at 14 years. We found no difference between the incidence of relapse in younger women compared with that in older women or between those who had their first smears taken before and after 1977. These findings do not support a previous suggestion that preinvasive disease is especially likely to be more aggressive in younger women.

It is interesting that the relapses occurred continually throughout the whole of the follow up period. Some of these relapses probably reflect persistence of the initial cervical intraepithelial neoplasia despite previous negative smears, but this seems unlikely for those occurring in later years in women who have had many previously negative smears, and reinfection with the papillomavirus has been found to be common.⁶ Again, persistence of the initial lesion would be expected to give an excess of relapses in the early years, which was not found in our study. This pattern of continuing relapse contrasts with the few relapses we found after surgical treatment of cervical intraepithelial neoplasia III.⁷ This supports a similar observation by Richart *et al*, who suggested that cryotherapy may stimulate an immune reaction to prevent reinfection with the papillomavirus.⁸

At present, some doctors recommend that mildly dyskaryotic smears should be treated with the utmost seriousness and that all women who have smears that contain dyskaryotic cells should be referred for colposcopy, surveillance by cytology being inadequate. Our results suggest that the strongest case for immediate colposcopy is to detect those women who present with mild dyskaryosis but who have an invasive carcinoma, as it is in these circumstances that cytology is probably least effective. One problem is that cancer can result not only in a misleading mild dyskaryosis but also in blood stained smears or smears containing "borderline" cells or considerable inflammatory changes. The number of such smears is legion. We have also been impressed by the experience required for accurate colposcopy, and indeed in our series more diagnostic errors resulted from colposcopy than from cytology. These would be multiplied if colposcopy were adopted virtually as a screening procedure. Our results also raise the question of cost effectiveness. In the 1347 patients followed up there

occurred only seven carcinomas. Frequent needless examinations would also be carried out, as mild dyskaryosis in many patients spontaneously regresses. Furthermore, we could find no evidence to suggest that a more rapidly progressive form of preinvasive disease that would indicate urgent colposcopy is present in younger women.

In agreement with Brown and Phillips our findings show that cytological review is quite safe provided that all patients who show a persisting smear abnormality are referred for colposcopy and biopsy.⁹ In view of the number of cervical intraepithelial neoplasia III lesions that we found to be associated with persisting mild dyskaryosis, however, we would now prefer to shorten the initial review period before biopsy to 12 to 18 months. Pregnancy also presents difficulties for cytological diagnosis. A further factor that may affect treatment policy is our confirmation that biopsy or laser treatment seems to confer protection against later infection by papillomavirus.

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Rheumatic symptoms after cardiac surgery: a prospective study

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Abstract

The incidence of different types of shoulder pain after open heart surgery was studied prospectively. Of 101 patients studied, 45 developed rheumatic symptoms during the first six weeks after the operation. Thirty eight patients reported pain in the region of the shoulder girdle with no loss of shoulder function (postpericardiotomy rheumatism). Three of these patients also had features compatible with the postpericardiotomy syndrome (fever, malaise, or pleuritic chest pain), and seven developed the syndrome without pain in the shoulder girdle. Of these 10 patients, one had generalised myalgia. Postpericardiotomy rheumatism alone was not associated with increased inflammation (measured by the erythrocyte sedimentation rate and concentration of C reactive protein); immunological tests including measurement of antibodies to cardiac muscle yielded inconclusive results. Replies to a postal questionnaire showed that symptoms of

postpericardiotomy rheumatism were present for over three months in 18 patients and for six months or longer in 14.

In view of the large number of patients now having open heart surgery postpericardiotomy rheumatism should be considered when patients report pain around the shoulders so that it is not misdiagnosed as angina.

Introduction

It has been known for 50 years that recovery from myocardial infarction may be complicated by shoulder pain of non-cardiac origin not associated with loss of shoulder function, which may start several months after the acute episode.^{1,2} This seems to be a separate entity to frozen shoulder or the shoulder-hand syndrome, in which shoulder movements are greatly restricted and the prognosis worse.^{3,4} Arthritis or arthralgia has been described as a feature of the

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postpericardiotomy syndrome⁵⁻⁸; early reports suggested that it was due to reactivation of rheumatic fever,⁹ but this was later disproved.⁹⁻¹¹ A recent report of the postpericardiotomy and postmyocardial infarction syndromes does not mention joint disease.¹² Dressler's original description of the postmyocardial infarction syndrome, which is virtually identical with the postpericardiotomy syndrome, does not include joint pain,¹³ but three cases of Dressler's syndrome with associated polyarthritis or polyarthralgia, mainly in the arms, have been reported.¹⁴ We describe the rheumatic symptoms reported by patients after open heart surgery. In postpericardiotomy rheumatism the pain is usually a persistent dull ache around the shoulder or arm, or both, or across the trapezius muscles. It is not associated with exertion—that is, it is not typical of angina—and there is no loss of shoulder function or range of movement. The postpericardiotomy syndrome consists of fever, malaise, or pleuritic pain or a combination of these.

Patients and methods

We studied 107 patients admitted for elective open heart surgery. Their ages ranged from 35 to 74 (mean 54.4 (SD 9.5)). Eighty four patients were having coronary artery bypass grafting, of whom six were also having aortic or mitral valve replacement and one a left ventricular aneurysmectomy. Seven patients were having mitral valve replacement alone, six aortic valve replacement, three closure of an atrial septal defect, and one a mitral valvotomy. The patients were interviewed and examined by one of us (KW) before operation and at a routine surgical outpatient visit about six weeks after operation. When possible patients were also seen about one week after operation, before discharge from hospital. They were asked specifically about rheumatic symptoms and features suggestive of the postpericardiotomy syndrome—for example, pleuritic chest pain, fever, and general malaise.

Blood samples were obtained on each occasion that patients were seen. A full blood count, erythrocyte sedimentation rate, and concentrations of C reactive protein, immunoglobulins, antibodies to cardiac muscle (with rat heart as substrate), rheumatoid factor (by the sheep cell agglutination test), and antinuclear factor were determined, and tests for formation of immune complexes were performed.

Many patients had to travel long distances to attend outpatient clinics, making rheumatological follow up difficult. A postal questionnaire about rheumatic symptoms was therefore sent six to 18 months after operation to all patients except those with the postpericardiotomy syndrome, who were seen separately.

Results

Of the 107 patients seen before open heart surgery, two died early in the postoperative period, one was withdrawn from the study because of severe embolic complications, and three were lost to follow up. All 101 patients who completed the study were seen at the six week follow up visit, 81 of them having already been seen one week after operation.

Forty five of the 101 patients reported the development of rheumatic symptoms during the first six weeks after operation (fig 1). All six patients who had an acute attack of gout had been taking or were taking diuretics compared with 14 (36%) of the patients without gout. For three patients this was their first attack.

POSTPERICARDIOTOMY RHEUMATISM

Forty patients developed pain around the shoulder girdle. One had had mild symptoms preoperatively

FIG 1—Rheumatic symptoms reported in first six weeks after open heart surgery related to postpericardiotomy syndrome

and a raised erythrocyte sedimentation rate, suggesting polymyalgia rheumatica; another developed pleuritic pains in the tip of the shoulder. Thus 38 patients had postpericardiotomy rheumatism (fig 1); their ages ranged from 36 to 74 (mean 53.7 (SD 10.2)). Patients with valvular heart disease and with ischaemic heart disease were equally affected. The time of onset of postpericardiotomy rheumatism was determined, pain of early onset being defined as that occurring before discharge from hospital (usually about one week after operation). Twenty three patients had pain of early onset, of whom 19 first noticed it in the first two days after operation as pain from the sternotomy lessened. Nineteen patients had pain of late onset, of whom 14 noted their symptoms during the second and third weeks after operation; two noted them in the fourth week, two in the fifth, and one in the seventh. In four patients in the early onset group pain later occurred on the opposite side, and they were therefore included in both groups. The table shows the site of pain in the patients. Four patients with early onset of pain and five with late onset had symptoms in two sites.

Number of patients with pain due to postpericardiotomy rheumatism after open heart surgery. Some patients had pain in more than one site

	Site of pain		
	Left shoulder	Right shoulder	Across trapezius muscles
Early onset* (n=23)	9	5	13
Late onset (n=19†)	11	9	4

*Before discharge from hospital after operation.

†Includes four patients with early onset pain who had late onset pain on other side.

At the follow up examination six weeks after operation two patients were found to have painful arcs on abduction of the shoulder which they had not been aware of. Seven patients who remained asymptomatic and eight who developed postpericardiotomy rheumatism had a history of pain around the shoulder girdle ($p=0.53$, Fisher's test of exact probability). In two of these patients postpericardiotomy rheumatism occurred at the site of previous shoulder pain.

Of the 62 patients who replied to the postal questionnaire, 13 (52%) of the 25 with postpericardiotomy rheumatism had had symptoms for at least three months and eight (32%) for six months or longer; a further six patients who had initially not had any symptoms had subsequently developed shoulder girdle pain. Many patients had consulted their doctors because of the pain, and eight were treated with simple analgesics, non-steroidal anti-inflammatory drugs, or physiotherapy.

POSTPERICARDIOTOMY SYNDROME

Ten patients developed features suggestive of the postpericardiotomy syndrome in varying degrees of severity. Nine patients complained of pleuritic chest pain, three having pleural effusions visible in chest x ray films; three had clinical evidence of pericarditis; and a further two had symptoms of pericarditis. All noted associated malaise. Although fever was documented in only one patient, others complained of a fever. In five cases symptoms warranted readmission of the patient. One patient required a short course of prednisolone for a pleural effusion, two responded well to indomethacin, and two were given anticoagulants despite negative results of ventilation-perfusion lung scans.

Five of the 10 patients had rheumatic symptoms: three had shoulder girdle pain indistinguishable from postpericardiotomy rheumatism (two having early and late onset of symptoms at different sites), one had referred pleuritic pain in the shoulder tip, and one had generalised myalgia (this man had characteristics of the postperfusion syndrome, with up to 15% atypical lymphocytes in peripheral blood¹⁵⁻¹⁸). The rheumatic symptoms of two patients continued up to one year after operation with no features of the postpericardiotomy syndrome other than fatigue: one had pain in both upper arms and across the trapezius muscles, and the other had knee pain and early morning stiffness with intermittent pain in the left shoulder. These symptoms were not associated with a raised erythrocyte sedimentation rate or concentration of C reactive protein.

LABORATORY INVESTIGATIONS

Figure 2 shows the mean concentration of C reactive protein before and one and six weeks after operation.

FIG 2—Mean concentration of C reactive protein before and after open heart surgery (normal <30 mg/l). Bars indicate 1 SD

Erythrocyte sedimentation rates before and after operation showed a similar pattern. The patients without symptoms and those with postpericardiotomy rheumatism had similar postoperative rises and falls in concentration of C reactive protein and erythrocyte sedimentation rate, but patients with the postpericardiotomy syndrome tended to have higher values six weeks after operation. There was no association between results of immunological tests and positive results of sheep cell agglutination tests and tests for antinuclear factor and antibodies to cardiac muscle. Neither the postpericardiotomy syndrome nor postpericardiotomy rheumatism was associated with antibodies to cardiac muscle: of the three patients who developed antibodies, one had postpericardiotomy rheumatism and two remained asymptomatic. No patients with the postpericardiotomy syndrome

developed antibodies to cardiac muscle, although one had high titres before and after operation.

Discussion

Forty five of our patients reported rheumatic symptoms during the first six weeks after open heart surgery, and a further six developed shoulder girdle pain in the next few months. As patients who develop symptoms are likely to reply to a postal questionnaire we assumed that those who failed to reply remained asymptomatic. Thus the incidence of shoulder girdle pain was about 50%. Thirty eight patients noted a dull ache around the shoulder girdle in the first six weeks after operation but did not have limitation of movement; this is similar to the symptoms occurring after myocardial infarction described by Edeiken and Wolferth.¹ None of our patients developed either a frozen shoulder or the shoulder-hand syndrome during the follow up period; both of these have been reported after myocardial infarction.^{3,4} Symptoms of postpericardiotomy rheumatism were mild. The main concern of patients who had had coronary artery bypass grafting was that the pain might be due to recurrence of angina, but in none of the cases was the pain related to exertion. Several patients consulted their general practitioners and were treated with non-steroidal anti-inflammatory drugs or physiotherapy, or both; others may have been reassured of the benign nature of their symptoms as a result of participating in this study.

Early onset postpericardiotomy rheumatism may be secondary to trauma to the chest wall or pericardium, or both. The causes of late onset peripostcardiotomy rheumatism are not known. Interestingly, the one patient who had a mitral valvotomy developed shoulder pain of late onset. Autoimmunity might explain postpericardiotomy rheumatism of late onset; although our routine immunological tests gave unhelpful results, we are investigating this further.

The postpericardiotomy syndrome is often difficult to diagnose definitely. Ten of our patients developed suggestive features, five having locomotor symptoms and three of these complaining of pain typical of postpericardiotomy rheumatism. Two patients with the postpericardiotomy syndrome and postpericardiotomy rheumatism still had arthralgia one year after open heart surgery.

We did not find the association between the postpericardiotomy syndrome and development of antimyocardial antibodies that has been reported by some workers.²¹⁻²⁵ Others have found that these antibodies are present in many patients with ischaemic heart disease and do not seem to have a pathogenetic role.²⁶⁻²⁹ Features identical with those of the postpericardiotomy syndrome can occur without obvious myocardial damage after trauma to the pericardium.³⁰⁻³³ The incidence of the syndrome after open heart surgery has fallen considerably with better pericardial toilet at operation, having been 20-40% in earlier studies.^{5,7-9,19,20} It may be more appropriate to look for antibodies to pericardium than to cardiac muscle.

In 1984, 10 000 patients in the United Kingdom had open heart surgery, and this number is increasing.³⁴ Our study suggests that up to a quarter of these patients could experience pain around the shoulders for three months or longer after operation. Awareness of the possibility of such pain enables clinicians to reassure patients and avoid further costly cardiological investigations.

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Perinatal brain damage: predictive value of metabolic acidosis and the Apgar score

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Abstract

To assess the predictive value for perinatal brain damage of acidosis at birth, alone or in combination with the Apgar score at 5 minutes, a cohort of 982 liveborn infants delivered over two months was studied prospectively. The umbilical cord was double clamped, and arterial acid-base values were successfully determined in 964 infants and lactate concentration in 931. Reference values defining acidosis (mean $\pm$ 2 SD) were obtained from a subset of 127 term infants who had no complications. The incidence of a low pH was 12% (111 out of 964), high base deficit 7% (70 out of 964), high lactate concentration 9% (83 out of 931), and low Apgar score at 5 minutes ($\leq$ 7) 3% (32 out of 982). Twelve of the 111 infants (11%) with acidosis had a low Apgar score, and 12 out of 29 infants (41%) with low Apgar scores had acidosis. At one year of age 35 infants were lost to follow up and 22 had an adverse outcome unrelated to asphyxia; 883 infants showed normal development but the possible sequelae of asphyxia were four deaths, slight abnormalities in 28 infants, and clear abnormalities in 10. The sensitivity and the positive predictive value of low pH for adverse outcome were, respectively, 21 and 8%, of high lactate concentration 12 and 5%, and of low 5 minute Apgar score 12 and 19%.

Metabolic acidosis determined in blood from the umbilical artery at birth is a poor predictor of perinatal brain damage.

Introduction

Perinatal asphyxia is an important cause of mortality and morbidity in the newborn infant and of neuro-

logical disability, mainly cerebral palsy, in later life. This view is widely accepted, although asphyxia as a pathophysiological and clinical concept is quite loosely defined. Its components are hypoxia, hypercapnia, and ischaemia, but both its incidence and role in outcome are difficult to evaluate because of lack of reliable diagnostic criteria.

The Apgar score at 5 minutes after birth has been used, and often misused, as a predictor of neurological damage due to asphyxia. The American collaborative perinatal project of 1959-66, however, showed poor sensitivity of the Apgar score in this respect: only 27% of children who later developed cerebral palsy scored below 7 at 5 minutes.¹ Metabolic acidosis reflects fetal distress and asphyxia, but its relation to the Apgar score is not close. In a cohort study in Oxford in 1982 only 19% of the infants with low Apgar scores had severe acidosis at birth and 27% of the infants with acidosis had a low score at 5 minutes.² Determining the pH of blood in the umbilical artery has become an important measure of the infant's condition at birth and is used routinely in many delivery hospitals. The importance of acidosis at birth has not, however, been related to long term outcome in a sufficiently large unselected population that has received adequate follow up. We assessed the predictive value of acidosis at birth and the 5 minute Apgar score for perinatal brain damage in a defined delivery cohort.

Subjects and methods

The study was approved by the ethical committees of both hospitals. It was based on the deliveries over two months (April-May 1984) in the department of obstetrics and gynaecology, a unit admitting

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