- ⁴ Kitchen WH, Yu VYH, Orgill AA, *et al.* Collaborative study of very low birthweight infants: correlation of handicap with risk factors. *Am J Dis Child* 1983;137:555-9.
- ⁵ Yu VYH, Bajuk B, Hollingsworth E. Neonatal intensive care for extremely low birthweight infants—a dilemma in perinatal medicine. *Aust Paediatr J* 1981;17:262-4.
- ⁶ Yu VYH, Kinlay S, Orgill AA, Bajuk B, Astbury J. Outcome of very low birthweight infants who required prolonged hospitalisation. *Aust Paediatr J* 1984;20:293-7.

Equating optimal temperatures for incubators

Sir,

Drs Sauer, Dane, and Visser in their paper¹ refer to the equation:

$$T_{\text{operative}} = 0.4 T_{\text{air}} + 0.6 T_{\text{wall}}$$

referencing a work by Hey and Katz. This particular equation can actually be traced to the classic paper of Hey and Mount.² The equation is regrettably a very widely cited mistake. In that paper it is stated that non-evaporative heat losses in infants in incubators are proportioned approximately 0.6:0.4, radiant: convective. Then the equation:

$$T_{\text{operative}} = 0.6 T_{\text{radiant}} + 0.4 T_{\text{air}}$$

is given. Only if $T_{\text{radiant}} = T_{\text{air}}$ can both these statements be true. That the first statement is correct is shown by the work of Wheldon³ and myself⁴ using different methods. The correct equation would then be:

$$T_{\text{operative}} = 0.65 T_{\text{air}} + 0.35 T_{\text{radiant}}$$

That is, heat loss is proportional to thermal gradient so non-evaporative heat loss = $(hr + hc)(T_{\text{skin}} - T_{\text{operative}}) = hr(T_{\text{skin}} - T_{\text{radiant}}) + hc(T_{\text{skin}} - T_{\text{air}})$, where radiant heat loss = $hr(T_{\text{skin}} - T_{\text{radiant}})$ and convective heat loss = $hc(T_{\text{skin}} - T_{\text{air}})$. Since in Hey and Mount's paper $T_{\text{skin}} - T_{\text{radiant}} = 5.3^\circ$ and $T_{\text{skin}} - T_{\text{air}} = 2^\circ$ if $hr(T_{\text{skin}} - T_{\text{radiant}}):hc(T_{\text{skin}} - T_{\text{air}})$ is 0.6:0.4 the $hr:hc$ is 0.36:0.64 or approximately 0.35:0.65.

¹In using Wheldon's hr and hc it is important to remember that $T_{\text{radiant}} = 0.4 T_{\text{mattress}} + 0.6 T_{\text{wall}}$ and T_{mattress} is approximately equal to T_{air} .

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Dr Sauer and co-workers comment:

The point raised by Dr LeBlanc concerns the application of the optimal temperature as found by us¹ in an incubator with an equal air and wall temperature to the clinical setting with different air and wall temperatures. The equation:

$$T_{\text{operative}} = 0.4 T_{\text{air}} + 0.6 T_{\text{wall}}$$

is derived by Hey⁵ from the concept of operative temperature developed by Winslow *et al.*⁶ From studies using a manikin in an incubator Wheldon³ calculated a relation between the heat transfer coefficient for convection and radiation of 0.6:0.4. Which of the two equations is being used, however, is of limited clinical importance: the

calculated environmental temperature might differ by 0.5°C. From our study the optimal environmental temperature can be estimated with a standard deviation of 0.7°C using simple clinical data. This variation might be related to the effect of posturing. From Wheldon's data³ it can be calculated that the optimal environmental temperature might change by 0.5 to 1°C when the posture is changed from a fetal to a spreadeagle position.

References

- Sauer PJJ, Dane HJ, Visser HKA. New standards for neutral thermal environment of healthy very low birthweight infants in week one of life. *Arch Dis Child* 1984;59:18-22.
- Hey EN, Mount LE. Heat losses from babies in incubators. *Arch Dis Child* 1967;42:75-84.
- Wheldon AE. Energy balance in the newborn baby: use of a manikin to estimate radiant and convective loss. *Phys Med Biol* 1982;27:285-96.
- LeBlanc MH. Relative efficacy of radiant and convective heat in incubators in producing thermoneutrality for the premature. *Pediatr Res* 1984;18:425-8.
- Hey EN. The relation between environmental temperatures and oxygen consumption in the newborn baby. *J Physiol* 1969;200:589-603.
- Winslow CEA, Herrington LP, Gagge AP. Physiological reactions of the human body to varying environmental temperatures. *Am J Physiol* 1937;120:1-22.

Coagulation defect of congenital tyrosinaemia

Sir,

I read with interest the paper by Evans and Sardharwalla.¹ The prolongation of Reptilase clotting time due to the patients' dysfibrinogenemia, in addition to other coagulation factor deficiencies with the exception of antithrombotic factor, was discussed without taking into consideration the noticeably low fibrinogen concentrations. With only one exception (case 2—see Table)¹ fibrinogen concentrations were lower than 1 g/l, which suggests that coagulation tests to determine fibrin formation should be interpreted very cautiously. Dysfibrinogenemia in liver disease has been shown in patients in whom fibrinogen concentrations were either taken into consideration² or recorded as being greater than 1 g/l.³

In addition, the diagnosis of congenital tyrosinaemia depended on raised tyrosine and methionine concentrations (there was sibling history and necropsy in case 3). Ninety nine per cent of tyrosine is degraded in cytosolic homogenetic pathways, including tyrosine transaminase which is affected in almost all chronic hepatic disease, especially cirrhosis.⁴ In addition, therefore, to serum tyrosine concentrations other studies related to hepatic enzyme values would be more convincing. There is some evidence that liver disease in this condition starts prenatally and precedes hypertyrosinaemia, which develops postnatally.⁵ We had a patient with hereditary tyrosinaemia whose serum tyrosine and urinary 2,4 dinitrophenylhydrazine concentrations respectively were five and 10 times those of the controls. Despite a low protein diet he developed a liver tumour at about 5 years of age,

and at necropsy hepatocellular carcinoma was found.⁶ This may indicate a serious prognosis for these patients.

Finally, I should like to point out that the statement: 'Galactosaemia was excluded by finding normal activity of red cell galactose-1-phosphate uridyl transferase'¹ does not exclude the possibility of galactokinase deficiency.⁷

References

- Evans DIK, Sardharwalla IB. Coagulation defect of congenital tyrosinaemia. *Arch Dis Child* 1984;**59**:1088-90.
- Green G, Thomson JM, Dymock IW, Poller L. Abnormal fibrin polymerisation in liver disease. *Br J Haematol* 1976;**34**:427-39.
- Lane DA, Scully MF, Thomas DP, Kakkar VV, Woolf IL, Williams R. Acquired dysfibrinogenemia in acute and chronic liver disease. *Br J Haematol* 1977;**35**:301-8.
- Nordlinger BM, Fulenwider JT, Ivey GL, *et al*. Tyrosine metabolism in cirrhosis. *J Lab Clin Med* 1979;**94**:832-40.
- Hostetter MK, Levy HL, Winter HS. Evidence for liver disease preceding amino acid abnormalities in hereditary tyrosinemia. *N Engl J Med* 1983;**308**:1265-7.
- Özalp I, Koçak N, Özsoylu Ş, Çağlar M, Kale G: Herediter tirozinemi (Bazı laboratuvar bulguları ile Wilson hastalığına benzeyen bir vaka). *Çocuk Sağ ve Hast Dergisi*. (In Turkish). 1978;**21**:115-20.
- Pickering WR, Howell RR. Galactokinase deficiency. Clinical and biochemical findings in a new kindred. *J Pediatr* 1972;**81**: 50-5.

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Role of aldosterone in late hyponatraemia

Sir,

I read with interest the report by Al-Dahhan *et al* on the beneficial effect of salt supplementation on the clinical course of low birthweight preterm infants.¹ On the basis of the lower urinary potassium:sodium ratio seen in supplemented preterm infants, the authors concluded that '... salt supplements cause some suppression of aldosterone secretion' and 'the tubule is capable of responding to the hormone'.

This statement may give the false impression that the role of aldosterone in the development of late hyponatraemia has not yet been studied. In fact, in recent years

several clinical studies have already been carried out in preterm infants showing that:

(1) In response to the renal salt wasting, negative sodium balance, and fall in the plasma sodium value, excessive increase occurs in the activity of the renin-angiotensin-aldosterone system.²⁻⁴

(2) The increased aldosterone secretion rate results in a rapid improvement of distal tubular sodium reabsorption and contributes to the re-establishment of positive sodium balance.^{5, 6}

(3) By giving supplemental sodium the activity of the renin-angiotensin-aldosterone system may be suppressed^{7, 8} even to the degree seen in neonates of the same postnatal age.⁸

The results presented by Al-Dahhan *et al*, therefore, provide only indirect evidence to confirm these previous observations.

References

- Al-Dahhan J, Haycock GB, Nichol B, Chantler C, Stimmler L. Sodium homeostasis in term and preterm neonates. III. Effect of salt supplementation. *Arch Dis Child* 1984;**59**:945-50.
- Honour JW, Valman HB, Shackleton CHL. Aldosterone and sodium homeostasis in preterm infants. *Acta Paediatr Scand* 1977;**66**:103-9.
- Sulyok E, Németh M, Tényi I, *et al*. Postnatal development of renin-angiotensin-aldosterone system, RAAS, in relation to electrolyte balance in premature infants. *Pediatr Res* 1979;**13**: 817-20.
- Sulyok E, Németh M, Tényi I, *et al*. Relationship between maturity, electrolyte balance and the function of the renin-angiotensin-aldosterone system in newborn infants. *Biol Neonate* 1979;**35**:60-5.
- Sulyok E, Varga F, Győry E, Jobst K, Csaba IF. Postnatal development of renal sodium handling in premature infants. *J Pediatr* 1979;**95**:787-92.
- Aperia A, Broberger O, Herin P, Zetterström R. Sodium excretion in relation to sodium intake and aldosterone excretion in newborn pre-term and full-term infants. *Acta Paediatr Scand* 1979;**68**:813-7.
- Siegel SR, Fisher DA, Oh W. Serum aldosterone concentrations related to sodium balance in the newborn infants. *Pediatrics* 1974;**53**:410-3.
- Sulyok E, Németh M, Tényi I, Csaba IF, Varga L, Varga F. Relationship between the postnatal development of the renin-angiotensin-aldosterone system and the electrolyte and acid-base status in the sodium chloride supplemented premature infants. *Acta Paediatr Acad Sci Hung* 1981;**22**:109-21.

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