SERIOUS HAZARDS OF TRANSFUSION

ANNUAL REPORT

1997 - 1998

Affiliated to the Royal College of Pathologists

British Blood Transfusion Society, British Society for Haematology
Faculty of Public Health Medicine, Institute of Biomedical Science
Institute of Health Service Managers
Public Health Laboratory Service Communicable Disease Surveillance Centre
Royal College of Anaesthetists, Royal College of General Practitioners
Royal College of Nursing, Royal College of Obstetricians and Gynaecologists
Royal College of Paediatrics and Child Health
Royal College of Physicians, Royal College of Surgeons, UK Transfusion Services

Published 9th March 1999

by The Serious Hazards of Transfusion Steering Group

National co-ordinators: **Dr Elizabeth M Love**
Ms.Kate Soldan (PHLS/CDSC)

Assistant national co-ordinator: **Mrs Susan Lowe: now Hilary Jones (SHOT office)**

Writing Group
LM Williamson, S Lowe, E Love, H Cohen,
K Soldan, D B L McClelland, DR Norfolk, J Revill, JAJ Barbara, D Birrell, A Todd
on behalf of the SHOT Steering Group

Requests for further information should be addressed to:

SHOT Office
Manchester Blood Centre
Plymouth Grove
Manchester
M13 9LL

Tel: **0161 251 4208**
Fax: **0161 251 4319**
Email: **hilary.jones@nbs.nhs.uk**
elizabeth.love@nbs.nhs.uk

ISBN **0 9532 789 1 3**

CONTENTS

1. MAIN FINDINGS AND RECOMMENDATIONS

2. FOREWORD – RECENT INITIATIVES TO IMPROVE TRANSFUSION SAFETY

3. TRANSFUSION RISK: PERCEPTION, REALITY AND PREVENTION

4. AIMS, EDUCATIONAL ACTIVITIES AND PUBLICATIONS

5. OVERALL ORGANISATION AND REPORTING SYSTEM

6. OVERVIEW OF RESULTS

7. INCORRECT BLOOD COMPONENT TRANSFUSED

8. ACUTE TRANSFUSION REACTION

9. DELAYED TRANSFUSION REACTION

10. TRANSFUSION-RELATED ACUTE LUNG INJURY

11. POST-TRANSFUSION PURPURA

12. TRANSFUSION-ASSOCIATED GRAFT VERSUS HOST DISEASE

13. TRANSFUSION-TRANSMITTED INFECTIONS

14. 'NEAR MISS' PILOT STUDY

15. FUTURE DEVELOPMENTS

16. REFERENCES

APPENDICES

1. Terms of reference and membership of Steering Group and Standing Working Group
2. Income and expenditure statement
3. Initial report form for non-infectious complications
4. 'Nil to report' card
5. CDSC/PHLS report form for infectious complications
6. Near-miss forms
7. Autologous transfusion form and questionnaire
8. Key points from BCSH Guidelines for the Administration of Blood and Blood Components.
9. Collated responses to questionnaires
    - (i) Incorrect blood component transfused
    - (ii) Acute transfusion reaction
    - (iii) Delayed transfusion reaction
    - (iv) Transfusion-related acute lung injury
    - (v) Post-transfusion purpura
    - (vi) Transfusion-associated graft-versus-host disease

GLOSSARY OF TERMS

ATR	Acute transfusion reaction
BCSH	British Committee for Standards in Haematology
BSH	British Society for Haematology
PHLS/CDSC	Communicable Disease Surveillance Centre of the Public Health Laboratory Service
CPA	Clinical Pathology Accreditation
DTR	Delayed transfusion reaction
FFP	Fresh frozen plasma
HTC	Hospital transfusion committee
IBCT	Incorrect blood component transfused
MCA	Medicines Control Agency
MSBT	Microbial Safety of Blood and Tissues Committee of the Department of Health
NBA	National Blood Authority
NEQAS	National External Quality Assurance Scheme
NHSE	National Health Service Executive
NICE	National Institute for Clinical Excellence
PTI	Post-transfusion infection (defined in Chapter 13)
PTP	Post-transfusion purpura
RCP	Royal College of Physicians
RCPath	Royal College of Pathologists
RhD	Rhesus D
TA-GVHD	Transfusion-associated graft-versus-host disease
TRALI	Transfusion-related acute lung injury
TTI	Transfusion-transmitted infection (defined in Chapter 13)

1. MAIN FINDINGS AND RECOMMENDATIONS

1. Participation

One hundred and ninety seven new reports were received, an increase of 16.5% over the 169 submitted last year. This was almost entirely accounted for by an increase in 'wrong component transfused' incidents from 81 to 110 new reports. Reports were submitted by 112/424 eligible hospitals, compared with 94 in 1996-97. Introduction of a 'nil return' card meant that hospitals with no serious events could register participation. Such cards were received from 164 hospitals, indicating that a total of 276/424 of hospitals (65%) are now taking an active part in SHOT. The importance of SHOT participation was emphasised in the recent Health Service Circular 1998/999 'Better Blood Transfusion'. Discussions have begun with the Clinical Pathology Accreditation (CPA) Scheme for consideration of ways to make SHOT participation a CPA requirement.

At present, blood administration errors may result in disciplinary action and therefore may discourage staff from drawing attention to weaknesses in the blood handling system. Identification and resolution of weak points in the 'blood handling chain' can be done best in an environment of 'no fault reporting', and it is this ethos which SHOT wishes to encourage.

Recommendations

(i) That consideration be given to making participation in SHOT a requirement for Clinical Pathology Accreditation, without breaching the anonymity of SHOT reporting.

(ii) That Trusts and regulatory bodies consider how best to encourage staff to report transfusion errors, and thus help management develop better means of ensuring correct blood administration.

2. 'Incorrect blood component transfused' incidents

Of 114 cases analysed (includes 13 previously reported), there were 2 deaths (1 solely due to the transfusion), and 20 cases of major morbidity, including 16 ABO incompatible transfusions, and 3 potential cases of RhD sensitisation in young females.

Analysis of causes revealed a significant number of primary errors in all areas of the transfusion process. In contrast to last year, laboratory procedures accounted for the highest percentage of first errors (36% compared with 29% in 1996-97), but patient sampling, collection of blood from the blood bank refrigerator, and administration of the component are all liable to potentially fatal errors.

Such errors are increasingly the subject of media interest and litigation. It is therefore timely to draw attention to a number of relevant recommendations from last year's report and offer some suggestions as to how they could be practically implemented.

Recommendation

(iii) Hospital systems should ensure that in-patients and out-patients can be positively identified at the time of both blood sampling and transfusion, especially in out-patient departments where formal patient identification documents may not be available .

One critical enabling factor for improving safety in the clinical transfusion process would be a universal requirement that each patient, at the time of first contact with the hospital or clinic be allocated a unique number that is effectively attached to the patient and is consistently used as the prime identifier for all

processes during the admission. This identifier would also be used for subsequent episodes and for tracing information about the patient through any of the hospital's information systems.

This goal is reflected in the current NHS Information Management and Technology strategy. Achievement of it would not only provide a basis for improving transfusion safety but also, for example, aid in the correct administration of drugs and attribution of diagnostic results.

The present report should provide a further impetus to the introduction of a requirement for the comprehensive use of unique patient identification systems, and the provision of resources for implementation of such systems.

Collection of blood from hospital blood banks is a common source of identification errors. The present report confirms the importance of these errors, which clearly lay the ground for further mistakes at the time of setting up the infusion of blood.

(iv) Hospitals should review their current system to ensure that errors in this area can be prevented. Appropriate staff training is essential. Standards should be set for a minimum identification requirement to be used when a blood component is collected.

Even in the absence of comprehensive identification and information systems, relatively inexpensive "stand alone" systems can offer excellent control of this part of the process, with the added benefit of registering the time that temperature sensitive products are removed from the refrigerator, thus extending the quality assurance of the blood storage conditions much closer to the point of clinical use.

(v) Blood issue control systems, which are already in use in some UK hospitals, should be formally evaluated for their efficacy and cost effectiveness, and further developed if necessary.

The bedside check is vital in preventing transfusion error.

(vi) Staff should be vigilant in checking information details of the blood component against those of the patient.

(vii) Every hospital should have a policy for formally checking the identity of the *patient* against the blood component label *at the bedside.*

Nursing observations during transfusion also show wide variations. National guidelines for the administration and monitoring of transfusion are being developed by the British Committee for Standards in Haematology (BCSH) in collaboration with the Royal Colleges of Nursing and Surgeons. While the new guidelines are an essential step, there is extensive evidence that the publication of guidelines is unlikely in itself to produce large changes in practice. Additional essential ingredients include strong and committed local leadership and management, adequately resourced and continuing training arrangements, and appropriate supporting systems to assist staff where necessary. These may include the use of computer based systems which already exist for the express purpose of supporting final pre transfusion checks and recording essential details of the transaction.

(viii) Computerised patient identification systems should be evaluated for efficacy and cost effectiveness, and further developed if necessary.

3. Immune complications of transfusion

The incidence of reporting of such incidents has remained at a similar level to last year, with 52 new acute/delayed reactions, 11 cases of post-transfusion purpura, 16 of transfusion-related acute lung injury and 4 of transfusion-associated graft-versus-host disease (all 4 fatal).

These are generally not the result of poor practice, although more detail will be sought for the next report on serological procedures in relation to failure to detect red cell alloantibodies. Investigation of such cases is not standardised and is often incomplete.

Some cases did not conform to the standard types of reaction generally recognised after transfusion. However, with the planned introduction of universal leucocyte depletion, and increasing availability of methylene blue FFP and eventually other virus inactivated components, it is important that ALL serious reactions which could possibly be related to transfusion are reported. Only in this way can important side effects of new processes be recognised.

Cases of PTP were generally recognised promptly, and investigated and treated quickly and appropriately. The inclusion of steroids in treatment does not appear to offer any advantage to the speed of recovery.

As in last year's report, TA-GVHD remains the commonest cause of death (4 cases). Taking the 2 years' reports together, the commonest single predisposing factor is B cell lymphoid malignancy, and a case can now be made that all such patients should receive irradiated components. A further case was seen in a cardiac surgery patient who received 'fresh' blood.

Full investigation of cases of TRALI and TA-GVHD require a joint approach between the hospital and the supplying Blood Centre, as donor testing is required. Development of standard protocols for investigation of suspected cases would aid diagnosis and establish underlying risk factors.

Recommendations

(ix) Clinicians are encouraged to report ALL types of serious unexpected reaction associated with transfusion.

(x) As for investigation of suspected transfusion-transmitted infection, Blood Services should develop systems for ensuring appropriate donor investigation of cases of suspected TRALI and TA-GVHD. This would also ensure complete and timely data provision to SHOT and minimise dual reporting.

(xi) Development of a standard protocol for investigation of TRALI and TA-GVHD would aid diagnosis. For TA-GVHD, consideration should be given to establishment of a single laboratory as a national reference centre, expert in techniques useful in this setting.

(xii) BCSH Guidelines for Irradiation of Blood Components should be reviewed for consideration of inclusion of patients with B cell lymphoid malignancy.

(xiii) The additional risks of 'fresh blood' should be borne in mind if such blood is ordered for cardiac surgery.

4. Transfusion-transmitted infections

Thirty-five reports of suspected cases were investigated. Of 26 completed investigations, only 4 fulfilled the criteria for confirmed probable transfusion-transmitted infection. The 4 cases comprised 2 hepatitis B transmissions (HBV), 1 hepatitis C (HCV), and 1 fatal case of bacterial contamination. Both HBV transmissions came from donors in the acute phase of infection, both of whom had risk factors which should have excluded them from donating. The newly diagnosed case of HCV transmission had been transfused in 1984, 7 years before the introduction of HCV donor screening. The fatality due to bacterial contamination was associated with *Staphylococcus aureus* in a platelet pool.

Recommendations

(xiv) National collation of data arising from these cases needs to continue over several years before a complete picture of the extent and nature of the infectious complications of transfusions can emerge.

(xv) Clinicians should report all post-transfusion infections diagnosed in their patients to their local blood centre for appropriate investigation. Blood centres should, in turn, complete an initial report form as soon as possible.

(xvi) National guidelines for the bacteriological investigation of adverse reactions associated with transfusion are available for hospitals. Hospitals should not destroy blood components implicated in post-transfusion reactions suspected to be due to bacteria, and should consult these guidelines or their local blood centre about the investigation of such cases.

(xvii) Methods and criteria used to exclude those individuals who have risk factors for transfusion transmissible infections from donating blood warrant continuing evaluation and development. Investigation of the reasons for non-exclusion of ineligible donors is also warranted.

5. Priority setting in blood safety

During the year since the publication of the last SHOT report, many pressures have come to bear on those responsible for the prevention of transfusion-transmitted infection. Considerations regarding the theoretical possibility of nvCJD transmission via blood have led to decisions to leucocyte deplete all blood components, and to source plasma for fractionation from outside the UK. This issue may in the end lead to more appropriate blood usage, and serious consideration of alternatives to donor blood. Nevertheless, it remains the case that there is no means by which the various elements contributing to overall blood safety are considered together, so that priorities can be set for action and direction of resources. Funding for prevention of infection and some immune complications eg TRALI requires national policy setting at Department of Health level and action by the Transfusion Services; prevention of TA-GVHD (the commonest cause of death in both SHOT reports) is covered by a BCSH guideline, while attention to blood administration processes are the responsibility of local management.

Recommendations

(xviii) Currently several organisations produce recommendations and guidelines aimed at assuring safety in different parts of the transfusion process. The need for a unitary source of clear and co-ordinated guidance for the various stakeholders in the transfusion process is now even more apparent. A unified body with overall responsibility for transfusion safety is recommended, to set priorities and direct resources for maximum patient benefit. Such a body could assist those responsible for clinical governance related to transfusion by beginning to provide a coherent system of standards and guidance covering both the " production" and clinical steps in the process.

6. Research into the dissemination, evaluation and outcome of the SHOT recommendations

Confidential enquiries such as SHOT can potentially achieve a great deal, but only if their findings are well promulgated, and result in changes in practice. It is often difficult to establish, in a voluntary reporting system covering only the most severe complications of transfusion, objective means of showing improvement in the overall transfusion process, since numbers of reports received are an insensitive measure of total performance. The following are suggestions for improving both accessibility to SHOT findings, and evaluation of changes in practice which may have resulted.

Recommendations

(xix) Establish and "market" a website with a well developed programme of objectives, starting with extending the availability of the report, determining the patterns of access to the site, and once staff resource is available, establishing some form of two way communication through a bulletin board, "Q&A" service etc.

(xx) Commission a simple questionnaire survey (possibly by phone and fax) to establish what proportion of addressees recall receiving the report or summary, and whether they can state what measures they have taken towards implementing any of the recommendations.

(xxi) Commission, perhaps in collaboration with Blood Group Serology NEQAS, a more comprehensive survey on a sample of institutions to establish a data set on their current laboratory procedures and the change processes and constraints. This would need to be in part done by site visits to validate written responses and to conduct relevant qualitative research through interviews with key individuals.

Recommendations (xx) and (xxi) above could and probably should be contracted to an independent organisation with a proven record of this type of health services research.

2. FOREWORD – RECENT INITIATIVES TO IMPROVE TRANSFUSION SAFETY

The Serious Hazards of Transfusion (SHOT) scheme, which receives and collates reports of death or complications of transfusion of blood or components on a voluntary confidential basis, is now well established. The first Annual Report covering 1996-97, in which 94 hospitals participated, showed that transfusion of a blood component to the wrong patient was the commonest problem, with 81/169 reports. In contrast there were only 8 reports of confirmed transfusion-transmitted infection. The findings indicated that blood itself is extremely safe, but drew attention to the need to direct resources towards the development of novel systems to ensure that it is correctly administered. In the meantime, promotion of secure blood handling procedures and training of all grades of staff concerned with the transfusion process was emphasised.

Notably, several recent initiatives to improve transfusion safety are in line with the findings and recommendations in the first annual SHOT report. To minimise the risk of incompatible transfusion, a guideline for blood handling and administration developed by the British Committee for Standards in Haematology will soon be published (for key points see Appendix 8). Following a Symposium organised by the UK Chief Medical Officers on Evidence-Based Blood Transfusion in July 1998, the implementation of hospital transfusion committees and participation in SHOT has been recommended in NHSE Circular HSC 1998/999 'Better Blood Transfusion'. The Clinical Pathology Accreditation Scheme, which accredits hospital blood banks, has recommended that appropriate procedures must be available for monitoring transfusion hazards. The National External Quality Assurance Scheme in Blood Group Serology has focussed in recent exercises on the detection and identification of multiple red cell alloantibodies, and improvement of red cell identification panels.

Prevention of transfusion-transmitted infection is addressed in a number of ways. Blood donor selection is now more rigorous with direct questioning to exclude potentially infectious donors, and selection criteria have been amended to exclude as a cell donor any individual who was resident as a child in a malarious area, unless shown to be negative for malarial antibodies. National guidelines have been developed for donor arm cleansing to minimise the risk of bacterial contamination of blood, and for the bacteriological investigation of adverse reactions associated with transfusion. Leucocyte depletion of blood, announced by the Department of Health as a purely precautionary measure against possible transmission of new-variant Creutzfeld-Jacob disease, is now being implemented.

This second Annual Report from SHOT, covering October 1997-September 1998, has incorporated the results of anonymised 'nil return' cards – the aim of this initiative is to obtain denominator figures against which the true risk of transfusion complications can be calculated. The results of a pilot study of 'near-miss' events, where an error is detected in time to prevent a mis-transfusion, have also been included. Increasing interest in autologous transfusion necessitates documentation of associated hazards, and a further recent initiative has been the extension of SHOT reporting to include serious hazards associated with autologous pre-deposit procedures. This will eventually be extended to cover other autologous procedures, such as cell salvage.

Once again, we would like to thank all those of you who took the time and trouble to send in reports and complete 'nil return' cards, which together have demonstrated participation in SHOT by two thirds of hospitals. This is tremendous progress in only two years, and reflects well on our established tradition of professional self-regulation. It is anticipated that the introduction of clinical governance will facilitate even wider participation in the future. We are moving towards the emergence of a complete picture of transfusion risk, which will enable informed decision-making on the setting of priorities and direction of resources for maximum patient benefit.

Dr Hannah Cohen MD FRCP FRCPath
Chair, SHOT Steering Group

3. TRANSFUSION RISK: PERCEPTION, REALITY AND PREVENTION

"It is not sufficient that the blood system be safe - it must be considered safe" (Krever Commission of Enquiry on the Blood System in Canada, 1997).

Since the onset of the AIDS epidemic in the early 1980s, public, medical and professional perception of blood transfusion has been dominated by the dread risk of viral transmission. There has been massive intellectual and financial investment directed at improved donor selection, testing for markers of infection and viral inactivation of blood components. The current risk of acquiring HIV by transfusion in the UK is less than 1 in 2,000,000 units transfused. By comparison, the chance of dying from being struck by lightning is around 1 in 1,000,000 and many common medical procedures have risks several magnitudes higher. Serious and fatal adverse reactions to drugs have been reported in 7% of patients in the USA[1].

However, transfusion now exists in a climate, familiar to industries such as nuclear power, where regulatory bodies and public opinion require us to pursue the unattainable goal of "absolute safety" or 'zero risk' at almost any cost. New and highly sensitive tests such as nucleic acid testing (NAT) for HIV, Hepatitis B and Hepatitis C will reduce, but not eliminate, the already low or remote risk of viral transmission at a price which would raise serious questions about cost-effectiveness in other areas of medicine.

In contrast to the unqualified success in reducing viral transmission, other important risks of transfusion have received much less attention or investment. Many more patients in developed countries die from receiving incompatible blood (usually due to mismatch of ABO blood groups) than contract HIV from transfusion. Data from the USA suggests that avoidable deaths from this cause occur in at least 1 in 600,000 transfusions[2] (likely to be a significant underestimate due to under-reporting). These deaths are just the tip of an iceberg[3], the submerged mass of which comprises at least 1 in 30,000 ABO-incompatible transfusions (most of which are not fatal), 1 in 12,000 incorrect units transfused (many, by chance, compatible) and an unknown number of "near miss" events where the error was discovered before transfusion. In the first SHOT Report "wrong blood into patient" episodes made up 47% of all reported events. Bacterial transmission by red cells and platelets also remains a significant hazard with a minimum estimated fatality of 1 in 100,000 transfusions in the USA[2]. Between 1986 and 1991, bacterial transmission accounted for 15.9% of all transfusion-related fatalities in the USA[4]. The first SHOT report recorded 3 bacterial infections in the total of 8 transfusion-transmitted infections reported in the UK in 1996/97. It is worth noting that autologous donors (who store their own blood before surgery) are also at risk from such hazards.

Whilst blood donation, testing and processing at the Transfusion Centre is highly regulated and quality assured, with tightly monitored automated sample identification systems the clinical transfusion process in hospitals remains poorly controlled and understood. Getting "the right blood to the right patient in the right place at the right time"[5] is a complex chain from patient blood sampling through laboratory testing to bedside administration. Errors can, and do, occur at all stages of the process. This SHOT report, like its predecessor, emphasises that "wrong blood into patient" incidents often involve multiple errors at several stages of the process.

Current efforts at prevention/improvement rely on the retrospective investigation of errors. They focus on apportioning blame and often add further checks and safeguards to an already complicated process[6]. This may, paradoxically, make the situation worse. Staff become reluctant to self-report errors, and interventions which seem obvious, such as bedside checking of patient and donor blood groups, may be dangerously unreliable in practice[7]. At the least, our traditional practices are ineffective as there is no evidence of a reduction in fatalities from ABO mismatched transfusion in the last 40 years[2].

How can we do better? Firstly, we need to avoid all unnecessary transfusions and much basic research is needed to establish evidence-based guidelines for transfusion in relation to common medical and surgical conditions. Clearly, we need more effective and sophisticated systems of error-reporting ("haemovigilance") of which SHOT can form the basis in the UK. There are important lessons to be learnt from other "error-critical" industries such as aviation and nuclear power. Whilst errors may be broadly classified as technical, organisational or human, human errors often result from organisational deficiencies. Identifying effective interventions relies on accurate "root cause analysis" of errors, sophisticated methodologies for which are well documented outside the medical sphere. A standardised system for the classification of adverse events is important and such a system for transfusion errors has recently been described in the USA[3]. Recording of near miss (near hit?) events is crucial as they have the same root causes as the much less frequent disasters and provide a rich source of incidents for analysis. An organisational culture of "no fault, no fear" reporting is essential. Successful haemovigilance systems should expect to see an increase in the number of reported events over time but a progressive fall in the "severity index" of the events[3]. This is an important fact for those interpreting successive SHOT reports to understand - a rising number of reported events is likely to be a measure of success. These are not new events, just previously unrecognised or unreported events. The crucial measure of success is a reduction in the proportion of clinically serious events.

Reporting and analysis of errors is only useful if it leads to effective interventions. High quality research is needed to improve our understanding of the clinical transfusion practice and interventions should be subject to rigorous clinical trial and audit. The focus should be on demonstrating improved clinical outcomes for patients. Areas of current interest include unique transfusion ID systems, mechanical devices to prevent mis-transfusion, systems to control access to "satellite" blood fridges and the use of computer-systems to supervise and monitor the whole process from sampling to transfusion. All of these have a cost which may well be amply repaid by a reduction in clinical disasters. A 'zero tolerance' approach to mis-labelled specimens[8] may provide a sense of reassurance, but will still fail to detect two transposed samples from patients whose blood groups are not known.

In summary, it is clear that the common perception of transfusion risks overemphasises the low residual risk of viral transmission whilst underestimating more frequent problems such as mis-transfusion and bacterial contamination. Haemovigilance schemes such as SHOT are essential in providing a knowledge base from which to design and test clinically effective interventions. We need to see much higher rates of event reporting, with constructive debate on the merits of voluntary and mandatory systems, and more sophisticated methodologies for classifying and analysing errors. Technical innovations, such as computerised clinical transfusion systems, will be important but organisational and cultural changes are equally important. Efforts to improve transfusion safety fit well into the "clinical governance" agenda which aims to ensure the quality of clinical care in the NHS. Effective Hospital Transfusion Committees will play a pivotal role at the local level (HSC 1998/999), and should ensure programmes of continuous monitoring and quality improvement in every institution. The knowledge derived from haemovigilance schemes like SHOT will also be important in providing the public, media, politicians and professionals with a clearer perspective on transfusion risks. It should also contribute to "realigning efforts with risks" in the field of transfusion safety[2], not least in the disposition of funds for R&D and service developments.

4 AIMS, EDUCATIONAL ACTIVITIES AND PUBLICATIONS

Aims. The Serious Hazards of Transfusion (SHOT) scheme was launched in November 1996. SHOT is a voluntary anonymised system which aims to collect data on serious adverse events of transfusion of blood components, and to make recommendations to improve transfusion safety.

Through the participating Royal Colleges and professional bodies, SHOT findings can be used to:

◊ inform policy within transfusion services
◊ improve standards of hospital transfusion practice
◊ aid production of clinical guidelines for the use of blood components
◊ educate users on transfusion hazards and their prevention.

Educational activities. Since the launch of the first Annual Report, SHOT has received widespread and very positive coverage both within the UK and overseas. The following are meetings during 1997-1998 in which members of the SHOT team have participated:

1997

October:	International Society for Blood Transfusion, Frankfurt
November:	Royal College of Nursing Transfusion Forum, York
December:	Haemovigilance Symposium, Athens

1998

March :	European Commission Workshop on Haemovigilance, Luxembourg
April :	British Society for Haematology Annual Scientific Meeting, Glasgow
	Institute of Biomedical Scientists Blood Group Serology Conference, Durham
	International Biomedical Science Symposium, Ireland
May :	5th NATO Blood Conference, Lisbon
June:	Royal College of Nursing Forum, York
July:	International Society of Blood Transfusion Scientific Meeting, Oslo
July:	UK Chief Medical Officers' Symposium on Evidence-Based Blood Transfusion
September:	British Blood Transfusion Society Annual Scientific Meeting, Nottingham Institute of Biomedical Sciences Scientific Meeting
November:	Royal College of Pathologists Transfusion Update Meeting Autologous Transfusion 3 years on, Royal College of Physicians of Edinburgh

In addition, Dr Lorna Williamson has been invited to co-chair with Dr Luc Noel, France, a Working Party on Haemovigilance on behalf of the International Society of Blood Transfusion.

Publications

- Williamson LM, Love EM. Reporting Serious Hazards of Transfusion: The SHOT Program. Transfusion Medicine Reviews 1998;**12**(1):28-35.
- McClelland DBL, UK SHOT Project, Love EM, Scott S, Williamson LM. Haemovigilance: Concept, Europe and UK Initiatives. Vox Sanguinis 1998;**74**(suppl.2):431-439.
- Williamson LM. Systems contributing to the assurance of transfusion safety in the United Kingdom. Editorial. Vox Sanguinis. In press
- Williamson LM, Lowe S, Love EM, Cohen H, Soldan K, McClelland DBL, Skacel P, Barbara JAJ. The Serious Hazards of Transfusion (SHOT) Initiative – Results of the First Year's Reporting. Submitted to British Medical Journal.

5. OVERALL ORGANISATION AND REPORTING SYSTEM

Organisation

The strategic direction of SHOT comes from a Steering Group with wide representation from Royal Colleges and professional bodies representing medical, nursing and laboratory staff. A recent welcome addition is a representative from the Institute of Health Service Mangers. The operational aspects of the scheme are the responsibility of a Standing Working Group, which is accountable to the Steering Group. The Terms of Reference of the Steering and Standing Working Groups, along with the current membership, can be found in Appendix 1. Two national co-ordinators are responsible for receiving and collating reports.

Minutes of Steering Group meetings are sent to the Department of Health for information.

The first two years' funding has come from the Transfusion Services within the United Kingdom and Ireland. Generous grants from the British Blood Transfusion Society and British Society for Haematology are gratefully acknowledged. An income and expenditure statement is presented at Appendix 2. Organisational and funding arrangements will be formally reviewed during 1999.

SHOT was affiliated to the Royal College of Pathologists in November 1997.

Scope and Reporting System

Participation in the scheme is entirely voluntary. National Health Service and private hospitals in the United Kingdom and Republic of Ireland, as provided by the NEQAS blood group serology scheme, as well as public hospitals in Guernsey, Jersey and the Isle of Man are invited to report.

SHOT invites reports of major adverse events surrounding the transfusion of single or small pool blood components supplied by Transfusion Centres (red cells, platelets, fresh frozen plasma, cryoprecipitate). It does not cover complications of fractionated plasma products (coagulation factors, albumin, immunoglobulin); as licensed medicinal products, these are already covered by the 'Yellow Card' system of the Medicines Control Agency.

During the period covered by this report, hospitals have been asked to report the following categories of adverse event:-

1. incorrect blood component transfused
2. acute transfusion reaction (including anaphylaxis)
3. delayed transfusion reaction
4. transfusion-associated graft-versus-host-disease
5. transfusion-related acute lung injury
6. post-transfusion purpura
7. bacterial contamination
8. post transfusion viral infection
9. other post-transfusion infection e.g. malaria

Reporting of transfusion-transmitted infections

Suspected cases of transfusion-transmitted infection are reported, using local procedures, to supplying blood centres. Blood centre involvement is essential to ensure rapid withdrawal of other implicated components and appropriate donor follow-up. These cases are then reported by blood centres to the National Blood Authority/Public Health Laboratory Service Communicable Disease Surveillance Centre (NBA/PHLS CDSC) post-transfusion infection surveillance system. If the SHOT office is notified directly of an infectious hazard, the hospital haematologist and transfusion centre are approached by the co-ordinator to ensure that all relevant personnel have been informed and that the incident has been reported to NBA/PHLS CDSC.

Reporting of non-infectious adverse events

At hospital level, these are generally reported to the local clinician responsible for transfusion, usually a consultant haematologist. The incident is then notified to the SHOT office on the yellow 'initial report' form. For some complications, the local blood centre will have been involved in the investigation of the case. On receipt of a report, the assistant national co-ordinator allocates a number to the case, then issues a detailed follow-up questionnaire specifically designed for each hazard.

This enables confidential discussion of an incident between the SHOT office and the reporter if necessary. When incomplete information is received, the SHOT staff approach the local contact named on the report form. Once complete, the information in the questionnaire is entered in an anonymised way on to the SHOT database (see Fig 1).

The SHOT staff may offer to visit the reporting clinician, to assist with the completion of the questionnaire.

Confidentiality of data is fundamental to the success of the project.

Data are stored in a password-protected database in a secure location.

The help of the IT staff of the National Blood Service Northern Zone is gratefully acknowledged.

Once all the information has been gathered about an event and entered onto the database without patient, staff or hospital identifiers, all questionnaires, reporting forms and other paper records are shredded. SHOT does not provide details of individual cases, or any form of summarised data to any outside person or organisation, other than that provided in this report.

Limitations of the SHOT system

Reporting to the SHOT scheme is voluntary. We acknowledge that many incidents may go unrecognised or unreported, and that the reports analysed cannot provide a full picture of transfusion hazards.

The second year of reporting revealed further limitations in the questionnaires. These have been revised following consultation and after assessment of responses to the first report, the revisions have been adopted from 1 October 1998 for our third reporting year.

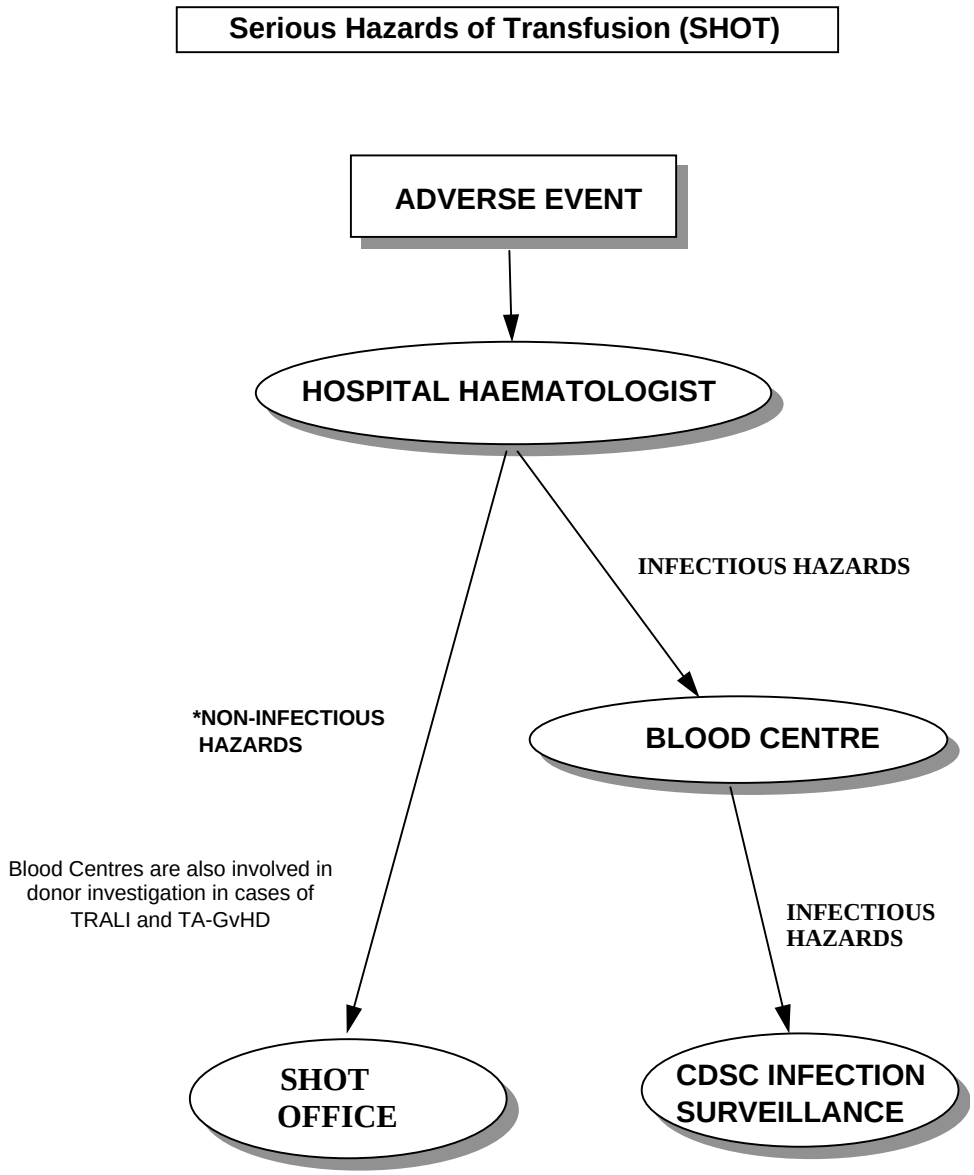
'Nil Returns' Card

Due to the anonymity of the scheme, denominator data from reporting hospitals was not provided in the first report. To ascertain the percentage of hospitals contributing to the SHOT reporting scheme this year, a nil returns card and covering letter (Appendix 4), was sent to the named consultant haematologist at all hospitals presently held on the SHOT mailing list (n = 424). The consultant haematologist was asked if he/she had reported any adverse events to SHOT during the period 01/10/97 to 30/09/98, or if no adverse events had been seen, to return the card as 'nothing to report'.

In an attempt to provide a denominator against which transfusion risk could be assessed, we also requested information on the number of red cell units transfused per annum and the number of units crossmatched per annum from hospitals sending either reports or 'nil return' cards. This card was also used to ask the hospital if it was interested in participating in a study of 'near miss' events (see Chapter 14). For this purpose a name and address was required, but all report cards were shredded as soon as the information was logged on to the database.

We intend to repeat this exercise quarterly to keep all hospitals informed of the latest initiatives in the SHOT reporting scheme and to prompt them to report any adverse events. The results of this exercise are detailed in Chapter 6.

Figure 1
SHOT reporting system flow chart



- Incorrect blood/component transfused
- Acute or delayed reaction
- Transfusion-related graft versus-host disease
- Transfusion related lung injury
- Post-transfusion purpura

6. OVERVIEW OF RESULTS

Ascertainment of data

The data in this report are derived solely from the initial report forms, and from subsequent analysis of questionnaires. All questionnaires were examined by the co-ordinators to identify inconsistencies in the information provided and, where these occurred, the reporting clinician was contacted for clarification of the event.

The SHOT reporting scheme for non-infectious complications of transfusion was launched on 18th November 1996. In the first SHOT annual report (1996-97) the incidents were reported by date of transfusion. The cases analysed occurred between 1st October 1996 and 30th September 1997 and were reported to the system by 31st December 1997. The report also included 14 incidents which occurred prior to October 1996, which were used to pilot the questionnaires.

After the launch of the first annual report it was evident that there would always be retrospective reporting and a delay in the return of completed questionnaires. In the SHOT 1997/98 report we have chosen to adopt the NBA/PHLS CDSC reporting system and to analyse data by date of initial report rather than by date of incident. The 1997/98 SHOT report therefore includes all initial report forms received between the 1st October 1997 and 30th September 1998. Due to the change in the presentation of data, the 1997/98 report includes 18 initial reports that were also included in the 1996/97 report. This 'double reporting' is unique to this report and will not be a problem in future years. To allow comparison with the previous report, incidence is based only on new cases received during the 1997-98 reporting year.

Overview of reports and 'nil return' cards received

Of the 424 hospitals registered in the SHOT scheme, 112 hospitals (26.4%) submitted initial report forms, compared with 94 (22.1%) during 1996-7. A further 164 hospitals (39%) sent a 'nil return', confirming their participation in SHOT and stating that they had had no adverse events to report during 1997-98. Eighty hospitals used the 'nil return' card to confirm that a report had previously been sent. Taking the 112 reporting hospitals with the 164 'nil returns', a total of 276/424 (65%) are now contributing to the SHOT initiative. Of the 244 nil returns card received, 151 hospitals expressed an interest in taking part in the 'near miss' project (see Chapter 14).

There were 197 new reports received during 1997-98, compared with 169 in the 1996-97 report, an increase of 16.5%. The largest section continues to be 'incorrect blood component transfused' with 110 new reports. The number of cases in each category is shown in Table 1, with the 1996-97 figures for comparison.

Table 1

Number of incidents reported in each category 1996/97 v 1997/98 **Reporting year 01/10/96 - 30/09/97 = date of transfusion;** **reporting year 01/10/97 - 30/9/98 = date of initial report received.**			
	1996/97	**1997/98 (total)**	**New cases 1997-98**
IBCT	81	121	110
ATR	27	30	28
DTR	27	27	24
PTP	11	13	11
TA-GVHD	4	4	4
TRALI	11	16	16
TTI	8	4	4
Total	**169**	**215**	**197**

IBCT: incorrect blood component transfused
ATR: acute transfusion reaction;
DTR: delayed transfusion reaction
PTP: post-transfusion purpura;
TA-GVHD: Transfusion associated graft-versus-host disease
TRALI: transfusion-related acute lung injury
TTI: transfusion transmitted infection.

Figure 2
Comparison of incidents reported in 1996/97and 1997/98.
Reporting year 01/10/96 to 30/09/97 = date of transfusion; reporting year 01/10/97 to 30/9/98 = date initial report received.

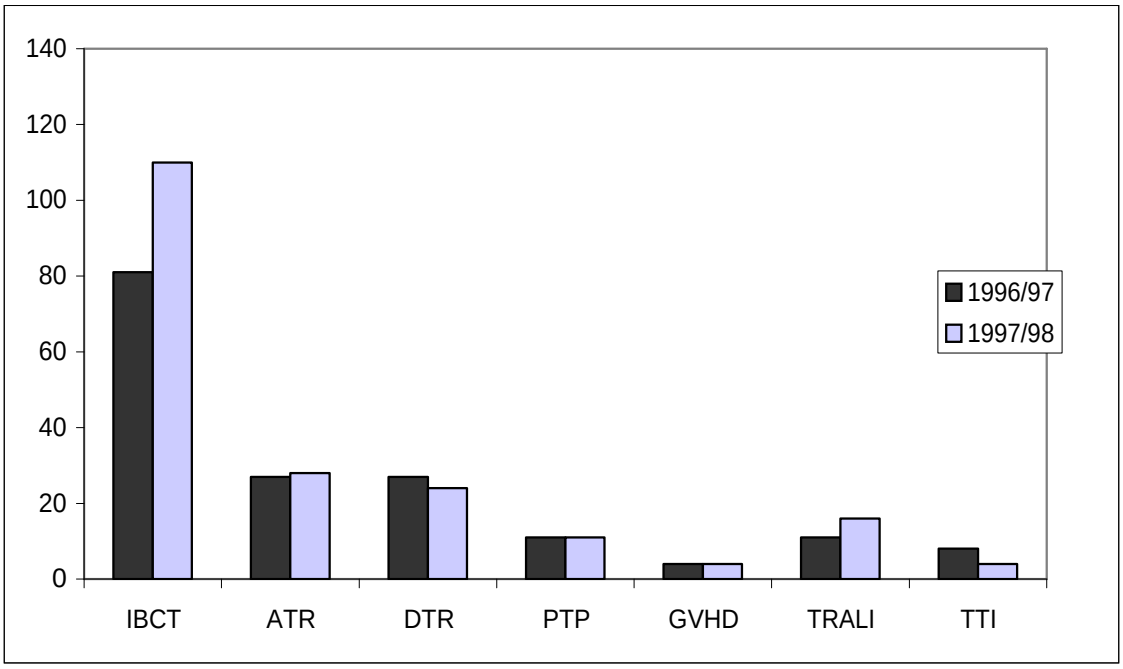
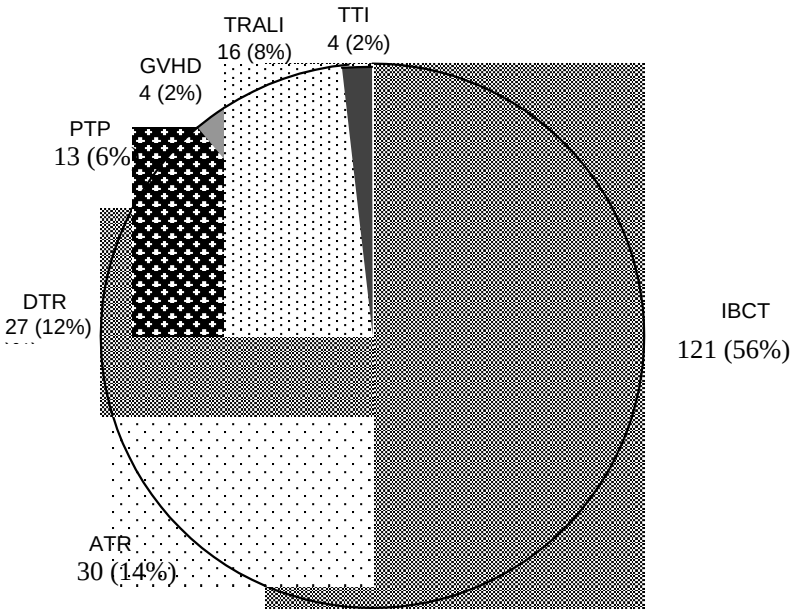


Figure 3

Overview of 215 cases for which initial report forms were received.



Analysis of questionnaires

A total of 199 questionnaires were analysed for this report, including 12 which were outstanding from initial reports received during 1996-97. There are a total of 20 new initial reports from 1997-98 from which questionnaires have not yet been received. These will be analysed in the next report.

Table 2

Summary of completed questionnaires received.

	IBCT	ATR	DTR	PTP	GVHD	TRALI	TTI	Totals
Total number of reports received	121	30	27	13	4	16	4	215
Questionnaires included in analysis	114*	26	27*	11	3	14	4	199
Questionnaires outstanding	9	4	2	2	1	2	0	20

* Includes two outstanding from 1996/97 report.

Figure 4

Overview of transfusion related mortality/morbidity data reported in completed questionnaires (n=199)

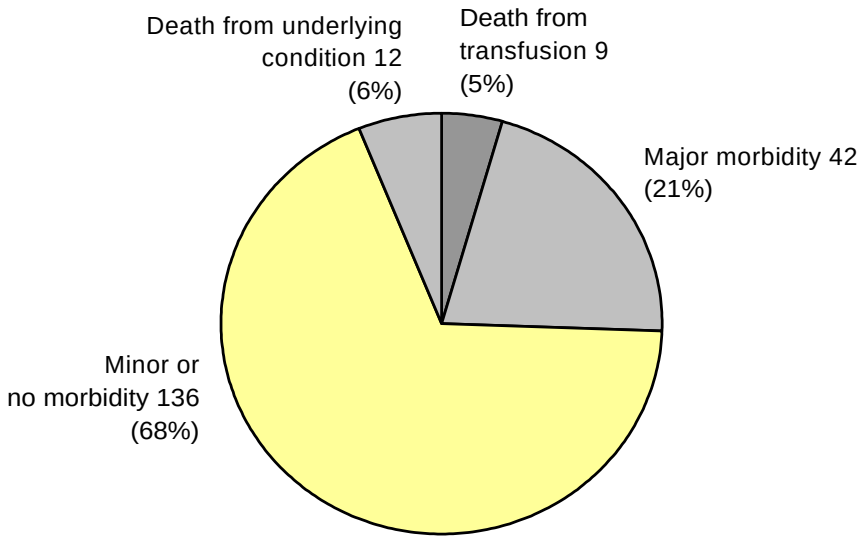


Table 3
Transfusion related mortality/morbidity according to the type of hazard reported (n=199)

	Total	**IBCT**	**ATR**	**DTR**	**PTP**	**TA-GVHD**	**TRALI**	**TTI**
Death attributed to transfusion	**9**	2	0	1	0	3	2	1
Major morbidity	**42**	20	1	4	2	0	12	3
Minor or no morbidity	**136**	88	19	21	8	0	0	0
Death due to underlying condition	**12**	4	6	1	1	0	0	0
Totals	**199**	114	26	27	11	3	14	4

Major morbidity was defined as the presence of one or more of the following:

◊ Intensive care admission and/or ventilation
◊ Dialysis and/or renal dysfunction
◊ Major haemorrhage
◊ Jaundice including intravascular haemolysis
◊ Persistent viral infection
◊ Acute symptomatic confirmed infection
◊ Potential Rhesus D sensitisation in a female of child-bearing age (or child)

Reporting delays

The following figures summarise the relationship between the time of transfusion and receipt of the initial report form and of the completed questionnaire. **These refer only to the 211 reports of non-infectious hazards reported to the SHOT office. For analysis of transfusion-transmitted infections, see Chapter 13.**

Figure 5
Calendar days between transfusion incident and initial report to SHOT (n=210)
Excludes three reports where the date of transfusion was not stated or known.

The median time for return of initial reports was 15 days, compared with 30 days in 1996-97.

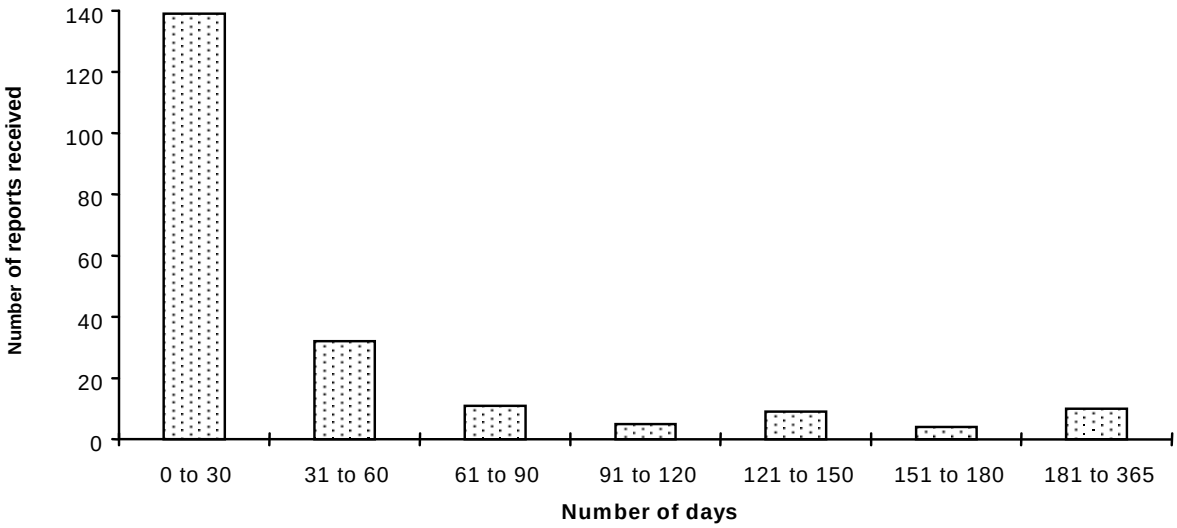


Figure 6
Calendar days between initial report and return of completed questionnaire (n=195)*
The median time between initial report and return of final questionnaire was 19 days, compared with 49 days in 1996-97. *Excludes 20 cases where a questionnaire has not yet been returned.

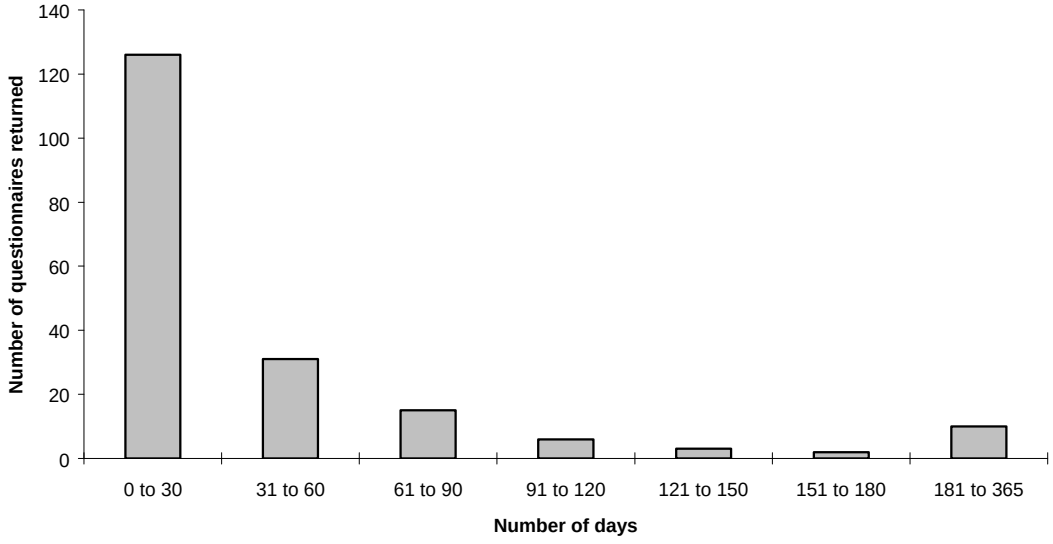


Table 4
Reasons for questionnaires outstanding

Pending - receipt expected	
Medical notes with consultant	3
Laboratory tests in progress	4
Recent report (< 30 days)	10
Case closed - questionnaire will not be submitted	
Clinician contacted 5 times to no avail	3
Total	**20**

All of the above departments were offered a visit by the SHOT staff to assist with completion of the questionnaire.

Of the 17 questionnaires outstanding from the 1996/97 report 5 cases were closed, and the remaining 12 are included in this year's analysis.

Overall transfusion activity and patient characteristics

In order to give some idea of the context in which hazard reports are taking place, Table 5 gives details of total blood component issues from the Transfusion Services in the UK and Ireland.

Table 5
Total issues of blood components from the Transfusion Services of the UK and Ireland in 1997 (to the nearest 1000)

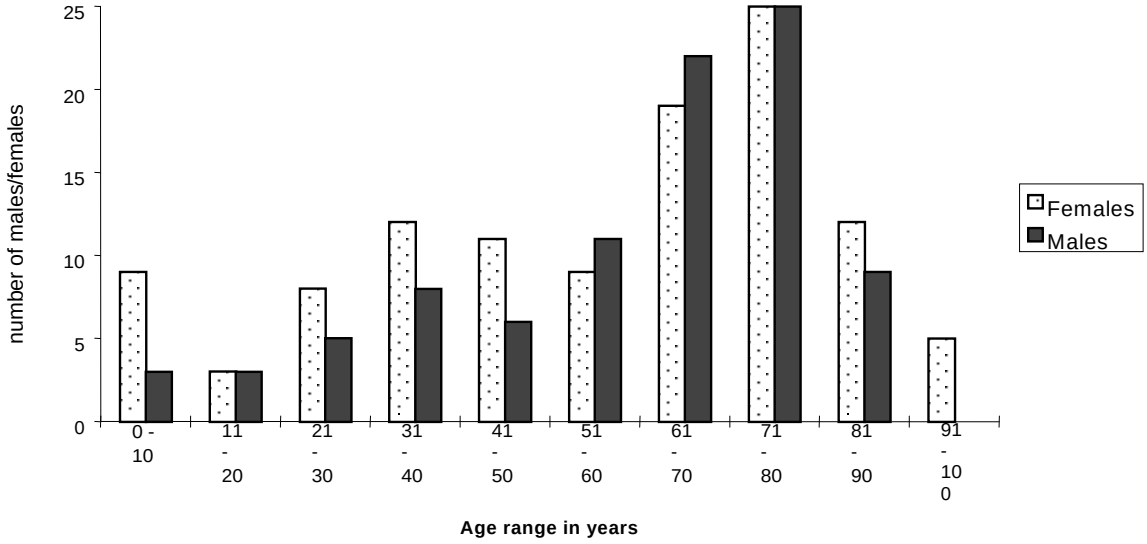
Red cells	2,750,000
Platelets	330,000
Fresh frozen plasma	438,000

Information was also sought, via the 'nil returns' cards, on the overall transfusion workload of individual hospitals contributing to the scheme.

Of 248 hospitals who replied, 226 gave data on number of red cell units issued, and 228 on number of units crossmatched. The returns received represent a total of 1,714,857 red cell units issued, and 2,725,609 units crossmatched. Thus these 248 hospitals (58% of those eligible) receive and handle 70% of all red cell units issued to hospitals.

Figure 7

Distribution of patients by age and sex at the time of transfusion (n= 204)*



Data excludes 7 cases where age was not stated.

	Males (n=96)	**Females (n=115)**
Age unknown	5	2
Age range	24 days - 90 years	2 days - 99 years
Median age	65 years	62 years

7. INCORRECT BLOOD COMPONENT TRANSFUSED

Definition.
This section describes all reported episodes where a patient was transfused with a blood component which did not meet the appropriate requirements or which was intended for another patient.

This category contained the highest number of reports, (110 of 197 new cases, 55.8%). This chapter analyses 114 questionnaires, consisting of 101 new cases, 11 cases included because of the change in report year, and two outstanding from initial reports received in the previous year.

In the fully reported cases, the majority of incidents involved either administration of a blood component intended for another patient (50 of 114, 44%) or laboratory errors (41 of 114, 36%). These incidents usually involved a series of mistakes and inadequate adherence to prevailing hospital documented policies and guidelines.

The data collated from all 114 questionnaires are presented in Appendix 9.

Sex of recipients	
Males	57
Females	67

Age of recipients	
Age range	2 days - 99 years
Median age	62 years

Components Implicated	Number of Cases
Red cells	98
Platelets	15
Fresh frozen plasma	9
Cryoprecipitate	2
*Anti-D immunoglobulin	3

* Adverse events to this plasma product are reported through the MCA yellow card system, but a decision has been taken to include these cases here, as they fall into the category of administration of a blood derivative to the wrong patient.

Table 6
Outcome of 114 incidents fully reported

Outcome	Number of incidents
Died of sequelae of transfusion	1
Died of sequelae of transfusion & underlying condition	1
Died of underlying condition	4
Recovered from complications of intra-vascular haemolysis	16
Survived with ill effects	4
Survived with no ill effects	88

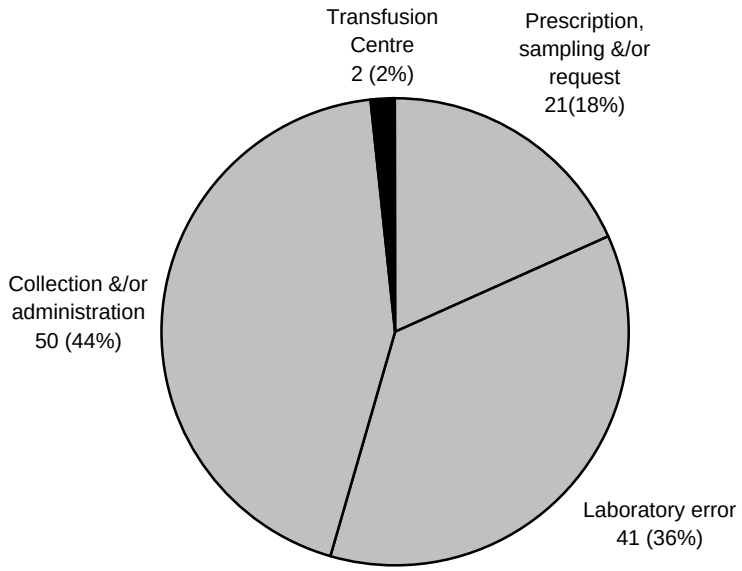
Analysis of reported errors

Where was the error reported to occur?

Errors fell into 4 categories	No. cases 1997-8	No. cases 1996-7
1. Prescription, request of component and/or obtaining the pre-transfusion blood sample	21	18
2. Laboratory errors - grouping, cross-matching or labelling	41	21
3. Collection from storage site and/or administration	50	34
4. Supplying Blood Centre	2	0

Figure 8

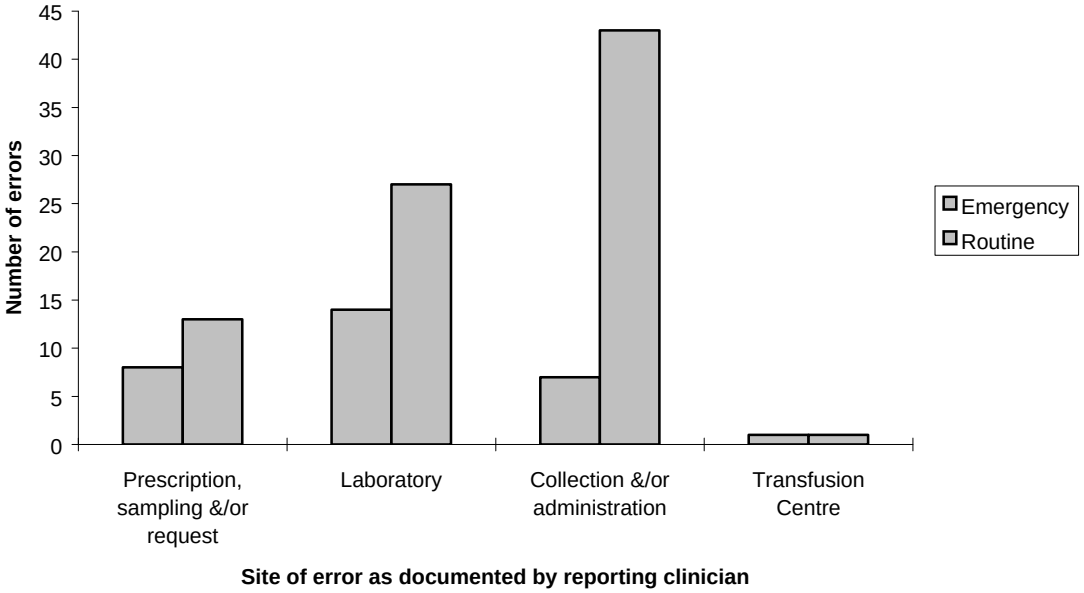
Distribution of errors as stated by the reported clinician



The questionnaire sought further information about the circumstances and the factors that may have contributed to these mistakes and adverse outcomes. The findings are presented in some detail, with the use of case studies where appropriate. The aim is to illustrate weak points in the process which have been identified by the reporting clinicians, in an attempt to help those responsible for training staff, or for the review and implementation of transfusion procedures, in order to identify areas for improvement.

Of the 114 complete reports, 84 errors related to routine non-emergency requests and 30 to emergency requests. Figure 9 shows the distribution of errors in routine and emergency transfusions.

Figure 9
Incidence of errors in the various stages of the process of emergency and routine transfusion.



Multiple errors contribute to many "wrong blood" incidents

Clinicians reported the particular error that had been recognised as the cause of the incorrect transfusion. However, closer analysis of the questionnaires revealed that in 31 (27%) of incidents the mistake had been preceded by other errors, such that in the 114 incidents fully reported a total of 159 procedural failures or omissions were identified.

Figure 10
Total number of errors per case (total cases =114; total errors = 159)

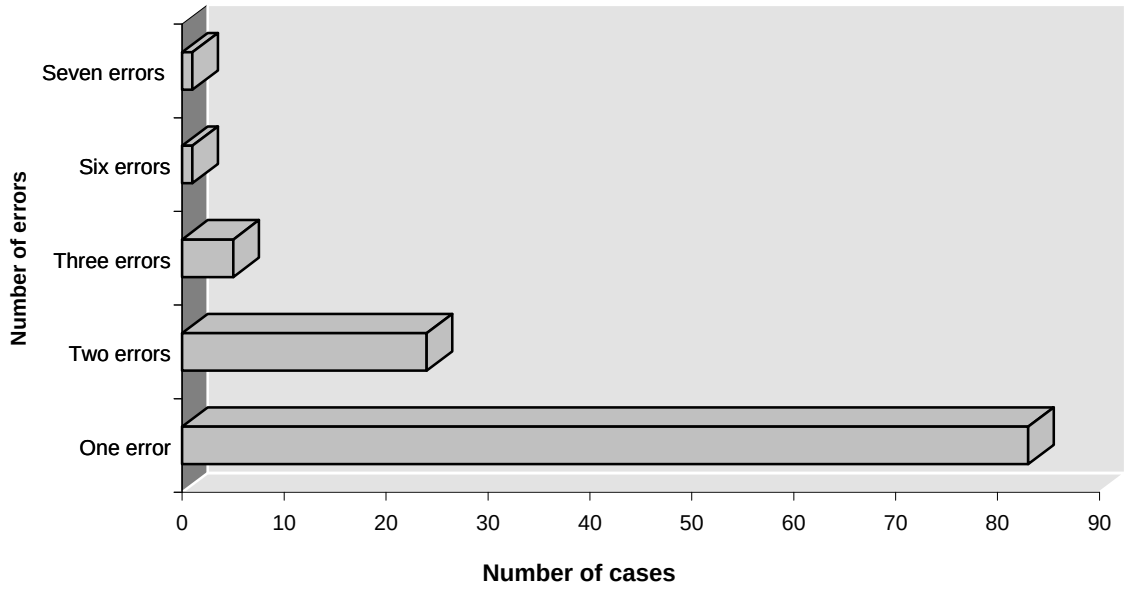


Table 7 illustrates the site of the initial procedural failure that was identified from analysis of the reports (column A), against the documented site of error as reported by the clinician (column B).

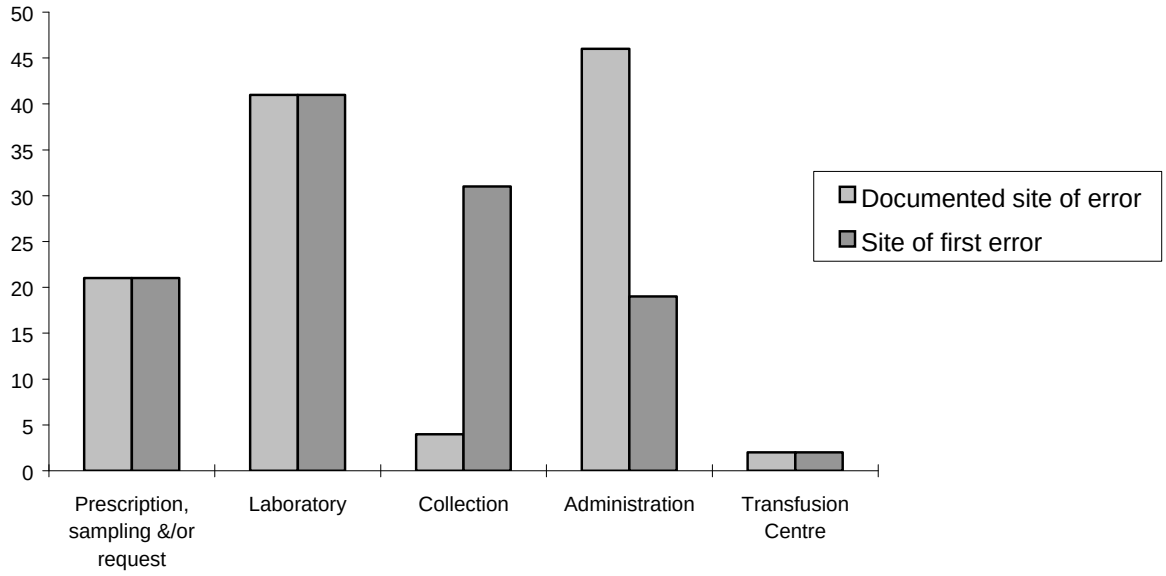
Table 7
Site of first error versus site of reported error (n=114)

Location	A Site of first error	B Documented site of error
Prescription, sampling and request		
1. Prescription of inappropriate &/or incompatible product by medical staff	3	3
2. Details on request form incorrect	7	7
3. Details on sample incorrect	8	8
4. Selection of incompatible products in emergency situations	3	3
Total	**21**	**21**
Blood bank laboratory		
1. Transposition of samples in laboratory	7	7
2. Historical group not checked	2	2
3. Blood incorrectly grouped	16	16
4. Blood incorrectly grouped & crossmatched	1	1
5. Component incorrectly labelled	7	7
6. Inappropriate component selected/issued	7	7
7. Clerical error	1	1
Total	**41**	**41**
Collection of component from hospital blood bank or other storage site		
1. Formal check for identity with patient omitted	15	1
2. Incorrect component collected	16	3
Total	**31**	**4**
Administration of product		
1. Component checked remote from the patient (eg at nurses station)	9	9
2. Misidentity of patient at time of administration	9	36
3. Formal identity check of product against patient omitted	1	1
Total	**19**	**46**
Transfusion Centre		
1. Unit of blood with haematocrit below specification	1	1
2. Unit of red cells wrongly genotyped	1	1
Total	**2**	**2**

In most hospitals the identity check of the component against the patient at the bedside is considered the final point in the checking procedure. Collection of an incorrect component was not identified as the key site of error by most reporting clinicians, as the onus of a correct component being transfused lies with the final bedside checking procedure. The questionnaire has been modified from 1st October 1998 to enable reporters to identify the site of first and subsequent errors.

Figure 11

Site of documented error which was recognised by the clinical team and reported to SHOT compared with site of first error.



The following analysis of 114 reports of wrong transfusions demonstrates a situation common to complex, multi-step processes, which involve many different individuals and which cross professional and managerial boundaries. Delivery of a reliable outcome constitutes a total quality management challenge, with the goal of ensuring that each person involved 'gets it right, first time, every time'.

1. Errors in prescription, requesting of blood, or patient sampling

Prescription errors

There were three cases where a clinician prescribed an inappropriate product. Two cases involved consultant anaesthetists selecting the wrong group of fresh frozen plasma from a theatre freezer due to incorrect serological reasoning (B RhD positive patient given O RhD positive FFP, and A RhD positive patient given O RhD positive FFP). In the third case a doctor knowingly took CMV seropositive platelets issued for one patient to use in an emergency on another patient who required CMV seronegative components. This in itself did not constitute an error, but the platelets were taken without any documentation or explanation to Blood Bank, contrary to the hospital's documented policies.

The request and supply of special components

There were 7 reports in which the correct component was not requested and/or issued. Four incidents involved the transfusion of non-irradiated components where irradiation was required. Two other cases occurred where CMV seronegative components were appropriate but untested components were provided. All of these errors occurred due to inadequate information being put on the request form, or poor communication between different specialities.

One patient was receiving fludarabine and therefore at risk of TA-GVHD. In another case the patient was awaiting autologous peripheral blood stem cell (PBSC) harvest. The PBSC harvest was performed after transfusion of the non-irradiated platelets, and when the error was discovered the procedure had be repeated.

One telephone request error was documented involving a request from theatre. The name and hospital number of the previous patient in theatre was given to the blood bank, resulting in an incorrect unit of FFP being issued and transfused.

Errors in sampling

There were 8 incidents where the sample for crossmatch had been taken from another patient. In 7 of these cases the patient had not been grouped previously, and in 1 case the patient had a previous transfusion history (Case Study 1). In 7 of the incidents it was not documented whether the sample tube had been pre-labelled, although this question was in the questionnaire. In one case pre-labelled tubes were used (Case Study 2).

Case Study 1: a double error which removed a safety net.

Patient A required a routine group and crossmatch for elective surgery. The doctor took a sample from patient A and labelled the sample tube with patient B's details. Patient B had been grouped previously and a historical group was sought in the laboratory using manual records. However, his previous transfusion history was not available in the current file and this was not pursued further. This resulted in a group O RhD positive patient being transfused 50 mls of group A RhD negative red cells before an acute transfusion reaction alerted staff to the error. The patient survived with no ill effects.

Case study 2: Two incompatible transfusions due to multiple errors

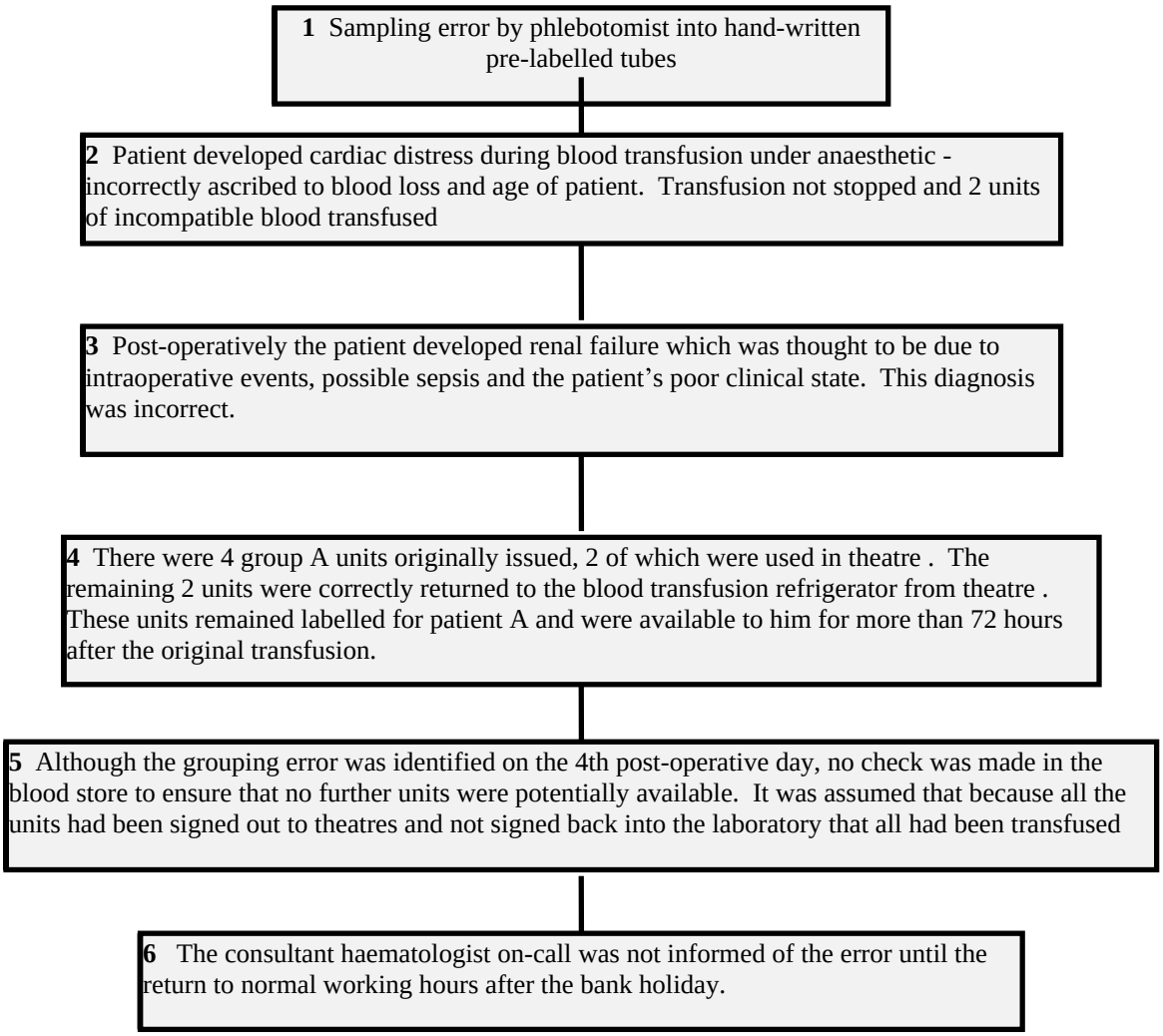
Patient A required routine group and crossmatch for elective surgery. The sample was taken by a phlebotomist into a pre-labelled hand-written tube with details of patient B (error 1). Patient A was bled and the sample put into patient B's tube, and vice versa. Neither patient had been grouped before. Patient A typed as blood group A RhD positive, and 4 units of group A RhD positive blood were crossmatched.

Patient A had his operation the following day. During the operation 2 units of blood were administered to replace intraoperative blood loss, and the patient was noted to have developed tachycardia, atrial fibrillation and moderate hypotension. This was ascribed to his age and relatively poor clinical status (error 2). On days 2, 3 and 4 post-operatively, patient A was unwell because of intermittent atrial fibrillation, the development of renal failure and mild jaundice. These changes were again ascribed to his surgery (error 3). On the 4th day post-operatively it was noted that he was anaemic with a haemoglobin of 7.6g/dL and another blood transfusion was ordered. The house surgeon re-bled the patient and sent a fresh crossmatch sample to the laboratory. The second sample was grouped as group O RhD positive and the transfusion IT system warned laboratory staff of an apparently discrepant result.

The house surgeon was contacted and sent a further sample which once again grouped as O RhD positive. Later that day a blood transfusion was set up. Half an hour into the transfusion the patient had a rigor. The transfusion was stopped and the house surgeon informed. The house surgeon noted that the blood was group A RhD positive, immediately disconnected the transfusion, and informed the transfusion laboratory. It was realised at this point that the previously issued group A RhD positive blood had not been removed from the blood bank (ie had been available to the patient for more than 72 hours after the original transfusion errors 4 & 5) and that this had been administered for a second time to the patient. It was recommended by the laboratory staff that the transfusion was continued with group O RhD positive blood which was administered without further evidence of reaction.

In conclusion, patient A received 3 units of incompatible blood. As a result of intravascular and extravascular haemolysis, he developed acute renal failure and cardiac problems which delayed his post-operative recovery. A further surprising aspect of this case was the delay in informing the responsible consultant haematologist of the error.

Flow chart of errors - Case-Study 2



2. Blood bank laboratory

Laboratory staff

Laboratory errors were not restricted to either inexperienced staff or to on call situations. Of the 41 laboratory errors reported (Figure 12), 25 incidents occurred during routine working hours. Twenty of these involved an experienced blood bank state registered MLSO and 1 an unsupervised MLA. Sixteen incidents occurred on-call, of which 7 involved regular blood bank staff, with the remaining 9 staff not regularly working in the blood bank.

Figure 12

Circumstances under which laboratory errors occur

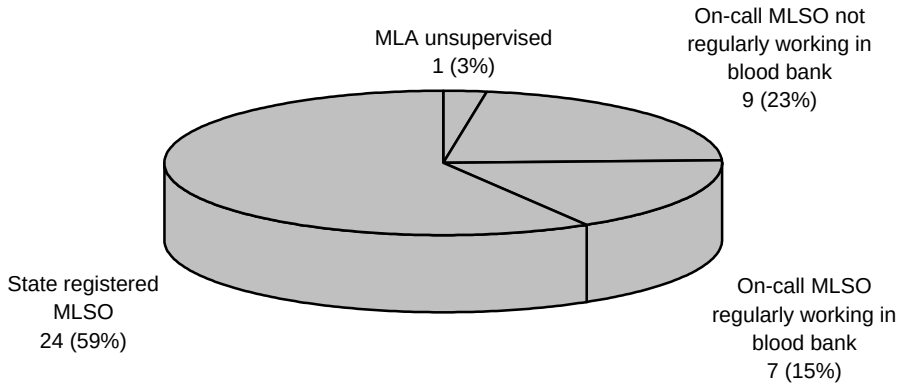


Table 8 details the grade of staff, type of error and whether the incident occurred during routine or on-call hours.

Table 8

Documented laboratory errors (n= 41)

Error	Total number of errors	State Registered MLSO routine hours regularly working in blood bank	State Registered MLSO on-call regularly working in blood bank	State Registered MLSO on-call not regularly working in blood bank	MLA unsupervised routine hours
A. Blood incorrectly grouped	**25**	14	5	6	
B. Blood incorrectly crossmatched	**1**		1		
C. Component incorrectly labelled	**7**	6	1		
D. Clerical error	**1**				1
E. Inappropriate component selected	**7**	4		3	
Totals	**41**	**24**	**7**	**9**	**1**

NOTES

A&B. Group and crossmatch errors (n=26)

- 7 errors were due to transposition of samples in the laboratory, 1 case resulting in the patients death.
- 16 errors in the performance of serological procedures, of which 1 was stated to be due to an exhausted MLSO at the end of a 24 hour on-call period.
- 1 instance of cross-matching error.
- In 2 cases the historical grouping record was not checked, which would have alerted the laboratory staff to the patient's antibody status.

Case study 3: errors in grouping and cross-matching

This was an obstetric emergency. The patient had been previously grouped as O RhD positive, the computer was in down time and therefore the historical group could not be checked (error 1). The patient was regrouped incorrectly as A RhD positive (error 2) and an immediate spin crossmatch failed to detect any incompatibility (error 3). Group A RhD positive units were issued and the Medical Laboratory Scientific Officer (MLSO) proceeded with a full crossmatch which revealed that the units issued were incompatible.

The labour ward was informed, by phone, to stop the transfusion immediately as the units issued were A RhD positive and the patient O RhD positive. There was a delay in the labour ward implementing the urgent message from the MLSO (error 4), by which time over 2 units had been transfused.

The patient was bleeding profusely, shocked and with disseminated intravascular coagulation. She required an emergency hysterectomy and 2 further laparotomies for control of bleeding. Under the circumstances the reporting clinician was unable to determine the contribution of the incompatible transfusion to the clinical picture.

C. Component incorrectly labelled (n=7)

- 2 errors - Red cells should have been irradiated, but although this was not performed, the laboratory paperwork indicated that it had been
- 1 error - label did not correspond with the unit number or the compatibility form
- 1 error - incompatible unit labelled and issued as compatible
- 1 error - laboratory label wrong with respect to donation number
- 1 error - 2 sample labels transposed in the laboratory, resulting in an RhD positive woman receiving anti- D immunoglobulin.
- 1 error- transposition of patient-specific compatibility labels

D. Clerical error (n=1)

- This related to a telephone request for FFP on a known patient who had been previously grouped and crossmatched (B RhD positive). The patient's historical record was checked, but the patient was misidentified as another due to the entry of an incorrect date of birth onto the computer. This culminated in the patient receiving a unit of A RhD positive FFP. The patient survived with no ill effects.

E. Inappropriate component selected/ issued (n=7)

- 1 error - inappropriate selection of component for patient with known antibodies
- 1 error - patient should have received leucocyte depleted blood, which was not issued
- 2 errors - where patients should have received irradiated products; in one case this was not communicated by the referring hospital
- 1 error - higher dose than required of anti D immunoglobulin was issued and given (2,500iu instead of 500iu)

- 1 error - Rh D positive platelets issued in error to an Rh D negative patient.

- 1 error - Patient grouped as B RhD negative. This group was not available and as red cells were required urgently, group O RhD negative red cells were issued. These stocks were then depleted so group O RhD positive red cells were issued. Then group O RhD positive fresh frozen plasma was issued and given in error.

3. Errors in withdrawal of blood components from storage location immediately prior to transfusion

As in the first report, withdrawal of an incorrect component from the storage location continues to be a substantial source of primary error, with 31 reported incidents.

In 2 cases the wrong component was handed over personally from blood bank staff to a porter, in 14 cases the wrong component was collected from a blood bank refrigerator and in 15 cases from a satellite refrigerator.

In 14 of these incidents the component was not checked for identity with the patient when it was taken from the refrigerator, and on 6 occasions a formal check had been performed but an incorrect component was still taken. In 11 cases, the collection details were not given.

In all these cases it appeared that the grade of staff checking the component did not influence whether a formal check was performed, nor whether the correct component was collected (Table 9). In 21 cases the component collected was incorrect with respect to name, date of birth and hospital number; in 5 cases it was incorrect with respect to date of birth and hospital number; in 1 case incorrect with respect to name and hospital number and in 1 case incorrect with respect to date of birth only. In 2 cases the completely incorrect type of component was collected.

Table 9
Formal check of component at the time of collection versus correct component collected: grades of staff involved (n=114)

Grade of staff	Formal identity check			Correct pack for patient		
	Yes	**No**	**Unknown**	**Yes**	**No**	**Unknown**
Qualified Nurse	19	5	17	33	8	
Unqualified Nurse	2	1	3	5	1	
Porter	19	11	13	30	13	
Theatre Staff	2		5	2	5	
*Other	3	2	1	3	3	
Unknown	2	1	8	9	1	1
Totals	**47**	**20**	**47**	**82**	**31**	**1**

* Other	Health care assistant	3
	Support worker	1
	Sent in taxi to SCBU	1
	Hospice driver	1

4. Administration of blood components - 'bedside' procedures

There were 50 reported cases where the final bedside check did not detect non-identity of the unit and patient. In most of these cases, two people were reported to have been involved in setting up and checking the transfusion. Table 10 shows the grade of staff setting up the transfusion in these cases.

Table 10
Grades of staff involved in setting up transfusions in which the bedside check was incomplete (n=50)*

Grade of staff	Number of cases
2 Doctors	2
Doctor & qualified nurse	2
Midwife only	1
Qualified nurse & qualified nurse	34
Qualified nurse & unqualified nurse	3
Qualified nurse & unknown	4
Doctor & unknown	2

*excludes 2 cases where the grade of staff was not reported

One explanation regularly stated for 'misidentity of patient at time of administration' (10 cases) was the practice of checking one or more component(s) remote from the patient, leading to transposition of components and compounded by omission of a final identity check at the bedside.

Case study 4 – the dangers of checking units away from the bedside

This incident occurred during a period of nursing night duty. Three patients were having blood transfusions on the same ward. One was in progress, while the other 2 patients were waiting for red blood cells to arrive from the blood bank. The red cells arrived for patient A. The senior state registered enrolled nurse (SREN) checked the component against patient A's notes, with the night sister at the nurses' station (error 1).

The night sister was bleeped by another ward and left the SREN to put up the transfusion (error 2, this hospital's nursing policy states 2 qualified nurses are required to put up and check a transfusion). The final patient identity check was not performed at the bedside resulting in patient A, (blood group O RhD positive), receiving group A RhD positive red blood cells (error 3).

When the SREN realised her error, she contacted the on-call locum and bleeped the night sister. When the locum arrived on the ward, he advised the nursing staff not to notify the on-call haematologist (error 4). Patient A received no investigations appropriate to an ABO incompatible transfusion (error 5). The on-call locum explained that '50mls of blood would not do any harm'(error 6). He then spigoted off the unit that had been partially given in error to patient A and reconnected it to patient B, the intended patient (error 7). Both patients survived with no ill effects.

Case Study 5 – fatal case of non-identity missed by bedside checking

In one case a health care assistant collected an incorrect component with respect to name, date of birth and hospital number, from a satellite refrigerator. The formal identity check at the bedside was not adequately performed resulting in a group O RhD positive patient receiving 2 units of group A RhD negative red cells.

The patient developed a fever, haemoglobinuria hypotension and cardiac problems which culminated in his admission to the intensive care unit. The patient died as a result of this incompatible transfusion.

Use of identity wristbands

In 12 incidents where an incorrect component was transfused, the patient had no identity wristband. Five cases occurred on the ward, 1 in theatre, 4 in out-patients and 2 in the accident and emergency department. In 2 cases group O Rh D positive patients received group A Rh D positive red cells and suffered the complications of intravascular haemolysis.

5. Transfusion centre errors

There were 2 documented transfusion centre errors.

- One was where a red cell unit was typed as Ss retrospectively, having been issued as homozygous. The transfusion centre notified the hospital blood bank by phone, by which time the unit had been transfused. The patient survived with no ill effects.

- The second case involved an exchange transfusion for neonatal jaundice. The laboratory staff noticed a falling MCV from 86 to 66 post exchange. This led to a discussion with the transfusion centre. The donor was recalled and found to have severe iron deficiency anaemia, with a haemoglobin of 7g/dL 1 week post donation. This donor should not have passed the copper sulphate donor screening test for anaemia. There were no adverse sequelae in the patient.

How was the error first recognised?

Of the 114 cases of an incorrect component transfused

- 11 were identified due to an acute transfusion reaction. Five of these were ABO incompatible transfusions (red cells); 3 ABO and RhD incompatible (red cells); in 2 cases the units were incompatible due to patient antibodies:- an O RhD positive Jk (a-b+) patient transfused O RhD positive Jka positive red cells, and an A RhD positive patient with anti-E transfused A RhD positive E positive red cells. In 1 case the blood groups were not stated.
- 38 incidents were detected by the ward staff.
- 51 incidents were detected by laboratory staff. One of these involved failure to detect anti Fy^a in a previously transfused patient admitted as an emergency with haematemesis. A Fy^a positive, and therefore incompatible, unit was supplied. Despite an acute reaction (hypotension and fever) the transfusion was continued and the patient went on to develop evidence of delayed haemolysis. Retrospective crossmatch easily detected the anti Fy^a and hence the cause of the delayed reaction.
- 7 errors were detected by theatre staff.
- 6 errors were identified by the patient or the patient's relative.
- 1 error was noted by the Transfusion Centre.

Where transfusion of the incorrect component was not associated with a reaction the error was detected in a variety of ways, for example:

- A patient, who had regular transfusions for a non-malignant haematological disorder as an out-patient, stated in clinic 2 months post transfusion, that he had been given a unit of group A RhD positive blood and that he felt this may have accounted for his symptoms, and for his admission to the Intensive Therapy Unit (Table 12, Case 52). The patient was group O RhD positive and in retrospect had probably suffered the complications of intravascular haemolysis due to this error.

- Two units of red cells were checked remotely from each of two patients and then transposed. An acute reaction in one of the patients alerted ward staff to their error and the transfusion was stopped on the second patient. The red cells and patients implicated were group O RhD positive and group B RhD negative; this error therefore exposed 1 patient to the risks of an ABO incompatible transfusion and the other to the risks of RhD sensitisation.
- In 1 case, when the patient required a second transfusion 4 days post-operatively the second grouping was different from the original group (Case Study 2).

Outcome

Of the 114 cases fully investigated, there were 41 ABO incompatible transfusions, 16 Rh incompatible transfusions, 5 ABO + Rh incompatible transfusions and 1 incompatible transfusion due to a missed anti Fya antibody (Tables 11 and 12) plus 6 instances where the blood groups of patient and/or unit was not stated.

- 1 patient died as a result of the transfusion. This was an O RhD positive patient who received 2 units of A positive red cells and required intensive care admission with cardiac problems (Case Study 5).

- 1 patient died as a result of an ABO incompatible transfusion combined with his underlying condition. The patient was group O RhD positive and received 4 units of A RhD positive red cells. The patient was admitted as an emergency with gastro-intestinal bleeding. He developed rigors, hypotension, renal failure and coagulopathy which combined with his underlying condition necessitated admission to the intensive care unit.

- 16 patients recovered fully or partially from the effects of intravascular haemolysis. Fourteen of these were ABO incompatible transfusions, 1 was due to an undetected Fy^a antibody at crossmatch, and 1 to an ABO and Rh incompatible transfusion.
  One of these patients, who recovered from intravascular haemolysis, required both ITU admission and dialysis and was discharged with renal failure. This was an 95-year old lady who had been admitted for a total hip replacement. The incorrect transfusion resulted in her no longer being able to live independently.
  Another patient who suffered the complications of intravascular haemolysis, had not been prescribed a transfusion. This patient was confused with a reduced conscious level at the time of the unintended and mismatched transfusion (O RhD positive patient given group A RhD negative red cells).

- Of the 16 patients receiving RhD incompatible transfusions, 3 were females aged 27 years, 5 years and 10 months respectively. It was not known at the time of reporting if these females had developed anti-D.

- There were 6 reports where the blood group was stated as unknown. In 1 of these the patient suffered from rigors and haemoglobinuria after only 50-100mls of red cells, and it is assumed that this transfusion was ABO incompatible. It is noteworthy that no investigations appear to have been performed on this case.

- Four patients were recorded as having died of their underlying condition. In one of these neither the blood group of the patient nor the incorrect component were stated. In another, the patient was paralysed and ventilated in the intensive care unit at the time of the incorrect transfusion (an ABO incompatible transfusion of 2 units of red cells due to laboratory grouping error).

Table 11

Sequelae of incorrect component transfused according to whether there was ABO and/or Rhesus incompatibility (n=108)* For further details please refer to Table 12.

Sequelae	Asymptomatic	Minor reaction	Major morbidity	Death
ABO incompatible	22	2	15	2
Rh incompatible	13	0	3	0
ABO + Rh incompatible	3	1	1	0
ABO + Rh compatible	45	0	1**	0
Totals	**83**	**3**	**20**	**2**

- excludes 6 cases where the blood group was not stated
- ** Fy^a incompatible

Major morbidity was classified as the presence of one or more of the following, attributed to the transfusion:

- Intensive care admission and/or ventilation
- Dialysis and/or renal dysfunction
- Major haemorrhage
- Jaundice including intravascular haemolysis
- Potential risk of RhD sensitisation in a female of child-bearing age (or child)

Minor reaction: The patient suffered symptoms/complications attributed to the transfusion but these did not require ITU admission or dialysis and the patient recovered rapidly.

Asymptomatic: No symptoms were directly attributed to the transfusion. Death due to the underlying condition or from other causes are included in this category (n=5)

Table 12
Sequelae of ABO and/or Rhesus incompatible transfusions, and an incompatible transfusion due to undetected Fy[a] antibody (n=63)

Patient ABO & Rh group	IBCT ABO & Rh group	Blood component	Volume IBT transfused	Symptoms/ complications	ITU ventilation &/or dialysis	Outcome
1. A neg	A pos	platelets - apheresis	1unit	potential Rh sensitisation female 27 years	none	survived with potential long term effects
2. B pos	A pos	FFP	1 unit	none	none	no ill effects
3. O pos	B pos	red cells	<50mls	none	none	no ill effects
4. A neg	O pos	red cells	3 units	none	none	no ill effects
5. A pos	AB pos	red cells	3 units	none	none	no ill effects
6. B pos	O pos	FFP	2 units	none	none	no ill effects
7. A pos	O pos	FFP	>100mls	haematological changes/ coagulopathy	already on ITU	no ill effects
8. O pos	A pos	red cells	50-100mls	loin pain hypotension haematological changes/ coagulopathy	none	intravascular haemolysis; recovered
9. O neg	O pos	red cells	1 unit	possible Rh sensitisation female child 5 years	none	survived with potential long term effects
10. A neg	A pos	red cells	50-100mls	none	none	no ill effects
11. O neg	AB pos	red cells	<50mls	fever	none	no ill effects
12. O neg	O pos	red cells	9 units	developed anti D male, 71 years	none	no ill effects
13. A pos	B neg	red cells	50-100mls	none	none	no ill effects
14. O neg	O pos	red cells	1 unit	none	none	no ill effects
15. O neg	A pos	red cells	2units	none	none	no ill effects
16. O pos	A pos	red cells	3 units	difficult to ascertain if any of the complications were due to the incorrect transfusion - patient shocked and bleeding profusely (case study 3)	ITU admission	intravascular haemolysis; recovered
17. O pos	A pos	red cells	<50 mls	none	none	no ill effects
18. AB pos	O pos	FFP	2 units	none	none	no ill effects
19. A pos	B pos	red cells	<50mls	fever hypotension	none	intravascular haemolysis; recovered
20. A pos strong Fya antibody	A pos	red cells unselected	2 units	fever hypotension post transfusion fall in Hb jaundice	already on ITU	intravascular haemolysis; recovered

Patient ABO & Rh group	IBCT ABO & Rh group	Blood component	Volume IBT transfused	Symptoms/ complications	ITU ventilation &/or dialysis	Outcome
21. O pos	A pos	red cells	<50mls	bronchospasm hypotension rigors fever	none	intravascular haemolysis; recovered
22. B pos	A pos	red cells	3 units	none	none	no ill effects
23. O neg	O pos	red cells	<50mls	none	none	died of underlying condition
24. A pos	B pos	red cells	2 units	difficult to ascertain if any of the complications were due to the incorrect transfusion - patient shocked and bleeding profusely	already on ITU	died of underlying condition
25. B neg	O pos	FFP	4 units	unknown	unknown	no ill effects
26. A neg	O pos	platelets - apheresis red cell pedipack	2 units 1 unit	possible Rh sensitisation female infant 10 months	unknown	survived with potential long term effects
27. O pos	A pos	red cells	1 unit	haemoglobinuria haematological changes/ coagulopathy	none	intravascular haemolysis; recovered
28. O pos	A pos	red cells	4 units	rigors hypotension haemoglobinuria haematological changes renal failure	ITU admission	died due to incompatible transfusion and underlying condition
29. O pos	A pos	red cells	<50mls	none	none	no ill effects
30. A neg	A pos	red cells	1 unit	none	none	no ill effects
31. O neg	O pos	red cells	2 units	none	already on ITU	no ill effects
32. O pos	B pos	red cells	<50mls	none	none	no ill effects
33. O pos	B pos	red cells	1 unit	none	none	no ill effects
34. O pos	A pos	red cells	<50mls	none	none	no ill effects
35. O pos	A neg	red cells	>100mls	hypotension haemoglobinuria haematological changes/ coagulopathy renal failure	none	intravascular haemolysis; recovered
36. O pos	A pos	red cells	2 units	haemoglobinuria	already on ITU	intravascular haemolysis; recovered
37. O pos	A pos	red cells	<50mls	none	none	no ill effects

Patient ABO & Rh group	IBCT ABO & Rh group	Blood component	Volume IBT transfused	Symptoms/ complications	ITU ventilation &/or dialysis	Outcome
38. O pos	A pos	red cells	>100mls	haemoglobinuria hypotension loin pain rigors fever haematological changes/ coagulopathy	none	intravascular haemolysis; recovered
39. O pos	A pos	red cells	2 units	hypotension atrial fibrillation cardiac problems renal failure electrolyte imbalance	none	intravascular haemolysis; recovered
40. B pos	O pos	FFP	>100mls	none	already on ITU	no ill effects
41. B pos	O pos	FFP	1 unit	none	none	no ill effects
42. A neg	A pos	red cells	1 unit	none	none	no ill effects
43. O pos	A pos	red cells	1unit	haemoglobinuria electrolyte imbalance fever haemoglobin-aemia hyper-bilirubinaemia	none	intravascular haemolysis; recovered
44. O pos	A pos	red cells	2 units	none	none	no ill effects
45. B neg	O pos	red cells	<50mls	none	none	no ill effects
46. O pos	B neg	red cells	<50mls	rigors fever	none	no ill effects
47. B neg	A pos	red cells	<50mls	fever rigors loin pain	none	intravascular haemolysis; recovered
48. O pos	A pos	red cells	1 unit	dark urine rigors haemoglobinuria ventilatory problems	none	intravascular haemolysis; recovered
49. O neg	O pos	red cells	2 units	none	none	no ill effects
50. A neg	A pos	red cells	2 units	none	none	no ill effects
51. O pos	A pos	red cells	3 units	poor increment in Hb post transfusion hyper-bilirubinaemia	none	no ill effects

Patient ABO & Rh group	IBCT ABO & Rh group	Blood component	Volume IBT transfused	Symptoms/ complications	ITU ventilation &/or dialysis	Outcome
52. O pos	A pos	red cells	1 unit	hypotension bronchospasm haemoglobin-uria fever, rigors, cardiac & ventilatory problems	ITU admission	intravascular haemolysis; recovered
53. O pos	A pos	red cells	2 units	fever haemoglobinuria hypotension cardiac problems	ITU admission	died of sequelae of transfusion
54. A neg	A pos	red cells	<50mls	none	none	no ill effects
55. A pos	O neg	FFP	1 unit	none	none	no ill effects
56. O pos	A neg	red cells	<50mls	none	none	no ill effects
57. O pos	A neg	red cells	>50mls	none	none	no ill effects
58. A neg	O pos	red cells	1 unit	none	none	no ill effects
59. AB neg	B pos	red cells	2 units	none	none	no ill effects
60. O pos	A pos	red cells	2 units	rigors fever	none	intravascular haemolysis, recovered
61. B neg	A neg	red cells	2 units	hypotension fever cardiac problems renal failure	ITU admission dialysis	intravascular haemolysis; recovered
62. A pos	B pos	red cells	1 unit	none	none	no ill effects
63. A pos	B pos	platelets, pooled	<50mls	none	none	no ill effects

PROCEDURAL REVIEW

Because of the anonymous nature of reporting, it has not been possible to analyse this data by number of hospitals. However, of 114 incidents analysed, 50 questionnaires stated that as a result of review of the incident locally, changes had been made. The commonest (30 cases) was review of or modification to existing procedures, with, in some cases, changes to written guidelines, protocols or standard operating procedures. Ten reports stated that there would be additional training for staff, and 7 said that entirely new systems (both manual and computerised) had been or would be introduced. Two incidents gave rise to a request for more staff, and one incident resulted in the suspension of a staff member.

Twenty nine incidents had been reviewed by the Hospital Transfusion Committee, and a further 56 such reviews were pending. For the remaining 29 hospitals, no local transfusion committee existed.

SUMMARY OF FINDINGS

1. Three prescription errors were reported, 2 of which were due to incorrect serological reasoning by consultant anaesthetists.

2. Seven request errors were noted, 6 involved the request and supply of 'special components', 1 involved a telephone request where incorrect information was given.

3. There were 8 cases where the crossmatch sample was taken from the wrong patient resulting in major morbidity in 1 patient. This incident involved the use of hand-written pre-labelled sample tubes (Case Study 2).

4. The historical transfusion record was not always checked prior to component issue (Case Study 1).

5. Errors in grouping, crossmatching, labelling and selection of a component were reported. Seven of the grouping errors were due to transposition of samples in the laboratory; one incident resulted in the patient's death.

6. The withdrawal of the wrong pack from its storage location, usually the hospital blood bank, continues to be an important source of primary error, with 31 such incidents reported. The grade of staff collecting a component ranged from qualified nurses to a support worker. In one incident the collection of an incorrect component culminated in the patients death (Case Study 5)

7. **The most important single cause contributing to incorrect transfusions was the lack of a formal identity check of the component with the patient at the bedside. There were 50 such cases, 1 incident resulting in the patient's death. One common explanation stated was the practice of checking one or more component(s) against the paperwork only, remote from the patient, eg at the nurse's station.**

8. Lack of patient hospital identity wristbands or other formal means of identification led to an incorrect component being transfused on 12 occasions. Two of these cases lead to complications of intravascular haemolysis.

9. In 1 reported case a component was given to a patient for whom blood transfusion had not been prescribed at all. The patient was confused with a reduced conscious level at the time of the unscheduled transfusion and suffered complications of intravascular haemolysis.

RECOMMENDATIONS

This year's recommendations are essentially the same as those in the SHOT 1996/97 report.

1. Selection and issue of components for transfusion should only be performed by staff specifically trained in serology.

2. Request systems for blood and components should ensure prescription, issue and administration of the correct component. These should cover 'special requirements' and telephone requests, and should clarify the respective responsibilities of medical and blood bank staff.

3. Pre-labelled sampling tubes should not be used.

4. Access to previous transfusion records in the laboratory containing grouping information should be available at all times and used as appropriate .

5. Blood banks should review procedures and systems including enforcement of the current guidelines and standards available, in addition to training to prevent errors of sample handling and technical errors.

6. Hospitals should review their current system to ensure that errors in the collection of blood from the blood bank can be prevented. Standards should be set for a minimal formal identification requirement when a component is collected. Novel identification systems are available, but have resource implications. However, these systems merit evaluation and development.

7. The bedside check is a vital step in preventing mis-transfusion. Staff should be vigilant in checking identification details of the component against those of the patient. Every hospital should have a policy for formally checking the blood component at the bedside. This is already stated in the Handbook of Transfusion Medicine[9], and is currently being addressed by the British Committee for Standards in Haematology (for the key points of the forthcoming BCSH Guideline on blood handling, see Appendix 8).

8. Hospital systems should ensure that in-patients and out-patients can be identified at the time of both sampling and transfusion, especially in out-patient departments where specific patient identification documents may not be available.

9. Blood components should always be administered against a written prescription.

8. ACUTE TRANSFUSION REACTIONS

Definition
Acute transfusion reactions were defined in this report as those occurring at any time up to 24 hours following a transfusion of blood or blood components excluding cases of acute reactions due to an incorrect component being transfused as these are covered in Chapter 7.

This category accounted for 14% of non-infectious hazards reported.

Thirty initial reports (28 new) were received and 26 completed questionnaires returned. Due to the change in reporting of cases by date when the initial report was received rather than the date of incident (for an explanation please refer to chapter 6) 2 cases reported in the 1996/97 Annual Report are also reported here. The data collated from the returned questionnaires are shown in Appendix 9.

This chapter highlights the main findings from the 26 completed questionnaires.

Overall there were 6 deaths in this group , 1 following FFP and 5 following platelets, but all deaths were from underlying causes and none were attributable to complications of transfusion.

Sex (30 reports)	
Males	17
Females	13

Age	
Age range	3 days - 92 years
Median age	65 years

Components implicated	No. of cases
Red cells (RBC)	13
Fresh frozen plasma (FFP)	8
Platelets	5

1. Reactions in which red cells were implicated

There were 13 cases and all survived without long term sequelae. Reactions could be broken down into 4 categories as follows:

Reaction type	Number of cases
Haemolytic	3
Non-haemolytic febrile	8
Hypotensive	1
Anaphylaxis	1

Haemolytic reactions and their clinical sequelae

There were 3 cases in this group as follows:

- A 77 year old female with known auto immune haemolytic anaemia, who suffered a febrile reaction and exacerbation of auto immune haemolysis during the transfusion.

- A 72 year old female with known cold haemagglutinin disease (CHAD), who suffered an exacerbation of haemolysis less that 2 hours after a transfusion of red cells filtered at the bedside. Complement activation as a result of fresh plasma transfusion or other mechanisms may exacerbate haemolysis in CHAD.
- A neonatal top-up transfusion of leucodepleted red cells was associated 8-24 hrs later with a rising bilirubin level and poor haemoglobin increment. No specific cause was found and the hospital queried whether the age of the red cells may have been implicated. British Committee for Standards in Haematology guidelines state red cells for small volume top-up transfusions may be used at any time throughout the approved shelf life[10].

Non-haemolytic febrile transfusion reactions (NHFTR)

SHOT does not specifically set out to gather data on uncomplicated NHFTRs as these generally are not classified as serious transfusion complications. However 8 reports fell into this category and all survived without sequelae. Reactions occurred during the transfusion in 4 cases, less than 2 hours after the end of transfusion in 2 cases and between 2 and 7 hours after in 2 cases. Following investigation, 4 patients were found to have leucocyte/HLA antibodies, in 2 patients no antibodies were found, and results were not available for the remaining 2 cases.

Hypotension

A 34 year old male bone marrow transplant donor suffered a hypotensive reaction during transfusion of autologous whole blood through a bedside leucodepletion filter. The reaction recurred after stopping and re-starting the transfusion. This case was referred to in the SHOT Annual Report 1996/97. Hypotensive reactions to platelets, associated with the use of negatively charged bedside filters and treatment with ACE (angiotensin converting enzyme) inhibitors as anti-hypertensive therapy is a recently recognised transfusion complication[11]. The patient reported here was not receiving treatment with ACE inhibitors. Recent reports of this condition have included patients receiving red cells, as in this case[12].

Anaphylaxis

An 86 year old female suffered hypotension, dyspnoea and fever during transfusion and was treated with adrenaline in addition to steroids and antihistamines. Investigation revealed antibodies to plasma proteins (anti Gm) and subsequent transfusion with washed red cells was well tolerated.

2. Reactions in which fresh frozen plasma (FFP) was implicated

There were 8 reports in this group with one death from the underlying condition unrelated to the transfusion reaction. Reactions, which all occurred during the transfusion, could be broadly broken down into 3 categories:

- Anaphylaxis/anaphylactoid
- Allergic (not anaphylaxis)
- IgA antibodies

Anaphylaxis/anaphylactoid reactions

There were 4 patients in this category and their reactions were characterised by the development of hypotension in association with a rash and/or pruritis with respiratory complications in 2 (dyspnoea in 1 patient and increasing ventilatory pressure in another, anaesthetised, patient). All cases were treated with steroids and antihistamines and 2 patients received adrenaline.

Allergic reactions (not anaphylaxis)

Three patients were deemed to have suffered allergic reactions. One patient with thrombotic thrombocytopenic purpura treated by plasma exchange developed a cough and restlessness and was diagnosed as having anaphylaxis. However, as there was no evidence of hypotension the authors considered this to be an allergic reaction, not anaphylaxis. A second patient developed a rash and pruritis. The third patient who received several units of FFP following an obstetric haemorrhage developed dyspnoea and swelling of her tongue.

All 3 patients were treated with steroids and antihistamines. In addition adrenaline and bronchodilators were given in the first case.

IgA antibodies

One patient developed fever and unspecified pain during FFP infusion and was found to be IgA deficient with antibodies to IgA. He was treated with steroids and antihistamines. He died from underlying pathology (bladder cancer).

Of the reactions to FFP only 3 were reported to the local Blood Centre although 6 were reported to the local Hospital Transfusion Committee. In general, investigations as to the causes of the reactions appeared lacking. It is concluded that such reactions could be better characterised and investigated since the symptoms encountered were in some cases severe, requiring treatment with adrenaline, a potentially hazardous therapy. Also it is conceivable that some of the respiratory reactions could have been a result of transfusion-related acute lung injury (see Chapter 10) but could not be attributed to this cause in the absence of appropriate investigations.

3. Reactions in which platelets were implicated

- There were 5 cases in this group, 3 of which occurred during the transfusion, one 2-7 hours after and one 24 hours after transfusion of platelets. All 5 patients died from underlying pathology. All cases were reported to the local Blood Centre and 4 were reported to the local Hospital Transfusion Committee.

- Results of investigations were generally lacking although in all but one case post-reaction blood samples had been taken.

- A 55 year old group A patient developed acute haemolysis 24 hours after receiving 2 units of group O apheresis platelets. Anti A was eluted from the red cells and was attributed to passive transfer. Although transfusion of platelets across the ABO barrier is permissible, BCSH guidelines state that if group O donors are used for group A, B or AB patients it is important to ensure that the donors do not have high titre anti A or anti B[13]. It is not known whether this guideline was applied in this case.

- A second patient suffered severe pruritis between 2 and 7 hours after transfusion of platelets through a bedside leucodepletion filter. The reaction was not investigated but loosely attributed to “cytokine

release". It was recommended that future platelet transfusions be washed or given through a neutrally-charged leucodepletion filter.

- The 3 reactions which occurred during transfusion of platelets could not be easily categorised. Two reactions were characterised by the development of chest pain. The first of these occurred in a 70 year old male with haematological malignancy who was given platelets via a bedside leucodepletion filter. Although no features of the previously described "platelet-filter interaction" [11] were seen, the future use of washed platelets or neutral-charge filters was recommended. The second patient, a 40 year old male also with haematological malignancy, in addition suffered lower limb pains, tachycardia, drowsiness and hypotension. The third reaction, in an acute surgical patient with disseminated intravascular coagulation, consisted of hypotension during the transfusion of unfiltered pooled platelets.

Response times

In general the medical officer informed of the reaction attended the patient promptly and took appropriate action. including contacting a haematologist where necessary.

Observations

Nursing observations showed quite wide variation, however every 15 minutes appeared to be the most popular interval (see Table 13)

Table 13

Frequency of nursing observations

Frequency of observations	**Number of cases**
5 minutes	1
10 minutes	1
15 minutes	8
30 minutes	2
60 minutes	3
Continuous (high dependency patients)	4
Nil	2
No information available	5
Total reporting	**26**

Reporting to Blood Centres and Hospital Transfusion Committees

This was highly variable, as can be seen from the following table:

Table 14

Reporting of reactions to local Blood Centre (BC) and Hospital Transfusion Committees (HTC)

Reported to:	**Number**
HTC only	3
BC only	11
HTC and BC	9
Neither	3
Total	**26**

Comments

- 8 reports of non-haemolytic febrile transfusion reaction, comprising the majority of reported reactions to red cells, were received. It was not the original intention of the SHOT scheme to seek data on reactions of this type which are not generally regarded as serious. However it is essential that clinicians feel able to report all reactions which they may consider serious.
- Hypotensive reactions have previously been described in patients who are being treated with angiotensin converting enzyme (ACE) inhibitors and who are transfused with platelets via a negatively-charged bedside leucodepletion filter. The reactions are attributed to activation of the kallikrein system and inability to break down bradykinin which is highly vaso-active[11]. Whilst the reactions described in the current report do not strictly fit this description, it is important that a mechanism exists to report all serious and unusual reactions. In this way, previously unrecognised complications of transfusion may be brought to light. This is particularly relevant given that from later this year all blood in the UK will be leucocyte depleted, and new unexpected symptoms might arise. This was seen in the USA, when a particular type of filter caused an unusual and severe iritis and visual impairment ('Red Eye syndrome') which came to light after sporadic reports were centrally collated by the Food and Drugs Administration.
- Reactions to FFP showed greater prominence in this report in comparison to 1997. These reactions, in common with some of those to platelets, were in general incompletely characterised and investigated.
- There was considerable variation in nursing observations. The forthcoming BCSH Guideline on blood handling will provide a recommended scheme (see Appendix 8).
- There continues to be variation in the investigation of acute transfusion reactions.

Recommendations

- Clinicians should continue to report all serious and unusual reactions as only in this way will previously unrecognised complications of transfusion, and particularly of novel components, be brought to light.
- Reactions to FFP could be better characterised and investigated since the symptoms encountered may be severe, requiring treatment with adrenaline, a potentially hazardous therapy. Some respiratory reactions attributed to FFP could represent transfusion-related acute lung injury but can only be attributed to this cause if appropriately investigated.

9. DELAYED TRANSFUSION REACTIONS

Definition

Delayed transfusion reactions were defined in this report as those occurring more than 24 hours following a transfusion of blood or blood components. In practice these are almost invariably delayed haemolytic reactions due to the development of red cell allo-antibodies

This category accounted for 13% of non-infectious hazards reported.

27 initial reports were received and 27 completed questionnaires were returned. Due to the change in reporting of cases by date when the initial report was received rather than the date of incident (for an explanation please refer to Chapter 6), three cases reported in the 1996/97 Annual Report are also reported here. The data retrieved from the returned questionnaires are shown in Appendix 9. This chapter highlights the main findings from the 27 completed questionnaires.

Sex	
Males	9
Females	18

Age	
Age range	27 - 91 years (1 unknown)
Median age	71

Components implicated	No. of cases
Red cells (rbc)	27

In all cases allogeneic red cells were implicated. The development of 46 newly detectable red cell allo-antibodies was recorded in 27 patients who suffered delayed haemolytic transfusion reactions (DHTR).

There were 2 deaths in this group of patients, 1 from underlying disease and 1 from the combined effects of underlying disease and transfusion complications.

Five patients were noted to have pre-transfusion red cell allo-antibodies and were assumed to have received red cells lacking the appropriate antigen. In 1 of these it cannot be ruled out that pre-existing anti Kna, an antibody not considered to be of clinical significance, may have masked the presence of other allo-antibodies in a multiply transfused patient.

In one patient, a pre-transfusion antibody screen was omitted and immediate-spin cross-match only performed, despite the fact that the transfusion was not an emergency. Multiple red cell allo-antibodies were detected post-transfusion.

In another patient an unspecified antibody to a low frequency antigen was apparently detected pre-transfusion and it was unclear from the records whether this was in fact the anti Jka detected post-transfusion and responsible for the DHTR.

Two patients were previously known to have clinically significant red cell antibodies some years earlier. However, these were not detectable immediately prior to the transfusion, were not disclosed by the patient and the information was not available in blood bank records to guide the selection of appropriate red cells.

In 20 patients the transfusion was said to be routine and in 7 urgent. At least 1 patient and possibly 2 were transfused for iron deficiency anaemia.

Table 15 shows the breakdown of new post-transfusion red cell allo-antibodies according to antigen specificity and Table 16 gives details of these antibodies for individual patients.

Table 15

New post-transfusion red cell allo-antibodies in 27 patients: according to antigen specificity

Antibody group	Number	Sole antibody
Kidd (Jk)		
Jka	17**	9
Jkb	1	
Duffy (Fy)		
Fya	4	2
Kell		
K	3*	
Rhesus		
D	2	2
C	2	
c	3*+	2
E	8+	1
e	1	
MNSs		
M$	1	
S	1	
Lutheran		
Lua	1	
Lewis		
Lea	1	
Weak cold agglutinin$	1	
Total	**46**	**16**

* 1 each previously known but not disclosed

+ 1 each enzyme-only in presence of multiple antibodies, not responsible for DHTR

** 2 possibly present pre-transfusion

$ Unlikely to be of clinical significance

Table 16

New post-transfusion red cell allo-antibodies in individual patients

ID	Antibody(ies)	Comment
1	K + Jka	Anti K known 10 years previously, not disclosed
2	Jka + E	? masked by pre-existing anti Kna
3	E + Jka + Fya	Antibody screen omitted pre-transfusion
4	Jka	? missed as antibody to "low frequency" antigen pre-transfusion
5	Jka	
6	Fya	
7	E + wk cold agglutinin	
8	E + Jka	
9	Jka	Hospital unable to detect post-transfusion (presumed found at reference centre)
10	Jka	
11	Jka	
12	C + E	Pre-transfusion anti S
13`	Jka	
14	D	Primary sensitisation to Rh D in an RhD negative male
15	E + Jka + Lua + Lea	Sequential development of antibodies
16	Jka (+ K later)	Responsible antibody = Jka
17	c	Known 1980, not disclosed
18	Jka	
19	Fya	Pre-transfusion anti K
20	Jka (+ M)	Responsible antibody = Jka. Pre-transfusion anti E + Fya
21	Jka + c + E	Enzyme-only anti c + E
22	Jka + K	
23	Jka	
24	E + Fya + Jkb	Pre-transfusion anti K
25	D	? Primary sensitisation to RhD in a RhD negative male
26	c (+ HLA)	
27	C + e + S	

Clinical sequelae

Symptoms and signs could be divided into 4 categories as follows:

- **Group 1** Asymptomatic (± positive direct antiglobulin test (DAT) ± spherocytes)
- **Group 2** Falling haemoglobin (↓Hb) / positive DAT/ spherocytes (2 of these parameters)
- **Group 3** ↓Hb + jaundice ± positive DAT ± spherocytes
- **Group 4** As group 3 + renal impairment

Group 1
There were 3 patients in this group (cases 1, 19, 22). All survived without sequelae.

Group 2
There were also 3 patients in this group (cases 6, 8, 26) and again all survived without sequelae.

Group 3
There were 17 patients in this group (cases 2, 3, 4, 7, 10, 11, 13, 15, 16, 17, 18, 20, 21, 23, 24, 25, 27) of whom 15 survived without sequelae, 1 died from underlying causes and the outcome in 1 was not stated.

Group 4
There were 4 cases in this group (cases 5, 9, 12, 14) of whom 2 survived with renal failure, 1 died from the combined effects of underlying disease and DHTR and 1 survived without sequelae.

The above results are detailed in Table 17.

Table 17 Grouping of cases by clinical sequelae of DHTR

Group 1		Group 2		Group 3		Group 4	
ID	**Antibody**	**ID**	**Antibody**	**ID**	**Antibody**	**ID**	**Antibody**
1	K + Jka	6	Fya	2	Jka + E	5+	Jka
19	Fya	8	E + Jka	3	E+Jka+Fya	9$	Jka
22	Jka+K	26	c	4	Jka	12$	C+E
				7	E	14	D
				10	Jka		
				11	Jka		
				13	Jka		
				15	E+Jka+Lua+Lea		
				16*	Jka (+K)		
				17	c		
				18	Jka		
				20	Jka (+M)		
				21	Jka (+c+E)		
				23	Jka		
				24	E+Fya+Jkb		
				25	D		
				27	C+e+S		

* Died of underlying illness
+ Died of combined effects of underlying illness and DHTR
$ Survived with renal failure

Analysis of serological information

Limited information could be obtained from analysing the questions on serological methods and in some cases the questions were incompletely answered. The limitations of the current questionnaire have been recognised and the design of serological questions has been revised for the 1998/99 reporting year. Although it is not the intention of SHOT questionnaires to attempt to "police" the methodology used, and whilst no direct links can be established between NEQAS and SHOT data, it is hoped that by improving the quality of the questionnaire design, useful information can be obtained which will complement that obtained by NEQAS exercises.

Antibody screening

Table 18 gives information on the serological methods used for antibody screening in 22 of the 27 reported cases. The data is incomplete for the remaining 5 cases.

Table18
Summary of serological methods used for antibody screening

Screening Method	2 cell screen	3 cell screen	Total
Tube	3	6	9
Column	3	7	10
Tube and column		2	2
Microtitre		1	1
Total	**6**	**16**	**22**

Details of some of the antibody investigations were as follows:

Case 1 Historical anti K not detected pre-transfusion but detected by the same methodology post-transfusion.

Case 2: Anti Kna pre-transfusion could have masked pre-transfusion anti E+Jka in a multi-transfused patient (not shown in above matrix). The anti Kna had been detected previously by the Blood Centre and least incompatible units issued with no ill effects. It is presumed that this practice continued until a change in the strength of some serological reactions alerted the hospital to the possibility of additional red cell allo-antibodies.

Case 9: Anti Jka not detected by hospital post-transfusion despite positive DAT. The case was referred to the local Blood Centre which presumably detected and identified the antibody.

Case 17: Historical anti c not detected pre-transfusion but detected post-transfusion by the same method.

Without knowing more about the panels used it is not possible to draw any conclusions on the adequacy of the methodology.

Cross-matching

Interval between sampling and cross-matching

The interval between sampling and cross-matching is shown below for the 27 reports.

Interval between cross-matching and sampling (hrs)	No. of cases
0-47	22
48-71	2
72-96	1
> 96	0
Not known	2

There appears to be adherence to British Committee for Standards in Haematology guidelines[14]. However since the questionnaire did not ask about previous transfusion history this conclusion cannot be verified. This inadequacy in the questionnaire has subsequently been corrected for the 1998/99 reporting year.

Cross-matching methodology

No useful information could be elicited due to the current design of this section of the questionnaire. The format has been modified for the 1998/99 reporting year.

Reporting to Blood Centres and Hospital Transfusion Committees

20/27 (74%) cases were reported to local Blood Centres whereas only 12/27 (44%) were reported to Hospital Transfusion Committees. The design of the questionnaire was such that it was not possible to know whether the latter figure represents lack of reporting per se or lack of a Hospital Transfusion Committee. This question has been re-designed for the 1997/98 reporting year.

Comments

- There was little evidence of poor laboratory practice. In the majority of cases DHTRs occurred as a result of the development of new red cell allo-antibodies and could not have been prevented, as the antibodies were undetectable at the time of the original antibody investigation in previously sensitised patients. Exceptions to this were 1 case where the pre-transfusion antibody screen was omitted and limited cross-matching performed, one case of possible mis-identification of an antibody to a low frequency antigen and 1 case where the hospital failed to detect anti Jka post-transfusion despite a positive DAT. In the first case the hospital took immediate steps to review procedures and instigate re-training.

- The antibody specificities encountered as causes of DHTRs were as expected from the literature[15] and showed a preponderance of anti Jka (17/46 or 37% of all antibodies, 17/27 or 63% of patients).

- The onset of DHTRs ranged from 1 to 28 days (median 7 days). A delay of 28 days is unusual for DHTRs which are normally the result of re-stimulation to an antigen to which the patient was previously sensitised. In this case the antibody was a result of primary sensitisation to Rh D in a RhD negative male who received a massive transfusion of RhD positive blood, a well accepted practice under defined circumstances.

- In 2 cases historical clinically significant antibodies were undetectable pre-transfusion, were not disclosed by the patient and the previous records were not available to guide the selection of suitable blood.

- Only 44% of cases were reported to Hospital Transfusion Committees.

Recommendations

- Access to previous transfusion records may alert to historical clinically significant antibodies which are undetectable at the time of cross-match

- Careful questioning of patients regarding previous transfusion and the possible existence of patient antibody cards should be stressed

- There should be greater utilisation of Hospital Transfusion Committees as a forum for discussion of such cases.

10. TRANSFUSION-RELATED ACUTE LUNG INJURY

Definition
Transfusion-related acute lung injury was defined as acute dyspnoea with hypoxia and bilateral pulmonary infiltrates occurring during or the 24 hours after transfusion, with no other apparent cause.

Sixteen initial reports were received, of which 14 questionnaires were returned in time for inclusion in this year's annual report. These numbers take into account 2 sets of duplicate reports, where data on the same patient was sent independently from both the clinician and the Blood Centre investigating the case. The 14 analysed cases involved 6 females and 8 males, with a median age of 60 years (range 5-82). Five patients were under 30 years , including 2 children aged 5 and 12 years. Collated responses to questionnaires are reported in Appendix 9.

There were 2 fatalities in which TRALI was at least partly implicated. Of the 12 others, 10 fully recovered from the episode (although 1 died later from other causes), 1 recovered with impaired respiratory function, and in 1 case the outcome was unstated.

The first fatality (Table 19, Case 2) involved a child of 12 years, who had undergone unrelated bone marrow transplantation for chronic myeloid leukaemia. At the time of transfusion the child had persistent fever unresponsive to antibiotics, poor respiratory function, and was already being treated with methyl prednisolone. Platelets were transfused to cover removal of a central venous line, following which the child became increasingly breathless, with hypotension and reduced pO_2. Chest X ray revealed widespread diffuse alveolar shadowing, so ventilation was commenced, but the child subsequently died. Serology of the donors was reported by the clinician as negative, although the relevant laboratories have no record of the case.

The second fatality (Table 19, case 14) occurred in a man of 82 years with underlying chronic obstructive airways disease and hypertension, who was transfused with platelets and red cells because of underlying myelodysplasia. He developed dyspnoea, originally thought to be cardiac in origin, but cardiac investigations revealed good left ventricular function. He was treated with diuretics, but his renal function deteriorated, and he died several days later of renal failure. Serology on the platelet donor was negative, but the red cell donors were not investigated.

GLOSSARY OF TERMS FOR TABLE 19

HLA **Histocompatibility locus associated**

MAIPA **Monoclonal antibody immobilisation of platelet antigen (test)**

LCT **Lymphocytotoxicity (test)**

ELISA **Enzyme linked immunoabsorbent assay**

LIFT **Lymphocyte immunofluorescence (test)**

GIFT **Granulocyte immunofluorescence (test)**

Table 19
Leucocyte antibody investigations in 14 TRALI cases

No.	Patient	Donors			Results on positive donors
		Number tested	Number negative	Number positive	
1	Multispecific HLA	3	2	1 (?)	Inconsistent results.
2	Appropriate investigations not done				-
3	Anti-lymphocyte IgG (postnatal)	23	21	4	4 pos crossmatch; 2/4 HLA Class I; both female donors of FFP/plts.
4	Appropriate investigations not done				-
5	HLA type A1,2: B8,18. HLA Class I antibodies 12% (incl A33)	2	1	1	Antibodies to HLA B15, 21 and some cells expressing B52, B53, B18.
6	Neg pre-transfusion; identical results to donor following transfusion	5	4	1	Multiparous female – IgG antibodies reacting with lymphocytes and granulocytes (LCT/MAIPA neg - ? specificity)
7	HLA antibodies neg	No data			-
8	Serology neg	11	10	1	Antibodies to lymphocytes and granulocytes by ELISA.
9	Serology neg	6	5	1	Positive in LIFT and crossmatch with patient.
10	Serology neg	3	2	1	Positive in GIFT and crossmatch with patient
11	Anti-HLA A2 + A28 (68)	2	2	0	-
12	Serology neg	2	1	1	Antibodies to granulocytes/HLA Class I
13	Serology neg	28	22	6	Granulocytes antibodies in 6 plus HLA Class I antibodies in 1.
14	Not tested	1	1	0	Platelet donor neg; red cell donors not tested.

Comments

- The investigation and diagnosis of TRALI can be difficult. Incomplete investigations were a feature of many cases reported here, with donors of transfused components either not investigated at all, or only partially. All cases analysed here met the case definition above. However, some authors include positive donor serology in the case definition; this was confirmed in 9/14 cases analysed here.

- The most common underlying diagnosis was haematological malignancy (6 patients), followed by emergency surgery/haemorrhage (4 patients), elective surgery with abnormal coagulation (3 patients) and warfarin reversal (1 patient). Five patients had underlying respiratory disease, 3 had underlying cardiac disease and 2 had sepsis. One case turned out also to have constrictive pericarditis. These pre-existing features may have rendered the patient more vulnerable to modest degrees of respiratory impairment.

- Six patients received red cells alone, 2 received platelets alone, and 1 received FFP alone. The other 6 patients were transfused with multiple components, including cryoprecipitate in 3. On the basis of timing of symptoms, the component implicated in the episode of TRALI was red cells in 7 cases, platelets in 3, FFP in 2, and cryoprecipitate in 1 (unclear in 1). The source of the platelets was apheresis in 2 cases, and pooled platelets in 1 case.

- Cases were generally reported because of respiratory deterioration (dyspnoea and reduced pO_2) during (7 cases) or soon after transfusion (7 cases). This was accompanied by fever in 4 cases, and hypotension in 5. Chest X ray in 11 cases showed appearances of diffuse alveolar shadowing or were reported as pulmonary oedema.

- Nine patients were admitted to ITU, of whom 8 were ventilated for periods of 1-10 days. Two patients was already on ITU when symptoms began. Specific treatments were steroids, usually in the form of dexamethasone, in 7 patients (2 further patients were already receiving methyl prednisolone), an anti-histamine in 5, and intravenous immunoglobulin and diuretics in 1 patient each.

- Of the non-fatal cases, 1 died later of the underlying condition, 9 made a full recovery, and 1 recovered with impaired respiratory function (not stated in 1).

Recommendations

- Many cases are reported to SHOT promptly after the event - this is to be welcomed, but means that serological investigation of the donors, which is relevant to the diagnosis of TRALI, is not yet available. Such investigations require liaison with the Blood Centre which supplied the component. It is therefore suggested that Transfusion Services ensure that mechanisms are in place for local collation of laboratory investigations on each case, for forwarding to SHOT when complete. This would also minimise duplicate reporting.

- Interpretation of results would be aided by a national protocol for the investigation of suspected cases of TRALI.

- Prevention of TRALI would require additional selection and/or testing of blood donors, particularly where plasma-rich components (platelet concentrates and FFP) are to be prepared. Such a strategy needs to be considered against other priorities for improvement of blood safety, and the possible impact on donor availability.

11. POST-TRANSFUSION PURPURA

Definition:
Post-transfusion purpura was defined as thrombocytopenia arising 5-12 days following transfusion of red cells associated with the presence in the patient of antibodies directed against the HPA (human platelet antigen) systems.

Of 13 reports received between 1/10/97 and 30/9/98, 2 had been analysed as part of last year's report, but are included here as part of the change of reporting year. Two questionnaires which were received after 1/10/98 will be included in next year's report. The 9 new reported cases thus do not represent a major change from last year's figures (11 reports and questionnaires). Collated responses to questionnaires are reported in Appendix 9.

There were no fatalities attributed to PTP, but 2 cases died of their underlying condition. Neither met the case definition as described above, and could be considered as only possible cases of PTP. In the first case, thrombocytopenia (20-49 x 10^9/L) was an incidental finding 5-9 days following transfusion. No platelet alloantibodies were detectable, and the patient (aged 74) died from underlying causes (fractured neck of femur). The second case, profound thrombocytopenia (< 10 x 10^9 /L) developed 5-9 days after a red cell transfusion given as part of supportive care for a chronic haematological malignancy in an 89-year old; again, no platelet alloantibodies were detectable, using a commercial antibody detection kit (GTI-Pack).

Of the 11 cases analysed for this report, all were female, with a median age of 61 years (range 39-89).

Comments

- All cases had had previous pregnancies, but none <5 years prior to the implicated transfusion. There was no definite history of neonatal thrombocytopenia in any case, but 1 woman had had both an intrauterine and a neonatal death, the latter having congenital anomalies.

- 4 cases had been transfused, at times ranging from several months to 20 years prior to the transfusion.

- All cases followed red cell transfusion (in 1 case of buffy coat-depleted red cells), given in association with elective (3 cases) or emergency surgery (3 cases). As noted in last year's report, 4 patients had pre-existing gastro-intestinal haemorrhage. In only 2 cases was there fever associated with the transfusion.

- In all cases except 1 (< 5 days), thrombocytopenia was noted 5-15 days following transfusion. This was an incidental finding in 2 cases (platelet nadirs 58 and '20-40' x 10^9/L), but was accompanied by purpura and/or minor haemorrhage in 7 cases and major gastrointestinal/vaginal haemorrhage in the remaining two cases. In all symptomatic cases except 1, the nadir of platelet count was <10 x 10^9/L.

- In 8/9 serologically positive cases, the platelet alloantibody was of HPA-1a specificity (also with an auto-antibody to platelet glycoprotein Ia/IIa in 1 case). In the remaining case, the antibody specificity was HPA-1b in a patient genotyped as HPA-1a homozygous.

- All symptomatic cases were treated with intra-venous immunoglobulin, accompanied by random platelets in 4 cases, HPA-1a negative platelets in 2 cases and steroids in 3 cases.

- One asymptomatic case recovered spontaneously; 5 cases had recovered to a platelet count of > 50 x 10^9/L by day 3; 3 further cases took 12-14 days to recovery.

Recommendations

- All cases reported were investigated and managed promptly and appropriately with intravenous immunoglobulin. The place of steroids in the management of PTP is uncertain, and on the evidence from this and last year's report (a total of 18 cases) does not lead to more rapid recovery of platelet count, and cannot be recommended for routine use. The only 2 cases treated with steroids in this report had major haemorrhage; whether this was related to the steroids is uncertain.

12. TRANSFUSION-ASSOCIATED GRAFT-VERSUS-HOST DISEASE

Definition
Transfusion-associated graft-versus-host-disease was defined as the development of the classical symptoms of fever, rash, liver dysfunction, diarrhoea and pancytopenia occurring 1-6 weeks following transfusion, without other apparent cause. The diagnosis was usually supported by skin/bone marrow biopsy appearances and/or the presence of circulating donor lymphocytes.

Four cases were reported, the same number as last year, although one case reported during 1997-8 had been transfused in 1996. All 4 were male, aged between 60 and 72, and all 4 cases died of TA-GVHD. Questionnaires have been received for the first three (collated in Appendix 9), which are described individually.

The first case (transfused in 1996) was transfused with red cells following a coronary artery bypass graft, and had no known underlying immune defects. The red cells were <5 days old, although not specifically requested as 'fresh'. Between 10 and 14 days after the transfusion, he developed clinical and laboratory features of TA-GVHD, with histology of skin biopsy and post-mortem tissues consistent with the diagnosis. Investigation of the donors revealed 1 HLA homozygous donor who shared a haplotype with the patient [donor: A1, B8, DR3; patient A1, B8, DR3(17), DR6(13)]. The patient succumbed to infection before therapy could be commenced.

The second case had autoimmune thrombocytopenia, treated with oral prednisolone, but no other immunosuppressive therapy. He was transfused with red cells and buffy-coat derived platelets because of gastro-intestinal haemorrhage. TA-GVHD developed 5-9 days later, confirmed by skin and bone marrow biopsy. Treatment with methylprednisolone was not effective, and he died of infection within 3 days.
The patient's HLA type was A2, A3; B8, Bw6; DR15, DR17. The donors were not HLA typed.

The third case had been treated 2 years previously for B cell non-Hodgkin's lymphoma with CHOP chemotherapy and local radiotherapy. The lymphoma had since remained in remission. He was transfused with red cells because of recent gastrointestinal haemorrhage and developed TA-GVHD 15-19 days later, confirmed by skin biopsy. Despite treatment with methylprednisolone and anti-lymphocyte globulin, he died of infection. As in the first case, there was HLA haplotype sharing between a homozygous donor and patient (donor: HLA- A1, B8, Cw7, DQ2; patient: HLA- A1, A31; B7, B8; Cw7; DR 17; DQ 2).

The fourth case had Waldenstrom's macroglobulinaemia, a low grade B cell lymphoid malignancy. He was transfused with red cells from 6 donors, who are still under investigation. This case will be reported completely in next year's report.

Comments

- Considering the 4 cases reported this year together with the 4 described in last year's report, the most common single remaining risk factor for TA-GVHD appears to be the presence of B cell lymphoid malignancy, present in 4 of 8 cases. HLA haplotype sharing between donor and recipient was an additional feature in 1 lymphoma case reported this year ; in the lymphoma cases reported last year, it was not possible to determine from the investigations undertaken whether there was also HLA haplotype sharing between donor and recipient. B cell lymphoma is not a current indication for gamma irradiated cellular components in the current BCSH guidelines on TA-GVHD prevention[16].
- HLA haplotype sharing between a homozygous donor and the patient was also a feature in the cardiac surgery patient reported this year. Cardiac surgery is a major risk factor for TA-GVHD in Japan, where HLA haplotype sharing in combination with transfusion of large volumes of fresh blood have been identified as compounding factors. In the case reported here, the blood used was <5 days old, but not requested as such.

- Treatment of autoimmune disease with oral steroids is not currently recognised as a specific risk factor for TA-GVHD.

Recommendations

- In view of the rarity of this condition, investigation of such cases would benefit from a single, nationally agreed protocol, which could be made available through blood transfusion centres. As with TRALI, mechanisms should be developed within Transfusion Services to ensure liaison with hospitals and complete donor investigation in TA-GVHD cases. Consideration should also be given to identifying a single national laboratory for investigation of suspected cases, so that diagnostic experience can be accumulated.
- BCSH guidelines for TA-GVHD prevention should be reviewed to consider whether B cell lymphoid malignancies should now be added to the indications for irradiated components. This would require calculation of the risk to such patients, taking into account the likelihood of transfusions also being from HLA homozygous donors sharing a haplotype with the patient.
- The additional risks of TA-GVHD from 'fresh' blood should be borne in mind if such blood is considered for cardiac surgery.

13. TRANSFUSION-TRANSMITTED INFECTIONS

Introduction

Infectious complications following transfusion differ from non-infectious complications in several ways that may affect the ascertainment and investigation of incidents. The onset of symptoms related to a transfusion-transmitted viral infection may occur from several weeks to years after the date of the transfusion. Reports of infections transmitted by transfusion in a particular year can therefore accrue over the subsequent year(s). The number of cases ascertained by the end of any period of time is therefore expected to be an incomplete picture of the infections transmitted during that period. Acute infections, such as bacteraemias, that tend to be clinically apparent and diagnosed within days of receipt of the infectious transfusion, may be relatively complete but chronic viral infections will be underrepresented.

In addition, the occurrence of disease, or the observation of serological markers of infection, in individuals who have donated blood can lead to the ascertainment of transfusion-transmitted infections by tracing and testing of recipients exposed to components collected from donors during potentially infectious periods. Recipients may be asymptomatic at this time and only identified by this investigation.

Post-transfusion infections (PTI) may be due to an infected (or contaminated) transfusion or infection may have been acquired from another source. Investigation of markers of infection in an implicated donation, or in subsequent samples from the donors of implicated donations, can confirm transfusion as the probable cause of infection, thus confirming the infection as transfusion-transmitted (TTI). Alternatively, the need to investigate other possible sources of infection may be identified. The blood service must therefore be informed about implicated transfusions so that investigations can be conducted to confirm or refute the suspicion that the implicated transfusion(s) may have been infectious. This is essential to prevent further transmission(s) by other components and/or by chronically infected donors. Such investigations may involve microbiological testing of many donors and may take several months to complete.

A surveillance system to collect standardised information about infections suspected to have been transmitted by transfusion was introduced in the British Isles (excluding Scotland) and the Republic of Ireland by the National Blood Authority and the Public Health Laboratory Service Communicable Disease Surveillance Centre (PHLS/CDSC) in October 1995. A parallel system is in place in Scotland; no confirmed cases were reported in Scotland during this report year.

Methods

Participating blood centres (see above) reported all post-transfusion infections of which they had been informed to the NBA/PHLS CDSC infection surveillance system. The criteria for identifying infections eligible for reporting as post-transfusion infections were either: a) the receipt of the transfusion had been confirmed and the infection in the recipient had been confirmed (by detection of antibody, antigen, RNA/DNA or culture) and there was no evidence that the recipient was infected prior to transfusion, or, b) the receipt of the transfusion had been confirmed and the recipient had acute clinical hepatitis of no known cause (including no evidence of acute HAV, HBV, HCV, EBV or CMV infection in post-transfusion samples to date). If other possible sources of infection were known for a post-transfusion infection, an initial report was still requested.

Information about the recipient, the recipient's infection and the transfusion(s) implicated as the possible source of infection formed the basis of the initial report. Subsequently, after appropriate investigations had been completed, details about the findings of the investigation, were reported. (PTI report forms are in Appendix 5).

A post-transfusion infection was classified as a transfusion-transmitted infection (TTI) if the following criteria were met at the end of the investigation:-

- the recipient had evidence of infection post-transfusion, and there was no evidence of infection prior to transfusion

and, either

- at least one component received by the infected recipient was donated by a donor who had evidence of the same transmissible infection,

or

- at least one component received by the infected recipient was shown to have been contaminated with the agent of infection

Data received by 31/12/98, about incidents of transfusion-transmitted infections initially reported by blood centres between 1/10/97 and 30/9/98, were included in this report. Data received about incidents reported during the previous two years of the surveillance system are included in a cumulative table.

Unless the investigation was closed due to the identification of a probable source of infection other than transfusion, investigations that were closed without being able to conclusively investigate the source of the post-transfusion infections were classified as post-transfusion infections of undetermined source.

Results

Thirty-five initial reports of post-transfusion infections were made by blood centres during the report year. An additional 6 reports were received about post-transfusion reactions that were suspected to be due to bacteria but for which no evidence of bacterial infection (or endotoxin) that could have caused the reaction was sought and found in the recipient or implicated component (i.e. the incidents did not satisfy the criteria for a post-transfusion infection as stated above, but may have been reactions of bacterial origin). Reports were received from 12 of the 21 blood centres (between 1-7 cases each) participating in the surveillance system. These 12 centres collect approximately 87% of the donations tested by blood centres participating in the surveillance system.

Figure 13 shows the classification of reports during the report year. Of the 35 post-transfusion infections initially reported by blood centres to the surveillance system between 1/10/97 and 30/9/98, 4 (11%) were classified, after appropriate investigation, as transfusion-transmitted infections. Table 20 shows the transfusion-transmitted infections reported to the surveillance system between 1/10/97 and 30/9/98 by year of transfusion: Two were transfused during the report year, and 2 were transfused prior to the report year.

Figure 13
Classification of post-transfusion infections (and post-transfusion reactions) initially reported between 1/10/97 and 30/9/98

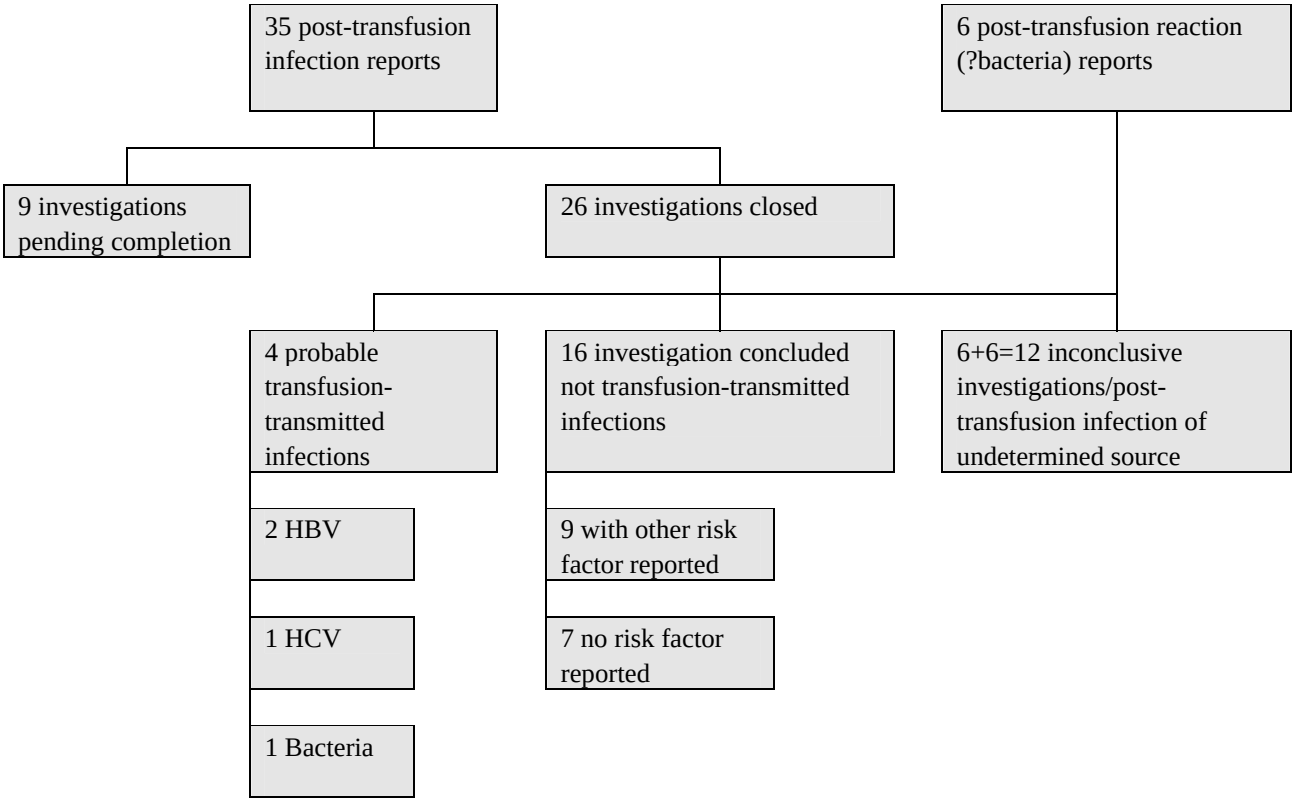


Table 20
Transfusion-transmitted infections reported between 1/10/97-30/9/98 by year of transfusion. The number of incidents are shown, with the total number of identified infected recipients shown in brackets.

Year of transfusion	pre-1997	1997	1998 (to end Sept)	Total
Infection				
HBV	1(1)[a] [1991]	1(1)	-	2(2)
HCV	1(1) [1970-85]	-	-	1(1)
Bacterial	-	-	1(1)[b]	1(1)
Total	2(2)	1(1)	1(1)	4(4)

Notes [a]One household member who was caring for the recipient has been diagnosed with acute HBV.
[b]Infection was implicated in the death of the recipient.

Details of transfusion-transmitted infections

A. Infections for which donation testing is mandatory

Hepatitis B virus

Two transfusion-transmitted HBV infections were reported. One recipient (26 year old male) had acute HBV infection five months after transfusion of a red cell unit (one of 14 red cell units given over a year) that was found, by testing of the archived sample of the donation, to be anti-HBc negative but HBV DNA positive. At the time of the investigation, the donor recalled having viral symptoms and abdominal pains 5 months post-donation and was found to be anti-HBs positive. The probable source of the recipient's HBV infection was concluded to be an HBV infectious, though HBsAg and anti-HBc negative donation collected from a repeat donor during early acute infection.

One recipient (59 year old male) was found to be an HBsAg and HBeAg positive HBV carrier 6 years after transfusion with 8 red cell units. One of the donors was found to have markers of resolved HBV infection and it was also discovered that this donor had developed acute HBV (confirmed by the local laboratory) 3 months after donating the implicated donation. No archived sample of the donation was available for further testing. The probable source of the recipient's HBV infection was concluded to be an HBV infectious, but HBsAg negative, donation collected from a new donor during acute infection. Secondary transmission seems to have occurred as a household member who was caring for the infected recipient was diagnosed with acute HBV at the same time as the recipient's diagnosis.

Both of the donations implicated in these two transfusion-transmitted HBV infections were collected from donors who subsequently disclosed risk factors for HBV infection that should, according to donor selection criteria in place at the time, have been recognised as making them ineligible for blood donation. Further investigation is needed to identify the reasons why these donors were not recognised as ineligible for donation.

Hepatitis C virus

One transfusion-transmitted HCV infection was reported. A patient (52 year old male) was found to be anti-HCV and HCV RNA positive during investigation of chronic liver disease. The patient had been transfused with at least 4 red cell units more than 7 years prior to the introduction of anti-HCV testing of blood donations in September 1991. One of the donors was found to be anti-HCV positive when a subsequent donation was tested at another blood centre. This donor's previous donations were entered into the HCV lookback programme and at the start of the lookback process one red cell unit was identified as a component involved in this post-transfusion infection investigation. The probable source of the recipient's HCV infection was concluded to be an HCV infectious donation collected from a repeat donor prior to anti-HCV testing.

HIV

No transfusion-transmitted HIV infections were reported during this year.

B. Infections for which donation testing is not mandatory

Bacteria

One transfusion-transmitted bacteraemia was reported. One recipient (32 year old female) developed a bacteraemia after transfusion with red cells and platelets and died two days after the transfusion. *Staphylococcus aureus* was isolated from the recipient and from skin and nasal swabs from one of donors who contributed to the platelet pool.

Details of post-transfusion infections not found to be transfusion-transmitted infections

Six (17%) post-transfusion infections (1 bacteraemia, 1 HBV infection, 4 HCV infections) were classified as post-transfusion infections of undetermined source due to incomplete investigation of the transfusion(s) implicated as the source of infection. For sixteen (46%) post-transfusion infection reports (9 HBV infections, 5 HCV infections, 1 dual HBV and HCV infection and 1 HIV infection), investigation was completed and no evidence was found to implicate transfusion as the source of infection. A possible source of infection other than transfusion was known for 9 of these infections (HBV: previous transfusion (details incomplete), surgery (x2), travel to country of high endemicity, birth in country of high endemicity, liver transplant; HCV: birth & travel in country of high endemicity, transfusion abroad, injecting drug use).

Time to reporting

For the 4 transfusion-transmitted infections, the intervals between transfusion and diagnosis of the infection in the recipient was 1 day (*Staphylococcus aureus*), 17 weeks (acute HBV), 6 years (HBV carriage) and 12 years (HCV). The intervals between diagnosis and blood centres being informed that the infection was suspected to be associated with transfusion were 2 days, 72 days, 110 days and 30 days. The intervals between the blood centre being informed and the completion of the initial surveillance report form were 40 days, 44 days, 63 days and 214 days.

Underreporting

The cases ascertained by this surveillance system were diagnosed, suspected to be attributable to transfusion, communicated to the blood service, and reported by a blood centre to the surveillance centre. At any one of these steps, other post-transfusion infections may have been missed and the extent of underreporting of post-transfusion infections is therefore unknown. The proportion of post-transfusion infections that are reported each year may vary as other factors such as testing performed on transfusion recipients, awareness of transfusion as a possible source of infection, reporting of information to blood centres and reporting of information from blood centres to the surveillance centre vary. In June 1998 all participating blood centres were contacted and asked to confirm that the number of reports they had made to the surveillance system was the total number of post-transfusion infections that they had been informed about, or to report outstanding reports as soon as possible.

Previous year

During the previous reporting year (i.e. 1/10/96 to 30/9/97) 8 transfusion-transmitted infections were reported (see SHOT Annual Report 1996-97 for details of these cases). None of the post-transfusion infections reported during the 1996-97 year that were pending full investigation at the time of the 1996-97 SHOT annual report have been subsequently concluded to have been transfusion-transmitted infections.

One post-transfusion HCV infection investigation that was initially reported in the 1995-96 report year, and was classified as undetermined at the time of the 1996-97 SHOT report was, during the 1997-98 report year, updated to become a transfusion-transmitted infection when an untraced donor returned to donate blood in another region and was found to be anti-HCV positive. This donor's previous donations were entered into the HCV lookback programme and at the start of the lookback process one component was identified as a component involved in this post-transfusion infection investigation.

Table 21 shows the cumulative number of transfusion-transmitted infections reported by the end of September 1998.

Figure 14 shows the number of reports received by year of report since October 1995.

Table 21

Cumulative total transfusion-transmitted infections: reported between 1/10/95-30/9/98 by date of transfusion. The number of incidents is shown with the total number of identified infected recipients in brackets.

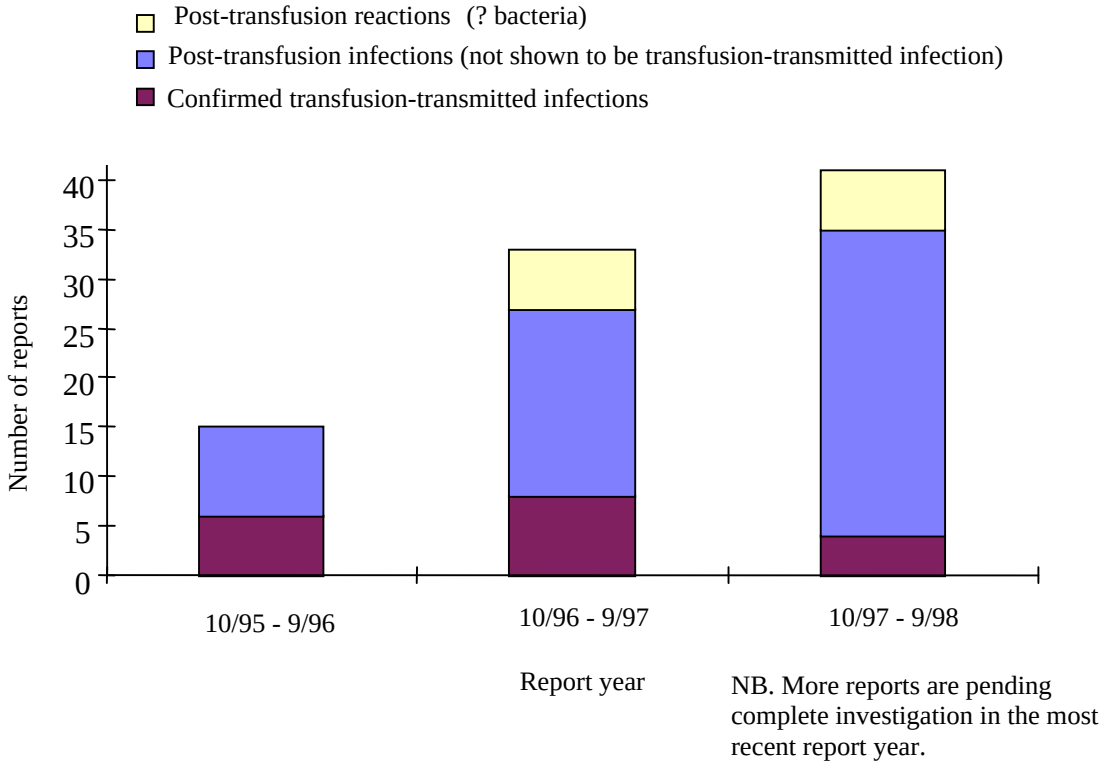
Year of transfusion	pre-1995	1995	1996	1997	1998 (to end Sept)	Total
Infection						
HAV		-	1(1)	-	-	1(1)
HBV	1(1)[a]	1(1)	1(1)	1(1)	-	4(4)
HCV	4(4)[b]	-	5(5)	-	-	5(5)
HIV		-	1(3)	-	-	1(3)
Bacterial		1(1)	1(1)	3(3)	1(1)[c]	6(6)
Malaria		-	-	1(1)[c]		1(1)
Total	5(5)	2(2)	5(7)	5(5)	1(1)	18(20)

Notes: [a]One household member who was caring for the recipient has been diagnosed with acute HBV.

[b] Transfusions prior to anti-HCV testing of blood donations.

[c] Infection was implicated in the death of the recipient.

Figure 14 : PTI reports by report year



NB. More reports are pending complete investigation in the most recent report year.

Comments

- Reported transfusion-transmitted infections are rare, and only 4/35 (11%) suspected cases were confirmed during this 12-month period of reporting. A further 31 cases of post-transfusion infection were reported to have been investigated. Almost half (46%) of the PTI reports during this year have been shown to not be caused by transfusion; for 17% of the reports the investigation was inconclusive and for the remainder (26%) the investigation is still ongoing.

- Six cases of post-transfusion reactions suspected (but not confirmed) to be due to bacteria were also reported. Conclusive investigation of a suspected bacteraemia in a transfusion recipient relies heavily on the collection and handling of relevant samples at the hospital where the transfusion was performed. This means that absence of evidence of an infection (or toxin), in donations given to recipients who had post-transfusion reactions that were suspected (on clinical presentation) to be due to bacteria does not equate with evidence of absence of a transfusion-transmitted infection (or toxin).

- The intervals between transfusion and diagnoses of transfusion-transmitted infections were long - many weeks, months or years. Infections transmitted by transfusion between 1/10/97 and 30/9/98 will continue to be ascertained by the surveillance system as diagnoses are made in the future.

- The intervals between blood centres being informed of post-transfusion infections and completing an initial report form were long and should be reduced in order to ensure that information reaches the surveillance centre as soon as possible.

- Two transfusion-transmitted infections (2 HBV infections) were due to donations collected from donors during marker negative “window periods” following recent infection. Both donors had risk factors for acute HBV that should have led to their exclusion from blood donation.

- Two transfusion-transmitted infections (1 HCV infection, 1 bacterial) were due to collection of a donation from a donor with an infection for which no routine microbiological testing was in use routinely at that time.

- No reported transfusion-transmitted infections were due to errors in the microbiological testing, or release, of blood donations.

- One transfusion-transmitted infection reported during this year resulted in the death of the recipient.

Recommendations

- National collation of data arising from these cases needs to continue over several years before a picture of the extent and nature of the infectious complications of transfusion can emerge.

- All post-transfusion infections diagnosed in patients should be reported by the clinician to the local blood centre for appropriate investigation. Blood centres should, in turn, complete an initial report form as soon as possible.

- National guidelines for the bacteriological investigation of adverse reactions associated with transfusion are available for hospitals. Hospitals should not destroy blood components implicated in post-transfusion reactions suspected to be due to bacteria, and should consult these guidelines and the local blood centre about the investigation of such cases.

- Methods and criteria used to exclude those individuals who have risk factors for transfusion transmissible infections from donating blood warrant continuing evaluation and development. Investigation of the reasons for non-exclusion of ineligible donors is also warranted.

- Staff handling blood components should familiarise themselves with their normal range of appearance, and inspect packs for leaks or unusual colour/turbidity which might suggest bacterial contamination. Components which appear unusual in any way should NOT be transfused, but returned to the blood bank[9].

14. NEAR MISS PILOT SCHEME

Definition
Any error which, if undetected, could result in a wrong blood group, or issue of an incorrect or inappropriate component, but which was recognised before transfusion occurred

The types and frequency of errors causing transfusion of the incorrect or inappropriate blood or components are now becoming clearer. Underlying these problems however, are errors, potentially serious if undetected, which are recognised during the checking and validation procedures built into each stage of the transfusion process at various stages.

Awareness and recognition of these detected errors ('near miss' events) could provide useful information to enable modification of procedures and testing protocols, thereby reducing the potential problem areas which contribute to an incorrect transfusion.

A small pilot scheme for 'near miss' events has been trialled by 4 hospitals over an 8 month period, and will be extended during the next year to cover approximately 20 volunteer hospitals. It is intended to run this extended scheme for a period of 6 months to assess the magnitude of problems and obtain a more comprehensive survey of where these arise.

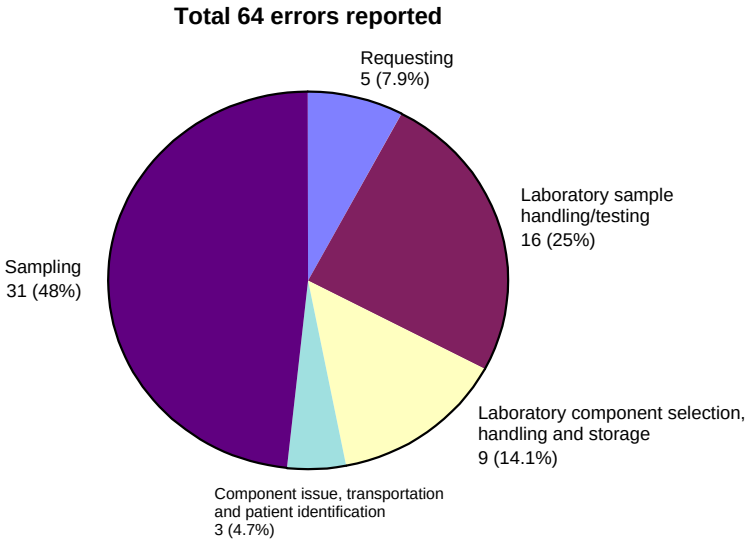
As it was anticipated that the frequency of 'near misses' would be several times higher than the frequency of adverse events reported to SHOT, it was felt that a very brief and straightforward reporting system was essential to encourage compliance. A series of 5 forms (see Appendix 6), has been drawn up, each form covering a particular area of activity. The details are entered by ticking appropriate sections on the relevant form, with minimal text entry required, and the completed form is then returned to the SHOT Office for retrospective collation and analysis. No further documentation is involved.

The 5 activity areas covered on the Near Miss report forms are:

1. Sample errors
2. Request errors
3. Laboratory sample handling/testing errors
4. Laboratory component selection, handling and storage errors
5. Component issue, transportation and patient identification errors

The results from the initial pilot scheme are reported below.

Figure 15
Overview of site of 'near miss' errors.



Sample errors

31 sample errors were reported

- 15 incidents occurred where the sample tube was labelled for the intended patient but where the blood had been taken from someone else.
- On 13 occasions the correct patient was bled but another patient's details put on the sample label. It is not known if addressograph labels were implicated. This question will be included on the form in future.
- 16 phlebotomy errors were stated to involve a doctor, 9 involved nursing staff and 4 involved phlebotomists. The staff involved in the other 2 incidents were not stated.
- 23/31 errors occurred during routine laboratory hours.
- 29/31 errors were detected within the laboratory, usually by comparison with previous computer records of the patient involved.

Sample errors accounted for 31/64 (48%) of total errors notified. These are of serious concern as they are likely to represent the tip of the iceberg, in that incorrect samples from patients with the same blood group, or samples from patients not previously tested, will not be detected.

Request errors

- 3/5 resulted from lack of clarity of telephone requests to the laboratory. One case involved incorrect patient selection from a pick list on the ward terminal of the hospital computerised information system.

Laboratory sample handling/testing errors

- 9/16 reports recorded errors in RhD typing of the patient, both false positive and false negative. Two of these same errors also led to an incorrect ABO group result. The causes of these errors were not clear.
- On 5 occasions an incorrect sample was used for testing.
- In 1 case a unit of blood, found to be incompatible, was incorrectly issued for use
- 11/16 errors occurred during routine working hours.

Laboratory component selection, handling and storage errors

Nine errors were recorded in this group

- In 2/9 reports, selected blood units by the Blood Centre were incorrectly phenotyped. In one instance a K+ unit was supplied in error, and in the other the luggage labels attached to the bags and bearing phenotype information were transposed on 2 units.
- 1 out of date unit of FFP was issued in error but detected by the ward checking procedures.
- 1 error occurred where component labels were attached to the wrong bags.
- On 2 occasions random donations were issued instead of CMV seronegative components.
- On 3 occasions blood was stored incorrectly in a remote refrigerator but the error was recognised before transfusion. The fact that blood had been removed from a remote refrigerator and returned after an excessive period of time was recognised by the MLSO in 2 of the cases, whilst in the other case blood had been stored in a ward refrigerator not suitable for blood storage.
- 6/9 errors occurred during normal laboratory working hours.

Component issue, transportation and patient identification errors

- Only 3 problems were recognised in this area despite this being a significant cause of errors in the 1996-97 SHOT report.
- All errors reported were due to collection of a component for a wrong patient; on one occasion the identical error was repeated with a second unit for the same patient
- Porters were stated to have collected components from a laboratory blood bank on 2 of the occasions.
- All the problems occurred out of routine laboratory hours and involved collections from the main blood bank refrigerator.

Summary

- These reports are from a very small number of selected hospitals and may therefore not be representative of a larger survey.
- A disturbing number of phlebotomy errors were detected. This is a potentially serious problem as it is known that in approximately 50% of cases there will be no historical record to compare, or by coincidence, an identical ABO and RhD grouping result will be obtained.
- A significant proportion of laboratory errors resulted in an incorrect RhD group.
- Only 3 errors of collection from the laboratory were noted, despite this being a significant cause of mis-transfusion in the first SHOT report. It must be appreciated that only 4 hospitals were studied and by chance these sites may have had less opportunity for this type of error.
- A larger selection of hospitals of various sizes and special interests, will be included in the proposed next stage of the ‘near miss’ study, in order to try to obtain a more representative picture.

15. FUTURE DEVELOPMENTS

1. Participation

The first SHOT report has been generally well received, and introduction of the 'nil return' card has demonstrated that 65% of hospitals now participate in SHOT, in only our second year. This is a tribute to the positive attitude of haematologists and blood bank staff towards improvements in transfusion safety. For most hospitals, SHOT has been a welcome initiative, but over 30% of hospitals are not yet participating. It is interesting to note that the National Confidential Enquiry into Perioperative Deaths (NCEPOD) also has a approximately 30% non-participation rate, after 10 years of reporting. The debate on whether participation in SHOT and other confidential enquiries should be compulsory will no doubt continue once the National Institute for Clinical Excellence (NICE) is established, and mechanisms for Clinical Governance in place. The importance of SHOT was emphasised in the recent Health Service Circular to Trusts on Better Blood Transfusion (HSC1998/999). In the meantime, it would in theory be possible to make participation in SHOT a requirement for CPA accreditation, using an anonymised receipt system to maintain confidentiality. This approach was recently endorsed by the SHOT steering group, and CPA will consider this issue in due course.

The other aspect of the 'nil return' card was to ask for data on workload. We appreciate that this is an additional burden on participants, but it has allowed us to begin to create the all-important 'denominator figure' against which risk of transfusion hazard can be calculated. This will eventually allow transfusion risk to be more accurately placed alongside other medical and life risks, an important step in allocating health care priorities.

2. ' Near miss' pilot exercise

As reported in the previous chapter, this pilot ran well, and demonstrated the utility of the forms. It is clear that much can be learnt from 'near-miss' analysis, particularly on how mis-transfusion is prevented by timely detection of errors. There has been a gratifying response from hospitals wishing to participate in a longer study; these will be allocated shortly and hospitals contacted with information packs.

3. Reporting of hazards of pre-deposit autologous donation

It has been recognised that risks from autologous procedures cannot at present be assessed alongside those of allogeneic blood. For this reason, SHOT took the opportunity at the Royal College of Physicians Update Consensus Conference on Autologous Transfusion in November 1998, to launch the questionnaire designed to capture major adverse events associated with pre-deposit autologous transfusion (see Appendix 2). This has been promulgated with the help of the Autologous Transfusion Special Interest Group of the British Blood Transfusion Society, and thanks are due to them for their support.

The next phase of this initiative will involve the development of questionnaires to capture serious events associated with intra-operative cell salvage and peri-operative haemodilution, techniques which are set to increase over the next few years.

4. Putting SHOT on the web

As last year, this report will be made available to all hospital transfusion laboratories, and summaries widely distributed to participating professions. As we approach the millennium, we are considering additional ways in which SHOT information can be made more readily available to a wider audience. As a beginning, we are in discussion with the Royal College of Pathologists Information Technology Department to discuss whether this report could be added to the RCPath website. We will also consider longer term options, such a separate web site, and the possibility of interactive material for educational purposes. Much of this kind of development will of course depend on availability of resources.

5. SHOT in the era of Clinical Governance and the role of the National Institute for Clinical Excellence

At the time of writing, SHOT is a professionally independent body, affiliated to the Royal College of Pathologists, and funded largely by the UK Transfusion Services, with generous help from the British Society for Haematology and the British Blood Transfusion Society. Other Confidential Enquiries, which are Department of Health funded, will become part of the NICE structure. It remains to be seen what options SHOT will have once NICE is established, and whether its long term future is best secured by joining the other Confidential Enquiries within NICE. Whatever the future holds, SHOT can clearly demonstrate a high degree of professional support for its activities, and our willingness to work with other professional organisations to promote best transfusion practice.

At a local level, it may well be that once systems for local Clinical Governance are in place, managerial support for resources required to implement SHOT recommendations may be easier to obtain. We are pleased to welcome a representative from the Institute of Health Service Managers on to the Steering Group, and will be seeking advice from hospital managers regarding the best way to promulgate SHOT recommendations through hospital management structures.

16. REFERENCES

1. Lazarou J, Pomeranz B, Corey P. Incidence of adverse drug reactions in hospitalized patients. JAMA 1998;**279**;1200-1205
2. Aubuchon JP, Kruskall MS. Transfusion safety: realigning effort with risks. Transfusion 1997;**37**:1211-1216.
3. Kaplan HS et al. Identification and classification of the causes of events in transfusion medicine. Transfusion 1998;**38**:1071-1081.
4. Goldman M, Blajchman MA. Transfusion-associated bacterial sepsis. Transfusion Medicine Reviews 1991;**5**:73-83.
5. McClelland DBL et al. Reducing risks in transfusion: process and outcome. Transfusion Medicine 1996;**6**:1-10.
6. McClelland DBL. Treating a sick process. Transfusion 1998;**38**:999-1103.
7. Ingrand P et al. Reliability of the pre-transfusion bedside compatibility test: Association with transfusion practice and training. Transfusion 1998;**38**:1030-1036.
8. Lumadue JA, Boyd JS, Ness PM. Adherence to a strict specimen-labelling policy decreases the incidence of erroneous blood grouping of blood bank specimens. Transfusion 1997;**37**:1169-1171.
9. Handbook of Transfusion Medicine. Ed. DBL McClelland. HMSO: London. 1996.
10. British Committee for Standards in Haematology Blood Transfusion Task Force (Chairman: D Voak): Guidelines for administration of blood products: transfusion of infants and neonates. Transfusion Medicine 1994;**4**:63-69.
11. Shiba M, Tadokoro K, Sawanobori M, Nakajima K, Suzuki K and Juji T: Activation of the contract system by filtration of platelet concentrates with a negatively charged white cell-removal filter and measurement of venous blood bradykinin level in patients who received the filtered platelets. Transfusion 1997;**37**:457-462.
12. Popovsky MA. Communication to USA Food & Drugs Administration Blood Products Advisory Committee, 1998.
13. British Committee for Standards in Haematology, Working Party of the Blood Transfusion Task Force (Convenor: MF Murphy): Guidelines for platelet transfusion. Transfusion Medicine 1992;**2**:311-318.
14. British Committee for Standards in Haematology Blood Transfusion Task Force (Chairman: JAF Napier): Guidelines for pre-transfusion compatibility procedures in blood transfusion laboratories. Transfusion Medicine 1996;**6**:273-283.
15. Robertson D. Davenport: Haemolytic transfusion reactions. In Transfusion Reactions. Ed. Popovsky MA, AABB Press 1996. 1-44.
16. British Committee for Standards in Haematology Blood Transfusion Task Force. Guidelines on gamma irradiation of blood components for the prevention of transfusion-associated graft-versus-host disease. Transfusion Medicine 1996;**6**: 261-271.