A POSTOPERATIVE COMPLICATION IN A PATIENT WITH A MITRAL COMMISSUROTOMY

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MANY PATIENTS have now had the operation of mitral commissurotomy for mitral stenosis following rheumatic fever. Of these, many are now surviving for a long time, so that they are living long enough to encounter the same surgical risks as the ordinary population. When these people are presented for surgery, they may require special care rather than management in a routine manner. From this viewpoint, I wish to discuss the following event.

Mrs. N. A., who had a mitral commissurotomy in 1956, was presented for partial salpingectomy. The indications for partial salpingectomy were that she had been in heart failure during her third pregnancy and that she found it difficult to look after the three children she had at home. She was not in congestive cardiac failure but was easily tired. She became short of breath occasionally on exertion. The blood pressure was 110/70 mm. Hg and the pulse rate was quite regular at 78 per minute. The internist had noted slight mitral insufficiency with a Graham-Steell murmur or slight aortic incompetence.¹ The haemoglobin was 12.5 G, the urine was free of sugar and albumin, her weight was 40 kg., and the anaesthetist who saw her preoperatively classified her as a "fair candidate."

Premedication on the morning of operation consisted of the following: (1) sodium pentobarbital 100 mg. orally at 9:45 a.m.; (2) morphine sulphate 10 mg. and hyoscine hydrobromide 0.2 mg. by intramuscular injection at 10:15 a.m. At 11:20 a.m. anaesthesia was induced with 250 mg. of a 2.5 per cent solution of sodium thiopentone. After 60 mg. of succinylcholine chloride, the patient was intubated with a No. 9 Magill cuffed orotracheal tube. The induction was smooth and trouble-free. The anaesthesia was maintained with oxygen and cyclopropane using a 0.1 per cent succinylcholine chloride drip in normal saline for relaxation with controlled respiration in a closed circuit with a carbon dioxide absorber. The operation, a partial salpingectomy and appendectomy, started at 11:30 a.m. and ended at 12:55 p.m. There were no complications and blood loss was minimal. The fluids given were: (1) normal saline 100 ml. containing 100 mg. of succinylcholine chloride; (2) a bottle of 5 per cent dextrose-water containing 500 ml., which was still running slowly when the patient entered the postanaesthetic room. A further 1000 ml. of 5 per cent dextrose-water was ordered to be given slowly. The patient recovered quickly and left the postanaesthetic room at 3:30 p.m.

At 4:00 a.m., this lady developed congestive cardiac failure with: (1) orthopnoea, with a respiratory rate of 28 per minute; (2) râles all over the chest; (3) tachycardia with a pulse of 130 per minute and B.P. 115/65; (4) neck vein distension. She was given Digoxin 1 mg. intravenously, sodium mercaptomerin 80 mg. (2 c.c. Thiomerin) by intramuscular injection, and Pethidine 100 mg. by

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intramuscular injection, and was placed in an oxygen tent. The Digoxin was continued with 0.25 mg. given daily and, on this regime, she recovered from her cardiac failure within the next few days. An E.C.G. at the time of this episode showed right ventricular hypertrophy with dilatation of the right ventricle but no atrial fibrillation. A chest X-ray showed some pneumonitis in the left first and second interspaces anteriorly. This was treated with 200,000 units Penicillin G Procaïn and 0.25 gm. Streptomycin twice a day for 4 days and Chloramphenicol 1000 mg. twice daily for 5 days. An X-ray of the chest was clear 6 days later.

DISCUSSION

One must ask why this patient developed congestive heart failure postoperatively, and whether any treatment or precaution would have prevented this. In commissurotomy patients 61 per cent have a good result at 2 years and 51 per cent of these have a good result at 5 years. Age is no bar to operation but atrial fibrillation, cardiac enlargement, and mitral incompetence impair the chances of these patients so that two-thirds are well at 2 years and only one-third are well at 5 years following operation.² As well there exist some other factors producing poor results and these are aortic and tricuspid valve disease, pregnancy, bacterial endocarditis, systemic hypertension, myocardial failure, chronic lung disease, and especially bronchitis and emphysema.³

In addition some patients may die from assorted lesions, such as cerebral and pulmonary embolus, alcoholic cirrhosis, and sudden arrhythmias. The rate of deterioration of all patients seems to be steady at 5 per cent per year.⁴ Speaking physiologically, it is stated that "cardiac output in relation to oxygen consumption remains below normal both at rest and during exercise" in patients with a mitral commissurotomy. Thus, it appears that valvulotomy, even when successful in relieving symptoms, does not necessarily restore normal haemodynamics.^{5,6} Another thought is that these patients may have an expanded central blood volume which could make them more susceptible to pulmonary oedema. Moir and Gott using isotopic iodine-131 in human serum albumin investigated this but were not able to demonstrate an increase in central blood volume in patients with mitral stenosis, though this may have been due to the experimental technique. However, they did ascertain that: "All of the normal subjects and the patients with mitral stenosis showed a rise in cardiac output after exercise together with a decrease in Mean circulation time. However, the Mean increase in Cardiac Output was significantly greater in normal subjects than in patients with mitral stenosis."⁷ Of course these results refer to mitral stenosis and not to mitral commissurotomy patients but it is a reasonable speculation that some of these findings are correct for a patient with a mitral commissurotomy.

Returning to our own case, it is possible that our patient suffered congestive heart failure since we did not give digitalis preoperatively, even though she was not showing atrial fibrillation or heart failure. A clue is given from the work of Kittle and Crockett, who concluded that the exhibition of digitalis and quinidine reduced the immediate postoperative incidence of atrial fibrillation in patients who had just had a mitral valvotomy, remembering, however, that our instance is not quite identical.⁸

Also it is possible that this patient came to grief through fluid overload. She had 100 ml. of *N* saline and 1500 c.c. of 5 per cent dextrose-water in the immediate postoperative period, which does not seem excessive at first glance. However, the figures are usually given for a 70-kg. adult, while our patient weighed only 40 kg., so she could have conceivably been given an overload. It is important to remember that, after any operation, the water and salt equilibrium is disturbed so that: (a) there is an impairment of water excretion by the kidney, (b) there is an impairment of sodium excretion by the kidney, (c) there is a temporary rigidity of kidney function so that the kidney is unable to adjust excretion to intake.⁹ Also there is a reduction in renal blood flow under light cyclopropane anaesthesia from 1200 ml./minute to 800 ml./minute.¹⁰

To conclude, our patient's troubles may have arisen from

1. Inefficient myocardium.
2. Fluid overload.
3. Lack of preoperative digitalization.
4. Pneumonitis postoperatively.

I hope this report may prove instructive to others who encounter similar patients in the future.

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RÉSUMÉ

Congestive cardiac failure as a complication postoperatively is reviewed in a special instance. This patient had had a mitral commissurotomy five years before, and the present procedure was a partial salpingectomy. This patient developed cardiac failure due possibly to: (1) inefficient myocardium, (2) fluid overload, (3) lack of preoperative digitalization, (4) pneumonitis postoperatively.

Some of the literature is reviewed.

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