insulated flexible wires (24-gauge) lead from the patient electrodes to the spring-loaded pushbutton clamps on the "block," which in turn are connected to the fuses. The unshielded wires have a sufficiently low capacitance even when six feet long to avoid harmful electrocautery current flow after a fuse is blown.

The fuse block may be fastened to the bed sheet with an attached heavy-duty pinch clamp. When a fuse blows during use, no attempt is made to locate or replace individual fuses. The "six-pack" fuse holder is easily replaced. If only three of the six monitor leads are being used, the same "six pack" may be simply turned over to provide three new fuses. Fuse testing and replacement are performed by supporting personnel, using a special resistance–continuity meter built for the purpose.

Several such fused cables have been in daily use in the Presbyterian Hospital operating rooms for more than a year.

CASE REPORTS

Reversal of Chloral Hydrate-associated Cardiac Arrhythmia by a Beta-adrenergic Blocking Agent

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Beta-adrenergic receptor blocking agents represent a new class of pharmacologic agents which have been used experimentally in treatment of a wide variety of disorders. At the present time one such agent, propranolol, is available for use in the therapy of idiopathic hypertrophic aortic stenosis, pheochromocytoma, and cardiac arrhythmias. Other beta receptor blocking agents are currently under clinical investigation. Since 1966 we have been using one, alprenolol (Aptine) in the treatment of cardiac arrhythmias. Successful treatment of a rare arrhythmia with this agent forms the basis of this report.

REPORT OF A CASE

A 48-year-old white man who weighed 258 pounds was admitted to the hospital emergency room at 9 PM in a comatose state 30 minutes after ingestion of approximately 18 g chloral hydrate. He had a history of an old myocardial infarction. During the attempted passage of a gastric tube the patient vomited, aspirated gastric contents, and became cyanotic. Cardiac arrest occurred. An endotracheal tube was inserted and external cardiac massage was begun immediately. The pattern on the electrocardioscope was that of ventricular fibrillation. At 9:15 PM the patient was given 88 mEq sodium bicarbonate intravenously and the heart was defibrillated. This produced on the oscilloscope (a direct-writer was not available) what was interpreted as sinus rhythm at a pulse rate of 130 to 150 beats/min, with frequent PVC's. Blood pressure was 130/100 mm Hg. At 9:25 PM the patient was given lidocaine, 60 mg, with subsequent reduction in frequency of PVC's, which lasted about 20 minutes, after which they became more frequent and multifocal. The blood pressure dropped to the 80–100 mm Hg systolic and at times was unobtainable. At 9:50 PM the patient was given an additional 80 mg lidocaine and 44 mEq sodium bicarbonate, which produced marked reduction in PVC's and a blood pressure of 120/80 mm Hg. The patient was not breathing spontaneously, and the endotracheal tube was attached to a positive-pressure ventilator. At 10:25 PM the vital signs were stable with blood pressure was 130/80 mm Hg; pulse rate 120 beats/min. The cardioscope was detached from the patient and preparations were made to transfer him to the Intensive Care Unit. The immediately subsequent events are obscure, but the personnel attending the patient reported that his

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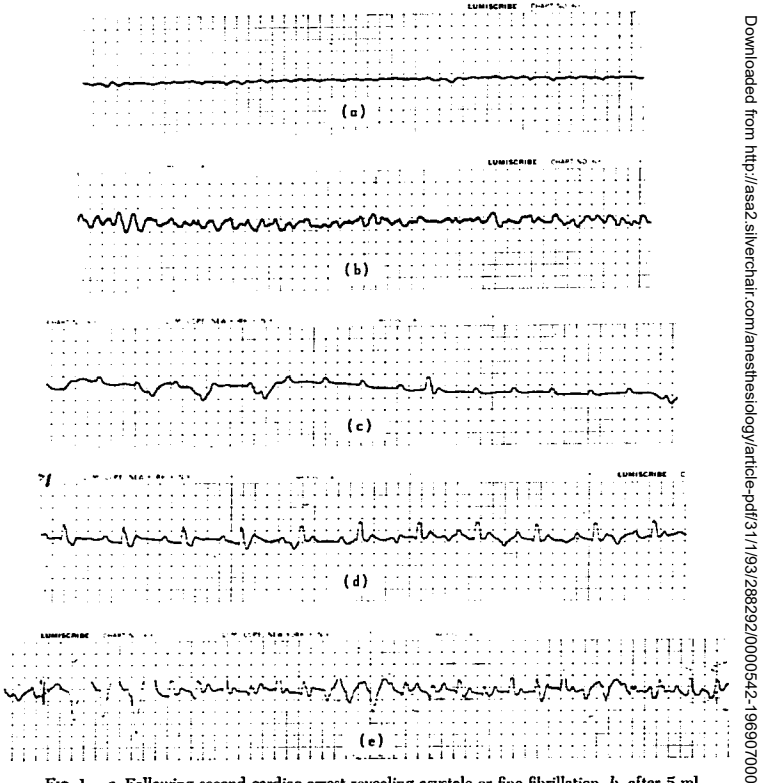
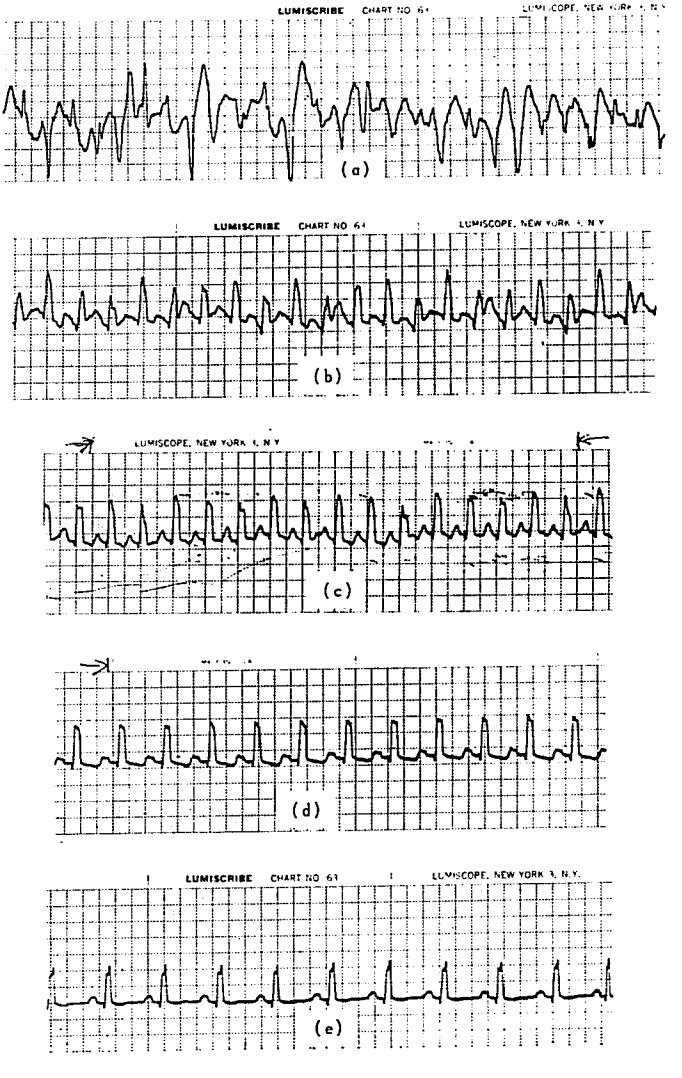


FIG. 1. *a*, Following second cardiac arrest revealing asystole or fine fibrillation, *b*, after 5 ml "disaster mix" showing coarse fibrillation; *c*, arrhythmia immediately after direct-current defibrillation; *d* and *e*, arrhythmia following administration of sodium bicarbonate.

status appeared satisfactory until 10:45 PM, when he again had sudden cardiac arrest. A direct writer was put into operation at this time and tracings were obtained. The electrocardiogram revealed the heart to be in asystole or very fine fibrillation (fig. 1*a*). At 10:49 PM the patient was given 5 ml of "disaster mix" (atropine, 2 mg; isoproterenol, 0.2 mg; phenylephrine, 4 mg; q.s. 20 ml saline), which produced a very coarse ventricular fibrillation (fig. 1*b*). He was given 88 mEq sodium bicarbonate at 10:52 PM, and the heart was defibrillated a few minutes thereafter. This produced atrial activity at a rate of 90 beats/min (fig. 1*c*), with gradually increasing ventricu-

FIG. 2. *a* and *b*, Arrhythmia during lidocaine therapy for 46 minutes, no blood pressure, continuous external cardiac massage; *c*, rhythm one minute after alprenolol, blood pressure 110/95 mm Hg; *d*, rhythm three minutes after alprenolol; *e*, rhythm ten minutes after alprenolol, blood pressure 130/80 mm Hg.



lar complexes appearing after another 44 mEq sodium bicarbonate was given at 10:58 PM and again at 11:20 PM. At this time, the electrocardiogram showed a 2:1 block with an atrial rate of 120 and ventricular rate of 60 beats/min (fig. 1*d*). Blood pressure could not be obtained, and external cardiac massage was continued. Shortly thereafter bursts of ventricular tachycardia appeared, followed by supraventricular tachycardia with multifocal PVC's alternating with ventricular tachycardia (fig. 1*e*). Therapy with intravenous lidocaine was started at 11:05 PM; the patient was given doses of 60–80 mg on four occasions during the next 46 minutes. The predominant rhythm during this time was supraventricular tachycardia with aberrant conduction (fig. 2*a*, *b*). There was no apparent cardiac output, and the patient was still being maintained with external massage. At 11:48 PM, approximately an hour after the second cardiac arrest, he was given 5 mg of the beta blocking agent, alprenolol. This dose was repeated six minutes later. Within a minute of the initial injection, the electrocardiogram began to show some regularity of rhythm and slowing of rate (fig. 2*c*). Blood pressure was 110/95 mm Hg at this time. Following the second dose, a definite sinus rhythm was noted at a rate of 100 beats/min, which gradually slowed over the next few minutes to 84 beats/min (fig. 2*d*, *e*). Blood pressure had risen to 130/80 mm Hg and cardiac massage was discontinued. Respiration became spontaneous; the patient continued to improve and was moved to the Intensive Care Unit.

Post-arrest complication amounted to an aspiration pneumonitis which was treated by intermittent positive-pressure ventilation, dexamethasone and antibiotics for ten days. Serial electrocardiograms during the recovery period showed an old inferior myocardial infarction with no change from tracings of four and six months previously. The patient was discharged completely asymptomatic on the eleventh postoperative day.

Comment

The standard pharmacology textbooks assert that adverse cardiac effects consisting of shortening of the refractory period and depressed contractility are produced only by toxic doses of chloral hydrate in patients with heart disease. Since the initial use of this drug as a hypnotic in 1869, few reports of its toxic myocardial effects in man have appeared in the literature.

In 1957 Muller and Fisch[1] described what they believed to be the first reported case of the adverse effect of the drug on the myocardium. Their patient was comatose about 45 minutes after ingesting 12 gm chloral hydrate. On admission an electrocardiogram showed atrial fibrillation with a ventricular rate of approximately 180 beats/min. Many of the beats exhibited aberrant conduction; occasional ventricular premature systoles were noted. Six hours later an electrocardiogram showed sinus tachycardia and slight depression of the ST segments in V5. Unfortunately, specific therapy for the arrhythmia was not mentioned in the report.

Gleich, Mongan and Vaules[2] treated a patient who was cyanotic and comatose approximately two hours after ingestion of 18 gm of chloral hydrate. Ten hours after admission the patient's blood pressure, which had been maintained at levels of 120/70 mm Hg with metaraminol given intravenously, dropped to 90/70 mm Hg. An electrocardiogram showed ventricular tachycardia, and 250 mg procainamide was given intravenously over a five-minute period. The cardiac rhythm became bigeminal after intravenous administration of an additional 100 mg procainamide it reverted to normal sinus rhythm. Three hours later the blood pressure became unobtainable and an electrocardiogram showed ventricular tachycardia with runs of ventricular fibrillation. An additional 850 mg procainamide was administered intravenously over the next two hours, with return of the cardiac rhythm to bigeminy and the blood pressure to 110/80 mm Hg. The patient was then given 500 mg procainamide intramuscularly every four hours and continued to receive the drug in decreasing dosages for the following six days with no further episodes of arrhythmia.

The arrhythmia in Muller and Fisch's case apparently was not life-threatening.[1] The complication in the patient reported by Gleich, Mongan and Vaules was considerably more serious, but was brought under control with conventional therapy in due time.[2] Our case was compounded by cardiac arrest, which probably enhanced the toxic effect of the drug and made the resultant arrhythmia refractory to lidocaine, one of the most dependable drugs for the treatment of acute ventricular arrhythmias. Absolute refractoriness could possibly be explained on the basis of the progressively increasing blood level of chloral hydrate during the period between the two cardiac arrests.

It now appears from many clinical reports that the beta-adrenergic blocking agents have

dual antiarrhythmic effects. In addition to their beta-adrenergic blockade there is a quinidine-like or local anesthetic-like action on the myocardium, which tends to increase the refractory period and decrease excitability. The latter property probably accounts for its effectiveness in the treatment of drug-induced arrhythmias, most notably digitalis, although Warner[3] recently has compiled a review of its increasing usage in the management of arrhythmias during anesthesia.

This report would add another drug to the growing list of arrhythmia-producers amenable to reversal by beta-adrenergic blockers. However, physicians should become thoroughly acquainted with the adverse effects as well as the contraindications of this group of drugs before attempting their use. It is recommended that well-accepted conventional forms of therapy be administered before resorting to the beta-adrenergic blockers.

Alprenolol (Aptine) (1-(0-allylphenoxyl)-3-iso propylamino-2-propranol), beta receptor antagonist, was supplied by Benjamin G. Covino, Ph.D., M.D., Medical Director, Astra Pharmaceutical Products, Inc., Worcester, Massachusetts. The author thanks Doctor Covino for his encouragement and advice in the preparation of the manuscript.

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Malignant Hyperthermia during Anesthesia

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Several reports describe the clinical signs, the prescribed treatment and the prognosis associated with malignant hyperthermia during anesthesia.[1-4] Wilson recently reviewed 40 cases. These were otherwise-healthy patients with a mean age of 21.7 years; the mortality was 73 per cent.[5]

We have treated a case of malignant hyperthermia which we present for several reasons: the signs of the syndrome were typical; there was a favorable response despite the severity of the disorder; the importance of early and vigorous treatment is demonstrated; the patient had been anesthetized twice before with no undesirable results.

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Case Report

On March 12, 1962, at the age of three months, the patient had her first operation for repair of a cleft lip. Premedication consisted of morphine, 0.2 mg, and scopolamine, 0.075 mg. Following oxygenation, 16 mg succinylcholine intramuscularly, and conscious intubation, anesthesia was maintained with nitrous oxide, ether and oxygen by means of an Ayer's T Tube. Procaine with epinephrine was used locally. Operation, anesthesia and postoperative course were uneventful.

On June 3, 1963, at the age of 18 months, the patient had her second anesthesia and plastic surgical procedure on the lip. Premedication was morphine, 0.5 mg, scopolamine, 0.1 mg, and secobarbital, 15 mg. Induction and intubation were accomplished with a cyclopropane–ether sequence. Succinylcholine was not used. Anesthesia was maintained with nitrous oxide and oxygen with an Ayer's T Tube.

On September 6, 1968, at the age of 6 years and 9 months, the patient was admitted for further correction of a cleft lip and nose deformity.