EQUATING OPTIMAL TEMPERATURES FOR INCUBATORS

SIR,

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

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

referring to Hey and Katz. This particular equation can actually be traced to the classic paper of Hey and Mount. The equation is regretfully a very widely cited mistake. In that paper it is stated that non-evaporative heat losses in incubators are proportionately 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 \( T_{\text{radiant}} = T_{\text{air}} \) in both these statements are correct. That the first statement is correct is shown by the work of Wheldon and myself using different methods. The correct equation would then be:

\[ T_{\text{operative}} = 0.65 T_{\text{air}} + 0.35 T_{\text{wall}}. \]

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.

In using Wheldon’s \(hr\) and \(hc\) it is important to remember that \(T_{\text{radiant}} = 0.4 T_{\text{matress}} + 0.6 T_{\text{wall}}\) and \(T_{\text{matress}}\) is approximately equal to \(T_{\text{air}}\).

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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 us 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 Hey from the concept of operative temperature developed by Winslow et al. From studies using a manikin in an incubator Wheldon 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 data 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.

Coagulation defect of congenital tyrosinaemia

SIR,

I read with interest the paper by Evans and Sardharwalla. The prolongation of Reptilase clotting time due to the patients’ dysfibrinogenemia, in addition to other coagulation factor deficiencies with the exception of antithaemophilic factor, was discussed without taking into consideration the noticeably low fibrinogen concentrations. With only one exception (case 2—see Table) 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 consideration or recorded as being greater than 1 g/l.

In addition, the diagnosis of congenital tyrosinaemia depends 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 homogentisic pathways, including tyrosine transaminase which is affected in almost all chronic hepatic disease, especially cirrhosis. 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 hypothyrosinaemia, which develops postnatally. We had a patient with hereditary tyrosinemia 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,

References

and at necropsy hepatocellular carcinoma was found. 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’ does not exclude the possibility of galactokinase deficiency.

References


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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. 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. 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.

3. By giving supplemental sodium the activity of the renin-angiotensin-aldosterone system may be suppressed even to the degree seen in neonates of the same postnatal age.

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

References


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