have not found this to be so.¹⁷ We have not used it, as its usefulness in preventing crisis has not been established. It has been suggested that bicarbonate should be used if the arterial pH shows some degree of acidosis.² But in developing countries, at least, facilities for blood gas analysis are rarely available.

Exchange transfusion has been recommended for the treatment of preoperative anaemia¹⁴ and as a safe method of managing anaesthesia and surgery.¹⁸ This is not practicable in regions where the incidence of haemoglobinopathies is highest, as the availability of blood is low. Furthermore, it is doubtful if it is necessary.

Urea has been used to prevent and treat sickle-cell crisis.^{19, 20} Urea breaks down hydrophobic bonds and thereby breaks down the crystals of haemoglobin S. This approach has been criticized because of the dehydration it may produce²¹ and the problems of giving a large urea load to patients whose kidneys are already badly compromised.²² This is currently the object of controlled trials.²² At present it does not seem to have a place in anaesthetic management.

It is clear that with straightforward anaesthesia and good post-operative care general anaesthesia is safe in patients with sickle-cell states without the need to resort to other measures.

Appendix

Since this series was completed a patient with sickle-cell anaemia has been anaesthetized while in crisis.

A girl, aged 10 years was admitted with a five-day history of abdominal pain, fever and vomiting. On examination she was dehydrated and pale and had a temperature of 102°F (38.9°C) and a pulse rate of 130/min. The abdomen was distended, rigid, and tender. The liver was not palpable but the spleen was very large. A provisional diagnosis of enteric fever or sickle-cell crisis with splenic infarct was made. Sickling was positive and haemoglobin electrophoresis showed haemoglobin SS.

Five days after admission the haemoglobin concentration fell from 6 to 4.7 g/100 ml. Her condition then deteriorated rapidly and a diagnosis of splenic abscess was made. She was given 2 units of non-sickling blood, and the next day splenectomy was performed under general anaesthesia. The spleen contained multiple infarcts and abscesses. Two units of blood were given during the operation.

She made a rapid recovery and was discharged two weeks after operation.

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Hospital Topics

Surgical Aspects of Bacterial Endocarditis

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Summary

Forty patients with a previous history of bacterial endocarditis were treated surgically between December 1967 and August 1971. Of 28 patients who had elective valve replacements there were four hospital deaths and one late death. Seven patients underwent emergency operation for intractable heart failure before completion of

antibiotic treatment, six survived operation and there was one late death. Six patients had operations for infection on pre-existing valve substitutes, of whom three were treated as emergencies. There were two hospital and no late deaths. 78% of all patients were alive and well four years to nine months after operation.

These results confirm that in addition to elective valve replacement surgery has an important role both in the treatment of intractable heart failure during the infective stage of bacterial endocarditis and in the eradication of infection on cardiac prostheses.

Introduction

Prognosis in bacterial endocarditis improved dramatically with the introduction of effective chemotherapy.¹ The development

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of valve replacement surgery a decade ago further increased survival among those patients who would otherwise have died from the haemodynamic consequences of the disease. Occasionally valve destruction proceeds so rapidly that heart failure supervenes before infection can be controlled. At first surgeons were reluctant to operate in a potentially infected field, and only recently have reports indicated that emergency surgical treatment carries a lower risk than persisting with medical treatment until definite eradication of infection can be assured.²⁻⁵

Cardiac surgery has also introduced new problems to the subject, and infective endocarditis on a valve prosthesis is difficult to cure by conventional means. The responsible organism is often a staphylococcus or a fungus, and mortality among these patients remains high.⁶⁻⁸

This report describes the management of 40 patients with a previous history of bacterial endocarditis who were treated surgically at the National Heart Hospital between December 1967 and August 1971.

Patients and Methods

Altogether 35 male and 5 female patients were studied. Their ages ranged from 12 to 68 years, with a maximal incidence in the fourth, fifth, and sixth decades. Seventeen patients had a previous history of rheumatic fever, six congenital aortic stenosis, and six infections on pre-existing valve substitutes. Twelve patients had no previous history of heart disease. One patient had a homograft aortic valve replacement after bacterial endocarditis on a rheumatic valve and later developed endocarditis on the homograft, which was replaced at a second operation. Forty-one operations were therefore performed.

INCIDENCE OF ENDOCARDITIS AND BACTERIAL DIAGNOSIS

Fifty-eight attacks of endocarditis were recorded among the 40 patients. Two patients had four attacks, one had three attacks, and 10 had two attacks. The remainder had a single episode of infection.

The diagnosis was confirmed by positive blood culture on 40 occasions; there were negative blood cultures in 12 instances and in six no data were available. The commonest reason for negative blood culture was the administration of antibiotics before the diagnosis had been suspected. The causative organisms, where known, are listed in Table I. Streptococcal infection accounted for 70% of the cases. There were four staphylococcal infections and two fungal infections. One patient had rickettsial endocarditis, the diagnosis being confirmed by a positive complement fixation test.

TABLE I—Causative Organisms among the 40 Cases

	No. of Cases		No. of Cases
<i>Streptococcus</i>	28	<i>Pneumococcus</i>	1
<i>Str. viridans</i>	23	<i>Haemophilus</i>	1
<i>Str. faecalis</i>	4	<i>Klebsiella cloacae</i>	1
<i>Str. sanguis</i>	1	<i>Rickettsia burneti</i>	1
<i>Staphylococcus</i>	4	<i>Diphtheroids</i>	1
<i>Staph. aureus</i>	2	<i>Atypical coliform</i>	1
<i>Staph. albus</i>	2	<i>Candida</i>	2

ANTIBIOTIC THERAPY

When the diagnosis was known most patients received a six-week course of the appropriate antibiotics. The exceptions were those in whom intractable heart failure supervened at an early stage of the illness, necessitating emergency operation before completion of a full course of chemotherapy (see below). Patients with negative blood cultures were usually treated with parenteral penicillin and streptomycin with or without procainicid.

INDICATIONS FOR OPERATION

There were three main indications for operation, enabling the patients to be classified into three groups.

Group 1.—Twenty-eight patients were operated on electively at varying intervals after successful eradication of the original infection. All were symptomatic at the time of operation and most had radiological evidence of increasing cardiomegaly and a deteriorating electrocardiogram. Sixteen had been in left ventricular failure and three were resuscitated from attacks of ventricular fibrillation two, three, and four days before operation.

Group 2.—Seven patients underwent emergency operation for intractable heart failure before completion of a full course of antibiotics. The duration of chemotherapy varied between 6 and 42 days with a mean of 21 days. All these patients were critically ill at the time of operation and had experienced at least one attack of frank pulmonary oedema.

Group 3.—There were six patients in whom the indication for operation was removal of one or more infected valve substitutes. The interval between first and second operations varied between four months and five years. All had been treated with at least one course of antibiotics. One had evidence of haemolytic anaemia due to a paraprosthetic leak and three of the six operations were conducted as emergencies because of recurrent pulmonary oedema.

Four of the 40 patients had definite systemic embolism at some stage of their illness, and this diagnosis was suspected in five others. In only one instance did embolism constitute an indication for operation. This was in a man who developed acute mitral regurgitation with pulmonary oedema after *Streptococcus viridans* endocarditis and ruptured chordae tendineae. Emergency mitral valve replacement was followed by left femoral embolectomy.

PREOPERATIVE PREPARATION AND OPERATIVE TECHNIQUE

No special preparation was given to patients having elective valve replacements. A course of intramuscular cloxacillin (500 mg six-hourly) and either streptomycin (0.5 g twice daily) or gentamicin (2 mg/kg body weight twice daily) was started the night before operation and continued for five days.

Patients undergoing emergency operation required careful preoperative management. The main problems encountered were those of left ventricular failure, dysrhythmias, and pulmonary dysfunction. Acid-base abnormalities were corrected where possible and some patients required inotropic support with an isoprenaline infusion. Anaesthetic advice was sought at an early stage, as patients with intractable pulmonary oedema were found to benefit from a short period of intermittent positive-pressure ventilation before operation. Catheters for monitoring venous and arterial pressures were placed under local anaesthesia, and general anaesthesia was not induced until surgeons, theatre staff, and heart-lung machine team were fully prepared to receive the patient. Operation was performed through a median sternotomy incision and normothermic cardiopulmonary bypass was established with arterial return to the ascending aorta. Bilateral coronary perfusion was used for aortic valve surgery, whereas most mitral procedures were conducted during anoxic arrest of the heart.

POSTOPERATIVE ANTIBIOTIC THERAPY

Patients in group 1 were given five to seven days' prophylactic antibiotic treatment with cloxacillin and either streptomycin or gentamicin. Cephaloridine (500 mg intramuscularly six-hourly) was given to three patients who were penicillin-sensitive.

No consistent policy was followed for patients operated on before completion of a regular course of antibiotics. Four received postoperative antibiotics for five days only, one for two weeks, and one, who was operated on only seven days after

starting chemotherapy, was given cloxacillin and streptomycin together with penicillin (30 megaunits intravenously daily) for six weeks after operation. The patient who had rickettsial endocarditis was treated postoperatively with tetracycline for four months. This drug was reinstituted one month later after a relapse and continued until she died one year after operation.

The patients in group 3 received antibiotics for five days postoperatively, the only exception being a woman with fungal endocarditis who, in addition, was given flucytosine (10 g by mouth daily) for four weeks after operation.

Results

The procedures performed and the results of operation in the three groups are given in Tables II-IV.

Group 1 (Table II).—Of the 28 patients who had elective valve replacements 15 had isolated aortic valve replacements, 9 had aortic and mitral valve procedures, 3 had ventricular

TABLE II—Results of Elective Operations in Group 1 (28 Patients)

	No. of Patients	Hospital Deaths	Late Deaths
Isolated aortic valve replacement:			
Homograft	10	1	
Starr-Edwards valve .. .	3		
Autograft	2		
Aortic and mitral valve replacement	6	3	
Aortic replacement and mitral valve repair	3		
Aortic valve replacement and closure of ventricular septal defect	3		1
Mitral valve replacement (homograft)	1		
Total	28	4	1

Autograft is replacement of aortic valve with pulmonary autograft and replacement of excised pulmonary valve with aortic homograft.

TABLE III—Results of Emergency Operations for Intractable Heart Failure in Group 2 (7 Patients)

	Duration of Preoperative Chemotherapy (Days)	Hospital Death	Late Death
Aortic and mitral valve replacement (Starr-Edwards valves) .. .	42		
Aortic valve replacement and resection of ascending aortic aneurysm ..	14		
Aortic valve replacement (Starr-Edwards valve) .. .	6	1	
Mitral valve replacement (fascia lata)	35		
Aortic and mitral replacement (Starr-Edwards valves) .. .	9		1
Mitral valve replacement (Starr-Edwards valve) .. .	9		1
Mitral valve replacement (Starr-Edwards valve) .. .	38		
Aortic valve replacement (Starr-Edwards valve) .. .	7		
Total	(Mean 21)	1	1

TABLE IV—Results of Operation for Replacement of Infected Valve Substitutes in Group 3 (5 Patients)

	Infecting Organism	Interval between First and Second Operations (Months)	Hospital Deaths	Late Death
Mitral Starr-Edwards prosthesis with fascia lata valve .. .	<i>Staph. aureus</i>	7		
Aortic homograft with Starr-Edwards prosthesis .. .	No culture	18	1	
Mycotic aneurysm aortic root, previous aortic and mitral Björk valves (E) .. .	No culture	4	1	
Aortic homograft with Starr-Edwards prosthesis (E) .. .	<i>Str. viridans</i>	60		
Mitral homograft with Starr-Edwards prosthesis (E) .. .	<i>Str. viridans</i>	28		
Aortic, mitral, and tricuspid Starr-Edwards prostheses ..	<i>Str. viridans</i> <i>Staph. aureus</i> , <i>C. parakrusei</i> , <i>C. parapsilosis</i>	22		
Total			2	

E = Emergency operation.

septal defects closed at the same time as aortic valve replacement, and 1 had an isolated mitral valve replacement with an inverted aortic homograft. There were four hospital deaths, three of which were in patients who had both mitral and aortic valves replaced, and one late death unrelated to heart disease. The mortality was thus 18%.

Group 2 (Table III).—In four instances operation was performed less than 15 days after the institution of chemotherapy. There was one hospital death. Another patient died one year after having mitral and aortic valve replacement for rickettsial endocarditis. In this case it was thought probable though not proved that the infection persisted postoperatively. The mortality in this group was 29%.

Group 3 (Table IV).—In three instances infection occurred on homograft valves, whereas in the other three cases prosthetic valves were involved. Three of the six operations were conducted as emergencies. One patient died at operation from rupture of a large mycotic aneurysm of the aortic root and another patient died five days postoperatively from ventricular fibrillation. There were no late deaths or reinfection. The mortality was 33%. Included in this group was an example of successfully treated fungal endocarditis. This occurred in a woman aged 37 who had her aortic, mitral, and tricuspid valves replaced with Starr-Edwards prostheses in February 1967. Eight months later she received treatment for streptococcal and staphylococcal septicaemias and subsequently developed candida endocarditis. This failed to respond to two courses of amphotericin B (1.7 g intravenously over six weeks and 1.5 g intravenously over eight weeks) and septic embolism ensued. In December 1970 under cover of flucytosine (10 g by mouth daily for 16 weeks) all three Starr-Edwards valves were removed and another three similar prostheses inserted. This cured the infection, and in September 1971 she survived a third operation for replacement of the second tricuspid prosthesis with a mounted homograft and repair of a mitral paraprosthetic leak.

PATHOLOGICAL FINDINGS

The conditions found among the affected aortic valves are given in Table V. Four valves had been almost completely destroyed by the infective process, there were fenestrated cusps in 13, and

TABLE V—Pathological Findings in Aortic and Mitral Valves

Aortic Valves		Mitral Valves	
Destroyed valve .. .	4	Vegetations .. .	10
Destroyed cusp .. .	5	Calcified valve .. .	8
Fenestrated cusp .. .	13	Non-calcified valve .. .	5
Ruptured chordae tendineae .. .		Perforation anterior leaflet .. .	6
		Aneurysm anterior leaflet .. .	3
		Diseased valve; no signs of infection .. .	5

vegetations were present in 10. Sixteen patients had appreciable mitral valve disease (Table V). In nine cases this was secondary to retrograde infection from aortic valve endocarditis.⁹ A conservative operation was possible in five instances, whereas valve replacement was required in the other 11. Other lesions encountered were ventricular septal defects (two congenital and one infective), myocardial abscesses (two cases), subaortic stenosis, aneurysm of the ascending aorta, aneurysm of a sinus of Valsalva, and mycotic aneurysm of the aortic root.

POSTOPERATIVE COMPLICATIONS AND FOLLOW-UP

Reoperation for haemorrhage and cardiac tamponade were required on one occasion each (Table VI). Three patients developed superficial infection of the sternotomy wound and sternal dehiscence occurred twice. One patient who had a ventricular septal defect repaired developed heart block which required pacing for three days and then reverted spontaneously

TABLE VI—Postoperative Complications

Early	No. of Patients	Late	No. of Patients
Haemorrhage	2	Infection	2
Tamponade	2	Mitral paraprosthesis leak ..	1
Wound infection	3	Aortic homograft valve failure	1
Sternal dehiscence ..	2	Early diastolic murmurs ..	5
Transient heart block ..	1	Haemolysis	1
Fever lasting more than 12 days	7		

to sinus rhythm. Seven patients had postoperative fever lasting more than 12 days. All had negative blood cultures and all resolved with time.

Among the late complications there were only two recurrences of endocarditis after surgery. In the patient with Q fever, who died one year after operation, probably the infection was never completely eradicated. Another patient developed late endocarditis on an aortic homograft valve inserted two and a half years previously and seven years after the first attack of endocarditis. There was one paraprosthesis leak, which was successfully repaired at reoperation. Of the 16 aortic homograft valves used one failed and required replacement two years after insertion and five developed early diastolic murmurs. In each of these patients the incompetence was judged to be trivial as the diastolic pressures were well maintained and they remained well without medical treatment. One patient in whom a rather small cloth-covered aortic Starr-Edwards prosthesis was inserted had evidence of a mild haemolytic anaemia, which was controlled with oral iron supplements.

The overall mortality in this series was thus 22.5%. Seven deaths (17.5%) occurred in hospital and 2 (5%) were late deaths. The remaining 31 patients (78%) were alive and well four years to nine months after operation.

Discussion

The natural history and prognosis of bacterial endocarditis have changed noticeably in recent years.^{10 11} The steady increase in the age incidence of the disease is confirmed by the present study, in which the maximal incidence occurred among patients in the fourth, fifth, and sixth decades. Effective chemotherapy has been largely responsible for the improved prognosis of the treated attack¹ but delay in diagnosis, particularly among the elderly, in whom atypical methods of presentation are common, may still result in irreversible valvular damage. The availability of valve replacement surgery at relatively low risk means that many of these patients with severely damaged valves can now be saved.¹² The overall mortality in the present group of 28 patients treated by elective surgery was 18%. These operations included nine double valve procedures and three in which ventricular septal defects were closed at the same time as valve replacement.

OPERATION DURING ACUTE STAGE OF ILLNESS

Difficult problems of management still occasionally arise when intractable heart failure supervenes before a regular course of antibiotics has been completed. This usually results from early valve perforation or destruction and clinical deterioration can be rapid. A clear description of the clinical features of the syndrome of acute aortic regurgitation was presented by Wise *et al.*,¹³ and it is important to appreciate that although coronary embolism and myocarditis secondary to the infection may contribute to left ventricular failure these patients usually die not from infection but from its mechanical consequences, as manifested by acute valvular regurgitation.

In 1965 Wallace *et al.*³ claimed the first successful surgical treatment of active bacterial endocarditis by valve excision and replacement, and in 1967 Windsor and Shanahan⁸ reported on another six patients treated surgically during the acute phase, of whom three died. These authors also emphasized the importance of intensive resuscitative measures before induction of anaesthesia and establishment of cardiopulmonary bypass. One of the

early fears of operating in a potentially infected field was that dissemination of infection would result in an increased incidence of postoperative sepsis or infection of the new valve substitutes. In 1967 Braniff *et al.*⁴ reported five successful consecutive aortic valve replacements for progressive heart failure during active bacterial endocarditis. Four of these operations were performed less than 28 days after institution of chemotherapy, but in each case culture of excised valves and abscess material was sterile. No postoperative infections were encountered and they concluded that early surgical intervention was justified in patients with life-threatening heart failure but that "a minimum of two weeks of an antibiotic regimen would be advantageous."

In our group of seven patients treated acutely four had preoperative antibiotic treatment of less than 15 days duration, and in only one instance was a positive culture obtained from vegetations or granulation tissue removed at operation. This occurred in the case of Q fever endocarditis, in which *Coxiella burnetii* was cultured from aortic root granulation tissue. The patient died one year later with evidence suggestive of persisting infection. Among the complete series of 40 patients there was only one other case of reinfection. This occurred late on an aortic homograft valve inserted two and a half years previously. Early postoperative sepsis was also no commoner than among the usual cardiac surgical population. Of the 33 patients who survived operation 26 were afebrile by the twelfth postoperative day and superficial wound infection occurred three times.

Another difficulty envisaged was that valve sutures might not hold in tissues softened by recent infection, and an increased incidence of paraprosthesis leaks was anticipated in the patients operated on during the active stage of the disease. This was confirmed by Stason *et al.*,¹⁴ who recorded four paraprosthesis leaks in 13 patients treated surgically less than six months after initiation of treatment for bacterial endocarditis. Two died and two survived reoperation. Sarat *et al.*¹⁵ recently reviewed a collected series of 59 patients operated on for active infective endocarditis and found that 15% developed postoperative paraprosthesis regurgitation. In our series, however, there was only one paraprosthesis leak, and this occurred in one of the seven patients in group 2.

Involvement of the aortic annulus and aortic wall by infection has been held to be especially unfavourable,¹⁶ and one patient in group 3 died at operation from rupture of a large subannular mycotic aneurysm of the aortic root. Nevertheless, Gonzalez-Lavin *et al.*¹⁷ recently reported five survivals among six patients with aortic valve endocarditis complicated by mycotic aneurysms of the aortic root. Signs of active infection were present in three of the patients at the time of operation.

PROSTHETIC ENDOCARDITIS

Though endocarditis on cardiac prostheses and valve substitutes is becoming relatively less common due to improved antibiotic prophylaxis during the operative period it remains an important problem owing to the large volume of cardiac surgery now being undertaken. The infecting organism is usually not a streptococcus, being more often a staphylococcus or a fungus. Identification of the organism may be difficult and even when known its eradication may be impossible until the foreign material acting as a nidus for infection has been removed.¹⁸

A distinction has been made between prosthetic valve endocarditis occurring early or late after surgery.^{6 7} The former results from contamination at the time of surgery with or without subsequent wound infection.¹⁹ Prognosis in this group of patients is poor and reoperation for removal of the infected prosthesis is usually necessary for cure of the infection. Two of the six patients in group 3 were in this category and both had sternal wound infections. One had a postoperative staphylococcal septicaemia and subsequently developed haemolysis and systemic embolism. Seven months after his first operation the Starr-Edwards prosthesis was successfully changed for a fascia lata valve and he had remained well 21 months later. The other

patient had a persistent fever and cardiac failure after replacement of aortic and mitral valves with Björk-Shiley prostheses. Reoperation four months later was performed as an emergency in the face of uncontrolled infection and the patient died from rupture of a large mycotic aneurysm of the aortic root which burst when the sternum was opened.

Endocarditis occurring late after cardiac surgery is usually caused by transient bacteraemias from procedures such as urinary tract manipulations or dental treatment,⁶ and the latter was responsible for two of the four cases we operated on with late infections. Patients with cardiac prostheses should therefore always be advised to take the same precautions against infection as those with congenital or rheumatic heart disease. The prognosis in this group is better than in those developing endocarditis soon after surgery, and provided the prosthesis is well incorporated the infection can sometimes be cured with antibiotics alone.²⁰ Partial detachment of a prosthesis or damage to a homograft valve secondary to infection requires surgical treatment on its own merits.

Coincident with the decrease in incidence of bacterial endocarditis after cardiac surgery has been an increase in fungal endocarditis. Ostermiller *et al.*⁸ reviewed the cases of 48 patients with fungal endocarditis reported in the literature between 1956 and 1970. They concluded that the chief aetiological factors were prolonged antibiotic therapy, general debility, and the use of steroids and found a mortality rate of no less than 89%. *Candida albicans* is by far the commonest causative agent⁸ and the diagnosis may be difficult to confirm.²¹ Fungi not normally pathogenic to man can also cause endocarditis.²² In this series the single case of fungal endocarditis occurred after prolonged intravenous antibiotic treatment for first streptococcal and then staphylococcal septicaemias. Successful eradication of the infection followed the combination of antifungal chemotherapy and excision and replacement of all three cardiac prostheses.

Conclusion

Our results indicate that in addition to elective valve replacement surgery has an important role both in the management of

intractable heart failure consequent on bacterial endocarditis and in the eradication of infection. The timing of operation is crucial, particularly where valve destruction occurs early, and in this event operation may be necessary before completion of antibiotic therapy.

We are grateful to our medical colleagues at the National Heart Hospital for their help in the management of these patients and for permission to report on them.

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Impressions of Cogwheel

A Divisional Study

FROM A SPECIAL CORRESPONDENT

British Medical Journal, 1972, 4, 602-604

In recommending the divisional structure as the best way of organizing medical work in hospitals the original Cogwheel Report¹ was probably thinking mainly of the clinical specialties. Physicians and surgeons of various persuasions would easily fit together in divisions of medicine and surgery. Indeed, some physicians had already thought about forming division-like structures themselves.² So, unless they were numerous enough to form divisions of their own, paediatricians might be expected to join the physicians and anaesthetists to join the surgeons. Nevertheless, because this tidy scheme left many specialists out of account in some hospitals groups pathologists, radiologists, clinical physiologists, physicists, pharmacists, and so on have been distributed into the other divisions or have been able to form divisions of their

own. In other circumstances the solution has been to group the "services" specialists together—in other words, the "second-line" experts to whom the "front-line" clinicians send requests for services. Sometimes anaesthetists have been included in this category.

I talked to Dr Delahay,* a pathologist who is a member of a services division which includes diagnostic radiologists (but not radiotherapists) and pathologists, and also biochemists, physicists, and pharmacists who are not medically qualified. This is therefore a heterogeneous group of people without many interests in common but serving the same group of requesting clinicians in several hospitals. Naturally, Dr. Delahay said, the discussions tend to become a trifle factional, but on the whole the meetings remain amicable. Medical physics, diagnostic radiology, and pathology all need large

* This name is fictional.