

of "spearing" tackles and the use of the head and helmet as a battering ram for injuries to the cervical spine.<sup>17</sup> The aim of registers for rugby injuries would be to achieve complete case ascertainment, which would require the number of players and the duration of play to be recorded. Also required are a universally acceptable definition of rugby injury and a standard format for recording injury, which could be completed by non-medical people.

What advantages would accrue from such registers? Firstly, they would provide the estimates of rates of different kinds of injury occurring during matches. The frequency, distribution, and nature of injuries could be followed over time. Secondly, they would allow associations with time, place, and person to be described. This information is essential in finding out more about the circumstances leading to injury. Case-control studies could then be carried out to provide estimates of the relative importance of other factors to injury, leading to a better understanding of the causes of injury and the ways to prevent them. Registers would also allow the effects of changes in the laws of the game on players' safety and the patterns of injury to be monitored over time. Establishing recurrence rates and the duration of incapacity would also be possible.

A picture of the "rugby playing population" could therefore be assembled, giving participating clubs or schools a summary of their players' injury profile in comparison with that of similar clubs or the rest of the region or country. This could directly contribute to coaching plans and the selection

of players and ultimately lead to a fall in the number of rugby injuries.

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- 1 Bannister R. Sport, physical recreation, and the national health. *BMJ* 1972;iv:711-5.
- 2 Sharp JCM, Macleod DAD. Injuries in competitive rugby football in Scotland. *Update* 1981;7:1355-61.
- 3 Dalley DR, Laing DR, Rowberry JM, Caird MJ. Rugby injuries: an epidemiological survey, Christchurch 1980. *New Zealand Journal of Sports Medicine* 1982;10:5-17.
- 4 Kay EJ, Kakarta P, Macleod DAD, McGlashan TPL. Oro-facial and dental injuries in club rugby union players. *Br J Sports Med* 1990;24:271-3.
- 5 Nathan M, Goodeke R, Noakes TD. The incidence and nature of rugby injuries experienced at one school during the 1982 rugby season. *S Afr Med J* 1983;64:132-7.
- 6 Addley K, Farren J. Irish rugby injury survey: Dungannon Football Club (1986-87). *Br J Sports Med* 1988;22:22-4.
- 7 Davies E, Gibson T. Injuries in Rugby Union football. *BMJ* 1978;ii:1759-61.
- 8 Sparks JP. Half a million hours of rugby football. *Br J Sports Med* 1981;15:30-2.
- 9 Durkin TE. A survey of injuries in a 1st class rugby union football club from 1972-1976. *Br J Sports Med* 1977;11:7-11.
- 10 Myers PT. Injuries presenting from rugby union football. *Med J Aust* 1980;iii:17-20.
- 11 Williams P. *Epidemiology of rugby injuries*. Cardiff: Five Nations Committee of the International Rugby Football Board, 1985.
- 12 Taylor TKF, Coolican MRJ. Spinal-cord injuries in Australian footballers 1960-1985. *Med J Aust* 1987;147:112-8.
- 13 Williams P, McKibbin B. Unstable cervical spine injuries in rugby—a 20 year review. *Injury* 1987;18:329-32.
- 14 Hoskins T. Rugby injuries to the cervical spine in English schoolboys. *Practitioner* 1979;223:365-6.
- 15 Silver JR, Gill S. Injuries of the spine sustained during rugby. *Sports Med* 1988;5:328-34.
- 16 Burry HC, Calcinaï CJ. The need to make rugby safer. *BMJ* 1988;296:149-50.
- 17 Torg JS, Truex R Jr, Quedenfeld TC, Burstein A, Speakman A, Nichols C. The National Football Head and Neck Injury Registry. Report and conclusions 1978. *JAMA* 1979;241:1477-9.

## Neonatal vitamin K<sub>1</sub>

### Prophylaxis for all

Almost 100 years ago a bleeding syndrome that was not due to trauma or an inherited bleeding disorder was described in newborn babies and called haemorrhagic disease of the newborn.<sup>1</sup> Some 50 years later deficiency of vitamin K was identified as the cause of gastrointestinal, nasal, and skin bleeding and bleeding after circumcision in healthy neonates, and the term haemorrhagic disease of the newborn was subsequently applied to bleeding from deficiency of vitamin K.

After studies had shown that vitamin K given to either the mother or the baby prevented severe hypoprothrombinaemia<sup>2</sup> prophylaxis with vitamin K (1 mg given intramuscularly) was widely used.<sup>3</sup> Doubts over whether all healthy babies needed intramuscular vitamin K, however, led to some units giving vitamin K orally and others abandoning prophylaxis altogether.<sup>4</sup>

In the United Kingdom a resurgence of haemorrhagic disease of the newborn was reported in 1983,<sup>5</sup> and changes in vitamin K prophylaxis and feeding practices were thought to account for this. Most of these cases (and those reported from other countries<sup>6</sup>), however, occurred not during the first week of life (so called "classical" haemorrhagic disease of the newborn) but between the 2nd and 26th weeks of life, peaking at weeks 4 to 6. Intracranial haemorrhage, a rare event in classical haemorrhagic disease, was found in more than half the cases. As in classical haemorrhagic disease, most of the infants had been exclusively breast fed.<sup>7</sup>

Further differences between classical and late haemorrhagic disease have been observed in follow up studies on

healthy newborn babies not given vitamin K prophylaxis.<sup>7</sup> Severe hypoprothrombinaemia or acarboxyprothrombinaemia (a marker for deficiency of vitamin K) was found in more than half of 5-6 day old infants but had resolved by week 5 to 6. Assays directly measuring vitamin K concentrations in plasma showed that because of limited transplacental transfer of vitamin K<sup>8</sup> and very low concentrations of the vitamin in fetal and neonatal livers<sup>9</sup> stores of vitamin K in the newborn are extremely small when compared with those in adults. The newborn baby therefore depends on an adequate supply of vitamin K in the first days of life, which must come exclusively from the diet—any contribution from the gut flora is unlikely.<sup>7</sup> Limited availability of maternal milk during the first days of life has been identified as the most important determinant of deficiency of vitamin K in healthy breast fed newborn babies.<sup>10</sup>

Delayed onset of lactation cannot, however, account for deficiency of vitamin K in breast fed babies 4-6 weeks old. Only rarely has the milk of mothers of affected babies been found to have particularly low vitamin K concentrations.<sup>7</sup> Malabsorption of vitamin K<sub>1</sub>, as shown in a baby with late haemorrhagic disease of the newborn and mild cholestasis,<sup>11</sup> might be a more important risk factor as subclinical liver disease was detected in a substantial proportion of cases of late haemorrhagic disease of the newborn.<sup>7</sup>

Cumulative case reports suggest that parenteral prophylaxis with vitamin K might protect against late haemorrhagic disease of the newborn whereas oral vitamin K is probably less effective.<sup>7</sup> Two papers published this week

confirm this impression (pp 1105 and 1109).<sup>12,13</sup> Information was collected on all cases of classical and late haemorrhagic disease of the newborn occurring over two years in Britain,<sup>12</sup> and the impact of prophylaxis with vitamin K was analysed by using a population denominator obtained from a study assessing the practice of giving prophylaxis with vitamin K in the United Kingdom.<sup>13</sup>

A surprising finding was the rarity of bleeding in the first week of life when no prophylaxis with vitamin K was given. This contrasts with a prevalence of biochemical deficiency of vitamin K over 50% in this population,<sup>7</sup> meaning that biochemical markers are of only limited value in assessing the relevance of deficiency of vitamin K in healthy newborn babies. Preventing haemorrhage rather than correcting biochemical abnormalities is the aim of prophylaxis; oral vitamin K was found to be sufficient to prevent classical haemorrhagic disease of the newborn.<sup>12</sup> Late haemorrhagic disease of the newborn occurred (with five cases of intracranial haemorrhage) despite oral vitamin K whereas parenteral prophylaxis seemed to protect against late disease. These data confirm observations from Switzerland,<sup>14</sup> Germany,<sup>15</sup> and Sweden.<sup>16</sup>

Should prophylaxis with vitamin K be given to all newborn babies and if so in what form? Without prophylaxis some 40 cases of intracranial haemorrhage due to deficiency of vitamin K in early infancy would be expected annually in the United Kingdom. Considering the consequences of intracranial haemorrhage for the affected children, their families, and society preventive action seems justified. At present parenteral prophylaxis with vitamin K seems the only safe option and is therefore recommended for all newborn babies.<sup>12</sup> As McNinch and Tripp point out, the potential side effects of parenteral prophylaxis with vitamin K deserve further study,

and new vitamin K preparations and more experience with regimens of repeated oral treatment may lead to changes in this advice.<sup>12</sup> Evidence of the efficacy of repeated oral vitamin K administration may come from Germany, if a recommendation to give parenteral vitamin K at birth or three oral doses of 2 mg of vitamin K<sub>1</sub> (at birth, during days 3-10, and during weeks 4-6) is implemented.<sup>17</sup>

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- 1 Townsend CW. The haemorrhagic disease of the newborn. *Archives of Paediatrics* 1894;11:559-65.
- 2 Dam H, Dyggve H, Hjalmar L, Plum P. The relation of vitamin K deficiency to hemorrhagic disease of the newborn. *Adv Pediatr* 1952;5:129-53.
- 3 Committee on Nutrition, American Academy of Pediatrics. Vitamin K compounds and the water-soluble analogues: use in therapy and prophylaxis in pediatrics. *Pediatrics* 1961;28:501-6.
- 4 Vitamin K and the newborn [editorial]. *Lancet* 1978;ii:755-7.
- 5 McNinch AW, Orme R L'E, Tripp JH. Haemorrhagic disease of the newborn returns. *Lancet* 1983;i:1089-90.
- 6 Lane PA, Hathaway WE. Vitamin K in infancy. *J Pediatr* 1985;106:351-9.
- 7 von Kries R, Shearer MJ, Goebel U. Vitamin K in infancy. *Eur J Pediatr* 1988;147:106-12.
- 8 Shearer MJ, Rahim S, Barkhan P, Stümmler L. Plasma vitamin K1 in mothers and their newborn babies. *Lancet* 1982;ii:460-3.
- 9 McCarthy PT, Shearer MJ, Gau G, Crampton O, Barkhan P. Vitamin K content in human liver at different ages. *Haemostasis* 1986;16:83-4.
- 10 von Kries R, Becker A, Göbel U. Vitamin K in the newborn: influence of nutritional factors on acarboxy prothrombin detectability and factor II and VII clotting activity. *Eur J Pediatr* 1987;146:123-7.
- 11 von Kries R, Reifenhäuser A, Goebel U, McCarthy PT, Shearer MJ, Barkhan P. Late onset of haemorrhagic disease of newborn with temporary malabsorption of vitamin K 1. *Lancet* 1985;ii:1035.
- 12 McNinch A, Tripp JH. Haemorrhagic disease of the newborn in the British Isles: a two year prospective study. *BMJ* 1991;303:1105-9.
- 13 Handel J, Tripp JH. Vitamin K prophylaxis against haemorrhagic disease of the newborn. *BMJ* 1991;303:1109.
- 14 Tönz O, Schubinger G. Neonatale Vitamin K Prophylaxe und Vitamin-K-Mangelblutungen in der Schweiz 1986-1988. *Schweiz Med Wochenschr* 1988;118:1747-52.
- 15 von Kries R, Göbel U. Vitamin K prophylaxis and late haemorrhagic disease of newborn (haemorrhagic disease of the newborn). *Acta Paediatr Scand* (in press).
- 16 Ekelund H. Late haemorrhagic disease in Sweden 1987-89. *Acta Paediatr Scand* (in press).
- 17 Sutor AH, Göbel U, von Kries R, Künzer W, Landbeck G. Vitamin K prophylaxis in the newborn. *Blut* 1990;60:275-7.

## Salt substitutes and potassium intake

### *Too much potassium may be disastrous for some*

Concern about the population's salt intake (itself a subject of dispute) has led to growing use of salt substitutes. In some of these substitutes, available in supermarkets and health food shops, salt is replaced by vegetable extracts and herbs with lactose as a bulking agent, but more commonly sodium chloride is replaced by potassium chloride. One of the most widely used salt substitutes contains 9 mmol of potassium per gram.<sup>1</sup> Replacing an average discretionary salt intake of 2.0-2.5 g/day would therefore increase a daily potassium intake of 60 mmol by about 20 mmol. There are three possible health benefits from such a change: protection against hypokalaemia, lowering of blood pressure, and protection against stroke independently of any effects on blood pressure. But there are also risks.

Hypokalaemia most commonly results from gastrointestinal losses or diuretic treatment. Gastrointestinal losses are likely to be short term and not seriously influenced by a small daily increase in potassium intake. Hypokalaemia induced by diuretics is associated with increased ventricular extrasystoles,<sup>2</sup> but there is no strong evidence that these are clinically important except in patients taking digoxin.<sup>3,4</sup> In the Medical Research Council's trial of treatment in mild hypertension supplementary potassium given to a subgroup of hypertensive patients increased their serum potassium concentrations but had no effect on 24 hour counts of extrasystoles.<sup>5</sup> In any case, the lower doses of thiazides now used in

treating hypertension have only a small effect on serum potassium values.<sup>6</sup> Mass treatment of the population to prevent iatrogenic hypokalaemia has little to commend it scientifically or ethically.

Arguments based on the effect of potassium in lowering blood pressure are stronger. Although carefully conducted trials have reached conflicting conclusions, a recent pooled analysis indicated that potassium supplementation reduced blood pressure in normotensive and hypertensive subjects by an average of 5.9/3.4 mm Hg.<sup>7</sup> The amount of potassium added, however, was over four times that likely to be achieved by discretionary use of salt substitutes (86 mmol/day). Furthermore, the possibility of publication bias in favour of studies with positive results<sup>8</sup> makes it likely that the fall in blood pressure was overestimated. Even so, quite small falls in population blood pressure may yield major returns. Interpopulation studies have shown a modest relation between the dietary sodium:potassium ratio and blood pressure. If it is assumed that this relation is causal and that the risk is reversible Rose has pointed out that an increase in dietary potassium of 20 mmol/day would lower systolic blood pressure by 4 mm Hg and that this would correspond to a 25% reduction in deaths attributable to raised blood pressure.<sup>9</sup> The 33% increase in potassium overall is not exorbitant. It would still be less than differences (of up to 50%)<sup>9</sup> between different social classes in the United Kingdom.