Irregular swellings of the axons were seen (fig. 3), and there was severe demyelination.

FIG. 2—Teased nerve preparations showing rosettes of oxalate crystals within perineurium and single leaflets of oxalate crystals within axon. (Osmium tetroxide. $\times 285$.)

Discussion

Our general necropsy findings did not materially differ from those in the literature except in the nervous system. The failure of clinical response to adequate dialysis has already been noted.⁸ In our patient peripheral neuropathy began at about the time intermittent haemodialysis was first started and progressed throughout life.

The most striking feature was the presence of microcrystals of calcium oxalate actually within axons (fig. 2). There is little doubt that such crystals, which are about the same diameter as the axon, would interfere with normal axoplasmic flow. The axonal migration of protein takes place in various peripheral nerves and in the central nervous system⁹ and is essential to the viability of distal parts of the axon. It is possible that a micro-

FIG. 3—Teased nerve preparation showing severe demyelination and irregular swelling of axons. (Osmium tetroxide. $\times 57$.)

crystal of calcium oxalate proximally placed would suffice to impair the function of an axon considerably more distally if axonal circulation were only incompletely obstructed. Demyelination of the peripheral nerve in our case was probably a secondary event.

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PRELIMINARY COMMUNICATIONS

Electrical Requirements for Ventricular Defibrillation

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Summary

Most deaths from ischaemic heart disease are sudden, occur outside hospital, and result from ventricular fibrillation. But defibrillators have only limited avail-

ability because of their size and weight. A miniature defibrillator has been developed. A single low-energy shock succeeded in removing ventricular fibrillation in 73 out of 82 episodes, and a further shock was successful in seven more episodes. Primary ventricular fibrillation probably always responds to low-energy electrical shocks, which challenges the conventional view that correction of ventricular fibrillation requires high-energy direct-current shock. Thus even smaller and lighter defibrillators are possible. Furthermore low-energy shocks cause less myocardial damage.

Introduction

Ischaemic heart disease is the major cause of premature death in the Western world. Over 60% of the deaths from acute coronary attacks among middle-aged and younger people occur within one hour of the onset of symptoms,¹ and more than 90% of these sudden deaths result from ventricular fibrillation.²⁻⁴ The limitations⁴⁻⁶ of prophylactic antiarrhythmic therapy in the prevention of sudden deaths are probably related to the

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definite but transient lowering of the ventricular fibrillation threshold immediately after the onset of coronary occlusion.⁷

Most sudden deaths from ischaemic heart disease occur outside hospital but they are preventable since the correction of ventricular fibrillation outside hospital is a practicable proposition.⁸ Paramedical workers are capable of correcting ventricular fibrillation,⁹⁻¹² and various prehospital coronary care schemes have been evolved.⁹⁻¹⁰⁻¹³⁻¹⁵ Furthermore, attempts have been made to identify individuals at risk of sudden death¹⁶⁻¹⁸; and the implantation of defibrillators has been considered,¹⁹ though there are apparently insurmountable problems.²⁰ Friedberg²¹ has suggested that those at risk should have a defibrillator available, even at home.

Major barriers to the widespread availability of portable defibrillators for use outside hospital by general practitioners, paramedical workers, and others are their cost and their bulk and weight; some defibrillators weigh 25 kg. A defibrillator weighing less than 3.2 kg (see fig. 1), however, has been designed and is being used in Belfast. Further reduction in the size and weight of defibrillators and the eventual production of a pocket defibrillator might be possible if the delivered energy was smaller than that currently considered necessary.

FIG. 1—Belfast miniature defibrillator.

It has been suggested that the energy delivered from current defibrillators may be insufficient to achieve defibrillation in large people.²²⁻²⁴ Thus Tacker *et al.* claimed that the energy delivered by most defibrillators—300 W s from a stored energy of 400—was unlikely to be effective in 50% of patients weighing 82 kg and showed it to be ineffective in 60% of patients weighing 90-100 kg.²² Nevertheless, a shock of this energy successfully defibrillated a patient weighing 145 kg,²⁵ and we have observed that shocks of 150-165 W s are effective in removing ventricular fibrillation. We therefore studied prospectively the efficacy of low-energy shocks using miniature defibrillators.

The Belfast Defibrillator

In the miniature defibrillator (fig. 1) the electronic components are incorporated in the paddles. The unit is powered by a 20.4-V rechargeable sintered plate nickel-cadmium battery. The 50- μ F capacitor when charged to 4 kV and discharged through a 50-mH inductor to a 50-ohm (Ω) load produces a monophasic pulse, critically damped (fig. 2). This waveform has been found in experiments to be the most effective in correcting ventricular fibrillation.²² The pulse

FIG. 2—Waveform of Belfast defibrillator.

rise time is 1.2 ms and its width 12 ms when the impedance is 50 Ω . From a stored energy of 400 Watt seconds (W s) 330 are delivered through a resistance of 50 Ω ; from a stored energy of 200 W s 165 are delivered. The battery is capable of delivering 30 shocks of 400 W s before recharge.

Patients and Methods

From August 1974 to January 1975 82 episodes of ventricular fibrillation were observed in 43 patients, 22 of whom had a single episode. Two types of defibrillator were used, each charged to 200 W s; from this stored energy the Belfast defibrillator (used in 57 episodes) delivered 165 W s. through a resistance of 50 Ω and the American Optical apparatus 150 W s. When the initial shock failed to remove ventricular fibrillation a further identical low-energy shock was applied immediately. The effect of each shock was observed on a continuous recording of the cardiogram on paper.

Of the 43 patients (33 men, mean age 60 years; 10 women, mean age 64 years) 18 had anterior and 19 diaphragmatic infarction, two had left bundle branch block with significant enzyme change, one had acute coronary insufficiency, and three had severe cardiac failure complicating chronic ischaemic or rheumatic heart disease. Twenty-five patients had had one or more previous myocardial infarcts. The duration of ventricular fibrillation before attempted removal varied from four seconds to 20 minutes; in 64 episodes it was two minutes or less. The time from the onset of symptoms to the onset of ventricular fibrillation in patients with acute infarction ranged from 0 minutes to 23 days, 44 episodes occurring within six hours and 16 episodes within one hour. Twenty-eight patients had 57 episodes of primary ventricular fibrillation (fibrillation occurring in the absence of cardiac failure or cardiogenic shock), and 15 had secondary ventricular fibrillation.

Results

Seventy-three episodes of fibrillation were converted to another rhythm by a single low-energy shock, while in seven a second low-energy discharge was successful. The outcome of the low-energy shock seemed to be unrelated to weight (see table). Ventricular fibrillation was removed in all patients weighing 91-110 kg. The weights of the patients who responded to a second shock varied from 62 to 81 kg. The two patients in whom low-energy defibrillation was unsuccessful weighed between 80 and 90 kg; both had secondary ventricular fibrillation. One had severe left ventricular failure complicating chronic ischaemic heart disease. Asystole from which the patient could not be resuscitated followed defibrillation by a high energy discharge. The second patient had had two episodes of myocardial infarction, a ventricular aneurysm, and bilateral bundle branch block; though defibrillation was achieved by high-energy discharge the patient died.

Incidence of Successful Defibrillation with Low-energy Shocks according to Body Weight

Body weight (kg):	50-60	- 70	- 80	- 90	- 100	- 110
No. of episodes ..	5	43	21	8	3	2
% Success ..	100	100	100	75	100	100

Discussion

It is usually assumed that at least 400 W s is needed to correct ventricular fibrillation.²⁶⁻²⁷ That our findings differ from those of others, such as Tacker *et al.*,²²⁻²⁴ needs explanation. The difference is not related to the waveform of the shock since those of the Belfast and the American Optical machines were both effective at low-energy levels. Tacker *et al.*²² did not report the time between the onset of symptoms of acute myocardial infarction and the appearance of ventricular fibrillation. Coronary occlusion results in a fall in ventricular fibrillation threshold which continues for 30-60 minutes,⁷ and the energy requirements for defibrillation are greatest during this time.²⁸ In our study 13 of the 57 episodes of primary fibrillation occurred within one hour of the onset of symptoms and low-

energy shocks were successful then. The initial shock was unsuccessful in 7% (two out of 28) of patients with primary fibrillation, but a second shock of identical energy was successful in both patients. The success of the second shock when the initial one has failed^{2,9} may be related to the decreased impedance with successive shocks.^{3,0} Apart from allowing smaller and lighter defibrillators these lower energy shocks cause less myocardial damage.

Our findings indicate the need for further investigation of the energy needed for defibrillation. Tacker *et al.*²⁸ claim that defibrillators should have a much higher output capacity than they do at present, but high capacity output instruments would be much heavier, more cumbersome, and more expensive and, therefore, less generally available. If it is confirmed that low-energy direct current shocks, particularly when repeated, will almost invariably correct ventricular fibrillation a cheap and generally available pocket defibrillator will be a possibility.

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SHORT REPORTS

Carcinoma of the Oesophagus with "Swallow Syncope"

Swallowing is an unusual cause of syncope. In all the patients so far described in which an oesophageal lesion was found, it was benign. We describe a patient in whom swallow syncope was the presenting symptom of carcinoma of the oesophagus.

Case Report

A 74-year-old man was admitted to Kingston General Hospital, Hull, in September 1974 with a two-month history of recurrent syncopal episodes, which occurred only while he was eating or drinking. After each attack he immediately felt well and denied any prior chest pains or palpitations. There was no relevant past history, apart from angina pectoris for 12 years, but, on further questioning, he admitted that for two months he had noticed a sensation of his food "sticking" at the lower end of his sternum.

Examination showed no abnormalities. The E.C.G. showed sinus rhythm with T wave inversion in the inferior leads. Other routine investigations were all normal. Continuous E.C.G. monitoring showed that whenever the patient ate or drank he developed definite sinus bradycardia (often as slow as 15 per minute) and on one occasion sinus arrest lasting six seconds. This was always associated with a feeling of lightheadedness. Atropine 1.2 mg, intravenously given 10 minutes before meals prevented these episodes.

A barium swallow showed a large, dilated oesophagus with an irregular, fixed filling defect at the lower end. Fibreoptic endoscopy was performed (after the insertion of a temporary demand transvenous pacing catheter)

and a friable polypoid growth was seen encircling the lower end of the oesophagus. Biopsy of the growth showed a poorly differentiated adenocarcinoma which had infiltrated the submucosa of the oesophagus.

At operation oesophagoscopy was repeated with the pacemaker switched off. The obstruction was seen at 40 cm and when the oesophagoscope was passed beyond this point the patient developed a profound sinus bradycardia. This recovered completely as soon as the oesophagoscope was withdrawn above this level. Partial oesophagogastric anastomosis was then performed via a left thoracotomy. Postoperative recovery was uneventful. Three months later he was eating and drinking without discomfort. An E.C.G. recording at that time showed sinus rhythm with no change in heart rate on swallowing.

Discussion

Data on the 14 patients previously described with syncope on swallowing are summarized in the table. Syncope in relation to oesophageal carcinoma has not been reported before. In most reported patients syncope was due to disturbances of atrioventricular conduction; in only a minority of patients could digitalis be implicated as a contributory factor. In one patient His-bundle studies showed the block to be above the bundle of His.¹ In only one patient has syncope been attributed to sinus bradycardia with sinus arrest.²

Most authors postulate a vagovagal reflex mechanism. The abolition of the symptoms by atropine in our patient, also reported by others, supports the vagus as the efferent pathway; a vagal afferent pathway is also likely, though the possible role of the sympathetic nerves has not been explored.

Clinical and Pathological Features of Described Cases of Swallow Syncope

Reference	Age	Sex	Pathology	Known ischaemic heart disease	Observed Arrhythmia
MacKenzie, 1906	25	M	Unknown	None	A-V block
Weiss and Ferris, 1934	64	M	Oesophageal diverticulum	None	Complete heart block, ventricular arrest
Weiss and Ferris, 1934	59	F	Unknown	None	Sinus arrest
Iglauer and Schwartz, 1936	55	F	"Spastic oesophagus"	None	Complete heart block
Correll and Lindert, 1949	67	M	Oesophageal diverticulum	None	A-V block with slow irregular rhythm
James, 1958	58	M	Oesophageal diverticulum	Yes	A-V block leading to asystole
Deuchar and Troughton, 1960	63	M	Diffuse oesophageal spasm	None	Ventricular asystole
Pedersen, 1963	63	M	Achalasia of the cardia	Not stated	Atrial fibrillation and ventricular asystole
Kopald <i>et al.</i> , 1964 ²	48	M	Diffuse oesophageal spasm	None	Nodal or sinus bradycardia, sinus arrest with nodal escape
Tolman and Ashworth, 1971	45	M	Oesophageal stricture	Not stated	Sinus bradycardia
Sapru <i>et al.</i> , 1971 ³	56	F	None	None	Ventricular asystole
Levin and Posner, 1972 ⁴		M	Carcinomatous meningitis with demyelination of the vagus nerve. No oesophageal pathology.	None	
Lichstein and Chadda, 1972 ¹	85	M	None	None	A-V block
Alstrup and Pedersen, 1973 ⁵	64	F	None	Yes	A-V block
			Diffuse oesophageal spasm	None	A-V block