LETTERS / BRIEWE

Occupational lung disease

To the Editor: The fundamental error in attempts to determine the prevalence or incidence of disease is underascertainment, or denominator difficulty. The next most important problem is uncertainty as to the size of the population at risk, or denominator difficulty. The mining industry has until very recently relied heavily on migrant labour, and the number of men involved is very large indeed. The diseases of greatest concern are pneumoconiosis and tuberculosis, both of which may have long latent periods before disease becomes obvious. Recognition of the individual miner or ex-miner's disease depends heavily on the awareness of the doctors and nurses at the local clinic or hospital. If this is a mine hospital he is fortunate, as a rule, but once he leaves the mine it is unusual for occupational chest disease to be recognised or reported. There are thousands of undetected cases of compensatable occupational chest disease in the rural areas of South Africa and in the adjoining states from which migrant labourers are recruited.

At the risk of seeming to perseverate, may I repeat and amplify some ad hoc studies of hospitals in the rural areas. Many hospitals have never (according to the records of the Medical Bureau for Occupational Diseases (MBOD)) reported a single case. One hospital reported a single case in 1989, but none before or since; another 7 cases between 1969 and 1989; a third 20 cases between 1971 and 1992.

At two hospitals in which a determined attempt has been made (with the full co-operation of the medical superintendents), very large numbers of cases have been reported. In the first no case had been reported before 1990, and since then 139 have been reported. In the second no case had been reported before November 1991 -- since then 204 have been reported. More importantly, communities are beginning to organise around the issue and in the catchment of the latter hospital there are two established health committees identifying ex-miners and transporting them to hospital for radiographs and medical examination. In two

other areas committees are about to be formed.

It is a sobering experience to view the radiographs of men my age, and to remember that when I was a privileged schoolboy and medical student they were beginning their long and arduous careers in the mining industry and are now breathless on exertion and often destitute. I believe firmly that there is a reservoir of occupational chest disease in the rural areas sufficiently large to change the situation described by Dr Leger1 appreciably, and for the worse.

The editorial plea² for a 'shift of emphasis towards issues more closely related to the practical needs of the community' is appropriate and deserves strong support. The issue of compensation for work-related disease and disability is a very practical issue for a lot of people in the rural areas. May I, through your columns, continue to nag my colleagues. The fact that the journal now reaches every doctor in active practice in the country makes it un voiture critiquer par excellence.

Of the roughly 7 540 cases reported to the MBOD during the period November 1991 - September 1992, 204 or just over 2,7%, have been reported from a single rural district hospital.

Dr Leger has stimulated a very interesting series of letters. Before anyone is able to reach a conclusion as to the size of the neglected epidemic of occupational lung disease, extensive research is required in the rural areas of southern Africa from which migrant workers originate.

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- Leger J-P. Occupational diseases in South African mines a neglected epidemic? $SA fr Med \tilde{\jmath}$ 1992; 81: 197-201. Lee NC. The $SAM\tilde{\jmath}$ more a shift of emphasis (Mini-editorial). $SAfr Med \tilde{\jmath}$ 1992; 82 (Oct): (i).

Ascorbic acid causes spuriously low blood glucose measurements

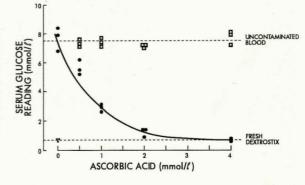
To the Editor: By chance, we found that a commercial vitamin C-containing glucose drink for athletes sharply lowered the blood sugar concentrations of volunteers, while a pure glucose solution, containing the same amount of sugar as the commercial product, clearly raised it.

To investigate this further we administered vitamin C (0,30 mmol/kg body mass) to 13 volunteers, and measured the ensuing blood glucose and insulin levels. To our surprise, the results showed unequivocally that ascorbic acid had no effect on the concentration of glucose or insulin in the blood (Fig. 1). On reviewing our methodology, it became clear that we had used a different method of glucose determination than before. In the original study we had used Dextrostix; the Beckman Astra System was used the second time.

We therefore added ascorbic acid to the serum of 3 healthy subjects (= 3,5 mM ascorbate). The blood glucose readings (mean ± SE) using the Beckman method were only minutely lowered by this procedure (0,23 ± 0,06 mmol/1; P < 0.01).

Ascorbate had a major effect on the Dextrostix readings. One millimolar ascorbate more than halved the serum glucose reading, and 2,0 mM almost completely counteracted the colour change brought about by 7,70 mM glucose (Fig 2). Ascorbic acid is a strong reducing agent which competes with the dye in Dextrostix for the H₂O₂ produced by the action of glucose oxidase on glucose. The Beckman Astra System uses an oxygen electrode to measure the amount of H2O2 formed, and is therefore hardly affected by ascorbate.

Dice and Daniel1 reported a nearly three-fold reduction in apparent 'insulin requirement' by a juvenile-onset diabetic subject (20-year-old Dice himself) on ingestion of 62



Mean (± SE) changes in serum glucose concentration measured by the Beckman Astra System, after administration of ascorbic acid (50 mg/kg: body mass) to 13 healthy subjects, 3 hours after a standard meal. The 13 control subjects imbibed only the vehicle in which the ascorbic acid was dissolved.

mmol (11 g) ascorbate per day. If they used Dextrostix, as seems likely, then diabetics taking vitamin C are in very serious danger of being misinformed about their blood glu-- the more so, since Dice's glycosuria also seemed to disappear with vitamin C administration.1 If this was due to the use of a vitamin C-sensitive urinary glucose method, there is no fail-safe against this particular form of chemical misinformation. (Benedict's reagent cannot be used as back-up as it reacts false positively with ascorbate.)



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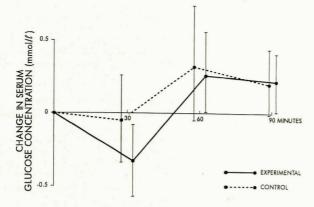


FIG 2 In vitro influence of ascorbic acid on the serum glucose reading obtained with the Dextrostix method. All the determinations were carried out on the same serum sample, except those indicated with crosses, which depict the dedicated spectrophotometer's readings using a fresh, dry Dextrostix. The solid circles depict the effect of various concentrations of ascorbic acid on the serum glucose reading. The open squares depict the readings obtained when only the solvent (physiological saline) for the ascorbic acid concentrate was added to the serum.

Dice reported no untoward effects from the reduction of his insulin therapy over 23 days (other than massive glycosuria and hyperglycaemia on termination of the experiment).1 A diabetic patient with an intercurrent infection, who is taking vitamin C in large doses, might not be so fortunate.

We do not advise against the use of ascorbate by diabetics. We merely recommend awareness of the problem, especially since the literature on Dextrostix claims that the test, being an enzymatic one, is specific for glucose.

We thank the subjects for their co-operation. We are indebted to Messrs D. Els and M. Voget of the Department of Chemical Pathology, Tygerberg Hospital, for performing the serum glucose and insulin measurements.

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Dice JF, Daniel CW. The hypoglycemic effect of ascorbic acid in a juvenile-onset diabetic. Int Res Commun System 1973; 1: 41.

ntravascular intra-uterine transfusions for severe fetal iso-immunisation — a new echnique in South Africa

To the Editor: Since 1989 we have used ultrasound guidance for cordocentesis to determine the degree of fetal haemolysis and intra-uterine intravascular transfusion (IUT) in the treatment of the severely affected fetus.

Amniotic fluid optical density at 450 nm is measured when the maternal anti-D titre rises above 1:16. A value of greater than 0,3 in midtrimester is an indication for fetal transfusion, while one of less than 0,2 is followed up by repeated amniocentesis. Between 0,2 and 0,3, fetal haematological values (using cordocentesis) are measured. We transfuse when the fetal haemoglobin concentration is below 10 g/dl.

A freehand technique of ultrasound-guided IUT is used. Essentials for success include: (i) an adequately sedated mother; (ii) experience in cordocentesis (including an experienced assistant); (iii) an aseptic technique; (iv) the availability of suitably prepared and tested blood; (v) a surgical team and theatre on standby in case of fetal distress; (vi) fetal neuromuscular blockade if movement is a problem; (vii) accurately calculated transfusion volume; and (viii) meticulous monitoring during and after the procedure.

To date we have performed 44 cordocenteses in 25 pregnancies and attempted 35 IUTs in 18 of these cases. In 9 cases fetal haematological values were normal and IUT was not required. The range of gestational age at the time of the first IUT was 18 - 34 weeks. The number of IUTs per patient ranged from 1 to 5.

Table I summarises our results. One of the 2 failures ended in an intraperitoneal transfusion and the other fetus was aborted because of persistent bradycardia after the diagnostic cordocentesis. (This necessitated immediate caesarean section.)

There were 5 intra-uterine deaths. Three were considered to be IUT-related and 2 to be unavoidable, as the fetuses were moribund before the procedures. The risk factors associated with fetal death were: (i) severe hydrops fetalis; (ii) first transfusion before 26 weeks; (iii) persistent fetal movement during the procedure; and (iv) repeat procedures within 7 days. The first two factors correlate with the severity of the

TABLE Outcome of IUT

Pregnancies	18	
IUTs attempted	35	
Technically successful	33	
Failures	2	
Live births	13	
Fetal deaths	5	

disease, but the latter two may be avoided by improved technique.

We conclude that ultrasound-guided cordocentesis and IUT are valuable additions to the options for management of haemolytic disease of the fetus. They may be performed at any institution where the necessary ultrasound expertise and equipment are available and there is access to adequate laboratory, blood bank and theatre facilities. Long-term follow-up of the neonates is encouraging.

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- Parer JT. Severe Rh isoimmunization current methods of in utero diagnosis and treatment. Am J Obstet Gynecol 1988; 158:
- Weiner CP, Williamson RA, Wenstrom KD, Sipes SL, Grant SS, Widness JA. Management of fetal hemolytic disease by cordocentesis. *Am J Obstet Gynecol* 1991; **165:** 546-553.

 Keckstein G, Tschürtz S, Schneider V, Hütter W, Terinde R, Jonatha W-D. Umbilical cord hematoma as a complication of
- intrauterine intravascular blood transfusion. Prenat Diagn 1990;
- Radunovic N, Lockwood CJ, Alvarez M, Plecas D, Chitkara U, Berkowitz RL. The severely anemic and hydropic isoimmune fetus: changes in fetal hematocrit associated with intrauterine death. Obstet Gynecol 1992; 79: 390-393.