THE POSTCOMMISSUROTOMY SYNDROME

BY

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The term "postcommissurotomy syndrome" (P.C.S.) was first used by Soloff et al. (1953) to define an illness with a "distressingly high incidence" (43 out of 183 cases) after mitral valvotomy. The main symptoms were sternal pain with radiation to the left chest and pyrexia, the onset was mostly abrupt either days or weeks after operation and the duration varied from ten days to four weeks with frequent further relapses. The syndrome occurred either alone or in various combinations with other postoperative complications; though the mortality was low (3 out of 43) there was an adverse influence on the outcome of the operation in more than one-third of the cases. Among the many hypotheses advanced for the pathogenesis they favoured the idea of rheumatic reactivation induced by surgical trauma. Bercu (1953), who observed the P.C.S. in 10 per cent of his patients, agreed that it may suggest rheumatic activity though trauma to the heart may be more important; the prognosis in his cases was not different from that of the uncomplicated cases. January et al. (1954) with a similar incidence used the term "postvalvotomy syndrome"; they did not consider the symptoms as wholly characteristic of rheumatic fever. McAllister (1953) suspected a low-grade infection of the lingula as its cause. Dressler (1954) for whom the syndrome consists of the triad, pericarditis, pleurisy, and pneumonitis, compared the P.C.S. with idiopathic recurrent pericarditis and tried to establish a rheumatic etiology for both. Verheugt et al. (1954) did not mention the syndrome by name, but described its signs as temporary rheumatic activity because of its successful treatment with amidopyrine and the rise in antistreptolysin titre in some of their patients. Elster et al. (1954), after a painstaking study of the P.C.S. in 16 patients, doubt if this is due to rheumatic fever, bacterial infection, pulmonary infarction, or direct surgical trauma; for them it represents a self-limited form of pericarditis and pleuritis induced by trauma of surgery in patients with rheumatic heart disease.

No original study of the P.C.S. has so far appeared in British publications. The comprehensive papers on mitral valvotomy by Baker et al. (1952), Logan and Turner (1953), Holmes et al. (1953) do not mention it. Wood (1954), however, devoted a short space to postoperative pericarditis with fever, observed in 10 per cent of his cases, which subsides permanently and leaves no serious consequences.

During the last four years the P.C.S. was frequently observed in our hospital. The early cases were not recognized: it was thought that too-tight pericardial suture or the incorrect pH of the novocaine solution injected into the pericardial sac might be responsible for the pericarditis and that the chest pain and pyrexia were due to postoperative auricular thrombosis. Some of our patients were admitted to other hospitals during relapses and diagnosed as left basal pneumonia; this shows that the syndrome is not generally known.

A study was therefore undertaken of all our patients with P.C.S. to describe their symptoms and to find out more about the origin of their illness. Admittedly a rheumatic flare-up could be expected after valvotomy; the finding of Aschoff nodes in 20–40 per cent of the excised auricular appendages by others and the positive laboratory tests for rheumatic activity and for
adrenocortical hypofunction following operation (Cordeiro et al., 1954), suggested that valvotomy was frequently performed during rheumatic activity. Nevertheless a rheumatic hypothesis for P.S.C. sounded unconvincing because of the rare incidence—2 per cent in our series—of overt rheumatic recurrences after valvotomy. More attention has therefore been devoted in this investigation to the local postoperative conditions which may constitute the possible cause of the P.C.S.

Material and Investigation

Out of 100 patients who had mitral valvotomy, 22 were selected for study; in these either the early or late postoperative period was complicated by a prolonged illness with pyrexia, pericarditis, left pleural effusion and tendency to relapse. There were 5 men and 17 women. The ages ranged from 26 to 52 with an average age of 37. Altogether 26 episodes were observed in these patients; 19 patients were seen during their illnesses while in 3 data from other hospitals were available. Special investigations included chest films at least twice weekly; frequent electrocardiographic recordings in many; culture and microscopy of the pleural effusion, white cell counts, and E.S.R. in some.

The cases fell into two groups.

Group I comprised 14 patients with 16 episodes. Here the illness followed closely upon the operation (11 cases) or started after a time lag of 5–11 days after operation (3 cases). The average duration of the illness as calculated in days of pyrexia was 17 days with a range of 8–34 days (Fig. 1A). Two patients (Cases 2 and 3) had relapses 2 and 2 1/2 months later lasting 21 days and 2 months respectively (Fig. 2).

Group II comprised 8 patients with 10 episodes. Here the patients left hospital in less than 3 weeks and the illness appeared at intervals after operation varying between 4 weeks and 4 months (Fig. 1B). The period of illness was much shorter than in Group I (Fig. 1A); it ranged between 4 and 14 days with an average duration of 9 days. Two patients (Cases 7 and 12) had relapses after 3 and 7 weeks.

Since the syndrome in Group I followed so closely upon the postoperative period, the nature and duration of the normal postoperative sequelæ in 35 patients with uncomplicated course were also studied.
THE POSTCOMMISSUROTOMY SYNDROME

After the first few days the incisional pain subsides; at the end of the first week or even earlier the patient sits out; he becomes ambulatory after 10–14 days and after three weeks is ready for convalescence. There may be some postoperative pyrexia which seldom lasts longer than four days. The effusion from the left chest drains away during the first 24 to 48 hours after which the intercostal drainage tube is removed. In most of the patients some effusion reforms, but aspiration is rarely necessary, and at the end of the second week the chest film shows only pleural thickening but no effusion in 75 per cent of the patients. In the remainder the effusion absorbs during convalescence. In the few isolated instances where aspiration was performed after the first week the liquid was usually straw-coloured. Though the patient rarely complained of severe central chest pain, a transitory pericardial rub was heard in 65 per cent of cases during the first week. Electrocardiographic evidence of pericarditis during this period was found in 4 out of 10 patients where early electrocardiograms were recorded (Fig. 3). A left pleural rub was heard frequently. In two instances a right pleural rub was heard without the patient complaining of pain and without signs suggesting pulmonary infarction. Polymorphonuclear leucocytosis and raised E.S.R. were frequently present and persisted beyond the febrile period.

THE POSTCOMMISSUROTOMY SYNDROME (P.C.S.)

The symptoms were similar to the normal postoperative sequelæ; they were identical in Group I and Group II though they were more severe and of longer duration in Group I, particularly during relapses (Cases 2 and 3).

Pyrexia was moderate and continuous; it varied from 99°–100° F. with occasional spikes to 101°–102° F. Its duration was variable (Fig. 1A and B); when the temperature fell to normal it rarely remained so but continued around 99° F. for some days. Alternatively the fever remitted for a day or two, then started again (Fig. 2). Rigors were not seen except in one patient where the
Fig. 3.—Uncomplicated mitral valvotomy. Typical changes of pericarditis in the early postoperative period, regressing after 2 weeks, disappearing after 8 weeks.

**TABLE I**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Rub</th>
<th>Pain</th>
<th>EC.</th>
<th>X-rays</th>
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<tbody>
<tr>
<td>1</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>No</td>
<td>Severe</td>
<td>Probable</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>Severe</td>
<td>Probable</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>Yes (post-op.)</td>
<td>No</td>
<td>Yes (relapse)</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>No</td>
<td>No</td>
<td>Probable</td>
<td>No</td>
</tr>
<tr>
<td>9</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
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<tr>
<td>12</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
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<td>13</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>14</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>15</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>16</td>
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<td>Severe</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>17</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
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<td>18</td>
<td>Yes</td>
<td>No</td>
<td>—</td>
<td>No</td>
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<td>19</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
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<tr>
<td>22</td>
<td>No</td>
<td>Yes</td>
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<td>No</td>
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</tbody>
</table>

effusion became infected. Persistent pyrexia as a symptom was important for it distinguished P.C.S. from the normal postoperative sequelae.

**Pericarditis.** The signs and symptoms of pericarditis are pericardial rub, central chest pain, electrocardiographic S–T and T wave changes, and sudden increase in size of the heart shadow in the chest film. These signs were present alone or in various combinations; the most frequent association was that of pericardial rub with electrocardiographic abnormalities. A pericardial rub was more often observed than pain (Table I), and in fact the two were often dissociated. In
Fig. 4.—Case 1. Typical changes of pericarditis at 19 days regressing after 9 weeks; record still abnormal at 22 weeks.

Fig. 5.—Case 4. Combined digitalis and pericarditis changes producing S–T and T pattern resembling ventricular strain in V5–V6. Preoperative digitalis changes affect V5–V6 only while postoperative pericarditis changes extend as far as V3.
contrast to the fleeting postoperative rub in the uncomplicated cases it persisted for more than four days in most of those patients. In Cases 15 and 17 it changed site, shifting from the lower sternal region to the base of the heart. In Case 14 it was still present two months later.

Electrocardiographic changes, though often obscured by digitalis effect, were recognized in 15 out of 19 cases. While the patterns of pericarditis alone were prominent in the limb leads (Fig. 4), when combined with digitalis effect, the chest leads were more helpful (Fig. 5). Here again, in contrast with the uncomplicated cases (Fig. 3) the changes persisted for many weeks (Fig. 4) or appeared during convalescence (Fig. 8).

Central chest pain was not a prominent feature in our series. It was present in 6 patients and was severe in 3 only, being the reason for readmission in 2 (Cases 2 and 3). It had the usual character of pericardio-pleural pain; it was stabbing, made worse by coughing, deep breathing, or any movement, and when severe was responsible for a fast and shallow respiration easily confused with the dyspnea of congestive heart failure. Pain over the left base of moderate intensity was present in 3 instances and was erroneously attributed to the wound pain of the thoracotomy. Occasional pain in the left shoulder tip suggested diaphragmatic involvement. Pain on the right side of the chest was not observed.

The radiological sign of pericarditis—the sudden increase in size of the heart shadow—was observed in two instances only (Fig. 6). The presence of left basal effusion obscuring the left cardiac border made it impossible to estimate the heart size in the others. Pericardial aspiration did not yield any fluid in the two patients in whom it was attempted.

**Fig. 6.—Case 1.** Sudden increase of heart shadow at 7 days due to pericarditis.

**Pleural Effusion.** The persistence of left basal effusion for weeks or months or its recurrence after apparent absorption constitutes, with pyrexia, the leading feature of the P.C.S. In the chest film the fluid reaches its maximum, the upper border of the posterior aspect of the sixth to eighth rib in the mid-clavicular line, between the fourth and seventeenth postoperative day and when aspirated reforms quickly. Following absorption of the fluid pleural thickening remains in the left costophrenic angle where small amounts of fluid may again collect in association with pyrexia and further relapse (Fig. 7 and 8).

Paralysis of the left dome of the diaphragm through accidental damage to the phrenic nerve during operation may have been an additional factor in Cases 10 and 13 retarding recovery.

In 4 patients (Cases 2, 14, 15, and 19) loculation of the effusion occurred between the pericardium and pleura in the anterior part of the chest (Fig. 9). One of these (Case 2) was the most
Fig. 7.—Case 3. Left pleural effusion still present at 5 weeks at discharge; partial clearance during convalescence; reappearance at 12 weeks during relapse.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Appearance</th>
<th>Cultures</th>
<th>Aspiration</th>
<th>Amount (oz.)</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Days after operation</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Pure blood</td>
<td>Sterile</td>
<td>Rep. 6–25</td>
<td>Few oz.*</td>
</tr>
<tr>
<td>3</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>10, 14, 19</td>
<td>40, 6, 14</td>
</tr>
<tr>
<td>8</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>9</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>6</td>
<td>Few oz.</td>
</tr>
<tr>
<td>10</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>8</td>
<td>24</td>
</tr>
<tr>
<td>11</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>14</td>
<td>Few oz.</td>
</tr>
<tr>
<td>13</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>15</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Purulent</td>
<td>Haem. staph. aureus</td>
<td>17</td>
<td>Few oz.*</td>
</tr>
<tr>
<td>18</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>22</td>
<td>40</td>
</tr>
<tr>
<td>19</td>
<td>Bloodstained</td>
<td>Sterile</td>
<td>7</td>
<td>Few oz.*</td>
</tr>
<tr>
<td>22</td>
<td>Straw-coloured</td>
<td>Sterile</td>
<td>60</td>
<td>13</td>
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</table>

* Aspirated from anterior loculi.
severe of our cases; the P.C.S. lasted almost 3 months with a few weeks intermission between two bouts of illness. Cases 14 and 19 were of moderate severity. The fourth (Case 15) recovered in 19 days, 4 days after the pocket, which was infected, was emptied.

Laboratory data of the effusion were available in 11 patients (Table II). The fluid was blood-stained in 10 where the aspiration was performed between the fifth and twenty-second postoperative day. It was straw coloured in Case 22, two months after the operation, and purulent in Case 15 on the seventeenth day, when culture revealed the presence of haemolytic *Staphylococcus aureus*. In the others the cultures were sterile. The cellular composition suggested inflammatory exudate with a prevalence of neutrophils.

The Lungs. Left basal effusion obliterates the underlying lung fields in the chest film. When the fluid absorbs crepitations may be heard but at this stage pleural thickening obscures the lung

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**Fig. 8.—Case 12. Normal postoperative course.** Appearance of left pleural effusion at 7 weeks, with changes of pericarditis in the electrocardiogram. Complete radiological clearance and normal record at 19 weeks.
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changes. It is likely that under extensive effusions pulmonary basal consolidation develops. This was proved in our only direct observation (Case 4) in a patient who 7 months after mitral valvotomy followed by P.C.S. underwent a second thoracotomy for aortic stenosis: there were dense pleural and pleuro-pericardial adhesions over the left base and the lower third of the left lung showed carnification.

Fig. 9.—(A) Case 2. Hæmorthorax after sling operation for mitral incompetence with anterior loculus formation at 9 weeks. Clearance with retraction of left chest. (B) Case 19. Anterior loculus formation after mitral valvotomy at 2 weeks; after aspiration at 3 weeks; complete clearance at 10 weeks.

The haematological findings were too variable to be of diagnostic help. The leucocyte count at the height of the syndrome varied between 12,000 and 40,000 with 73–82 per cent neutrophils and bore no relation to the severity of the syndrome. The E.S.R. in the postoperative cases was unreliable; in the relapsing cases it was moderately raised to 30–40 mm.

DIAGNOSIS

The P.C.S. has some features in common with rheumatic pericarditis and has been attributed to it. The pericardial involvement, the persistent pyrexia, the tendency to relapses, the increased E.S.R. are present in both conditions. However, there are many more dissimilarities.
Preceding streptococcal infection
Arthritis
Carditis
Constitutional upset
Outcome
Salicylate effect
Aschoff bodies

Rheumatic Fever
frequent
present
present
great
possibly fatal
invariable
probable

P.C.S.
ever
absent
absent
slight
good prognosis
absent
not found

Latent streptococcal infection could be ruled out since all patients had routine penicillin treatment after operation.

The absence of articular involvement was particularly striking. Rheumatic relapses, though rare, may occur after valvotomy and joint pains in them are the rule (illustrative case M.S.). Rheumatic recurrence may also be associated with the P.C.S. in rare instances (illustrative case E.P.) and in these salicylate effect may be manifest; but joint pains and articular swellings are not part of the P.C.S.

The pericarditis of rheumatic fever is a partial manifestation of generalized cardiac involvement. The pericarditis of the P.C.S. is a local process that leaves the valves and the myocardium unaffected. Prolongation of the Q–T interval and delay in atrio-ventricular conduction were not observed in the electrocardiogram. Out of 13 patients of our series with sinus rhythm before operation 5 developed auricular fibrillation after it and of these sinus rhythm could be restored in two. The incidence of 23 per cent in whom auricular fibrillation became established is therefore similar to that in patients without P.C.S. Postoperative congestive heart failure was observed in 3 patients and this incidence (14%) is actually the same as in the group without P.C.S. The effect of digitalis and mercurial diuretics was similar in those with and without P.C.S. and when fever abated, often spontaneous diuresis set in. No further valves became affected during the illness.

The absence of Aschoff bodies in the auricular appendages of our series is the least impressive argument against rheumatic activity. Opinion among pathologists is so divided as to what should be regarded as an “active” Aschoff body (Enticknap, 1953; McKeown, 1953) that their presence or absence is more a subjective impression than an objective finding (Hinson, 1955). In our hospital, where strict criteria have been applied such a finding was exceptional (2%).

Pulmonary infarction also causes severe chest pain with basal effusion and may be confused with the P.C.S. Its sudden onset with shock, blood-stained sputum, signs of thrombophlebitis in the leg veins, and the electrocardiographic patterns of acute right ventricular strain are the differentiating features. The diagnosis is easy if radiological changes develop on the right side; but if these are on the left side and are obscured by the postoperative effusion they may be impossible to detect. If the diagnosis is doubtful, anticoagulant treatment may be given; this is followed by rapid improvement if inarction is the cause, but it leaves the P.C.S. unaffected. Both conditions may on course coexist.

Cerebral embolus complicated P.C.S. in Case 3. We believe that the “psychosis” described as part of the syndrome by Soloff et al. (1953) may be due to this.

Prognosis. This is remarkably good. The patients do not look unduly ill and however long the illness lasts they eventually recover. There was a marked contrast in many of our patients between the extensive objective signs of pericarditis, pleural effusion, and fever, and the slight constitutional upset. They lost little weight during the illness and the appetite remained fair.

Treatment. All available antibiotics were tried in the early cases, before we knew much of the syndrome, in the hope of influencing the basal pulmonary consolidation which we thought might be the cause of the fever. Like others we have not seen any conclusive results. Neither were we impressed with the effect of aspirin or salicylates except in the patient with joint pains. In an illness with such a variable course and duration any effects attributed to treatment have to be weighed critically. If the relapse is short any treatment may be regarded as effective; in fact
patients with short relapses treated in other hospitals were considered to have had good responses to penicillin treatment.

**DISCUSSION**

The P.C.S. appears as the accentuation, prolongation, and recurrence of the normal postoperative sequelae. In view of the slight systemic upset it causes and its relative benignity, local rather than general causes have to be sought to explain its persistence and the subsequent relapses.

The surgical technique was the same in all our cases. After amputation of the auricular appendage and closing of the auricular wound the pericardium was loosely stitched with interrupted sutures leaving a free communication between pericardial cavity and left pleural space. Thus the effusion draining through the intercostal tube is derived partly from the pericardial sac. Under normal conditions it is hæmorrhagic during the first few days, but becomes serous and straw-coloured after the first week if it still persists. In the P.C.S. the effusion was blood-stained in all the 10 patients in whom it was aspirated after the fifth day. Prolonged oozing from the auricular wound into the pleuro-pericardial space may be an important cause since blood in the serous cavities is regarded as a powerful irritant (Holmes Sellors, 1954). It may set up a foreign body reaction in the pericardium, with which Wood (1954) compares the syndrome.

Case 2, who developed a frank hæmothorax after a sling operation for mitral incompetence had the longest illness of all and recovered eventually with a retraction of the left chest.

It may be possible that rheumatic subjects show an increased irritability of their serous membranes to extravasated blood. Massell et al. (1937) were able to produce subcutaneous nodules, similar, if not identical in histological structure to rheumatic nodules (Mote et al., 1937) by subcutaneous injection of blood in 90 and 50 per cent of their patients with clinical and subclinical rheumatic fever respectively: only in 3 per cent of normal controls did nodules develop.

Another important local factor that may coexist is the pocketing of the effusion; though this was observed in only 4 cases, the aspiration of the pockets in 2 resulted in immediate improvement and rapid recovery. These pockets were anteriorly situated; the insertion of the drainage tube in the posterior axillary line might be responsible for their site. The dense adhesions around them made their detection difficult.

The local factors that we consider as the cause of the P.C.S. have hitherto escaped attention. A common opinion seems to be that the P.C.S. is due to rheumatic activity. Clearly a confusion exists in earlier reports between P.C.S. and genuine recurrences of rheumatic fever after valvotomy as described by Soulié et al. (1954) which we believe are two distinct entities that can be differentiated. The clinical condition that most resembles P.C.S. is idiopathic recurrent pericarditis (Dressler, 1954). Patient H.S., now aged 43, is our only case of this kind under observation. From 1936 till 1953 he had eight relapses lasting from one to three weeks, with pericardial rub, mild pyrexia, minimal rise of E.S.R. and slight electrocardiographic changes. No rheumatic or tuberculous ætiology could be established in this otherwise very fit man.

The strongest argument advanced in favour of rheumatic activity is the absence of P.C.S. after operation in congenital heart disease. Our material of congenital cases is too small to be used for comparative study, but in one case clinical and electrocardiographic signs of pericarditis with associated rapid increase in size of the cardiac silhouette followed Blalock anastomosis. The electrocardiograms published by Brock and Campbell (1950) after infundibular resection for infundibular stenosis are similar to our Fig. 3 and may be interpreted as due to pericarditis. The only operation for congenital heart disease that is similar in surgical trauma to mitral valvotomy is that for auricular septal defect and here an illness similar to that of the P.C.S. has been observed (Likoff, 1954).

Until more reliable laboratory tests for rheumatic activity are forthcoming the P.C.S. should be considered as due to local trauma, with postoperative hæmorrhage and loculus formation as the main causative factors. Though it should be recognized as a clinical entity, its importance should not be overrated; it may be prolonged, but spontaneous recovery is the rule and it has no
adverse influence on the outcome of the operation. The additional discomfort it causes in 22 per cent of the patients after mitral valvotomy is a small price to pay for the lasting benefit conferred by the operation.

**SUMMARY**

The postcommissurotomy syndrome (P.C.S.), an illness characterized by pyrexia, pericarditis, left pleural effusion, and tendency to relapses, was observed in 22 out of 100 patients who had mitral valvotomy. It followed closely upon the operation in 14 patients (Group I) or appeared after a time lag of four weeks to four months following operation in 8 patients who had a normal postoperative course (Group II). Two patients in each group had further relapses.

Pyrexia was moderate and continuous and lasted from 8 to 34 days in Group I and from 4 to 14 days in Group II.

Pericarditis was mostly revealed by the electrocardiogram and the presence of a pericardial rub. Pain was infrequent and radiological signs were exceptional.

Left pleural effusion persisted for weeks or months and recurred after apparent absorption. It became loculated in 4 patients; emptying of the pockets in 2 led to rapid recovery. The effusion was blood-stained in 10 out of 11 patients, when aspirated between the fifth and twenty-second postoperative day. The cultures with one exception were sterile.

Relapses, with similar symptoms and duration, occurred 3–10 weeks after the first bout of illness.

Comparative study in uncomplicated cases showed that a few days of pyrexia, left pleural effusion, which becomes serous after the first week and absorbs within a fortnight, and transitory signs of pericarditis are part of the normal postoperative course. The P.C.S. thus appears as the accentuation, persistence, and recurrence of the normal postoperative sequele. It is caused by postoperative oozing from the auricular wound, as shown by the blood-stained effusion, responsible for a recurrent pleuro-pericarditis. Loculation of the effusion and left basal lung consolidation may constitute additional factors. Though the P.C.S. has some features in common with rheumatic pericarditis and has been attributed to it, it should be distinguished from the latter by the absence of preceding streptococcal infection, the absence of arthritis, of carditis, of a positive salicylate effect, features that were present in the few instances of overt postoperative rheumatic recurrence. There is no available specific treatment. The disease, however prolonged, causes little systemic upset, has a good prognosis and has no adverse influence on the outcome of the operation.

**ILLUSTRATIVE CASES**

*Postcommissurotomy syndrome.* A woman, aged 50 years, had been breathless on effort for two years, increasingly so for six months, with orthopnea for six weeks. On examination she was dyspneic on slight exertion. She had mitral stenosis with slight incompetence, and auricular fibrillation but no peripheral signs of congestive heart failure. Radioscopy showed mitral shaped heart with some enlargement. At operation (12/2/52) tight stenosis was found and a good split was performed.

*Postoperative course.* The patient was semi-comatose for three days with transient weakness of the left side of the body (? cerebral embolus). There was persistent pyrexia from the first to fourteenth postoperative day with no response to penicillin (Fig. 2). The electrocardiogram showed evidence of pericarditis. Blood-stained effusion was aspirated from the left pleural cavity on the tenth day (40 oz.), fourteenth day (6 oz.) and nineteenth day (14 oz.). The effusion became straw coloured when aspirated on the twenty-fifth day (26 oz.). There was no congestive heart failure and there were no joint pains at any stage.

Progress at home was satisfactory for the next seven weeks when she developed pyrexia and complained of severe pain across the front of the chest. When re-admitted to hospital left pleural effusion was present (Fig. 7). Aspiration yielded 12 oz. of straw-coloured fluid; the electrocardiogram showed persistent abnormalities of pericarditis. Irregular fever continued for eight weeks with no response to penicillin or chloramphenicol. There was no congestive heart failure and she never complained of joint pains. In the eighth week she became mentally confused and developed some weakness of the left arm and leg with complete recovery in two days (cerebral embolus). Subsequent progress has been good and she has experienced a fair increase of effort tolerance maintained till the present time.
THE POSTCOMMISSUROTOMY SYNDROME

Postcommissurotomy Syndrome with Rheumatic Recurrence. A woman, aged 46 years, had rheumatic fever when aged 14 with a recent recurrence at the age of 42. For 10 years she has been short of breath on effort and was admitted to hospital on three occasions for treatment of congestive heart failure. Recently orthopnoea set in and she had haemoptysis on one occasion. On examination she was dyspnoic on slight exertion. There were signs of mitral stenosis with pulmonary hypertension, a good opening snap and auricular fibrillation, but no peripheral signs of congestive heart failure. Radioscopy showed generalized cardiac enlargement with large left auricle and right ventricle and pulmonary congestion. At operation (18/2/52) the mitral valve was found to be tightly stenosed and calcified; a good split was performed but slight incompetence was produced.

Postoperative course. The patient was febrile from the fourth to twelfth postoperative day with no response to penicillin or chloramphenicol. Thereafter she was apyrexial for three months, but during this period she experienced no subjective improvement. She had to be admitted as an emergency three-and-a-half months after operation on account of severe precordial pain and fever. She had an extensive pericardial rub and one week later she developed generalized joint pains. There was a leucocytosis of 34,000 and she developed congestive heart failure. On treatment with salicylates and mercurial diuretics she improved and she was discharged from hospital after two months. Her subsequent course was complicated by recurrent congestive heart failure and severe dyspnoea. She died in congestive failure 18 months after operation.

Rheumatic Recurrence Two Years after Valvotomy. A woman, aged 27 years, had chorea from 8 to 10 years of age but no rheumatic fever. She had been breathless on effort for 18 months; recently she had repeated large haemoptyses and two attacks of pulmonary oedema. Her effort tolerance has been much restricted since, on account of orthopnoea. On examination she was in sinus rhythm with no peripheral signs of congestive heart failure. There was mitral stenosis with considerable pulmonary engorgement on radioscopy, but little cardiac enlargement. At operation (1/8/50) severe stenosis was found and a good split performed.

Postoperative course. Recovery was uneventful with great improvement and she became able to lead an unrestricted life for two years. In January 1953 she developed rheumatic fever and was treated at another hospital for one month, with two further months in bed at home. Thereafter she remained well until October, 1954, when orthopnoea again became evident and an aortic diastolic murmur was detected. In November, 1954, auricular fibrillation set in; it could not be converted to sinus rhythm by quinidine. In spite of control of the ventricular rate with digitalis, the effort tolerance deteriorated and orthopnoea increased. Cardiac catheterization showed P.C.V. mean pressure 35 mm. Hg and P.A. mean pressure 55 mm. Hg. Re-stenosis was diagnosed; this was confirmed at a second valvotomy (12/4/55), when a good split was performed. Great subjective improvement followed and this is still maintained at the present time.

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