The major E.C.G. changes were a very pronounced general low voltage and a marked prolongation of the electrical systole (figs. 1 and 2). The low voltage was most pronounced immediately after the onset of the cardiac symptoms, improved slowly thereafter, and disappeared after 11 weeks; it was noticeable mostly in the peripheral and left precordial leads. In contrast to the low voltage the Q-T interval was only slightly prolonged immediately after the onset of the acute attack and reached its maximum five days later (0.62 sec—that is, a prolongation of 59% over the normal for the given heart rate). The Q-T interval had returned to normal three weeks later although the voltage was still low. Another noticeable feature was the negative T wave in leads I, II, aVL, and V₂-V₆, suggesting a subepicardial lesion. This inversion of the T wave lasted 11 weeks.

During the first four weeks after the acute attack the patient complained of extreme weakness and occasional anginal symptoms. The chest radiograph remained normal. In the course of the next few weeks her symptoms gradually subsided and she became able to walk again without difficulty for short distances. She was discharged from the clinic 11 weeks after the onset of the cardiac symptoms. At that time the E.C.G. showed no difference compared with the findings before the starvation period (fig. 1).

Comment

Prolonged total starvation is generally well tolerated by obese but otherwise healthy persons and has been maintained for periods up to 249 days without any complications (Thomson *et al.*, 1966). On the other hand, sudden death as a con-

sequence of total caloric restriction of relatively short duration has been reported in persons with pre-existing signs of heart failure or myocardial damage (Spencer, 1968). Moreover, Garnett *et al.*, (1969) recorded the sudden death from ventricular fibrillation of a healthy woman aged 20 seven days after the termination of 30 weeks of total starvation for the treatment of obesity—12 weeks longer than in the present case. Shortly before death the E.C.G., which had been normal one month previously, showed low voltage and prolongation of the Q-T interval, which apparently were not caused by a disturbance of the electrolyte levels. The patient had already regained her ideal weight. Garnett *et al.*, suggested that the changes in the E.C.G. and the sudden death of their patient were due to gross protein loss causing massive destruction of the cardiac myofibrillae.

We suggest that in grossly obese but otherwise healthy persons therapeutic starvation may be carried out under close supervision for three to four weeks. The fast should then be followed by a low-calorie diet, with possibly one or two days of total fasting per week. If necessary starvation may be repeated later for a similar period.

References

- Garnett, E. S., Barnard, D. L., Ford, J., Goodbody, R. A., and Woodhouse, M. A. (1969). *Lancet*, 1, 914.
 Spencer, I. O. B. (1968). *Lancet*, 1, 1288.
 Thomson, T. J., Runcie, J., and Miller, V. (1966). *Lancet*, 2, 992.

Spontaneous Oesophageal Rupture after Carbachol Therapy

P. COCHRANE

British Medical Journal, 1973, 1, 463-464

For many years carbachol has been used to promote emptying of a distended urinary bladder. More recently Goodman and Gilman (1970) have drawn attention to untoward side effects of the drug. A case of oesophageal rupture after the administration of carbachol is described here.

Case Report

A 58-year-old man was admitted to hospital with a history of having vomited some reddish-black material two hours previously after a subcutaneous injection of carbachol given to relieve urinary retention. The vomiting was immediately followed by severe central chest pain and increasing breathlessness. His doctor arranged his admission to hospital with a presumptive diagnosis of myocardial infarction.

On admission he was restless, breathless, and in considerable chest pain, with a pulse rate of 100/min and a blood pressure of 140/90 mm Hg. Generalized inspiratory and expiratory rhonchi were audible. An electrocardiogram showed no evidence of a myocardial infarction but this was thought to be the most likely diagnosis, with dissection of the aorta as an alternative. Two hours later, when he had become even more breathless and cyanosed and congestive changes were present on chest x-ray examination, a pulmonary embolus was suspected. Over the next 12 hours the condition was unchanged and a

diagnosis of ruptured oesophagus was made after a bloodstained, left-sided pleural effusion had been aspirated. By this time surgical emphysema was present in the neck, and radiography showed mediastinal emphysema and a left-sided hydropneumothorax. At a left-side thoracotomy 22 hours after the onset of symptoms the left pleural cavity was found to be filled with black fluid, the lung and chest wall covered with fibrin, and the mediastinum quite distended. The pleura was opened and the lower 8 cm of the oesophagus dissected, revealing a 3-cm tear in the posterior wall. This was repaired.

On the third postoperative day the suture line gave way, and an oesophageal leak was confirmed by a Gastrografin swallow. Left pleural drainage was maintained and on the ninth day oesophagoscopy confirmed this complication. Later a right-sided pleural effusion was also drained. On the 12th day a tracheostomy was performed because the chest condition was deteriorating, and the patient was maintained with intermittent positive pressure ventilation. This was discontinued on the 17th day and the tracheostomy tube was removed six days later. Unfortunately, on the 24th day he developed a pyrexia of 40°C associated with a left-sided empyema and he died after a cardiac arrest the next day. Permission for a postmortem examination was refused.

Comment

The term "spontaneous" oesophageal rupture should not imply a previously normal oesophagus or an absence of other precipitating factors (Anderson, 1957). Perhaps "post-emetic" is a more descriptive term, though the rupture may occur without vomiting after physical straining, childbirth, and seizures (Tesler and Eisenberg, 1963). In 80% of cases spontaneous oesophageal ruptures occur in men in their fifth or sixth decades, typically after food or alcoholic overindulgence. It still has a mortality of 30%, partly owing to delayed diagnosis and to pre-existing oesophageal disease (Wychulis *et al.*, 1969). In the present case failure to appreciate the characteristic symptom-sequence of vomiting, chest pain, and dyspnoea led to delay in diagnosis. Pre-existing oesophageal disease was not noted at the operation but a postmortem examination was not performed.

Carbachol is a cholinergic drug which until recently has been used in the emergency treatment of acute urinary retention. It

Walton Hospital, Liverpool L9 1AE

P. COCHRANE, M.D., M.R.C.P., Senior Medical Registrar

has become unpopular because of its many side effects, which include vomiting, defaecation, abdominal pain, perspiration, salivation, bradycardia, and hypotension (Goodman and Gilman, 1970). The present case of spontaneous oesophageal rupture may well have been caused by the emetic action of the drug increasing the intraoesophageal pressure. In addition to its emetic property carbachol has a direct cholinergic stimulatory action on the smooth muscle of the oesophagus (Ingelfinger, 1958), which may also have contributed to the rupture by increasing intraoesophageal pressure. The transient chest and abdominal pain which may occur after an injection of the drug may reflect this smooth muscular spasm of the oesophagus and bowel respectively, and carbachol is contraindicated when mechanical obstruction of the bowel or urinary bladder is suspected. Nevertheless, I have been unable to find any reports of

rupture of the bladder or bowel after the administration of the drug. At present carbachol should be considered only for the treatment of intestinal and urinary atony.

I wish to thank Mr. J. K. B. Waddington for his advice in the preparation of this report and allowing access to the operation notes.

References

- Anderson, R. L. (1957). *American Journal of Surgery*, 93, 282.
 Goodman, L. G., and Gilman, A. (1970). In *Pharmacological Basis of Therapeutics*, 4th edn., p. 472. London, MacMillan.
 Ingelfinger, F. J. (1958). *Physiological Review*, 38, 533.
 Tesler, M. A., and Eisenberg, M. M. (1963). *Surgery and Gynecology*, 117, 1.
 Wychulis, A. R., Fontana, R. S., and Payne, W. S. (1969). *Diseases of the Chest*, 55, 190.

Erysipelothrix Septicaemia without Endocarditis

R. H. TOWNSHEND, A. E. JEPHCOTT,
M. HASAN YEKTA

British Medical Journal, 1973, 1, 464

Only 19 cases of septicaemia due to *Erysipelothrix rhusiopathiae* have previously been reported in the world literature. The case reported here, the tenth to survive, is unique in that there was no evidence of endocarditis.

Case Report

A 52-year-old woman who worked in a fish-and-chip shop was admitted to hospital with a history of an attack of epigastric pain, dyspnoea, sweating, and vomiting seven weeks previously which was succeeded by lassitude, occasional shivering and sweating attacks, and increasing dyspnoea. One week before admission she developed right-sided pleural pain and fever which did not respond to tetracycline and co-trimoxazole. She was a pale, anxious woman with extensive psoriasis and badly bitten fingernails. Her heart was normal and the blood pressure 110/75 mm Hg. Basal rales and crepitations were heard in both lungs, more pronounced on the right. Blood examination showed Hb 10.7 g/100 ml and W.B.C. 10,700/mm³. The chest radiograph showed increasing basal shadowing.

Bronchitis was diagnosed, and treatment was started with tetracycline followed by penicillin 1 megaunit six hourly with streptomycin 0.75 g daily. There was no lasting improvement and the drugs were stopped after only four days. A chest radiograph then showed appearances typical of pulmonary infarction on the right side and a probable infarct on the left side. Though there was no evidence of venous thrombosis repeated small pulmonary infarcts were thought to be the probable cause of the illness. The patient rapidly improved on anticoagulant treatment with warfarin. The pyrexia settled after three days and remained normal for three weeks.

After three weeks, despite satisfactory anticoagulant control, the pyrexia recurred. The white cell blood count, which had fallen to 7,000/mm³, rose to 15,000/mm³ with a neutrophil leucocytosis. Further chest radiographs and tomographs showed changes typical

of infarction, and the spleen became palpable. Three successive blood cultures grew *Erysipelothrix rhusiopathiae*, the isolates being morphologically, culturally, and biochemically indistinguishable from this organism as described in standard reference works (Wilson and Miles, 1964; Cowan and Steel, 1965) and in individual reports (Klauder *et al.*, 1943; Procter, 1965). Sputum culture was negative.

Treatment with penicillin 1 megaunit six hourly was started and the warfarin stopped. The pyrexia settled completely the next day and did not recur. Still no heart murmur was heard and phonocardiograms were normal. Phlebograms of both legs showed normal veins up to the inferior vena cava, and no dysgammaglobulinaemia was found by immunoelectrophoresis. Penicillin was stopped after four weeks and the patient was discharged from hospital. Three months later she was readmitted with a further episode of cough and fever. Each of four blood cultures was sterile, and she improved rapidly without treatment. She has remained well since with only occasional, mild attacks of bronchitis. Chest radiographs have shown gradual clearing of the basal opacities.

Comment

Skin infections due to erysipelotheix are common among meat and fish workers, causing erysipelotheix. Systemic infections are rare. In all the 10 fatal cases reported (Layani *et al.*, 1966; Morris *et al.*, 1965; Mandal and Mallock, 1971) endocarditis was present and damage to one or more heart valve was found in those examined postmortem. Endocarditis was also diagnosed clinically in all the patients who survived, and heart murmurs were present in every case. Our case is without precedent in that no heart murmur was heard at any time and the septicaemia was accompanied by repeated episodes of pulmonary infarction.

The portal of entry of the infection remains unknown. No cuts or abrasions were noted, but anyone constantly cutting up fish must receive scratches, and nail biting may have been a contributory factor. The focus of infection was probably somewhere in the venous system or in the right side of the heart, but not in the heart valves. Infected emboli presumably broke loose to cause the pulmonary infarcts.

References

- Cowan, S. T., and Steel, K. J. (1965). *Manual for the Identification of Medical Bacteria*, London, Cambridge University Press.
 Klauder, J. V., Kramer, D. W., and Nicholas, L. (1943). *Journal of the American Medical Association*, 122, 938.
 Layani, F., May, V., and Machon, F. (1966). *Presse Médicale*, 74, 1733.
 Mandal, B. N., and Mallock, J. A. (1971). *New Zealand Medical Journal*, 73, 355.
 Morris, C. A., Schwabacher, H., Lynch, P. G., Cross, C. D., and Dada, T. O. (1965). *Journal of Clinical Pathology*, 18, 614.
 Procter, W. I. (1965). *American Journal of Medicine*, 38, 820.
 Wilson, G. S., and Miles, A. A. (1964). *Topley and Wilson's Principles of Bacteriology and Immunity*, 5th edn., London, Edward Arnold.

Lodge Moor Hospital, Sheffield S10 4LH

R. H. TOWNSHEND, F.R.C.P., Consultant Chest Physician
 M. HASAN YEKTA M.D., D.T.C.D., formerly Senior House Officer

Public Health Laboratory, Sheffield

A. E. JEPHCOTT, M.B., M.R.C.PATH., Consultant Bacteriologist