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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.

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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 antithrombin 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,