

presence of a specific deletion that would include the tumour suppressor gene.

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- 1 Cotter FE, Pritchard J. Clonality in Langerhans' cell histiocytosis. *BMJ* 1995;310:74-5. (14 January.)
- 2 Willman CL, Busque L, Griffith BB, Favara BE, McClain KL, Duncan MH, et al. Langerhans cell histiocytosis (histiocytosis X): a clonal proliferative disease. *N Engl J Med* 1994;331:154-60.
- 3 Knudson AG. Mutation and cancer: statistical study of retinoblastoma. *Proc Natl Acad Sci USA* 1971;68:620-3.
- 4 Dimenbergh RA, Brown KLB. Diagnostic evaluation of patients with histiocytosis X. *J Pediatr Orthop* 1990;10:733-41.
- 5 Egeler RM, de Kraker J, Voûte PA. Langerhans cell histiocytosis; 20 jaar ervaring in het Emma Kinderziekenhuis. *Ned Tijdschr Geneesk* 1993;137:955-60.
- 6 Gadner H, Heiger A, Ritter J, Göbel U, Janka GE, Kühl J, et al for the Mitglieder der DAL-HX-Studie. Langerhanszell-Histiozytose im Kindersalter-Ergebnisse der DAL-HX 83 Studie. *Klin Padiatr* 1987;199:173-82.
- 7 Matus-Ridley M, Raney RB, Thawarani H, Meadows AT. Histiocytosis X in children: patterns of disease and results of treatment. *Med Pediatr Oncol* 1983;11:99-105.

Palliative care in terminal cardiac failure

EDITOR,—In their review of recent advances in cardiology John McMurray and Andrew Rankin have nothing to report on the neglected subject of terminal cardiac failure.¹ There is little research on this and apparently little perception of the need for palliative care. This is exemplified by a survey I performed of 10 hospices in north east London and Essex. All responded either to a questionnaire or to telephone contact. One hospice had no inpatient beds, and another was a specialist AIDS unit. Of the remainder, four would consider admitting patients with a primary diagnosis of end stage cardiac failure. Only two of these had admitted patients with this condition in the past 12 months: one had admitted one and the other "less than five" in the past year. This seemed to be matched, however, by the demand for hospice beds for patients with cardiac failure. The maximum number of requests was "less than five." Three other hospices had only one or two inquiries a year. The others had no applications at all.

There seems to be virtually no demand for palliative care beds for patients with cardiac failure in the area surveyed. This may be because these patients receive treatment to control their symptoms from their general practitioners, cardiologists, or general physicians. It may also be related, however, to a lack of appreciation of the poor prognosis of this condition in patients unsuitable for transplantation. Research is needed to ascertain whether palliative care in a hospice setting improves the quality of these patients' last days.

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- 1 McMurray J, Rankin A. Cardiology. II. Treatment of heart failure and atrial fibrillation and arrhythmias. *BMJ* 1994;309:1631-5. (17 December.)

Nutrition and lung health

EDITOR,—We agree with M K Sridhar that antioxidants may ameliorate lung disease caused by exogenous oxidants such as cigarette smoke but would contend that the focus needs to be shifted from individual antioxidants to a more global measure of antioxidant defences.¹ There are several antioxidant vitamins, and many other

compounds such as urate, bilirubin, albumin, and non-protein thiols, whose contributions to the body's antioxidant defences may be neglected by measurement of either the dietary intake or the plasma concentration of an individual antioxidant.²

The ability to measure the body's antioxidant defences directly rather than the contributions of some of its individual components may provide a more accurate picture, one example being the assessment of the total plasma antioxidant potential, which measures the ability of plasma to retard in vivo a reaction mediated by oxidants.² Thus while several studies have shown reductions in plasma ascorbate concentrations in smokers, these may be due to either dietary effects or consumption of the vitamin by the increased oxidant stress of smoking.³

We have directly measured both plasma ascorbate concentration and plasma antioxidant potential in smokers.⁴ While both were reduced in smokers compared with non-smokers, there was no correlation between the plasma antioxidant potential and ascorbate concentration in individual subjects.⁵ Measurement of a single antioxidant molecule in plasma would therefore seem unlikely to provide an accurate assessment of total plasma antioxidant defences. Similarly, the effect of a single dietary intervention (increased dietary intake of ascorbate, α tocopherol, or β carotene) may be masked by other, unidentified dietary changes with opposite effects. Furthermore, substances such as ascorbate may be pro-oxidant in high doses, at least in some groups of patients.⁶

Under such circumstances it would not be surprising, as Sridhar notes, if a given dietary intervention produced no benefit or was shown to be harmful. We urge that antioxidant defences should be considered as an integrated whole rather than as an incomplete sum of partially measured, fragmented parts.

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- 1 Sridhar MK. Nutrition and lung health. *BMJ* 1995;310:75-6. (14 January.)
- 2 Miller NJ, Rice-Evans C, Davies MJ, Gopinathan V, Milner A. A novel method for measuring antioxidant capacity and its application to monitoring the antioxidant status in neonates. *Clin Sci* 1993;84:407-12.
- 3 Kallner AB, Hartmann D, Horning DH. On the requirements of ascorbic acid in man: steady-state turnover and body-pool smokers. *Am J Clin Nutr* 1981;34:1347-50.
- 4 Bacon PJ, Pickworth AJ, Jones JG, Menon DK. Plasma antioxidant activity in smokers. *Br J Anaesth* 1994;73:721-2P.
- 5 Bacon PJ, Pickworth AJ, Jones JG, Menon DK. The relationship between plasma antioxidant potential and vitamin C concentration in smokers and non-smokers. *Respir Med* 1994;88:815-6.
- 6 Hunt JV, Bottoms MA, Mitchinson MJ. Ascorbic acid oxidation: a potential cause of the elevated severity of atherosclerosis in diabetes mellitus? *FEBS Lett* 1992;311:161-4.

Care in a midwife managed delivery unit

May not be the best option

EDITOR,—V A Hundley and colleagues state that their objective was "to examine whether intrapartum care and delivery of low risk women in a midwife managed delivery unit differs from that in a consultant led labour ward."¹ They conclude that "midwife managed intrapartum care for low risk women results in more mobility and less intervention." Since 38% of the midwifery group became at high risk antenatally and had their entire intrapartum care in the consultant unit, however,

this conclusion cannot be drawn with confidence and it is fair to state that the objective was not achieved. To compare correctly intrapartum care and delivery of low risk women it would be necessary to exclude all those women who became at high risk antenatally and concentrate solely on the remaining low risk women admitted to the respective delivery units.

The finding of greater mobility in the midwifery group was hardly surprising since the 46% who delivered in the midwives unit had no access to fetal cardiotocography or electrocardiographic monitoring or epidural analgesia. Furthermore, to state that there was "less intervention" in this group is meaningless since intervention is not defined and rates of induction, augmentation, and operative delivery were identical.

We were intrigued by the finding of a reduced incidence of fetal distress in the midwifery group: no attempt is made to define this term, and the Apgar score, cord pH, and rate of operative delivery were identical in the two groups. In fact, the authors state that more babies required resuscitation in the midwifery group, which they attribute to greater use of naloxone. Use of opiates in both groups was identical. These findings are inconsistent.

Also highlighted is a lower rate of episiotomy in the midwifery group, while an increased incidence of third degree tears is ignored. A similar association was recently commented on by Henriksen *et al.*² Of 31 outcome variables analysed, only two are identified as significant: episiotomy ($P=0.04$) and neonatal resuscitation ($P=0.05$). Simple statistics implies that at least one of these was probably a chance finding.

We would be interested to hear from the authors about the frequency of problems in the third stage in the low risk group who required transfer to the consultant unit.

On the basis of the data presented we question the authors' conclusion that a midwife managed delivery unit is "the more effective option."

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- 1 Hundley VA, Cruickshank FM, Lang GD, Glazener CMA, Milne JM, Turner M, et al. Midwife managed delivery unit: a randomised controlled comparison with consultant led care. *BMJ* 1994;309:1400-4. (26 November.)
- 2 Henriksen TB, Bek KM, Hedegaard M, Secher NJ. Methods and consequences of changes in use of episiotomy. *BMJ* 1994;309:1255-8. (12 November.)

Analysis is invalid

EDITOR,—V A Hundley and colleagues have missed the opportunity to contribute usefully to the debate about the safety of non-consultant intrapartum care because their analysis of their work is flawed.¹ They state that their objective was "to examine whether intrapartum care and delivery of low risk women in a midwife managed delivery unit differs from that in a consultant led labour ward." To achieve this they randomised low risk women at booking, provided all the women with the same standard antenatal care, and compared various maternal and fetal outcomes between the two groups on an "intention to book" basis. But such a comparison is invalid because the two groups were no longer comparable (that is, no longer consisted of only low risk women) at the time (the onset of labour) of the intervention.

An analogy might help. Say one wanted to investigate the antihypertensive effect of a new drug in women with newly diagnosed hypertension. Women are screened (a single measurement of blood pressure), classified as hypertensive on the basis of the result, and immediately randomised to receive a new drug or placebo three