KEY ISSUES IN CHILD HEALTH SURVEILLANCE*

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The past hundred years have seen dramatic improvements in child morbidity and mortality rates. A major factor has been the near eradication of many of the infectious diseases that cause illness including measles, tuberculosis, whooping cough, poliomyelitis, diphtheria and scarlet fever. However, a close monitoring of such infectious diseases remains a priority. This paper highlights presentations given to the second Joint Symposium of the Royal College of Paediatrics and Child Health (RCPCH) and Royal College of Physicians of Edinburgh (RCPE) in November 1999, focussing on the result of monitoring for existing and newly emerging disease.

MONITORING EMERGING INFECTIOUS DISEASE IN SCOTTISH CHILDREN

The impact of infectious disease was such that by 1900 the infant mortality rate in Glasgow was 143 per 1,000.[1] Since then there has been a remarkable improvement in child health, and in the late 1950s and early 1960s many believed that the chapter of infectious diseases, particularly among children, was finally closing. However, as a direct consequence of an outbreak in 1964 of typhoid affecting over 500 adults and children in Aberdeen, a national centre for the control and prevention of infectious diseases in Scotland, the Communicable Diseases (Scotland) Unit, was established in 1969. Now known as the Scottish Centre for Infection and Environmental Health, it conducts surveillance and research, working with other national agencies to provide operational support, advice and education in matters relating to infectious and environmental hazards.

Despite the decline in the incidence of many traditional infectious diseases in children, there are still several 'hot' issues to be addressed, not least the cost-effectiveness of antenatal screening and infectious disease control.

ANTENATAL SCREENING

In England and Wales, recent guidance from the Department of Health recommended that all antenatal clinic attendees should be offered and recommended an HIV test.[2] In Scotland, where the characteristics of HIV transmission have been different from those in the rest of the UK, a decision as to whether or not the same approach should be adopted has still to be taken. However, analysis suggests that antenatal screening may prevent only one additional transmission a year compared to 50 in England and Wales,[3] where such an overall HIV-related policy is considered cost-effective.[4]

Universal antenatal hepatitis B virus (HBV) screening within the UK is only now being widely performed, though the Department of Health has stipulated that this should be generally available by April 2000.[5] Greater Glasgow Health Board, however, has undertaken universal screening for several years. Between 1994 and 1997, 68 infections were detected in antenatal attendees, 25% being HBeAg positive and therefore of greatest risk of transmitting infection to the child. With only two-thirds of at risk children completing the immunisation course, it is clear that once pregnant women are diagnosed as being HBsAg positive, effective immunisation protocols should be implemented; this cannot be expected to happen without considerable planning and organisation.

Though antenatal screening can be effective in reducing transmission of HIV and HBV, what are the implications for hepatitis C virus (HCV) infectivity in Scotland? Firstly, is there such a problem? It is clear that HCV is on the increase with up to 80% of intravenous drug injectors in Glasgow being HCV-antibody positive. Using data collected via unlinked anonymous testing, transmission would be in the order of 20–30 cases a year of the expected 400 HCV-infected antenatal attendees.[6] However, unlike HIV and HBV, as yet there is no compelling case to offer antenatal HCV screening as no treatment can be given to prevent mother-to-child transmission, and the majority of infected children do not suffer from HCV-related disease during their childhood or early adulthood.

INFECTIOUS DISEASE CONTROL

Over the past 20 years Scottish children in line with the rest in the UK have benefited greatly from the routine national immunisation programme. The introduction in 1988 of MMR vaccination led to a remarkable decline in the incidence of measles. However, the resurgence in 1994 of measles in the non-vaccinated teenage population demonstrated the need for vigilance.[7-9] Since concern over the safety of MMR was aired publicly in the media there has been a fall in the MMR vaccine uptake to a level below the 95% threshold which is considered to be needed to insure herd immunity. It is essential that clinicians emphasise that MMR is safe and there is no scientific evidence to the contrary.

Benefits of other immunisation programmes have been witnessed. The recent introduction of the *Haemophilus b influenzae* (Hib) vaccine has virtually eradicated invasive Haemophilus infection in children. Also, public concern over recent rises in the incidence of meningococcal C disease has been tempered by the timely introduction of the new conjugate meningococcal C vaccine. With the Group C being the most predominant pathogen, it is estimated that, annually (in Scotland alone), 160 cases and eight deaths will be prevented by the programme. Unfortunately, the great proportion of meningococcal cases in children under five is caused by the Group B pathogen, so it is extremely important that there is no complacency by parents and doctors alike in recognising the signs of the meningococcal meningitis and septicaemic disease.

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To assess the appropriateness of interventionist policies, outcomes need to be considered, both clinically and in cost-effectiveness terms. To achieve this successfully, appropriate data must continue to be collected in a routine and systematic way.

RARE DISEASE SURVEILLANCE: AN EFFECTIVE METHODOLOGY

Rare infections are by definition individually uncommon, but collectively they are an important cause of morbidity and mortality in childhood. With this in mind the British Paediatric Surveillance Unit (BPSU) was set up in 1986[10] with the support of the Public Health Laboratory Service and specifically its Communicable Disease Surveillance Centre (PHLS-CDSC), the Institute of Child Health (London), RCPCH (at that time the British Paediatric Association), the then Communicable Disease Surveillance Centre (Scotland) and the Faculty of Paediatrics of the Royal College of Physicians of Ireland.

Over the following 15 years the aims of the unit have had to change little. The aims are: facilitating public health and scientific research into uncommon childhood disorders and infections; allowing paediatricians to participate in surveillance whilst at the same time reducing the burden of case reporting; raising awareness within the medical profession of the less common disorders studied; and responding rapidly to public health emergencies.

The mechanism for active surveillance is simple. Each month those participating in the scheme, at present 2,000 consultant clinicians, are sent a report card listing the conditions currently under surveillance, usually no more than 12. A scientific committee approves appropriate conditions for surveillance following evaluation of study applications. Respondents return the card to the BPSU office, indicating the number of cases of each condition on the card, which they have managed during the preceding calendar month. Scottish paediatricians return their completed cards via the Scottish Centre for Infection and Environmental Health. The 'nothing to report' box is an essential feature of the system, allowing for the returning of the cards even if no cases are seen. Compliance, which averages over 90%, can then be monitored.[11] On receiving a case notification the relevant investigator is made aware that a case has been reported. A questionnaire is then dispatched to the reporting clinician; over 85% are completed and returned (Figure 1). Since its inception over 40 disorders have been surveyed including HIV/AIDS, congenital rubella, toxoplasmosis, haemolytic uraemic syndrome (HUS), acute flaccid paralysis (AFP) and Kawasaki disease, leading to the reporting of more than 12,000 cases.

Similar to any national multi-respondent surveillance scheme, it is unreasonable to expect 100% ascertainment; indeed, this may not be required to achieve the many aims of the study. However, awareness of potential areas of under-ascertainment is important; this can be due to incompleteness of the reporting database, cases seen by non-paediatricians, complicated case definitions or difficulty in disorder recognition. Investigators are therefore encouraged to use other complementary sources of reports to optimise ascertainment, provide validation and improve the accuracy of information. National surveillance for HIV and AIDS in children has, for example, ascertained data via the paediatricians, obstetricians, national haemophilia network and microbiologists (Figure 2). Other studies involved pathologists (Reye's syndrome and haemophagocytic lymphohistiocytosis) or microbiologists (Hib infection, HUS and AFP). Multi-ascertainment also allows for the use of capture-recapture techniques to support surveillance, thereby enabling total population numbers to be estimated.[11-13]

For a surveillance system to be considered successful, one needs to examine its public health impact. The BPSU has monitored diseases targeted by vaccination programmes, as well as examining late sequelae of vaccination, e.g. congenital rubella, subacute sclerosing panencephalitis, meningoencephaetis after MMR vaccine, AFP and Hib vaccine failures. This system also allows rapid response to health emergencies, e.g. monitoring for new variant Creutzfeldt-Jakob disease, and assessing the impact of changing the route of administration of vitamin K in newborn infants. Data collected have also supported the

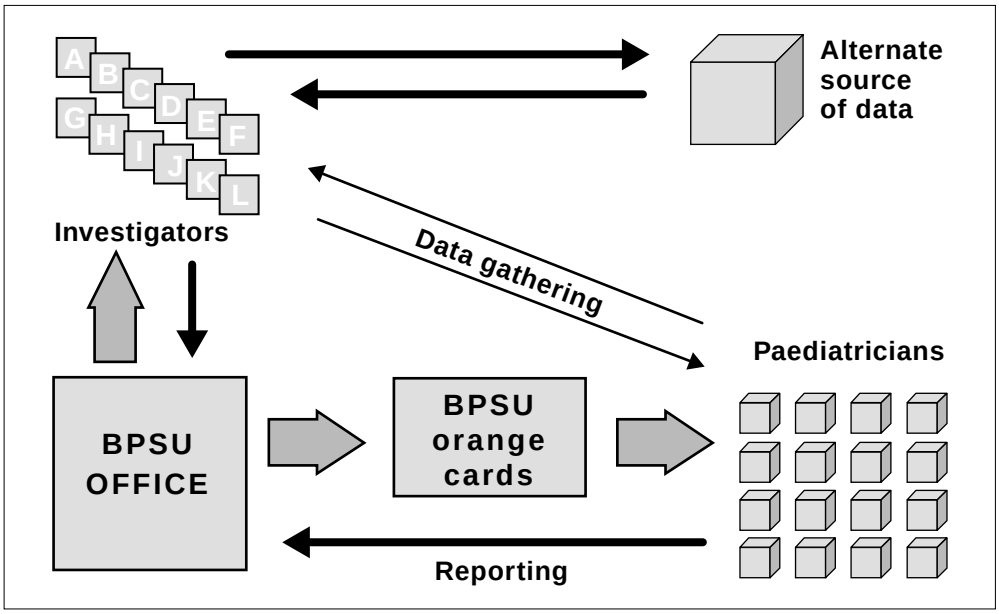

FIGURE 1
BPSU methodology for active surveillance.

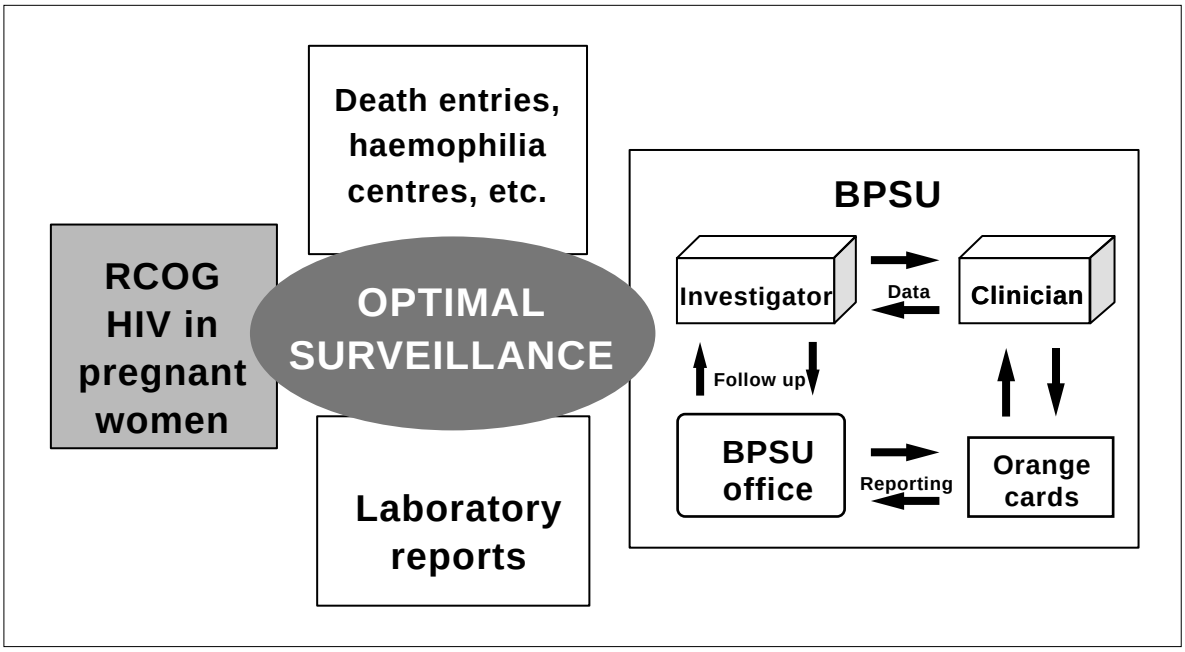

FIGURE 2
Surveillance – The Bigger Picture. HIV/AIDS in the UK.

need for national screening for congenital herpes and HIV[12-14] but not for congenital toxoplasmosis.[15]

The large output of publications and presentations, and the development of a web site (http://bpsu.rcpch.ac.uk) highlight the educational impact of the BPSU. Such output raises awareness of rare disorders within the medical profession and beyond. Of great importance is that a close liaison is maintained with parent groups, notably through the organisation Contact a Family, which has allowed contact with those most affected by these disorders, the patient and their families.

Inevitably investigations into rare disease are hampered by the problem of generating a sufficient number of cases to derive meaningful data, therefore collection of cases will often be required from large and geographically diverse populations. Though the problem was addressed in the UK, there has been no effective methodology or organisation until recently to support the simultaneous collection across several countries of identical information on rare paediatric disease. Stimulated by contacts from investigators using the BPSU, counterparts in Germany, the Netherlands and Australia encouraged their paediatric organisations to develop similar active surveillance systems in 1992. Similar units in Switzerland, Malaysia, Papua New Guinea, Wales and New Zealand quickly followed.

To allow closer interaction between units, 1998 saw the establishment of the International Network of Paediatric Surveillance Units (INoPSU). Its mission is to promote 'the advancement of knowledge about uncommon childhood infections and disorders through the participation of paediatricians in surveillance on a national and international basis'. Specifically, its aims are to facilitate communication and co-operation between existing units (and investigators using these units), and assist in the development of existing and new units. Development of uniform diagnostic criteria, dissemination of new knowledge and the ability to rapidly mount international surveillance of emerging disorders are additional aims.

By the start of 1999 the ten established units had initiated collectively 147 studies on 103 different conditions (63 completed). Eight collaborative studies between units have been undertaken. Return rates of monthly cards ranged from 72–98%, and of questionnaires from 47–100%. Surveillance covers national populations (<15 years) of between 0.5 million (Latvia) and 14 million (Germany). Currently, over 8,540 paediatricians worldwide contribute monthly by reporting uncommon diseases in a population of over 47 million children.[11]

Collaborative projects on insulin-dependent diabetes mellitus in under-fives (the Netherlands, Britain, Germany), Hib vaccine failures (Britain, the Netherlands) and vitamin K deficiency bleeding (Germany and Britain) have now been completed.[16-18] Many of the units are now sharing surveillance protocols, e.g. on congenital rubella and HUS, which will allow for future data comparison. With several countries simultaneously collecting data using a recognised methodology, comparisons of disease incidence, management and outcome between geographical areas will be possible.

FOODBORNE INFECTIOUS DISEASES

Perhaps surprising in these times of greater awareness of the need for cleanliness in food preparation and appropriate storage, the past two decades have seen a steady rise in incidence of food-related illness. Concerns have been heightened further with the rise in the 1980s of two new infectious agents, *Escherichia coli* (*E. coli*) O157 and Bovine Spongiform Encephalitis (BSE), which has now been linked to new variant Creutzfeldt-Jakob disease (vCJD).

Recognised as an emerging infection which occurs worldwide, vero cytokinin-producing *E. coli* (VTEC) O157 has had its greatest impact in the UK, particularly Scotland. During the autumn of 1996 an outbreak in Lanarkshire affected over 500 people, leading to 21 deaths. Though first described in 1977, VTEC-producing *E. coli* is thought to have been around since the early 1960s. The first recognised

VTEC outbreak occurred in the US in 1982 and the first reported association between VTEC and HUS in children came from Toronto, Canada in 1983.[19] The first surveillance study, which confirmed the link between *E. coli* O157 and haemolytic uraemic syndrome (HUS) was undertaken by the BPSU between 1986 and 1988.[20] Though the commonest cause of renal failure in children, HUS has a number of potential aetiologies; the most important in the UK is O157 VTEC. Since 1988 the national annual incidence of reports of O157 VTEC has increased from eight cases to over 1,100. Aware of this increase and following the 1996 Scottish outbreak, the need for enhanced surveillance of HUS was highlighted in the Pennington Report.[21] The UK Collaborative Study of Childhood Haemolytic Uraemic Syndrome commenced at the end of February 1997 and has continued. Through the BPSU, paediatricians report cases of HUS, defined as those children presenting with renal impairment, anaemia and thrombocytopenia. BPSU ascertainment complements surveillance undertaken by clinical microbiologists, public health physicians and through a national case-control study. Data are collected using a structured questionnaire; routine faeces and serum samples were taken from patients and sent to local microbiological laboratories. Isolates and sera were then referred to the PHLS Laboratory of Enteric Pathogens and the Scottish *E. coli* O157 Reference Laboratory for microbiological confirmation and typing.

Between February 1997 and January 1999, 193 clinical confirmed cases were reported, 186 (96%) had a diarrhoeal prodrome, and of the seven with no diarrhoea, two had antibodies to *E. coli* O157 and two had streptococcal pneumonia. Twenty children had seizures and five died, though two of the five had neither evidence of O157 nor a diarrhoeal prodrome. Through stool and sera examination the main aetiological agent in the causation of HUS was found to be *E. coli* O157. Males and females were equally distributed, over 50% of the cases were under the age of four years and this age group appears to be more at risk of HUS. Relatively few cases were reported from urban areas such as London, Birmingham and Manchester with more cases found in the rural hinterland around these cities. Fifteen cases were reported following foreign travel, 13 from Spain. A seasonal pattern has been identified with the peak of diagnosis occurring in August, coinciding with the peak of *E. coli* O157 reports from laboratories.

It is important not to be blinkered into thinking this is just a foodborne disease. Considering *E. coli* O157 overall during the period of surveillance several national outbreaks occurred demonstrating other modes of transmission of this organism. Person-to-person transmission was reported, and the disease was shown to occur in five sets of the sibs (in total comprising 11 children). Outbreaks following food contamination have regularly been documented; apart from the Lanarkshire meat contamination, poor milk pasteurisation led in 1999 to an outbreak in Cumbria affecting 114 people; 25 were hospitalised and there were three cases of HUS. Poor pasteurisation was also the cause of an outbreak due to contaminated cheese in 1998. Contamination following contact with livestock can occur; in 1997 a child developed HUS after a visit to an open farm: *E. coli* O157 was identified in goat faeces from the farm. The importance of open farms having washing facilities for visitors is clear and should be an essential requirement. Transmission through untreated water sources, beach contamination and mud from a field have all led to outbreaks over the past couple of years. With *E. coli* O157 able to survive up to six months in mud and an infection level 100,000 times greater than cholera, it is easy to understand the ease with which contamination and transmission of this micro-organism can occur.

VTEC of several other serogroups have also been associated with the cases of HUS and though *E. coli* O157 is the main causative agent of HUS in the UK, this is not necessarily the same overseas. In Italy during 1988 *E. coli* O157 was the most common strain, but in recent years *E. coli* O26 has become the predominant strain; this is also the case in Denmark.

With the increasing globalisation of travel and food imports, changes in disease aetiology and the causation of disease can be expected in future. Global networks will be required to assess the development and spread of these organisms. Simultaneous surveillance of HUS in Britain, Australia, New Zealand and Germany is now being facilitated through INoPSU. Early results confirm that there are considerable geographic variations in stool isolates of shiga-toxin producing *E. coli*. In Australia O111:H predominates, with no reports of O157:H7 over the past five years. In the UK 90% of isolates and 70% in New Zealand are O157:H7. Conversely in Switzerland over 80% of isolates are non-O157:H7.

HUS incidence in Britain and Australia is similar at 0.7/100,000/year for those under 15, doubling to 1.5/100,000/year for under fives. This is half the rate shown by surveillance in New Zealand and Switzerland. If such differences continue it will be important to identify possible reasons for this: climate, rural setting, farming and animal feeding practices may well be possible causes. Interestingly, mortality in the latter two countries is nearly double that in the first two. These findings have important public health implications, particularly in highlighting the need for rapid and reproducible diagnostic tests for non-O157VTEC. It is clear that continued surveillance may allow us to relate clinical features and outcomes to specific serotypes.

BSE AND THE EMERGENCE OF VARIANT CJD

First described in 1920-1 by Creutzfeldt and Jakob as a disease affecting people in late middle age, the recent emergence of a new variant of CJD affecting the young has led to increasing worry over the public health implications for the UK. Concern arose in 1985 following the first recognised case of BSE, a similar transmissible spongiform encephalopathy in the British cattle herd.[22] Epidemiological and other studies later concluded that the most viable hypothesis was that ruminant-derived meat and bone caused BSE. The subsequent ruminant feed ban in 1988 brought BSE to public attention, enhanced by the Bovine Offal Prohibition Act in 1989 which banned certain specific bovine offals from the human food chain. The potential implications for public health were realised and following the 1989 Southwood Committee report,[23] the National CJD Surveillance Unit (CJDSU) was established in Edinburgh in May 1990, with the primary aim being to identify any change in characteristics of CJD that might be linked to BSE.[24] Based on the evidence collected by the CJDSU, 1996 saw the announcement of the discovery of a possible causal link between BSE and a variant of CJD.[25]

At the commencement of surveillance in 1990, there was no specific case definition and this was quite deliberate, as it could not be predicted what any resultant BSE-related

disease would be like in humans. Neurologists, pathologists and others reported anything that looked like CJD, and only about 50% of all referrals have turned out to be CJD. Knowing that sporadic CJD was mainly a disease of late middle age, it was therefore surprising that during 1995-6 small numbers of relatively young cases were referred to CJDSU. By early 1996, ten cases had developed what has become recognised as variant CJD (vCJD). Since then a total of 46 cases (21 males, 25 females) have been diagnosed with a mean age of death of 29 years and a mean age of onset of 28 years, with the earliest onset being 14 years.

Clear differences between classical CJD and vCJD have been established with mean survival in the former being four months from first symptoms, compared with 14 months in the latter. Though there is some overlap, a number of clinical features of vCJD differ from classical CJD. Such patients show a high prevalence and extended duration of psychiatric symptoms, and commonly present with depression, anxiety and withdrawal for up to six months before developing neurological abnormalities such as ataxia and involuntary movements. Currently the only way to conclusively diagnose the disease is with neuropathology that can be normally undertaken only after a *post mortem* examination.[26]

Abnormalities in vCJD patients have been identified on MRI brain scans: 75% of patients exhibit a high signal in the posterior thalamus. This may in the future assist with diagnosis when neuropathology is not available. A unique pathological appearance, including widespread deposition of florid plaques throughout affected areas of the brain, has also led to the belief that vCJD is a new disease.

Evidence of a link with BSE can be seen when comparing UK rates of BSE and vCJD with other European countries. In the UK 175,000 cases of BSE have been reported with 46 cases of vCJD. Comparison surveillance in Europe has identified approximately 3,000 BSE cases and only two further cases of vCJD, one from France and a second from Ireland, though this individual had lived in the UK. The firmest evidence of a link comes from transmission studies in mice.[27] Inoculation of these mice with vCJD showed near-identical transmission characteristics to that shown when BSE was injected into the mice. This strongly suggests that the agent causing vCJD is also the agent causing BSE.

As yet the future total number of cases of vCJD are unknown with estimates ranging widely from 130–80,000 cases or more. Much depends on the length of incubation of BSE in humans, and this is one reason for extending surveillance to children since the exposure level and factors are as yet to be determined.

EXTENDING vCJD SURVEILLANCE TO CHILDREN

With vCJD occurring mainly within the younger population, detection of vCJD in UK children has important implications for paediatrics and public health. To enhance current ascertainment of suspected vCJD cases, the BPSU with support from the Department of Health undertook to facilitate surveillance of vCJD in children. The project is co-ordinated from Addenbrookes Hospital with support from the PHLS in conjunction with the CJD surveillance unit in Edinburgh.

The presentation of vCJD differs from classical CJD and thus presentation of any cases in children may be difficult to predict. The strategy is therefore to detect suspected cases by looking at a broader group of conditions. This group needs to be large enough to include all possible cases of vCJD, hence surveillance was undertaken for a range of presentations under a combined term – Progressive Intellectual and Neurological Deterioration (PIND).

Surveillance commenced in May 1997 with clinicians being asked to report via the BPSU monthly orange card mailing. The surveillance cases definition asks for children aged under 16 years who presented with progressive neurological deterioration for more than three months with loss of already attained intellectual/developmental abilities and development of abnormal neurological signs. On receipt of a report, data are collected either by a telephone questionnaire or a site visit and then transcribed to anonymised proformas. An expert neurological advisory panel of seven paediatric neurologists discusses in detail each proforma for possible cases of vCJD.

For the 29 months to October 1999, 764 case reports have been received of which 570 cases have been discussed by the expert panel. Of these, 322 have been classified as PIND with a recognised cause. Confirmed diagnoses include the neuronal ceroid lipofuscinoses (40 cases), mitochondrial encephalomyelopathies (27 cases), GM2 gangliosidoses (27 cases), metachromatic leukodystrophy (19 cases), adrenoleukodystrophy (16 cases) and Niemann-Pick disease Type C (16 cases). The rest, which are still being investigated, are being followed up or do not meet the case-definition for PIND.

Important regional differences have been noted in the incidence of PIND cases – Yorkshire, the West Midlands and East London having the highest rates. It is suspected that this is due to high intermarriage rates in particular in ethnic and religious groups.[11] Though a total of 80 separate conditions have been reported as yet no definite/probable cases of new vCJD in children have been identified.[28] Medium term surveillance will be required in order to monitor the epidemiology of vCJD.

DIABETES – THE NEED TO MONITOR CONTROL

Life expectancy from diagnosis in young people with diabetes was 1.3 years in 1900. By the 1950s (35 years after the introduction of insulin) it had increased to 26.4 years. Presciently, even by 1923, Banting was emphasising the importance of normoglycaemia for the patient's well-being. However, from the 1930s to the 1970s, the emphasis was on as normal a lifestyle as possible ('Dispel not the happy illusions of childhood' – Goethe), whilst avoiding hypoglycaemia. By the 1960s, the question was being asked as to whether long-term complications (silently bequeathed by paediatricians with their patients to adult diabetologists) were inherent in the 'diabetic process', or could be postponed or prevented by control of the metabolic disturbance. Irrefutable epidemiological evidence (at least in those aged 13 years or over) from the American ten year prospective Diabetes Control and Complications Trial indicates that if good glycaemic control is achieved, in the long-term the risks of microangiopathic complications (retinopathy, nephropathy and neuropathy) are greatly reduced.[29] The question is now not why or whether to maintain good diabetic control, but how.

One of the outcomes of poor control is diabetic ketoacidosis (DKA). A rare but devastating complication of DKA is cerebral oedema, which appears to be sporadic and unpredictable. Although the aetiology of cerebral

oedema is not understood, possible contributory factors may be severity of DKA, the rate and/or quantity of intravenous fluid administration, a fall in plasma sodium concentration and hypoxia from bicarbonate administration. No sizeable case-control studies were conducted to support any of these theories, and even with optimum management by current standards, cases of cerebral oedema still occur.

A recent UK study was undertaken through the BPSU, with the aims of identifying factors in the clinical presentation and management of the child with DKA which may influence the development of cerebral oedema; to study the outcome of cerebral oedema in terms of mortality and morbidity and to determine the absolute risk of developing cerebral oedema among children with DKA.[11,30] To achieve these aims active surveillance was undertaken for a three-year period between October 1995 and September 1998, whereby cases of possible cerebral oedema were reported. During the middle two-year period (March 1996 to February 1998), all episodes of DKA in England, Scotland and Wales were ascertained through a separate reporting system established in Oxford. These would be used as the denominator to determine the risk of cerebral oedema, and as a pool of controls for a case-control study of factors predisposing to the development of cerebral oedema. Thirty-four cases of cerebral oedema were reported (median age of 11 years). In addition, six deaths during a period of ketoacidosis were not associated with cerebral oedema and occurred after the start of hospital treatment for DKA. Finally, there were also 26 cases of unexplained deterioration of conscious level but without other definite signs of raised intracranial pressure. Eight of the cerebral oedema group died, a 25% mortality rate; of the 26 survivors, 17 children had no obvious sequelae. The remaining nine children (35% of survivors) have a variety of new long-term disabilities. Outcome was not related to treatment received, and, in particular, all of those who died, and all but one with neurological sequelae, had received mannitol. However, all of those who had a respiratory arrest either died or had significant sequelae.

In examining the extent of DKA episodes, 225 consultants covering all units likely to see episodes of DKA reported over the two-year period: complete data were available on 2,941 episodes of DKA (1,845 old and 1,096 new diabetes). The incidence of cerebral oedema was 6.8/1,000 episodes of DKA, similar to that seen in the US, despite differences in the fluid and electrolyte management between the two countries. The risk was higher in newly diagnosed (11.9/1,000) than old (3.8/1,000) diabetes with a trend for increased risk in younger children. By matching controls from the DKA groups to each of the cerebral oedema it will be possible to compare factors in the presentation and clinical course of cases and controls, and determine factors, which may predispose to the development.

Many other questions remain concerning what level of glycaemic control in childhood and adolescence constitutes 'good' or 'good enough' control. Is the prevention of complications by the establishment of good controls in childhood and adolescence a realistic goal? How is it to be achieved in the context of normal physical and emotional growth, and 'normal' daily activities and family lifestyle? Will this be at the expense of more hypoglycaemia? If so, how frequently must hypoglycaemia occur, and how severe need it be in children of different ages before it is damaging to long-term cognitive functioning? Does more education, or learning new or improved skills in patients and clinicians, necessarily lead to better diabetic control? The risks for macrovasular disease seem unaffected by tight glycaemic control.

Many current approaches are in line for the delivery of improved care, but in recent years a major development for monitoring of outcomes, audit and in terms of epidemiological insights, has been the development of registers. The establishment of the Scottish Study Group for the Care of Young Diabetics in 1982, and the development of a register for all new diabetics was an audit tool and a source for professional advice, standard setting and statistical information.[31,32] Findings have included worse glycaemic control in young people over 12 years and in girls, with best control in the four to eight years range. An absent parent or an affected sibling is also associated with poorer control. Better control is obtained from three rather than two daily injections but many children frequently miss their insulin injections. Insulin type (self-titrated versus pre-mixed) and social deprivation are not significantly associated with the quality of glycaemic control.[33]

A minimum data-set to support the audit of children's diabetes services is now being established UK-wide to facilitate the development of service standards based on professional recommendations underpinned by evidence where available.

CONCLUSION

It should not be presumed that diseases previously considered as conquered should be totally consigned to the history books. Recent increases in cases of tuberculosis, and falling vaccination uptake leading to a rise in measles outbreaks in Europe, are witness to this. Whilst for the future vaccines may be available for other infectious disease such as chicken pox, meningococcal b infection, or rota virus, it is clear that the education of the young will be one of the most potent tools for disease prevention. Information and education may help prevent sexually transmitted disease, and better knowledge of food hygiene may also aid in prevention of infection such as Salmonella, Campylobacter and *E. coli* O157. Investment in educational strategies, along with the recognition that monitoring of current and newly emerging disease should be supported, will ultimately be shown to be of benefit to public health.

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Conflict of interest

R.M. Lynn is the BPSU scientific co-ordinator and one of the investigators undertaking HUS surveillance. The BPSU currently receives a grant from the Department of Health.

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