from non-amalgam sources. Our work in primates[3] revealed that dental amalgam releases much more Hg vapour, which is absorbed into body tissues, than anyone had expected. Experimental evidence[1-3] suggests that it would be prudent to avoid placing dental "silver" amalgam fillings in pregnant women.

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**Professor Larsson and Dr Sagulin write: "In the third paragraph of our letter, citing Vimy et al, we mistakenly gave the mercury uptake in µg/g rather than ng/g for liver and cerebrum".—ED.L.

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Transmission of sarcoidosis via cardiac transplantation

SIR,—We do not know what causes sarcoidosis. However, most attention has focused on a transmissible agent. We report here a case of possible human-to-human transmission via a heart transplant.

A 59-year-old man with end-stage ischaemic cardiomyopathy, hypertension, and a reduced left-ventricular ejection fraction after a large anterior myocardial infarction 19 months previously underwent heart transplantation. He was treated with maintenance immunosuppression in the form of cylosporin and azathioprine. Because of persisting rejection regular oral steroids were started at week 9. At follow-up in week 18 the patient complained of early morning fever and sweats but physical examination was unremarkable. His medication at that time was cyclosporin, azathioprine, prednisolone, hydralazine, metoprolol, and enalapril. A chest X-ray suggested a new, bilateral lower zone pulmonary infiltrate, and he was admitted for investigation.

Comparison with previous chest films revealed no changes and a computerised tomography scan of the thorax was normal. At bronchoscopy the bronchial tree appeared normal. Bronchial washings were negative on stain and culture for bacteria, including mycobacteria; cytological examination revealed no malignant cells. Transbronchial biopsy yielded two pieces of tissue both with diffuse interstitial granulomatous inflammation but no evidence of necrosis or malignant disease. Serum angiotensin-converting enzyme (ACE) levels and immunoglobulins were normal. Mantoux testing was negative, as were serological tests for cytomegalovirus, toxoplasmosis, histoplasmosis, syphilis, cryptococcus, and brucellosis.

The heart donor was a 19-year-old man admitted brain-dead after a self-inflicted gunshot wound to the head. The liver, kidneys, and corneas were also transplanted. At necropsy the donor had epithelioid granulomas in the pulmonary parenchyma and hilar lymph nodes but staining and culture of lung tissue did not yield microorganisms and talc crystals were not seen. The recipients of the corneas and kidneys have remained well. Biopsy of the liver at transplantation revealed several granulomas. A later liver biopsy for apparent rejection showed cytomegalovirus hepatitis only. This was treated and the recipient is now well.

The heart recipient was given no antimicrobial therapy and continues on cyclosporin, azathioprine, and prednisolone. A repeat transbronchial biopsy, done after 8 months, revealed minor perivascular fibrosis but no granulomas or inflammation.

The search for a transmissible agent in sarcoidosis has yielded inconclusive reports implicating microorganisms (mycobacteria,[1] non-diphtheria corynebacteria,[2] and viruses[3]), and there is epidemiological evidence for space-time clustering in some cases.[4] The diagnosis relies on the demonstration of typical pathological lesions in an appropriate clinical setting and on the exclusion of other causes. Our patient's short-lived febrile illness was probably an intercurrent viral infection and the X-ray and scan findings suggest that the biopsy changes were incidental. Serum ACE activity and the gallium scan were negative but these are not reliable indicators of sarcoidosis, especially in the presence of regular steroid therapy.[5] There were no microorganisms on stains or culture, and there was biopsy evidence of regression despite continuing immunosuppression. These findings do not exclude the possibility that the granulomatous changes were a response to a virus or other microorganisms transmitted in the donor tissues. Mitchell and Rees,[6] by inoculating animals with human sarcoid tissue, have demonstrated the possibility of a transmissible agent. It is thus feasible that receipt of an organ from a donor who has sarcoidosis may provoke a distant and disseminated response.

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Enhancement of recovery from psychiatric illness by methylfolate

SIR,—Commenting on our report (Aug 18, p 392) of a controlled trial of methylfolate in psychiatric patients with low or borderline red cell folate concentrations, Dr Leeming (Oct 13, p 953) suggests that the links between folate, monoamine, and biopterin metabolism are ripe for investigation in affective disorders. We agree. For many years we have emphasised these links,[1-4] and within the context of our clinic trial we have examined the relations between folate levels and cerebrospinal fluid (CSF) 5-hydroxyindolacetic acid (5-HIAA), homovanillic acid (HVA), and tetrahydrobiopterin (BH_4) in 34 patients with severe depression according to DSM III criteria (unpublished).

7 (21%) of the 34 patients had folate deficiency (red cell folate <150 ng/ml) and this subgroup had significantly lower CSF 5-HIAA (67·5 [SD 23·2] nmol/l) than the 27 patients with normal or borderline folate values (96·8 [43·7] nmol/l, $p<0·02$). For all depressed patients, red cell folate was significantly correlated with