Conclusions

There is no routine sampling of live birds or carcasses in chicken-packing stations in this country, and in view of the large number of birds slaughtered weekly such checks would be impracticable with existing facilities. However, there seems to be a case for more rigorous control of general hygiene throughout broiler production units and bacteriological control of animal feeding-stuffs. Outbreaks of food-poisoning can be more easily prevented if the proportion of infected birds is reduced to the level found by Tucker and Gordon (1968). The popularity of New York dressed birds is of particular concern. Such birds carrying food-poisoning organisms are potentially dangerous in shops and the kitchen.

The dispersal of infected litter to grazing land presents hazards to livestock which can in turn infect humans by direct contact or by contaminating milk. Measures which control disposal of litter from rearing units are worth further consideration.

We wish to express our gratitude to Mr. D. Breckon and the staff of the Public Health Laboratory, Chester, and the staff of the

Veterinary Investigation Laboratory, Liverpool, for technical assistance; also to Mr. J. S. Carrie and the public health inspectors of the Mid-Cheshire local authorities.

REFERENCES

- Dixon, J. M. S., and Pooley, F. E. (1961). *Mth. Bull. Minist. Hlth Lab. Serv.*, **20**, 30.
 Gordon, R. F., and Tucker, J. F. (1965). *Brit. poult. Sci.*, **6**, 251.
 Hobbs, B. C., and Allison, V. D. (1945). *Mth. Bull. Minist. Hlth Lab. Serv.*, **4**, 63.
 Jameson, J. E. (1962). *J. Hyg. (Lond.)*, **60**, 193.
 Kampelmacher, E. H. (1963). *Brit. vet. J.*, **119**, 110.
 Robinson, R. G. (1958). *J. med. Lab. Technol.*, **15**, 79.
 Rolfe, V. (1946). *Mth. Bull. Minist. Hlth Lab. Serv.*, **5**, 158.
 Semple, A. B., Turner, G. C., and Lowry, D. M. O. (1968). *Brit. med. J.*,
 Snoeyenbos, G. H., Carlson, V. L., McKie, B. A., and Smyser, C. F. (1967). *Avian Dis.*, **11**, 653.
 Taylor, Joan (1957). *Vet. Rec.*, **80**, 147.
 Tucker, J. F., and Gordon, R. F. (1968). *Brit. vet. J.*, **124**, 102.
 Van Schothorst, M., Guinée, P. A. M., Kampelmacher, E. H., and Keulen, A. (1965). *Reihe B.*, **12**, S.422.
 Vernon, E. (1967). *Mth. Bull. Minist. Hlth Lab. Serv.*, **26**, 235.
 Wilson, E., Peffenborger, R. S., Foster, M. J., and Lewis, K. H. (1961). *J. infect. Dis.*, **109**, 166.
 Wilson, J. E. (1945). *Vet. Rec.*, **57**, 411.

Red Cell Survival after Heterograft Valve Surgery

W. H. P. ROESER,* M.B., M.R.A.C.P.; L. W. POWELL,† M.D., M.R.A.C.P.; M. F. O'BRIEN,‡ F.R.C.S., F.R.A.C.S.

Brit. med. J., 1968, **4**, 806-808

Summary: Intravascular haemolysis was studied in 24 patients three to nine months after calf or pig valve heterografts had been inserted for severe valvular heart disease. No patient had haemolytic anaemia. In five of the 24 patients there was subclinical haemolysis, and in these five the haemolysis appeared to be related to residual aortic regurgitation or to the presence of other foreign material such as a Dacron aortic graft. The extent of postoperative haemolysis in these five patients was comparable to that observed preoperatively in patients with valvular heart disease.

The results support the belief that, in contrast to artificial valve prostheses, heterograft valves behave similarly to human valves as regards haemolysis.

Introduction

Intravascular haemolysis is a frequent complication of prosthetic valve replacement (Brodeur *et al.*, 1965; Rubinson *et al.*, 1966; Bell *et al.*, 1967), and in a significant proportion of cases frank haemolytic anaemia occurs, particularly with incompetent prostheses (Sayed *et al.*, 1961; Marsh, 1964; Stevenson and Baker, 1964). Random destruction of red cells due to valve turbulence and shearing forces created by a regurgitant flow of blood form the most likely basis for the haemolysis, and this theory has recently received experimental support (Nevaril *et al.*, 1968). Such mechanical trauma to the red cells is accentuated by contact with the foreign materials used in artificial prostheses. An immune basis in some patients has been suggested by the development of a positive Coombs antiglobulin test and the

fact that the haemolytic anaemia sometimes responds to treatment with corticosteroids (Pirofsky *et al.*, 1965).

Since 1966 formaldehyde-preserved calf and pig heterograft valves have been used to replace diseased valves and defective prostheses (O'Brien and Clarebrough, 1966, 1967). As these valves produce little turbulence (O'Brien and Clarebrough, 1966), and thus less mechanical damage to the red cells than artificial prostheses, it might be expected that intravascular haemolysis is uncommon in patients with heterograft valves. Though of obvious practical significance, it seems that this point had not been studied previously.

Methods

We have studied 24 patients who had calf or pig valve heterografts inserted at the cardiac unit of the Chermide Hospital, Brisbane. All had severe valvular heart disease, verified by preoperative haemodynamic studies. The haematological investigations were made three to nine months post-operatively. In addition, five of the patients were studied pre-operatively. Red cell survival was investigated with the ^{51}Cr technique after labelling a 10-15 ml. sample of the patient's red cells in vitro with 50-100 μCi ^{51}Cr (Veall and Vetter, 1958). The lower limit of normal for the $T_{1/2}$ ^{51}Cr -labelled red cells in this laboratory is 25 days. As further evidence of intravascular haemolysis, plasma haemoglobin was estimated in all patients. For red cell haemoglobin concentration and reticulocyte counts standard methods were used. The serum iron level and the iron-binding capacity of the serum were estimated by the method of Trinder (1956) adapted to the autoanalyser.

Results

Haemodynamic.—Three patients (Cases 1, 2, and 3) had significant residual aortic incompetence, but only one (Case 2)

* Teaching Registrar, Medical Professorial Unit.

† Senior Lecturer, Medical Professorial Unit.

Department of Medicine, University of Queensland, Royal Brisbane Hospital, Brisbane, Australia 4029.

‡ Cardiac Surgeon, Chermide Hospital, Brisbane, Australia.

complained of symptoms referable to his cardiac state. This patient has undergone reoperation, and his leaking aortic valve was recently replaced by a new heterograft valve. All other patients have returned to their former occupations.

Haematological.—Of the 24 cases reported five showed a reduced $T\frac{1}{2}$ for ^{51}Cr -labelled red cells postoperatively, and in two of these a slightly raised plasma haemoglobin level was found. Three of the five had residual aortic valve malfunction of severe (Cases 1 and 2) and moderate (Case 3) degree. The other two patients had additional foreign material in their circulation (see Discussion). In Cases 20–24 red cell survival was also studied preoperatively and was reduced in four. All five patients had a normal $T\frac{1}{2}$ for ^{51}Cr red cells postoperatively. None of the patients studied was anaemic, and the mild haemolysis noted in the above patients was well compensated. A significant reticulocytosis was observed in Cases 1 and 3 only and the peripheral blood smears showed no abnormal fragmented cells. Bone marrow aspirates were examined in eight cases. Case 1 showed normoblastic hyperplasia consistent with mild haemolysis while Cases 9 and 14 had reduced marrow iron stores. The other marrow aspirates were normal. The indirect and direct Coombs tests were negative in all cases. Measurements of serum iron and total iron-binding capacity were within the normal range in all cases. The patients showing a reduced red cell survival time were screened for abnormalities in glucose-6-phosphate dehydrogenase levels, osmotic fragility, haemoglobin electrophoresis, and Ham's acid-serum test. No abnormalities were detected in these investigations.

Discussion

Formaldehyde preservation of heterologous valves results in death of the tissue and subsequent loss of its endothelial lining *in vivo*. Haemodynamic studies have shown that the valves produce little turbulence (O'Brien and Clarebrough, 1967). Our results suggest that a normally functioning heterograft valve produces no detectable haemolysis. Of the 24 patients with heterografts in the present study only five had detectable haemolysis, and in four of these the $T\frac{1}{2}$ ^{51}Cr was over 20 days, indicating a very mild impairment of red cell survival. This contrasts with reports of artificial prostheses. Bell *et al.* (1967), in a series of 27 patients with artificial valve prostheses, found evidence of haemolysis in 20, while Brodeur *et al.* (1965) demon-

strated reduced $T\frac{1}{2}$ ^{51}Cr -labelled red cells in 8 of his 12 patients with Starr-Edwards valve prostheses, the range of values obtained being 9 to 23 days, with a mean of 18 days. Of these 12 patients 7 had normally functioning prostheses and four of them were found to have a reduced $T\frac{1}{2}$ for ^{51}Cr -red cells (18 to 23 days).

Rubinson *et al.* (1966) drew attention to the importance of valve incompetence in the production of traumatic haemolysis of red cells, and Nevaril *et al.* (1968) showed that the laminar shearing forces involved can be applied to the red cell *in vitro* and produce a haematological picture similar to that seen in patients with leaking valve prostheses. Three of our five patients with haemolysis (Cases 1, 2, and 3) had moderately severe aortic regurgitation. Case 1 is of particular interest since the degree of incompetence of his heterograft valve is similar clinically to that of his previous ball valve. However, whereas preoperatively the degree of haemolysis was severe and associated with overt haemolytic anaemia (see Table), the postoperative haemolysis was mild and well compensated. Case 2 had severe aortic incompetence after heterograft replacement and required further cardiac surgery. Nevertheless, his red cell survival was only mildly impaired and other indices of haemolysis were within normal limits.

Two of the patients with postoperative haemolysis (Cases 4 and 5) had additional foreign material in the circulation (a pacing catheter and a Dacron ascending aorta respectively). It is possible that this added factor was partly responsible for the reduced $T\frac{1}{2}$ ^{51}Cr red cells, and this aspect is under further investigation.

Valvular heart disease, particularly of the aortic valve, is now known to be associated with haemolysis of mechanical origin (Brodeur *et al.*, 1965; Eyster *et al.*, 1968; Roeser *et al.*, 1968). Brodeur *et al.* studied 21 cases of valvular heart disease and found evidence of haemolysis in 18. The $T\frac{1}{2}$ for chromium-labelled red cells in their series ranged from 14 to 21 days, and this is in accord with our own findings in a study of 22 preoperative patients. Similarly, Eyster *et al.* (1968) noted the presence of haemolysis in 6 out of 12 patients with aortic valvular disease. The extent of postoperative haemolysis in the five patients in our present study is comparable with that noted in patients in the above studies. These results thus support the belief that the role of heterograft valves as regards haemolysis is very similar to human valves. This applies equally to

Table of Results

Case No.	Age	Sex	Operation	Preoperative				Postoperative				
				Hb (g./100 ml.)	Plasma (Hb mg./100 ml.)	$T\frac{1}{2}$ ^{51}Cr (days)	Degree Incomp.*	Hb (g./100 ml.)	Retic. Count (%)	$T\frac{1}{2}$ ^{51}Cr (days)	Plasma Hb (mg./100 ml.)	Resid.* Incomp.
1	42	M	A.V.R., Starr valve removed; anatomy of ring distorted; poor placement of heterograft.	7.9	45.2	—	+++	13.9	3.0	22.5	10.4	+++
2	27	M	A.V.R.	13.9	3.6	—	++++	14.4	0.3	22	2.8	++++
3	53	M	"	12.2	7.1	—	+, A.S.	12.7	2.5	17	10.0	++
4	36	M	A.V.R. plus Dacron ascending aortic replacement	15.3	6.4	—	+++	15.3	1.8	20.5	2.0	+
5	45	M	A.V.R., permanent transvenous pacing catheter (heart block)	15.0	6.5	—	Pure A.S.	13.2	0.6	23	4.2	0
6	21	M	A.V.R.	12.1	3.6	—	+++	13.3	1.4	27	—	+
7	54	F	"	11.9	4.2	—	+, A.S.	12.5	1.0	25	2.0	(+)
8	45	M	"	14.8	4.4	—	+, A.S.	15.3	0.6	25	1.0	+
9	48	M	"	11.6	2.8	—	Pure A.S.	15.4	0.8	33	5.0	0
10	61	M	"	12.5	9.0	—	+, A.S.	12.8	1.6	26	5.4	0
11	42	M	"	13.1	2.0	—	+, A.S.	14.2	0.7	29	5.4	0
12	45	M	"	14.0	7.2	—	+, A.S.	14.6	1.2	32	6.2	0
13	42	M	"	16.4	4.5	—	+, A.S.	15.4	0.6	28	—	0
14	36	M	A.V.R. plus mitral and tricuspid annuloplasties	14.0	2.0	—	+++ M.I.	16.0	1.1	32	4.8	0
15	50	M	A.V.R., M.V.R.	16.1	7.5	—	+, M.S., M.I.	14.5	1.2	32	2.0	0
16	49	M	A.V.R.	14.3	11.8	—	+, A.S.	12.4	0.2	32	—	0
17	30	M	"	14.6	7.8	—	+, A.S.	12.4	1.0	25	2.0	0
18	57	M	"	13.7	8.2	—	+++	13.5	1.1	29	3.5	0
19	57	M	"	14.6	8.0	—	+, A.S.	14.8	0.8	25	1.0	0
20	59	F	"	14.8	5.0	27	Pure A.S.	14.0	1.2	32	0.8	0
21	35	M	"	13.4	2.8	24	+, A.S.	14.8	0.9	32	3.0	0
22	53	M	"	15.0	5.3	20	+, A.S., M.I.	16.2	1.8	25	4.0	0
23	35	M	"	15.2	3.6	22	+, A.S.	14.7	1.0	26	4.0	0
24	59	M	"	14.2	0.8	19	++++, A.S.	14.1	1.4	30	2.6	0

* Grading of incompetence: 0 = none, (+) = barely audible diastolic murmur, + = diastolic murmur without peripheral signs, ++ to +++ = increasing severity of peripheral signs.

A.V.R. = Aortic valve replacement. M.V.R. = Mitral valve replaced by aortic valve heterograft. A.S. = Aortic stenosis. M.S. = Mitral stenosis. M.I. = Mitral incompetence.

Normal ranges in this centre: Haemoglobin (Hb), 12.5–17.5 g./100 ml. (males) and 11.5–16.5 g./100 ml. (females). Plasma haemoglobin, 2–5 mg./100 ml. Reticulocyte count, 0.5–1.5%. $T\frac{1}{2}$ chromium-labelled red cells, 25–31 days.

the haemodynamically normal and malfunctioning valve. Hence heterografts do not appear to act as "foreign material" in the circulation. The results are also consistent with the view that a major cause of the haemolytic disorder after valve replacement by artificial valve prostheses is valvular turbulence with mechanical damage to red cells. In addition it appears that foreign materials other than prosthetic valves, such as pacing catheters or Dacron aortic grafts, may be implicated in mechanical haemolysis of red cells.

This study was supported by the National Health and Medical Research Council of Australia. We thank Dr. H. Smith, of the Department of Pathology, Royal Brisbane Hospital, and Dr. B. J. Stephens, of the Department of Pathology, Chermiside Hospital, for their co-operation and help, and Drs. G. H. Neilson and E. G. Galea for allowing us to study patients under their care.

REFERENCES

- Bell, R. E., Petuoglu, S., and Fraser, R. S. (1967). *Brit. Heart J.*, **29**, 327.
 Brodeur, M. T. H., Sutherland, D. W., Koler, R. D., Starr, A., Kimsey, J. A., and Griswold, H. E. (1965). *Circulation*, **32**, 570.
 Eyster, E., Mayer, K., and McKenzie, S. (1968). *Ann. intern. Med.*, **68**, 995.
 Marsh, G. W. (1964). *Lancet*, **2**, 986.
 Nevaril, C. G., Lynch, E. C., Alfrey, C. P., jun., and Hellums, J. D. (1968). *J. Lab. clin. Med.*, **71**, 784.
 O'Brien, M. F., and Clarebrough, J. K. (1966). *Med. J. Aust.*, **2**, 228.
 O'Brien, M. F., and Clarebrough, J. K. (1967). *Lancet*, **1**, 929.
 Pirofsky, B., Sutherland, D. W., Starr, A., and Griswold, H. E. (1965). *New Engl. J. Med.*, **272**, 235.
 Roeser, H. P., Powell, L. W., and O'Brien, M. F. (1968). Unpublished.
 Robinson, R. M., Morrow, A. G., and Gebel, P. (1966). *Amer. Heart J.*, **71**, 179.
 Sayed, H. M., Dacie, J. V., Handley, D. A., Lewis, S. M., and Cleland, W. P. (1961). *Thorax*, **16**, 356.
 Stevenson, T. D., and Baker, H. J. (1964). *Lancet*, **2**, 982.
 Trinder, P. (1956). *J. clin. Path.*, **9**, 170.
 Veall, N., and Vetter, H. (1958). *Radioisotope Techniques in Clinical Research and Diagnosis*. London.

The Drunk in Court: Survey of Drunkenness Offenders from Two London Courts

DENIS GATH,*|| B.M., M.R.C.P., D.P.M.; CELIA HENSMAN,† M.A.; ANN HAWKER‡
 MICHAEL KELLY,‡ M.D., D.P.M.; GRIFFITH EDWARDS,§ D.M., D.P.M.

Brit. med. J., 1968, **4**, 808–811

Summary: A total of 151 men charged with drunkenness with or without aggravations were interviewed immediately after their appearance before the magistrates. The survey was conducted in two Metropolitan courts; one in an area frequented by vagrants, and the other in a mixed middle-class and working-class area.

Few of the offenders were casual roisterers and the majority had a serious drinking problem. Half the offenders showed evidence of chemical dependence on alcohol as determined by morning shakes, morning relief drinking, amnesias, inability to stop drinking, and hallucinatory experiences.

The majority of the offenders were suffering from gross social isolation.

Existing ways of dealing with such men seem inadequate. A rehabilitation service is needed, with hostel accommodation, and particular attention should be paid to first offenders.

Introduction

The problem of society's response to the drunkenness offender is at present much under discussion. A Home Office working party is currently reviewing the question of "the treatment, within the penal system, of persons who habitually commit offences involving drunkenness." Its deliberations have been given urgency by Section 91 of the Criminal Justice Act, 1967 (H.M.S.O., 1967), which proposes that imprisonment for a "drunk and disorderly" offence be abolished as soon as alterna-

tive accommodation is available for the care and treatment of offenders. The first International Congress on the Drunkenness Offence was held in London in May 1968 (Camberwell Council on Alcoholism, 1968). Several speakers there reported a move in other European countries and the U.S.A. to treat the public inebriate as a sick person and to exclude him entirely from the courts and from the criminal process.

Every year in England and Wales there are approximately 75,000 convictions for drunkenness with or without "aggravations," a number which makes heavy demands on the resources of the police and courts. Yet little is known about the kind of person the drunkenness offender is, and the impact on him of arrest and conviction is little understood. The official statistics are limited to an analysis of drunkenness offences by geographical regions, and by sex and age of offenders (H.M.S.O., 1968). Offences of this kind are not recorded centrally, and there is no indication of what proportion of them are caused by habitual offenders and what proportion by "once only" offenders. Prison records are not helpful here, there being no reason to suppose that inebriates in prison are representative of drunkenness offenders as a whole. Previous surveys in the court setting have been carried out in London (Parr, 1962), Rochester, U.S.A. (Zax *et al.*, 1964), and Toronto (Giffen, 1966–8); these studies were of limited scope, as they did not include interviews with offenders but were based on scrutiny of court records, from which only simple demographic data can be drawn.

Recently the *British Medical Journal* (1968) drew attention to the dearth of research information concerning the kinds of people who are arrested for drunkenness, and emphasized that "the proportion of drunkenness offenders who are casual roisterers as opposed to people with serious drinking problems or sufferers from alcohol dependence remains obscure." The answer to this question is relevant to any discussion regarding reform of the law and the design of therapeutic alternatives to fine or imprisonment: the aim of the present investigation was to obtain information bearing directly on this question. More specifically, the aim was to interview a sample of men appearing

* Senior Registrar.

† Research Assistant.

‡ Senior Lecturer, Karolinska Institute, Stockholm.

§ Senior Lecturer.

Institute of Psychiatry, Maudsley Hospital, London S.E.5.

|| Present address: Department of Psychiatry, Queen Elizabeth Hospital, Birmingham 15.

From the Alcohol Impact Project, Addiction Research Unit, Institute of Psychiatry, De Crespigny Park, London S.E.5.