Hugh Downman and smallpox inoculation

Professor Dunn quotes Downman’s approval of Lady Mary Wortley Montagu in his fascinating account of the Exeter physician. Her contemporaries, however, were often less generous. This beautiful and literary lady contracted smallpox in 1715 and probably passed on from Timonius of Constantinople. They may well have heard an account of it from their own doctors. As Fellows of the Royal Society, they knew of the Turkish practice of “engrafting” smallpox. This is a practice that the Royal Society may well have heard an account of it passed on from Timonius of Constantinople. The following year she had the opportunity of travelling to Turkey with her husband who had been appointed ambassador to the Ottoman Empire. Receiptive towards Islamic culture she was struck by the relative absence of smallpox and learned that this was attributed to the deliberate, infecting of subjects with material from smallpox victims. In March 1718 she summoned the nurse who was Constantinople’s “general surgeon” for inoculation. The nurse pricked the wrist of Lady Mary’s young son with a needle, laid a tiny droplet of smallpox matter on the skin and mixed it with a drop of blood from the puncture. Some eight days later he became febrile and developed about 100 spots on his body. These quickly resolved without leaving scars.

Subsequently, the chequered success of variolation in the hands of English physicians, careless of the finer details of Turkish practice emphasised by Lady Mary, contributed to lifelong controversy. Most cruelly, her former friend Alexander Pope implied in one of his satires that she left people “pox’d by her love”, quite deliberately a defamatory double entendre as well as an attack on the safety of variolation.

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References

The use of sodium resounion in pseudoaldosteronism

We describe the use of sodium resounion in a patient with pseudoaldosteronism (PHIA). PHIA is a rare infantile abnormality characterised by raised plasma aldosterone, but mineralocorticoid resistance causing hyperkalaemia and hypertension. Severe recessive type 1 PHA is due to defective sodium reabsorption. PHA is due to defective sodium reabsorption (PHA). PHA is due to defective sodium reabsorption (PAT). PHA is due to defective sodium reabsorption (PAN). PHA is due to defective sodium reabsorption (PA). PHA is due to defective sodium reabsorption (PA).

Our patient presented aged 14 days with hyponatraemia (130 mmol/l) and hyperkalaemia (9.4 mmol/l). He made no response to hydrocortisone or furosemide. His plasma aldosterone level during crisis was extremely high (3820 pmol/l), confirming a diagnosis of PHA. Our patient’s sibling died neonatally with a presumptive diagnosis of PHA, suggesting autosomal recessive inheritance compatible with an ENaC defect. Our patient was managed on intermittent rectal calcium resounion when hyperkalaemic, and daily solution G (a preparation containing high levels of sodium (13.2 mmol/l)). The sodium requirement was 45 mmol/kg/day. Due to its unpalatability, solution G was given via gastrostomy. Despite this he had episodes of sudden collapse, precipitated by minor infections, with hyperkalaemia and life threatening hyperkalaemia, including a cardiac arrest. Discharge proved impossible.

After 18 months we changed his treatment to sodium resounion 0.25 g/kg twice daily via gastrostomy on advice from Professor Dillon (Great Ormond Street Hospital). Our patient

Infant to staff ratios and risk of mortality in very low birth weight infants

We were very interested to read the article by Callaghan and colleagues. They report a decline in mortality with less nurses caring for high risk infants over the first three days of life. This is a surprising finding, which is counter-intuitive to established neonatal wisdom. Indeed the authors quote a smaller study by Hamilton and colleagues, which found an increase in mortality with a reduced ratio of nurses to infants. Callaghan and colleagues’ findings may be true, with the most likely explanation of the deaths being excessive handling. Clearly, if this finding is replicated, then establishing the optimum number of nurses could lead to improved outcomes for high risk infants. It certainly warrants further study within the NHS and the United Kingdom.

It is widely assumed that increased numbers of nurses in the UK will improve the outcome of infants in neonatal care. Currently, in the National Health Service there is difficulty in maintaining adequate numbers of neonatal nurses, with many units having nurse staffing levels substantially below those recommended by the British Association of Perinatal Medicine. Unfortunately, this recommendation for more nurses is not based on a great deal of evidence, and the authors are to be praised for studying this topic.

Their results should, however be interpreted with caution. The health systems of the UK and Australia are different, most particularly in the proportion of centralised care and the ratio of nurses to infants. Callaghan et al make the point that the UK has a ratio of two very low birth weight (VLBW) infants to one nurse, whereas in Australia the ratio is approximately one to one. The UK Neonatal Staffing Study has recently looked at the 500 infants from 54 randomly selected units throughout the UK. This study did not show a clear relation between staff establishment and outcome, although it did show a linear relation between mortality and occupancy rates and a trend to increased risk of mortality with a lower nurse:infant ratio.

Callaghan et al discuss some of the weaknesses of their own study. There are also two factors that we wish to highlight. The first is that the authors have not looked in detail at the quality and abilities of the nursing staff. There is a wide variation in the abilities of staff, particularly when nursing agencies are used to provide nurses. As these staff may not work full-time or have much experience of the individual unit, they may be less efficient or able when compared to nurses who work full-time staff based on the unit.

The second factor is the method of determining nurse workload. Measuring the ratio of babies to staff is not an accurate assessment of nurse activity; a large number of very low birthweight babies often need less care than a small number of sicker babies. It is not clear from the paper how the authors dealt with the term infants, and whether these are included in calculating the ratio. Did the authors use the number of nurses per VLBW infant or per all babies in the unit? In addition, large babies can also generate a substantial workload if they are very unwell (for example, babies with persistent pulmonary hypertension of the newborn or congenital diaphragmatic hernia). Further studies measuring the true overall workload may give a better indication of the relation with outcomes.

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improved and the electrolytes became tightly controlled (see fig 1). Our patient was so much better that after two years inpatient stay, we managed to discharge him home on treatment. The sodium requirement is 19 mmol/kg/day, and he has had no further electrolyte decompensation.

We have treated six patients with recessive PHA in the past 10 years, previously treating them with calcium resonium rectally at times of hyperkalaemic crisis. This treatment has not controlled electrolytes, and two of our patients died. Our experience with recessive PHA is that the defect does not improve with age; one of our patients died at the age of 21 years, and the survivors remain on vast daily intakes of sodium. Sodium resonium has improved our current patient’s quality of life and allowed his discharge. Although use of sodium resonium in PHA was first described in 1984, it is not widespread. This case should prompt greater use of sodium resonium in PHA.

We must acknowledge the huge number of nurses and doctors that contributed to a successful outcome in this case.

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Once upon a time …

I very much enjoyed reading Storr and Rudolf’s review of literary perspectives on childhood. Appropriately enough, Charles Dickens loomed large. Oliver Twist is the first novel in the English language that takes a child as its central character. The book is also poenmatic and the early chapters are very much an attack on the working of the New Poor Law, a series of measures introduced to cut down the cost of the poor by precluding the able-bodied pauper from relief and by making the life of the workhouse as unappealing a prospect as possible. Dickens strongly objected to the fact that by consigning sexes to different quarters within the same workhouse, the need for family life among the poor and needy was totally disregarded. He satirised the new dietary provisions in Oliver’s asking for “... some more”, no doubt provoked by angry memories of his own childhood experiences.

Dickens was very clear that childhood experiences fundamentally influenced the way people behaved as adults. His description of a sick orphan returning a fresh air to his own room...a little quiet bed and a little platform over his breast on which to arrange toys...doll’s houses, woolly dogs, tin armies, Moorish tumblers, wooden tea-things, and the riches of the earth.” Dickens makes allowances for her mercenary and predatory nature in stating, “... observe how many things were to be considered according to her own unfortunate experience. Show her a wedding and she only saw two people taking out a regular licence to quarrel and fight. Show her a Christening, and she saw a little heathen personage... (who) was not in the least wanted by anybody, and would be shaved and bumbled out of everybody’s way, until it should grow big enough to shove and bang... Show her a live father and she saw a duplicate of her own father, who from infancy had been taken with fits and starts of discharging his duty to her, which duty was always incorporated in the form of a fist or leathem strap, and being discharged, hurt her. Our Mutual Friend portrays a London of wastelands, of disconnection and alienation, and a society dominated by financial speculation and commodity fetishism. As a cautionary tale warning against the moral dangers of greed and materialism it has lost none of its relevance.

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BOOK REVIEW

Child and adolescent psychiatry, 4th edition


It’s bigger, but is it better?

For me and my colleagues the answer is certainly, “Yes”.

Our Child and Adolescent Mental Health Service (CAMHS) now has all four editions of Child and Adolescent Psychiatry, “the child psychiatrist’s bible”, from the first edition, 1975 (Claver Oxford, 1975), to the second edition, 1985 (phone directory size); the paperback third edition, 1995 (Yellow Pages size, plus); and now this, the fourth edition, 2002, which is almost Data Sheet edition size. These awesome dimensions and the task of reviewing it (“you’ll have to read it, you fool” said a colleague) was somewhat off putting, but now that we’ve opened it and used it we don’t want to be without it. This book is the bible for the training of adolescent psychiatry textbook and a must have for any district hospital or postgraduate library. The sort of emotional behavioural and developmental problems encountered by paediatricians in hospital and in the community, in the interface between paediatrics and psychiatry, are covered in sufficient detail to be of real use to the clinician, whether he is going it alone or has the luxury of cross referral. The authors are experts in their fields, mainly eminent child and adolescent psychiatrists or psychologists from both sides of the Atlantic, but there are contributions from paediatricians in the chapter on soiling, for example.

Perhaps there is too much detail for the exam driven paediatrician in training, but then some only seem to grasp the value of developing some psychological frameworks later in their careers. Neither is this a quick fix alternative to the psychological component of the DCH and here again I suspect the size factor is likely to intimidate. Even SHOs in adult psychiatry who are now required by the Royal College to spend six months in child and adolescent psychiatry may be deterred and reach for a more MCQ orientated text in order to pass their exams.

For a hard pressed CAMHS team this book provides both the academic substrate and the clinical detail to be of real value in many everyday clinical scenarios. The reference lists are excellent and up to date and the index works, so that we have already checked out and accessed clinical information and papers we have been meaning to chase but never got round to. The new and esoteric stuff seems to be there as well as the genuine advances we have heard about on CPD days, such as the guidelines for assessment and treatment of childhood depression, clinically useful rating scales, and developments in the management of ADHD and autistic spectrum disorders, using an evidence based approach wherever feasible. What I have read have found fascinating and so it seems that, in this electronic age, there is still a place for a comprehensive reference book on the department shelf.

Much of all this is new and just not in the 1995 edition, which is why the book is so big and why your library should buy it.

M A Griffiths