Audit activity of trainees in the West of Scotland

Audit is becoming an increasingly important tool for use in medical practice under the auspices of clinical governance, and the expectation for trainees to participate in audit is increasing. The Royal College of Paediatrics and Child Health recommends that specialist registrar trainees perform yearly audit during their training and audits performed during Higher Specialist Training form part of the competency framework for specialty training.

One of the required characteristics of applicants for specialist registrar positions in the West of Scotland is participation in audit, and one of the desirable characteristics is active involvement in audit. It is therefore important that trainees are aware of the requirements of audit and are given enough time and support in which to carry it out.

Questionnaires were sent to all experienced senior house officers and specialist registrars in the West of Scotland training programme to assess audit activity.

Response rate was 83% in the specialist registrar group and 59% in the experienced SHO group.

In the specialist registrar group, 93% of respondents had performed an audit during their training although only 48% had completed an audit in each year of their training. Fifty two per cent of audits led to a change in practice, with only 16% being re-audited and therefore completing the audit cycle. Fifty two per cent of respondents graded the level of support given by senior staff to be less than satisfactory.

In the experienced SHO group, 92% of respondents had completed an audit with 53% actively involved in audit at the time of questioning. Forty two per cent of audits undertaken had led to a change in practice, with only 17% being re-audited. The most common reasons cited for those who had not performed an audit were insufficient time (100%) and lack of knowledge of a topic to audit (83%). Seventy six per cent of respondents felt that being given an audit topic and brief outline of how to carry this out at the beginning of a post would increase likelihood of completing an audit. Thirty per cent graded the level of support given by senior staff to be less than satisfactory.

Although the incidence of performing audit was high in the population questioned (92%), the incidence of completing the audit cycle was low (16%). Factors identified which may increase audit activity include increased support from senior staff, more time available for audit, and being allocated an audit topic and outline of how to carry this out at the beginning of a post.

Hypothermia following fever

A 15 month old child presented to A&E with a temperature of 39.2°C. On examination she was fully conscious, tachycardic, and tachypnoeic. Examination revealed crepitations at the left base. A chest x-ray confirmed the presence of a left lower lobe pneumonia. She was commenced on intravenous cefuroxime; initial results revealed white cell count 19.1 x 10^9/l (neutrophilia) and C reactive protein 126 mg/l.

On arrival to the ward she was found to be hypothermic (33.6°C). She had received paracetamol (15 mg/kg) and ibuprofen (5 mg/kg) in A&E. She had not been unduly exposed. This was her first presentation to hospital. In view of hypothermia with obvious sepsis a lumbar puncture was performed to rule out CNS involvement. This was entirely normal. Despite warming techniques she remained cold for the next 11 hours (fig 1).

Prolonged hypothermia provoked investigation of central causation. Thyroid function tests, cortisol, and computed tomography were normal. She recovered from her pneumonia and has been entirely well since.

In view of the temporal link between the antipyretics and the fall in temperature, it seems appropriate to consider causation. Both paracetamol and ibuprofen have previously been linked individually to hypothermia. Logically, giving both together may have a summative effect on decreasing temperature. Currently there seems to be a great hurry to “treat temperatures”, often using high doses of paracetamol combined with ibuprofen to reach the magic 37°C. However, the risks and benefits of fever should be weighed up. Fever induces host defence mechanisms preventing multiplication of organisms, but can also lead to febrile convulsions and increased cardiovascular demands.

Antipyretics are not without their problems and hypothermia may be one of these. Is hypothermia bad for you? Probably not in the short term, but generally we are not trying to induce it.

A recent case also describes hypothermia following a single dose of ibuprofen. This had a duration of four days.1 Ibuprofen has a half life of 2 hours and is unlikely to have such a prolonged effect. Hypothermic sepsis is uncommon in paediatrics but must also be considered in both cases.1 It is important to note that in neither case was a causal organism identified.

Mosaic Down’s syndrome prevalence in a complete population study

From 1 January 1997 to 31 December 2001 we performed a retrospective observational study on the incidence, accuracy of clinical diagnosis, and prevalence estimation of Down’s syndrome in a well defined population of 1.7 million in Northern Ireland.2 A total of 208 postnatal cases of Down’s syndrome were diagnosed: 197 trisomy (94.7%), 3 translocation (1.45%), and 8 mosaic cases (3.85%) (expected ratios 94% trisomy, 5% translocation, 1% mosaic). In a population of 114 307 live births, a minimum prevalence of 167.9 per 100 000 (or 1 in 595 births) was calculated.

The detection rate of mosaic variants is higher than quoted rates of 1–3%. This may be accounted for by inclusion of newly diagnosed adult cases in our study, but mosaic variants often do not have dysmorphic features and may not be identified in studies.

Ninety per cent of trisomy and 100% of translocation cases were diagnosed on clinical features alone, with karyotyping carried out for diagnostic confirmation. This figure fell to 37.5% for mosaic cases (p < 0.001), confirming the difficulties with the clinical diagnosis of mosaic Down’s syndrome, where few classical dysmorphic features are present.

The two mosaic cases diagnosed within seven days of life presented with simian creases, hypotonia, and characteristic facial features including epicantthic folds, up-slanting palpebral fissures, and protruding tongue. One patient had a sandal gap.

Three mosaic children were diagnosed after day 7. One clinically felt to be Noonan syndrome was diagnosed at 6 months. Another (diagnosed at 19 months) presented with developmental delay, without dysmorphic features, and the third had a sample sent at 7.5 years, as a check sample and not time of first diagnosis.

Three mosaic patients were diagnosed as adults. One was an inpatient at a regional specialist assessment centre for learning disabilities, and was previously known to have Down’s syndrome. A second presented at 18 years of age and was educationally subnormal with no dysmorphic features. The

References


Aspirin, Reye syndrome, Kawasaki disease, and allergies: a reconsideration of the links

Reye syndrome is very uncommon in Kawasaki disease patients despite the widespread use of aspirin. It is time to rethink the link between aspirin and Reye syndrome in the light of the rising prevalence of allergies for which the declining use of aspirin may be contributory.

The use of aspirin (ASA) has declined dramatically since the 1980s following reports linking its use to Reye syndrome. Since then, paracetamol has become the drug of choice for the treatment of fever or pain in children, and even in adults.1 Concurrently there has been an increase in the worldwide prevalence of the various allergic diseases, especially in industrialised countries.2-6 It may not be too bold a postulate that this increase in allergic diseases might be due (at least in part) to the decreased use of ASA, which has an anti-inflammatory action, suppresses subclinical or clinical inflammation. Paracetamol in contrast, has no such anti-inflammatory effects.

The current recommendations for the management of children with Kawasaki disease include treatment with high dose aspirin in the acute phase, and low dose aspirin during the period of thrombocytosis.7 For those with residual coronary problems, low dose aspirin is often given over an even longer term. In Japan alone, up to 200 000 children have received ASA for Kawasaki disease. Interestingly, only one case of Reye syndrome associated with Kawasaki disease has ever been reported, and only in the Japanese literature, giving an incidence of <0.005%.7

It is perhaps time to rethink whether there is any causal link between ASA and Reye syndrome. The relation between declining ASA use and increasing prevalence of allergies should also be more extensively evaluated. Paediatricians may want to consider ASA in place of paracetamol as their first choice antipyretic/analgesic in children, especially for those with a significant family and background history of atopy. If our prescribing habits change, we might yet see a decline in the prevalence of allergic diseases.
current deaneries, you eventually manage to transfer the NTN. The apparent lack of communication between the deaneries, the uncertainty over the NTN transfer, and personal ‘tumult’ over moving residence and starting in the vastly different world of laboratory science, cause intense personal anxiety. Despite the introduction ‘Calman’ scheme in Paediatrics in 1996 and the potential for inter-deanery transfers, it appears easier to move for personal reasons, such as a partner moving, than for obvious academic reasons.

During the later stages of your research time, you may be keen on specialising in paediatric intensive care medicine (PICM). The recent introduction of “grid numbers” within paediatric specialties has enabled trainees to receive regional subspecialty training, but at the cost of alienating SpRs that joined the Calman scheme prior to its inception, who have already undertaken a substantial proportion of their paediatric training. During this “transition” phase, some SpRs wishing to specialise face an uncertain future.

Second problem: With the current deanery wishing to recognise “some” of the research time, you are awarded your certificate of completion of specialist training (CCST), you do not have the minimum 24 months necessary to commit to a PICM grid post. You frantically write and speak to everyone involved with PICM training issues locally and nationally, including the Royal College of Paediatrics and Child Health (RCPCH), to resolve the matter. Countless e-mails later, you come to the realisation that you are very much alone in this “fight” to secure an academic subspecialty! The most frustrating aspect is that nobody, including the RCPCH, appears able to support you in your quest to obtain the necessary training, so that as a consultant you have all the necessary skills and experience. An additional problem may be that any clinical experience obtained in the hospital unit may not be recognised by the subspecialty committee to count as recognised training time in that subspecialty. At the end of the day, the deanery and specifically the post-graduate dean alone, dictates the time available for your training. After approximately eight months of uncertainty, I still await an extension to my CCST date. Furthermore, at the prime of my research, I have really questioned the cumbersome bureaucracy that seems to stifle the enthusiasm and aspirations of those who are prepared to make major changes to their own and their family’s lives to achieve their career goals.

Despite the Calman reconstruction of the middle grade staff training to “produce a shorter, more structured and organised training pathway”, that independent clinical competence as a consultant can be achieved much earlier than in the past in many disciplines,5 SpR training has not been shortened. Indeed, the average time to complete a five year CCST programme is over six years, with 47% of trainees having their CCST date reviewed.6 Postgraduate deans are obviously under “pressure” to implement the UK national directive to increase consultant paediatric training slots substantially over the next 5–10 years, which will require the delivery of suitably trained doctors. The danger is that the system nudges along the less motivated trainees, without necessarily encouraging those wishing to “branch out” and pursue interests in their chosen field. One such group that Calman appears to have neglected is academic trainees. The rapid decline in academic trainees and suitable posts in the UK must be of major concern to everyone.7,8 Despite this apparent demise, there appears to be genuine interest in undertaking research among paediatric SpRs, even if they do not have the necessary skills, training, or opportunity to undertake this within their Calman training post.

What are the possible solutions? Above all, academic trainees need be encouraged and nurtured. Anyone involved in research will become disillusioned and frustrated at some point, but to face endless bureaucracy and time constraining or diagnostic training and CCST, is both unnecessary and pointless. In the end, all that will happen is that trainees with real academic promise will be discouraged even before they start. If deaneries are to continue to manage SpRs with subspecialty and/or academic interests, there needs to be closer links with the RCPCH and in particular the relevant College subspecialty advisory committees. In addition, at a local level, a “mentoring” system for future academic trainees needs to be established, primarily to encourage research ideas and methodology, but also to provide a support network. If training issues are not addressed, the future of subspecialty and academic paediatrics in this country appears bleak.

On a closing note, although I know I am not unique in this situation, I really question whether I will ever make the same choices, if I knew the obvious limitations of the current training scheme!

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doi: 10.1136/adc.2004.057927

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Clinical improvement in cystic fibrosis following anti-tumourous chemotherapy

Clinical improvement in respiratory function in patients with cystic fibrosis (CF) has been reported following treatment with anti-tumourous chemotherapy. It has been suggested that long term upregulation of genes encoding proteins promoting multidrug resistance (MDR), including MDR P glycoprotein, may contribute to the reduced efficacy of the CFTR protein which is deficient in CF and may be the mechanism by which macrolides exert their effect in CF.9 Previous reports suggested an increase in MDR following chemotherapy in CF patients and called for more reporting of cases of CF patients undergoing chemotherapy.

We report a case of a male CF patient (homozygous ΔF508), aged 7 years, who underwent chemotherapy for acute myeloid leukaemia. He remained well during the six months of treatment, with no chest exacerbations. After cessation of treatment, nasal potential difference measurements showed a 6 mV response with a low chloride perfusion, unusual in CF, with typical CF baseline and A amiloride readings (~49 mV and 39 mV respectively). No increase in MDR P glycoprotein mRNA was detected from nasal brushings, compared to three other CF subjects. Six months post chemotherapy, he remained clinically well, with good lung function (FEV1 (96%) and FVC (110%): pretreatment FEV1 and FVC both 60%).

Interpretation of these data is clearly limited by the lack of pre-chemotherapy testing. In order that the potential effect of these drugs can be understood, we suggest a formal protocol for collection of quantitative data from cases of CF patients presenting with malignancies, both before and after the initiation of chemotherapy. In addition to records of clinical status, as performed by previous groups,1 we recommend: (a) pre- and post-treatment sweat tests (chloride and sodium values); (b) collection of nasal or (opportunistically) bronchial brushings for quantification of MDR P glycoprotein mRNA, by real time RT-PCR; and (c) nasal potential difference measurements. If the role of MDR proteins in mediating this effect is further substantiated in future CF patients receiving chemotherapy, there may be a role for the development of novel drugs which modulate MDR proteins.

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L-carnitine in cyclical vomiting syndrome

Cyclical vomiting syndrome is a disorder of unknown aetiology, characterised by recurrent stereotypic episodes of nausea and vomiting, with full recovery between attacks. The onset may be within the first three years of life, although UK studies have found the average age of onset to be 5.3 years. Childhood co-morbidities include motion sickness, constipation, and abdominal migraine. Exacerbations can be triggered by physical factors (infection, tiredness, pain, exercise, foods) and psychological stresses, both positive and negative. The attacks usually subside in adolescence, at which stage many develop migraine headaches.

Our 7 year old patient first presented with vomiting and constipation at 2 years of age. Over time, he suffered repeated stereotypical episodes. Extensive blood investigations and radiological studies were negative. He was diagnosed with cyclical vomiting syndrome. Attempts to control the frequent attacks were made with anti-emetics, prokinetics, antihistamines. None were effective, although sedation with lorazepam during exacerbations reduced the length of hospitalisation. Since diagnosis, he had been an inpatient only once with a vomiting episode. At age 6 years, he was empirically started on L-carnitine at 50 mg/kg daily. Pre- and post-treatment carnitine levels were normal, carnitine dependence may be an important metabolic requirement in cyclical vomiting syndrome patients because of a high metabolic turnover. As far as we are aware, this is the most striking improvement by L-carnitine ever reported in the literature. L-carnitine responsive cyclical vomiting syndrome is a possible subgroup or phenotype in the diagnostic workup of children with this syndrome, and should be considered an early treatment option in children suffering from intractable cyclical vomiting.

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doi: 10.1136/adc.2003.047183

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References


Childhood cancer survivorship—improving care and quality of life


Faced with another book about survivors of childhood cancer, the first thing to ask was, “Is this any different from the others? What does it add that other recently published books or the latest detailed reviews have not already said? Will it be just another list of late effects of therapy?” But this book is indeed different. It is a report from the National Cancer Policy Board in the United States as part of an initiative to address issues of concern for cancer survivors and does, I think, move forward the discussion about how the care for patients who have survived cancer should be provided. But how far?

The report determines that “an individual is considered a cancer survivor from the time of diagnosis through the balance of his/her life. Family members, friends and caregivers are also impacted by the survivorship experience and are therefore included in this definition.” It proposes seven recommendations—a policy agenda for the care of this unique group of individuals and gives a series of seven recommendations relating to clinical guidelines, minimum standards, improving awareness, education, future delivery of care, and research. It is not a list of late effects (although these are well summarised in chapter four) but rather looks forward to how the follow-up of patients who are survivors of childhood cancer can and should be addressed.

The three main themes explored in the report are provision of services, the education and training of professionals, and the areas where research needs to be conducted, not only the identification, prevalence, and aetiology of late effects, but exploring interventions that may ameliorate late effects and systematically evaluating treatment approaches and standards of follow-up care. Although this report is entirely based on practice in the USA, in many countries, including the UK, there is no organised, defined system of care for these patients. Furthermore as cancer therapy changes (intensiﬁcation of chemotherapy for some, minimal risk of any late effects of therapy for others), so the needs of this population continually change. The report explores different systems of care provision and recognises that there is no consensus on where care should be provided and by whom. There is a significant variation in cancer care across the country. Both in the USA and the UK, there is a need for evidence based clinical practice guidelines for the carers of survivors of cancer.

One of the strengths of this report is the background epidemiology and the chapter on the late effects. These data presented are, in part, based on the Childhood Cancer Survivor Study, a cohort study of 20 227 five-year survivors of childhood cancer diagnosed from 1970 to 1986 in the USA. The 10.8-fold excess in overall mortality requires reﬂection. Another key chapter is that on “delivering survivorship care”. Proposals for ideal follow up systems are given but, perhaps more
importantly, the authors reflect on the lack of evidence there is for appropriate systems of follow up and the need to explore novel approaches, such as distance networking through internet and telecommunications. The use of primary nurse-led care or care co-ordinators, from a variety of healthcare backgrounds is discussed. The challenge of crossing different sectors (medical, social care, education) to inform carers of the needs of this group of patients, optimise their care, and share responsibility for the needs of this patient group is also recognised.

One area of particular relevance not only to survivors of childhood cancer but also for other children with chronic diseases and indeed adults who have survived cancer, is the potential difficulties with future employment and life or health insurance. Although indeed adults who have survived cancer, is survivors of childhood cancer but also for this group of patients, optimise their care, and share responsibility for the needs of this patient group is also recognised.

There are some books that you wish you could have with you at all times. The well known format of the APLS manual is an A4 size book with plenty of free space, large diagrams, and comfortable font size. There was clearly no intention to compromise any of these for the sake of portability; the manual does not aspire to be pocketized. The APLS manual in paper form is published by BMJ Books and luckily this forward thinking publisher has now brought out several of their publications in electronic format. Once you buy the PDA version of the APLS manual it can be used on your PDA as well as on your desktop PC. BMJ Books use Mobipocket Reader for their eBooks. This is a very good choice because Mobipocket is the only one (of the three common PDA friendly formats that support encrypted eBooks) that runs on Palm OS, Pocket PC, Psion, Nokia, and Sony Ericsson P800. The PDA world is still divided into those with palm and those with Windows operating systems and it is important that the book is available for both platforms.

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M E M Jenney

Advanced paediatric life support: the practical approach, 3rd edition


The majority of readers of Archives of Disease in Childhood will know this book very well. My task here is not to appraise the content of the ebook, which is the same as the (now famous) paperback, but to test how well it serves its purpose when used in the ebook format, and specifically on a personal digital assistant (PDA).

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Hypothermia following fever

J Richardson and J Sills

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