PRELIMINARY COMMUNICATIONS

Ultrasonic Decalcification of Calcified Cardiac Valves and Annuli

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Summary

Heavily calcified annuli increase the incidence of complications after prosthetic valve replacement—heart block, separation of the aorta or the atrium from the ventricle, late aneurysm formation, paravalvular leak, and haemolysis. An ultrasonic calculus-disintegrator has been developed to remove calcific deposits. The instrument is portable, robust, easily sterilized, inexpensive, and provides nebulized water at the ultrasonic tip which keeps the tissues cool, helps to break up the calculus by cavitation, and washes the calcific debris into the sucker. Preliminary trial on excised calcific valves showed the ultrasound instrument to be capable of removing most forms of calcification. In clinical prosthetic replacement of valves it enabled good clearance of the annulus to be performed in six out of seven cases, in one of which earlier operation had been unsuccessful because of calcification. Two elderly patients with pure calcific aortic stenosis were successfully treated by debridement of the aortic valve with ultrasound.

Introduction

Replacement of cardiac valves in which calcification extends deeply into the annuli is, with current techniques, more often followed by complications than when the valves are uncalcified. The techniques of crushing and dissection of the calcific masses may damage adjacent cardiac conductive tissue (Gannon *et al.* 1966), detach the aorta or the atrium from the ventricle, or, if inadequate, predispose to paravalvular leaks (Wheat *et al.*, 1966; Danielson *et al.*, 1967; Favaloro *et al.*, 1967; Kastor *et al.*, 1968; MacVaugh *et al.*, 1969; Singh and Horton, 1971). A means of removing the calcification without adjacent damage or loss of suture-holding tissue is needed.

No satisfactory substitute for cardiac valves yet exists; McGoon's (1971) choice was soon discredited (Welch *et al.*, 1971; McEnany *et al.*, 1972). Valvotomy, even for calcified mitral valves, gives longer survival than replacement (Gerami *et al.*, 1971; Olinger *et al.*, 1971). The early use of debridement of the aortic valve, seen at a time when it had to be applied to all cases (Dong *et al.*, 1962), has since been justified (Hancock *et al.*, 1965). Although replacement is generally preferred (Hurley *et al.*, 1967) the advantages of retaining the patient's own tissue, with minimal risk of infection, thrombosis, haemolysis, or disruption, make operative debridement a realistic approach to calcific aortic stenosis (Enright *et al.*, 1971). Some means of increasing the safety, range of application, and thoroughness of this procedure is needed.

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Disintegration of Calcific Masses by Ultrasound

Ultrasound is vibration at frequencies above the human aural range. High-frequency vibration makes the inertia of small masses relatively more important than coherent forces, so it can be used to loosen dirt, push piles through clay, or break up cells. Hard, brittle material does not absorb and "damp out" these vibrations as does soft tissue, and can thus be broken up by them. This application was known in biblical times (Joshua VI, 20).

It has seemed for several years that the solution to the problem of heavy calcification of the cardiac valves could be an ultrasonic probe, preferably with some form of cooling and flushing to avoid thermal damage to the conduction bundle and to keep the operative field clear of debris. Dentists use such equipment to remove calculus from around the roots of teeth without damage to adjacent soft tissues. We thought that this apparatus might be a basis for development. Minor modifications have provided easier sterilization of both probe and water supply, enabling the normal dental unit to be applied to cardiac surgery. Modifications of the power available and the frequency of vibration are at present being tried, but we report here our experience with the standard unit, which provides 2·5 watts of power at frequencies around 25 kHz.* The apparatus is inexpensive, robust, and portable, and the handpiece can be held like a pen (see Fig. 2). There are many different tips available. We have found a slightly curved sharp tip best for the preparation of beds for valve replacement, and a similarly curved flat-ended one safer for the removal of calcification in valve debridement. Water is fed along the tip and turned into an aerosol mist by the vibration; this prevents damage to the paravalvular tissue from heat and carries away the calcific debris, which is removed by constant suction. The ultrasound induces a cavitation effect in the water which helps to break up the calculus.

Preliminary Trial

Valves excised during the course of prosthetic replacement were used. Calcification was either in solid masses displacing the tissues or in laminae closely interwoven into the fibres of the skeleton of the valves. The latter form was rather less brittle and seemed to contain more organic and less mineral material. Although we did not see solid masses of this form in the excised specimens later clinical experience showed that it could exist in pieces as big as a molar tooth extending deeply into the ventricular wall. The overlying endothelium always separated easily from the calcification.

Six valves—three mitral and three aortic—were decalcified by ultrasound. Solid, ripe condensations of predominantly mineral calcification were easy and quick to remove, but interstitial cartilaginous deposits were more difficult. This latter form, however, does not alone render valvular function inadequate or make the insertion of sutures difficult. None of the normal tissues of the valves were damaged, including fibrous tissue which had been closely mingled with the calcified material. Care had to be taken to avoid tears from the sharp-tipped probe. Only slight warmth developed in the tissues.

Clinical Trial

AID TO PROSTHETIC REPLACEMENT

Ultrasound was used for clearing deep-seated calcification from the bed of the valve in seven patients, four having mitral valve

* "Dental Sonic" scaling unit, Dental Supplies Ltd., London W.11.

replacement and three aortic valve replacement. In only one case was the technique unsuccessful; a large mass was present at the posteromedial commissure of a mitral valve, extending down into the ventricular wall. This mass was of the consistency of cortical bone rather than mineral chalk and did not break into rubble in the usual way when subjected to ultrasound. It was partially removed by conventional techniques to allow a prosthesis to be sewn into place, but the patient bled from the atrioventricular groove and required reoperation for haemostasis on the same evening. In all the other cases ultrasonic decalcification facilitated the clearance of heavy annular deposits, leaving strong, intact fibrous tissue for attachment of the prosthesis. In one patient a previous mitral valve replacement had been prolonged and inadequate because of deep-seated calcification of the annulus. A quarter of the circumference of the prosthesis had almost immediately become detached, and at the time of reoperation he was in intractable congestive cardiac failure. At the second operation the hindering calcific mass was quickly and easily removed with the ultrasonic probe, leaving a perfect bed for a new prosthesis, which was securely affixed.

COMPLETE DEBRIDEMENT

In two patients calcified stenotic aortic valves were treated by removal of the calcific material by means of ultrasound with, in addition, limited commissurotomy made possible by removal of calculus.

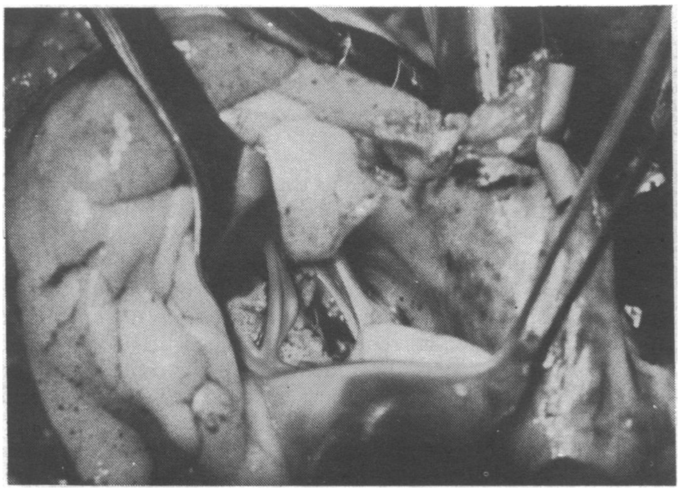

FIG. 1—Case 1. Heavily calcified, stenotic aortic valve. The cusps were firmly approximated by calcific deposits at their bases.

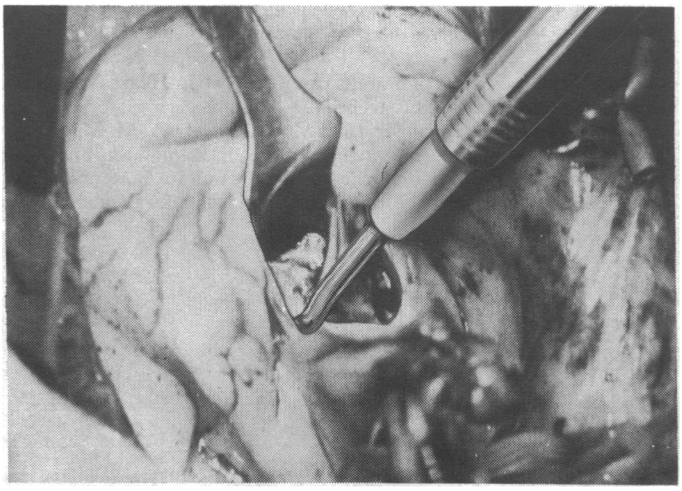

FIG. 2—Case 1. Probe of ultrasonic instrument being used to undermine a calcific plaque.

Case 1.—The patient, a 73-year-old woman, had developed grade 3 dyspnoea over the preceding two years and had recently had two attacks of nocturnal dyspnoea and angina decubitus. She had clinical evidence of aortic stenosis. *X*-ray examination showed pronounced ventricular enlargement and a large ascending aorta with calcification of the aortic valve. On 25 November 1971 aortic valve debridement was performed. The valve was tricuspid with fusion of the right and left coronary cusps and heavy deposits of calcific material in the fused commissure and bases of all the cusps (Fig. 1). The cusps could be separated by only ½ cm but they met well and there was no evidence of regurgitation. The calcific deposits were removed with the ultrasonic probe (Fig. 2), leaving raw but mobile cusp tissue (Fig. 3). The commissure between the coronary cusps could then be cut back by 4 mm. The cusps were intact and not perforated and the valve opened fully, permitting a good view into the ventricle. No regurgitation into the "vent" was seen after unclamping the aorta before defibrillation. Recovery was uneventful and there were no cardiac symptoms three months later. The pulse wave was normal, the blood pressure 190/100 mm Hg, and the aortic murmur was only a soft ejection souffle without thrill and no longer perceptible to the patient. There was no early diastolic murmur (Fig. 4). She was discharged without medication to South Africa.

Case 2.—This patient was a 64-year-old man whose symptoms had begun four years previously. He was admitted in left ventricular failure and angina decubitus. Examination and investigation showed aortic

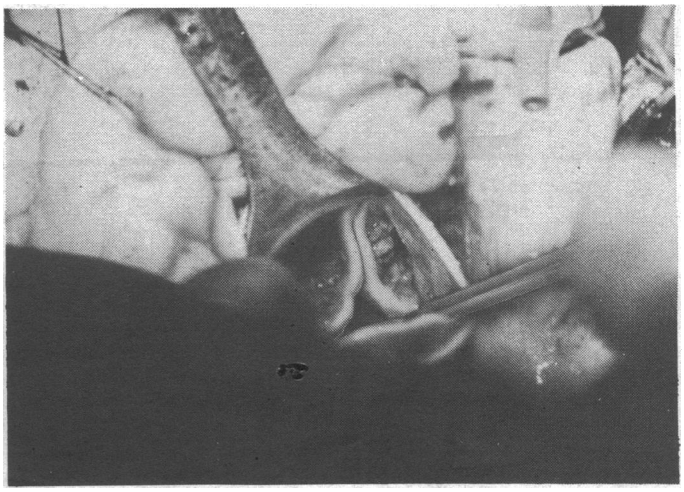

FIG. 3—Case 1. Result of ultrasonic decalcification. Normal-appearing valve with granular areas remaining where calcium had been.

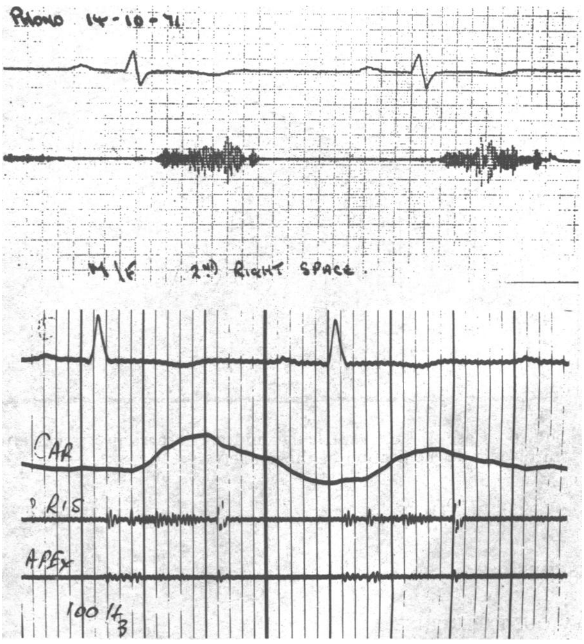

FIG. 4—Case 1. Phonocardiogram before (above) and after (below) ultrasonic decalcification of the aortic valve.

stenosis with left ventricular hypertrophy and strain. The aortic valve was moderately severely calcified and there was a gradient of 80 mm Hg across it (Fig. 5). On 3 January 1972 ultrasonic aortic decalcification was performed. The valve was bicuspid, with moderately heavy calcification at the bases of the cusps and in the rudimentary commissure at the midpoint of the anterior cusp. Some of the work was done with the sharp-pointed probe, which penetrated the base of the anterior cusp; this was easily closed with a single stitch, as the adjacent cusp tissue was undamaged by the ultrasound. Removal of the calcification enabled a limited incision to be made in the rudimentary anterior commissure; after this the valve opened wider and the cusps were adequately mobile. Recovery was uneventful, and two months after surgery the patient was living a normal life. Follow-up investigation showed a gradient of only 24 mm across the aortic valve (Fig. 6) and minimal regurgitation.

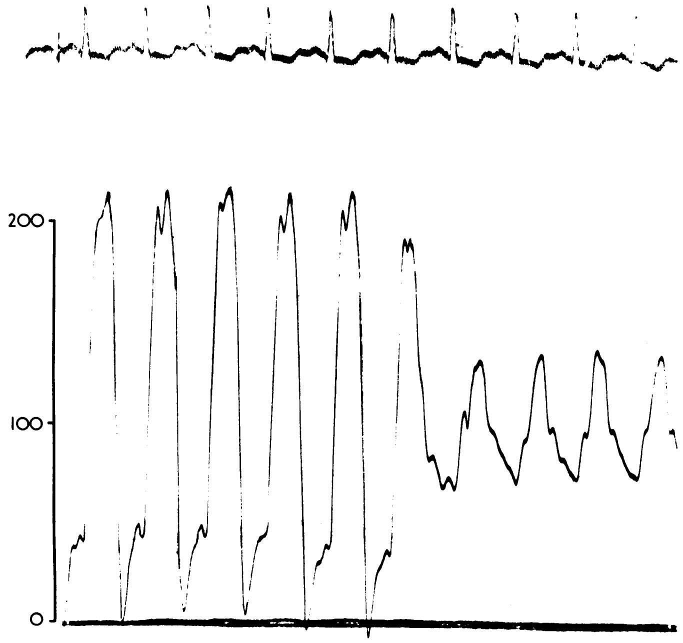

FIG. 5—Case 2. Left ventricular and aortic pressure traces before operation.

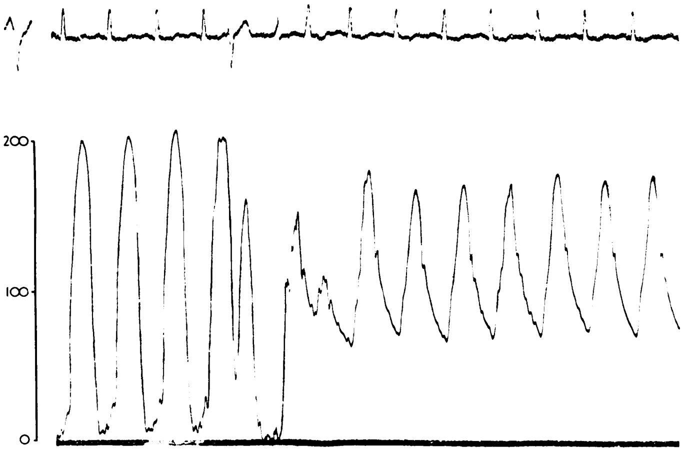

FIG. 6—Case 2. Left ventricular and aortic pressure traces after ultrasonic decalcification of the aortic valve.

Discussion

PREVIOUS EXPERIENCE

The advent of any new basic technique makes previous experience less relevant. Though the results of valvular replacement in the presence of heavy calcification are quite good the incidence of paravalvular leak or adjacent tissue damage is still higher than that in uncalcified cases. Reducing the incidence of prosthetic detachment by more extensive removal of calculus hitherto increased the likelihood of adjacent damage and heart block. More adequate and selective techniques of dealing with calcification were thus needed.

With previously available methods debridement compared favourably with replacement of the aortic valve at three years but not after five (Scannell and Austen, 1964). Advocates of debridement claimed that so long as some reduction, however little, in the gradient was achieved excellent palliation could be obtained (Enright *et al.*, 1971). Perhaps now more nearly complete elimination of gradients and consequent good long-term results can be achieved by ultrasonic restoration of valvular architecture.

CHOICE OF PATIENTS

Although the average age of some patients treated by aortic valve debridement was 48 years (Enright *et al.*, 1971), sufficient doubt of the long-term efficacy made us limit debridement to elderly patients. This policy was justified by those who otherwise favoured valve replacement (Scannell and Austen, 1966). The benefits of shorter cardiopulmonary bypass and myocardial ischaemia, the lower inertia of a natural valve compared with a ball prosthesis, and the lack of necessity for anticoagulation should be particularly valuable to older patients.

At present patients with valvular regurgitation are not thought suitable for debridement, but continuing favourable results may gradually change this restriction.

FURTHER DEVELOPMENT

The equipment now in use needs some modification to enable it to deal with the less mature, more organic bony form of calcification. This form is usually mixed with the fibrous stroma where, even if not fully removed, it does not preclude adequate movement of cusp or sound placement of stitch; but it can occur in solid masses. More power than the present 2·5 watts may be all that is necessary for its removal. To change the frequency of the ultrasound not only the generator but the cable, windings, and "core" of the handpiece must be altered. Trials of various frequencies are thus difficult, but eventually the optimum power and frequency should be built into the definitive cardiac version of this equipment. The ideal of a probe which can dissolve calcification as fast as the sucker can remove the debris cannot be too far away.

Ultrasound can be carried along the core of a catheter. The endoscopic destruction of calculi in the urinary and biliary systems is therefore within reach.

We are the recipients of a grant from the British Heart Foundation for the investigation of this technique. We are very grateful for the encouragement and help of Mr. B. P. Moore and Mr. A. M. Macarthur, on whose patients the equipment was used. Dental Supplies Ltd. kindly supplied and modified the apparatus and are now proceeding with investigations into the optimum power and frequency for our purposes.

References

Danielson, G. K., Cooper, E., and Ifuku, M. (1967). *Journal of Thoracic and Cardiovascular Surgery*, **53**, 858.
Dong, E., jun., Hurley, E., Hancock, E. W., Stofer, R. C., and Shumway, N. E. (1962). *Surgery*, **52**, 720.
Enright, L. P., Hancock, E. W., and Shumway, N. E. (1971). *Circulation*, **43**, Suppl. No. 1, p. 68.
Favaloro, R. G., Effler, D. B., Groves, L. K., Suarez, E., and Shirey, E. K. (1967). *Annals of Thoracic Surgery*, **3**, 503.

Gannon, P. G., Sellers, R. D., Kanjuh, V. I., Edwards, J. E., and Lillehei, C. W. (1966). *Circulation*, **33**, Suppl. No. 1, p. 152.
Gerami, S., Messmer, B. J., Hallman, G. L., and Cooley, D. A. (1971) *Journal of Thoracic and Cardiovascular Surgery*, **62**, 366.
Hancock, E. W., Hultgren, H. N., and Shumway, N. E. (1965). *Surgery, Gynecology and Obstetrics with International Abstracts of Surgery*, **120**, 770.
Hurley, P. J., Lowe, J. B., and Barrett-Boyes, B. G. (1967). *Thorax*, **22**, 314.
Kastor, J. A. *et al.* (1968). *Journal of Thoracic and Cardiovascular Surgery*, **56**, 289.
McEnany, M. T., Ross, D. N., and Yates, A. K. (1972). *Thorax*, **27**, 23.
McGoon, D. C. (1971). *British Heart Journal*, **33**, Suppl., p. 35.
MacVaugh, H., Joyner, C. R., Pierce, W. S., and Johnson, J. (1969). *Journal of Thoracic and Cardiovascular Surgery*, **58**, 291.
Olinger, G. N., Rio, F. W., and Maloney, J. V. (1971). *Journal of Thoracic and Cardiovascular Surgery*, **62**, 357.
Scannell, J. G., and Austen, W. G. (1964). *New England Journal of Medicine*. **270**, 96.
Scannell, J. G., and Austen, W. G. (1966). *Circulation*, **33**, Suppl. No. 1, p. 131.
Singh, H. M., and Horton, E. H. (1971). *Thorax*, **26**, 89.
Welch, W., Potvliege, P., and Primo, G. (1971). *Thorax*, **26**, 271.
Wheat, M. W., *et al.* (1966). *Annals of Thoracic Surgery*, **2**, 166.

MEDICAL MEMORANDA

Severe Hypothyroidism—an Early Complication of Lithium Therapy

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It is well recognized that treatment with lithium may cause thyroid abnormalities. Non-toxic goitre and impairment of thyroid function detectable by biochemical tests but without clinical manifestations have been reported. Overt hypothyroidism is uncommon. The following case is noteworthy in view of the rapid development and subsequent regression of clear-cut hypothyroidism during and after a short course of lithium treatment.

Case Report

A 48-year-old woman was admitted to hospital suffering from her fourth depressive illness in 10 years. Several milder depressive swings and hypomanic episodes had also been observed. She had no family or personal history of thyroid disorder. The admitting doctor queried the possibility of delayed tendon reflexes (not confirmed by other observers) and requested a P.B.I. estimation, which was 5·8 μg (normal 3·3-7·8 μg)/100 ml. There were no other symptoms or signs of thyroid disease. The E.S.R. was 3 mm in one hour. The depression remitted in hospital without specific treatment, but in view of the history of a recurrent affective disorder lithium carbonate was prescribed. Satisfactory blood levels were established without difficulty and she was discharged apparently well.

Seven weeks after beginning lithium, however, she complained of mild depression, tingling in the hands, and a hoarse voice. She returned a week later, when she was unequivocally myxoedematous, complaining of drowsiness and undue sensitivity to cold, with swelling of face, fingers, and ankles, moderate soft thyroid enlargement, a hoarse voice, exertional dyspnoea, bradycardia, and definitely delayed tendon reflexes. The P.B.I. was 0·8 μg/100 ml, and the E.S.R. 4 mm in one hour. She had stopped lithium three days previously. No treatment was prescribed, and she was closely observed as an outpatient. Two months later she was clinically euthyroid except for a mild improving bilateral carpal tunnel syndrome. Three months after stopping lithium the P.B.I. was 3·4 μg/100 ml, and after a further four months it had risen to 4·0 μg/100 ml. During the subsequent two years she required medical attention for mild depressive episodes, none of which necessitated admission. She otherwise remained well, and on examination 26 months after stopping lithium was without any symptoms or signs of thyroid disorder except for a unilateral mild carpal tunnel syndrome. At that time the P.B.I. was 4·2 μg/100 ml.

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Comment

Lithium administration may be followed, sometimes within a few weeks, by a decrease in the amount of thyroxine secreted by the thyroid (Sedvall *et al.*, 1968; Shopsin, 1970). A few patients develop goitre, usually as a "late" side effect occurring after months or years of lithium therapy (Schou *et al.*, 1968). There have been few reports of overt hypothyroidism. Wiggers (1968) reported the occurrence of clinical and biochemical myxoedema in a patient five months after beginning lithium. Shopsin *et al.* (1969) described the case of a middle-aged woman who was found to be hypothyroid with a goitre after taking lithium for two years. Investigation suggested that she suffered from an underlying chronic thyroiditis. Rogers and Whybrow (1971) described two cases of clinical hypothyroidism, one developing after 20 months and the other after three months of lithium therapy. Both patients, however, had received a variety of other psychotropic drugs, and in the second case symptoms suggestive of hypothyroidism were noted before treatment with lithium was begun.

While these studies suggest that the thyroid abnormalities associated with lithium therapy are not only unusual but relatively mild, this case shows that severe and potentially dangerous hypothyroidism can develop rapidly in a patient in whom there was no clinical or biochemical evidence before treatment began to suggest thyroid insufficiency. It adds pertinence to the request of the U.S. Federal Food and Drug Administration that thyroid function be assessed both before lithium treatment and at regular intervals while it continues. Perhaps this procedure should be adopted more widely in Britain.

I should like to thank Dr. R. H. Cawley, under whose care the patient was admitted, for permission to report this case and for helpful criticism.

References

Rogers, M. P., and Whybrow, P. C. (1971). *American Journal of Psychiatry*, **128**, 158.
Schou, M., Andisen, A., Eskjaer Jensen, E., and Olsen, T. (1968). *British Medical Journal*, **3**, 710.
Sedvall, G., Jönsson, B., Pettersson, U., and Levin, K. (1968). *Life Sciences*, **7**, 1257.
Shopsin, B., Blum, M., and Gershon, S. (1969). *Comprehensive Psychiatry*, **10**, 215.
Shopsin, B. (1970). *Diseases of the Nervous System*, **31**, 237.
Wiggers, S. (1968). *Ugeskrift før Laeger*, **130**, 1523.