Examples of audit in primary care paediatrics

Possible criteria for audit of urinary tract infection care

- 1 The accuracy of the diagnosis—that is, was an appropriately taken midstream urine specimen used to make the diagnosis, and was the result consistent with the diagnosis?
- 2 Was the infection appropriately investigated and followed up?
- 3 Was a suitable antibiotic used?

Structure—We may want to look at how good our children's facilities are. The best judges of that are the children and their parents, so it is best to find out how we are doing from a patient satisfaction survey. If worded properly, such a survey could use the parents' comments to create ideas to change the existing arrangements to be more "child friendly."

Process—We could choose an important topic such as urinary tract infection in childhood. The sort of criteria of concern might be those shown opposite.

All these criteria need to be fully discussed, justified by good published work, and then have standards applied to them. For example, the standard for taking a midstream urine specimen before treating a child with a suspected urinary tract infection would probably be agreed by most reasonable general practitioners to be 100%.

Outcome—This is the most difficult subject to assess in the small populations that we deal with in general practice. However, it is possible to find a few examples. One could be to look at emergency admissions of children with asthma as a criterion of control outcome. Having sampled a group, the number of emergency admissions in a year can be measured. Having created whatever change was in mind for the children's care, the audit can be run again to see if emergency admissions have reduced.

Audit is particularly useful in primary care for assessing the effectiveness of clinics. For example, auditing various aspects of asthma care before and after instituting an asthma clinic will help ascertain whether or not the clinic is worthwhile.

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Lesson of the Week

Heparin induced thrombosis: an important complication of heparin prophylaxis for thromboembolic disease in surgery

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Heparin may cause life threatening thromboses, which can be prevented by regular monitoring of the platelet count

Heparin induced thrombocytopenia with thrombosis, or the "white clot syndrome," is a rare but recognised complication of heparin therapy. The syndrome is idiosyncratic, immune mediated, and not dose dependent and therefore is equally likely to occur with prophylactic and therapeutic heparin dosage regimens. Despite published reports on the subject we were alarmed that many surgeons who regularly use heparin to prevent or treat thrombosis were unaware that heparin can induce thrombotic complications in susceptible patients. We present three typical case histories, a brief discussion of the condition, and some guidelines on its prevention and treatment.

Case 1

A 79 year old woman was admitted for a subcapital fracture of the right femur in April 1992. Prophylactic treatment with unfractionated calcium heparin was started on admission at a dose of 5000 IU three times daily. A Thompson hemiarthroplasty was performed 48 hours after admission. On the seventh postoperative day she underwent routine plethysmography, and as a result of the findings phlebography was performed. This confirmed extensive right femoral vein thrombosis (fig 1). She was fully anticoagulated with

intravenous sodium heparin. A full blood count at the time showed platelets to be $73 \times 10^9/l$ (normal $150-400 \times 10^9/l$). Two days later she developed bilateral signs and symptoms of acute leg ischaemia. An arteriogram was suggestive of emboli and she underwent left femoral embolectomy. At operation the naked eye appearance was that of white clot, which was later confirmed histologically.

Although good flow was re-established at operation, the leg became ischaemic again. Two weeks later the patient underwent below knee amputation. Her heparin was continued postoperatively until she was fully anticoagulated with warfarin. Twenty four hours after stopping heparin her platelet count had recovered to $112 \times 10^9/l$, and it was normal next day.

Case 2

A 70 year old man underwent elective right total hip replacement in May 1992. He was given calcium heparin prophylaxis at a dose of 5000 IU twice daily. There was no history of peripheral vascular disease or cardiac problems. In 1986 he had undergone left total hip replacement without prophylactic heparin cover.

Ten days postoperatively he developed clinical signs and symptoms of a deep venous thrombosis and phlebography confirmed right femoral vein throm-

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bosis. He was fully anticoagulated with sodium heparin. Six days later he developed acute ischaemia of the right leg. At that time the platelet count was $47 \times 10^9/l$. An arteriogram on the same day showed that both popliteal arteries were occluded (fig 2). Thrombectomy was performed and typical white clot was withdrawn. His platelet count remained low. Four days later his unfractionated heparin was stopped and anticoagulation with low molecular weight heparin started. After this his platelet count improved steadily and he had no more ischaemic episodes.

Case 3

A 72 year old woman underwent abdominoperineal resection of the rectum in July 1992. Prophylaxis with heparin was begun preoperatively, and the preoperative platelet count was $295 \times 10^9/l$. Seven days after operation the platelet count was $56 \times 10^9/l$. On the ninth day she complained of chest pain and shortness of breath, and pulmonary embolism was diagnosed by ventilation-perfusion scan. The platelet count was $19 \times 10^9/l$ and she therefore began a therapeutic dose of low molecular weight heparin (enoxaparin), and warfarin was instituted. Treatment with enoxaparin continued for four days.

On the 14th postoperative day she complained of left calf pain. This was treated symptomatically as she was already fully anticoagulated with warfarin. Five days later the leg became acutely swollen and blue. The platelet count remained $48 \times 10^9/l$. A filter was inserted into the vena cava as the thrombus was clearly extending despite full anticoagulation, and samples were taken for platelet aggregation studies. Later these showed heparin and enoxaparin dependent aggregation.

Over the next 10 days the platelet count gradually recovered. The leg, however, developed venous gangrene. Because of this and deteriorating renal function the leg was amputated by disarticulation through the hip one month after the original abdominal operation. The patient recovered but was wheelchair bound.

Discussion

Heparin induced thrombocytopenia is a very important idiosyncratic drug reaction. It is said to occur in 5% of patients receiving heparin.¹ The risk of haemorrhage in a patient who becomes thrombocytopenic while receiving heparin is obvious. Bleeding usually results in withdrawal of heparin, in line with advice from the Committee on Safety of Medicines.

Thrombotic events associated with thrombocytopenia are less well recognised and are not mentioned in the current *British National Formulary* or in the datasheets from manufacturers. They may occur in 20% of patients with heparin induced thrombocytopenia and present life threatening complications.² The resulting mortality has been reported as high as 25%.³ If the cause is not recognised patients may receive increased doses of heparin, exacerbating the situation.

Three cases occurring in four months is broadly in line with the figures quoted above, and highlighting the problem in the Nottingham hospitals has resulted in other cases being brought to our attention. Although all the patients were treated with the same brand of heparin (Calciparine), no two patients received heparin from the same batch. There was no appreciable overlap in the concomitant medication, and the only relevant change in practice was that in orthopaedic surgery heparin tended to be given for the entire inpatient stay rather than for a standard five days. This was a result of reports of late thrombosis after stopping prophylaxis.^{4,5}

FIG 1—Case 1. Phlebogram showing extensive femoropopliteal venous thrombosis

FIG 2—Case 2. Arteriogram showing bilateral popliteal arterial thrombosis

The most likely "cause" of the cluster of cases was prior ignorance of the condition.

Thrombotic events of all types can be associated with heparin induced thrombocytopenia, including myocardial infarctions and thrombotic strokes. More rarely, thromboses can occur in mesenteric, renal, adrenal, and spinal arteries.⁶ Myocardial infarctions and strokes are relatively common among elderly surgical patients receiving heparin prophylaxis—for example, those having major orthopaedic surgery for hip fracture or joint replacement—and only a small proportion are due to heparin induced thrombocytopenia with thrombosis. Thrombotic complications that are heparin associated are therefore likely to be underdiagnosed unless surgeons are aware that heparin can rarely cause or exacerbate thrombotic episodes.

The thromboses associated with this condition consist mainly of platelets and are therefore pale because red cells have not become enmeshed within them by fibrin. This is the origin of the term white clot syndrome, which is widely used in the United States and Canada.

DIAGNOSIS

The diagnosis of heparin induced thrombocytopenia is usually made on the basis of the exclusion of other causes of thrombocytopenia or retrospectively from a rapid rise in the platelet count after the withdrawal of heparin. Aggregation of control platelets by the patient's serum or plasma in the presence of heparin is a fairly simple test. It can be performed in most haematology laboratories and may be used to predict whether the platelets will cross react with low molecular weight heparins. The methods used for such testing vary from simple aggregation to more complex tests that can be found in haematological publications.⁷

TREATMENT

Arterial thromboses resulting from heparin should be treated on their own merits. As they are not associated with atherosclerotic lesions the thrombus is frequently amenable to removal with an embolectomy catheter. When the indication for heparin is purely prophylactic it should be stopped and replaced by oral anticoagulation if the patient remains at high risk of venous thromboembolism. If the patient has already sustained a severe deep vein thrombosis the situation is

more difficult. In the first instance low molecular weight heparin may be substituted for unfractionated heparin after performing the appropriate platelet aggregation studies. When results are negative a favourable outcome is likely, but cross reaction occurs in one third of cases.⁸ Although this may imply that low molecular weight heparins carry a smaller risk of thrombotic complications, this requires confirmation after longer clinical experience with the newer agents.

Oral anticoagulation should be started so that it may become effective in 48 to 72 hours. If the platelet count does not recover or further arterial thrombi occur then all forms of heparin will have to be abandoned. Fibrinolytic agents such as streptokinase and urokinase have been used but are not options in the first few days after surgery because of their potentially devastating effects on wound healing. This is particularly important in joint replacement, where wound breakdown may presage serious complications. Ancrod, a defibrinating agent, has been used, particularly in Canada, and is said not to affect mature cross linked fibrin or cause bleeding problems.⁹ In the immediate postoperative period interrupting the vena cava with a filter to prevent fatal pulmonary embolism may need to be considered if anticoagulation is contraindicated.³

Recommendations and conclusion

We have three main recommendations. Firstly, all patients receiving heparin for more than five days should have a baseline platelet count performed and a further count five to seven days after treatment was initiated. Secondly, all patients who sustain a deep

venous thrombosis despite heparin prophylaxis must have a platelet count performed, and the possibility of heparin induced thrombosis should be considered before they are anticoagulated with the same drug. Thirdly, patients who have previously been exposed to heparin require a platelet count sooner after re-exposure than five days as prior sensitisation may result in the rapid onset of complications.

Surgeons using heparin prophylaxis must be aware of the serious complications of heparin induced thrombocytopenia.

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Crisis in medical education?

"Something is seriously wrong with medical education in Britain." The pessimistic opening salvo which introduces this stimulating book by Stella Lowry is based on impressions gleaned from conversations with deans of teaching hospitals, the General Medical Council's recent review of medical education, and a lengthy national consensus inquiry organised by the King's Fund. It seems that there is widespread agreement that our present medical school curricula are grossly overcrowded with factual information and that our students are inhibited from developing into creative, critical thinkers and "problem solvers." There are, of course, problems even with these distinguished sources of information. Much of the evidence on which their conclusions are based is anecdotal, and none of them, at least to my knowledge, has ever looked at a control group of graduates from other fields; I doubt if any students worth their salt are ever satisfied with a degree course. Those who take medical education seriously have never been happy about what they are doing. Our present soul searching is reminiscent of what George Pickering was saying 30 years ago, and, for that matter, of Thomas Sydenham's views of medical education in Oxford in the seventeenth century—one of his contemporaries recorded that Sydenham thought it better to send a man to Oxford to be instructed in shoemaking than to learn how to practise medicine. However, the fact that each generation of clinical teachers seems to make a mess of things is certainly not a reason for complacency.

One of the major frustrations for medical educationalists is that there is no way of measuring the end result. . . .

Furthermore, we are trying to design a course to prepare young people for activities which range from a full time career in basic science, through hospital medicine or general practice, to the directorship of a pharmaceutical

company. And it is inappropriate to take undergraduate teaching out of the context of the overall training of doctors. It is no use developing a generation of creative, critical thinkers only for many of them to become disillusioned by a ridiculously long period of apprenticeship or the mind numbing requirements of some of our specialist examinations.

Another major problem for medical education is that it has to adapt itself to the constantly changing scene of clinical science and practice. Sitting through a recent selection committee for a consultant physician, at which the entire conversation revolved round purchasing and providing, and at which clinical practice, teaching, and research were rarely mentioned, I wondered whether our graduates of the future would be better prepared for their professional lives by spending their postgraduate years in a business management college rather than striving to obtain a diploma from one of the medical royal colleges, let alone developing clinical and pastoral skills. It is clear that we cannot go on adding more and more to our courses. Most medical schools have simply grafted on each new development as it has come along; molecular biology, communication skills, ethics, and business management are just a few of the dishes that have been added to an overly rich diet in recent years. No wonder many of our young doctors are not, to borrow one of C P Snow's favourite academic labels, graduating in a state of "smiling euphoria."

. . . But although I agree with most of what Dr Lowry has to say I remain to be convinced that there is a crisis in medical education; there will only be a genuine crisis when those who teach medical students are convinced that they have got it right.—SIR DAVID WEATHERALL in *Medical Education*, by Stella Lowry, 1993. Obtainable from the BMJ bookshop, price £6.95.