tate ventricular fibrillation (Fig. 7). In this study 7 (35%) of the 20 patients who required atropine for bradyarrhythmia and hypotension developed an inappropriately rapid heart rate, despite careful titration of the dosage.

Studies directed toward the correction of the autonomic disturbance are likely to be more rewarding than the investigation of the prophylactic value of antiarrhythmic agents with an action similar to lignocaine (Lown and Wolf, 1971). Lignocaine may be an unsatisfactory antiarrhythmic agent in the presence of bradyarrhythmia (Lown and Vassaux, 1968; Han, 1969). There is also evidence that it is unsatisfactory in the presence of tachycardia (Geddes *et al.*, 1972).

Among patients who survive the risk of sudden death autonomic disturbance may be important in relation to the size of the area of infarction and therefore to the incidence of cardiogenic shock and pump failure. Patients who come under intensive care early in whom bradycardia and other dysrhythmias are quickly corrected have a low mortality from shock and pump failure (Pantridge, 1970). Furthermore, the incidence of shock among those who get early intensive care is very significantly lower than among those in whom intensive care is delayed more than three hours (Adgey *et al.*, 1971).

There has been clinical and experimental support for the view that the area of infarction may be increased by hypotension after coronary occlusion (Pantridge, 1970; Maroko *et al.*, 1971). Tachycardia also increases the severity and extent of the myocardial ischaemic injury (Maroko *et al.*, 1971). It is therefore interesting that practolol was valuable in controlling the manifestations of sympathetic overactivity which occurred in 46% of patients in this study.

Conclusion

The data presented here show that trying to correct autonomic disturbance after acute myocardial infarction is rewarding. Seventy-two of the 74 patients were aged under 70 and 65 of

them survived the three-week period in hospital—an overall mortality of 9.7%. A mortality of more than 26% might have been expected since 30 of 117 patients in the Bristol study who sought medical help within one hour died (Mather *et al.*, 1971), and it is known that most deaths in the first hour occur within the first 30 minutes (McNeilly and Pemberton, 1968).

References

- Adgey, A. A. J., Geddes, J. S., Mulholland, H. C., Keegan, D. A. J., and Pantridge, J. F. (1968). *Lancet*, 2, 1097.
 Adgey, A. A. J., *et al.* (1971). *Lancet*, 2, 501.
 Bezold, A. von. (1867). *Untersuchungen Physiologisches Laboratorium zu Wurzburg*, 1, 95.
 Ceremuzynski, L., Staszewska-Barczak, J., and Herbaczynska-Cedro, K. (1969). *Cardiovascular Research*, 3, 190.
 Ebert, P. A., Allgood, R. J., and Sabiston, D. C. (1967). *Surgical Forum*, 18, 114.
 Friedberg, C. K. (1972). *Circulation*, 45, 179.
 Fulton, M., Julian, D. G., and Oliver, M. F. (1969). *Circulation*, 40, Suppl. No. 4, p. 182.
 Geddes, J. S., Webb, S. W., and Pantridge, J. F. (1972). Unpublished.
 Han, J. (1969). *American Journal of Cardiology*, 24, 800.
 Han, J., and Moe, G. K. (1964). *Circulation Research*, 14, 44.
 Han, J., de Jalon, P. G., and Moe, G. K. (1964). *Circulation Research*, 14, 516.
 Han, J., Millet, D., Chizzonitti, B., and Moe, G. K. (1966). *American Heart Journal*, 71, 481.
 Levine, H. J. (1969). *American Journal of Cardiology*, 24, 826.
 Levy, M. N., and Zieske, H. (1969). *Journal of Applied Physiology*, 27, 465.
 Lown, B., and Vassaux, C. (1968). *American Heart Journal*, 76, 586.
 Lown, B., and Wolf, M. (1971). *Circulation*, 44, 130.
 McNeilly, R. H., and Pemberton, J. (1968). *British Medical Journal*, 3, 139.
 Malliani, A., Schwartz, P. J., and Zanchetti, A. (1969). *American Journal of Physiology*, 217, 703.
 Maroko, P. R., *et al.* (1971). *Circulation*, 43, 67.
 Mather, H. G., *et al.* (1971). *British Medical Journal*, 3, 334.
 Pantridge, J. F. (1970). *Quarterly Journal of Medicine*, 39, 621.
 Pantridge, J. F., and Adgey, A. A. J. (1969). *American Journal of Cardiology*, 24, 666.
 Pantridge, J. F., and Geddes, J. S. (1966). *Lancet*, 1, 807.
 Pantridge, J. F., and Geddes, J. S. (1967). *Lancet*, 2, 271.
 Peterson, D. F., and Brown, A. M. (1971). *Circulation Research*, 28, 605.
 Richter, C. P. (1959). In *The Meaning of Death*, ed. H. Feifel, chapt. 18. New York, McGraw-Hill.
 Samaan, A. (1935). *Journal of Physiology*, 83, 332.
 Sarnoff, S. J. (1970). Carl J. Wiggers Award Lecture, American Physiological Society.
 Yu, P. N. (1972). *Circulation*, 45, 189.

MEDICAL MEMORANDA

Treatment of Bacterial Endocarditis: Complicating Haemodialysis

LEOPOLDO F. SALDANHA, RALPH GOLDMAN,
KENNETH ADASHEK, DONALD G. MULDER

British Medical Journal, 1972, 3, 92-93

Several reports (Brescia *et al.*, 1966; Goodman *et al.*, 1969; Kurtz, 1969; Ribot *et al.*, 1971; Ribot and Frankel, 1971) have brought attention to the occurrence of bacterial endocarditis as a complication of chronic haemodialysis. We report an additional

case of acute aortic insufficiency secondary to *Staphylococcus aureus* bacterial endocarditis treated successfully with antibiotics and emergency aortic valve replacement. The most probable source of infection was the arteriovenous Teflon-Silastic shunt.

Case Report

The patient was a 33-year-old man in whom a diagnosis of lupus nephritis had been made in 1967. Despite treatment with prednisone, he became progressively uraemic, and haemodialysis was initiated in July 1968.

The arteriovenous shunt was relatively free of complications until December 1970, when surgical revisions were necessary because of poor blood flow and infection. Fever developed and antibiotic treatment was initiated. He had anorexia, orthopnoea, paroxysmal nocturnal dyspnoea, and chest "heaviness" when recumbent for two weeks before admission. On the day of admission he awakened with sudden severe substernal pain. When seen he was in acute respiratory distress. The blood pressure was 140/60, the pulse 90/min. and irregular, and the temperature 40.5°C. There were inspiratory rales in the lung bases. On auscultation a gallop rhythm and a systolic ejection murmur at the aortic area were heard, radiating along the left sternal border and

University of California, Los Angeles, School of Medicine, Los Angeles, California 90024, U.S.A.

LEOPOLDO F. SALDANHA, M.D., Fellow in Nephrology

RALPH GOLDMAN, M.D., Professor of Medicine

KENNETH ADASHEK, M.D., Resident in Medicine (Present address: U.S.P.H.S., San Diego, California)

DONALD G. MULDER, M.D., Professor of Surgery

into the neck; a grade III/VI diastolic decrescendo murmur was heard at the base and left sternal border, with a diastolic rumble at the apex. There were "water-hammer" pulses and a small subungual splinter haemorrhage in the left index finger. The electrocardiogram showed first-degree atrioventricular block, left ventricular hypertrophy, and occasional premature ventricular contractions. A chest roentgenogram showed cardiomegaly and pulmonary congestion. Several blood cultures yielded coagulase-positive *Staph. aureus*, and vancomycin was started. Despite treatment he developed bilateral pneumonic infiltrates, persistent fever, and refractory congestive heart failure.

Operation was performed on the 14th day after admission. The ruptured right cusp of the aortic valve was totally involved in an acute inflammatory process. The deficient leaflet tissue was thickened, oedematous, and boggy, and at the base of the cusp there was an abscess which had burrowed a centimeter or more into the ventricular myocardium. After radical debridement a No. 10 Starr-Edwards valve was inserted with difficulty.

Material from the abscess was found to contain a few Gram-positive cocci, but cultures were negative. Histological examination showed changes of active bacterial endocarditis in a scarred, calcified valve.

There were many complications in the postoperative period, including pulmonary insufficiency requiring tracheostomy and assisted respiration, postcardiotomy syndrome, recurrent pleural effusions, right-sided empyema, and pneumothorax secondary to a bronchopleural fistula. He was discharged after four months in hospital. He died suddenly one year after the operation while on dialysis. At necropsy there was no evidence of active endocarditis and the aortic prosthesis appeared competent.

Comment

The presence of an arteriovenous fistula seems to predispose to experimental and clinical infections. Bacterial endarteritis alone or in association with endocarditis has been reported in congenital, traumatic, and surgically-created internal and external arteriovenous fistulas (Smith *et al.*, 1969). Hence the use of the internal arteriovenous fistula for dialysis does not eliminate the risk of infection, although the incidence is much less than with the external shunt (Ralston *et al.*, 1971). One of the patients in the initial series of Brescia *et al.* (1966) died of aortic and mitral bacterial endocarditis. The source of infection was unknown.

Eight other episodes of bacterial endocarditis have been reported to have developed in seven patients as a complication of chronic haemodialysis (see Table). Two involved the aortic valve alone, three the mitral valve, two the aortic and mitral valves, and one the tricuspid valve. One patient successfully

underwent two valvular operative procedures in the space of one year. Only two of the remaining six cases survived. The tricuspid valve involvement was an unexpected necropsy finding. *Streptococcus viridans* and *Staph. aureus* were the infective organisms in most patients. Infection of the external arteriovenous shunt was identified as the source of infection in four of the cases. Two other cases of cardiac valve replacement in patients undergoing haemodialysis were described by Lansing *et al.* (1968). The first patient had enterococcal aortic valve endocarditis as a complication of peritoneal dialysis, but the aortic insufficiency was diagnosed only when he was on maintenance haemodialysis. The other patient had rheumatic mitral insufficiency. A case of rheumatic aortic valve replacement was reported by Lewis *et al.* (1971). All three had tolerated cardiac surgery very well.

Bacterial endocarditis still causes a high mortality despite the appreciable improvement since the advent of antibiotics. The major cause of death is intractable congestive heart failure (Wise *et al.*, 1971). The surgical results have improved after valve replacement in patients with endocarditis, including those in whom infection has not been controlled preoperatively. The presence of uraemic complications, including the bleeding tendency, defective wound healing, anaemia, and the multiple drug therapy for arterial hypertension in most patients increases the risk of operation. Yet, with proper care, haemodialysis patients can tolerate major operations, including cardiac surgery.

It appears that patients on maintenance haemodialysis have a reasonable chance of survival after heart valve replacement. This treatment should be considered in the presence of intractable heart failure resulting from bacterial endocarditis.

References

Brescia, M. J., Cimino, J. E., Appel, K., and Hurwicz, B. J. (1966). *New England Journal of Medicine*, 275, 1089.
Goodman, J. S., Crews, H. D., Ginn, H. E., and Koenig, M. G. (1969). *New England Journal of Medicine*, 280, 876.
Kurtz, D. M. (1969). *New England Journal of Medicine*, 280, 1302.
Lansing, A. M., Leb, D. E., and Berman, L. B. (1968). *Journal of the American Medical Association*, 204, 134.
Lewis, E. J., Cavallo, T., Harrington, J. T., and Cotran, R. S. (1971). *Human Pathology*, 2, 185.
Ralston, A. J., Harlow, G. R., Jones, D. M., and Davis, P. (1971). *British Medical Journal*, 3, 408.
Ribot, S., and Frankel, H. J. (1971). 5th Annual Meeting of the American Society of Nephrology, Washington, D.C., p. 63.
Ribot, S., Gilbert, L., Rothfeld, E. L., Parsonnet, V., and Jacobs, M. G. (1971). *Journal of Thoracic and Cardiovascular Surgery*, 62, 59.
Smith, C. M., Morton, J. D., and Thompson, R. D. (1969). *Journal of the American Medical Association*, 210, 131.
Wise, J. R., jun., *et al.* (1971). *Lancet*, 2, 115.

Summary of Reported Cases of Bacterial Endocarditis as a Complication of Chronic Haemodialysis

Author	Age and Sex	Valve Involved	Organism	Source of Infection	Treatment	Outcome
Brescia <i>et al.</i> (1966)	28 N.S.	Mitral and aortic	N.S.	Unknown	N.S.	Died
Goodman <i>et al.</i> (1969)	32 M.	Aortic	Enterococcus	Dental procedure	Antibiotics	Died
Kurtz (1969)	45 F.	Aortic	<i>Str. viridans</i>	Dental procedure	Antibiotics	Survived
	22 M.	Tricuspid*	N.S.	External arteriovenous shunt	Antibiotics	Died
	33 F.†	Mitral	<i>Staph. aureus</i>	External arteriovenous shunt	Antibiotics and valve replacement	Survived
Ribot <i>et al.</i> (1971)	N.S.	Mitral (paravalvular leak)	N.S.	External arteriovenous shunt	Surgical repair	Survived
		Mitral	<i>Staph. aureus</i> and <i>Str. viridans</i>	External arteriovenous shunt	Antibiotics	Survived
Present report	N.S.	Mitral and aortic	<i>Str. viridans</i>	N.S.	N.S.	Died
	33 M.	Aortic	<i>Staph. aureus</i>	External arteriovenous shunt	Antibiotics and valve replacement	Survived

* Necropsy finding.
† Two episodes in the same patient.
N.S. = Not stated.