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E. J. IMMELMAN, M.B., CH.B., F.C.S.(S.A.).

J. H. PEACOCK, M.D., CH.M., F.R.C.S.

K. E. F. HOBBS, M.B., B.S., F.R.C.S.

S. K. MITRA, M.A., M.S.
Department of Surgery.

A. C. HUNT, M.D., M.C.PATH.
Department of Pathology.

J. B. BOWES, M.B., B.S., F.F.A. R.C.S.
Department of Anaesthetics.

University of Bristol.

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Medical Memoranda

Atrial Flutter with Block—Contraindication to use of Lignocaine

Brit. med. J., 1968, **2**, 223–224

Much interest has recently been aroused in the use of lignocaine as an antiarrhythmic agent (*Lancet*, 1967). This drug has been shown to be of value in the treatment of ventricular arrhythmias and atrial ectopic beats occurring after myocardial infarction, cardiac surgery, and direct-current cardioversion of atrial fibrillation (Hitchcock and Keown, 1959 ; Minuck, 1965 ; Spracklen *et al.*, 1968). Lignocaine has also been recommended in the treatment of digitalis-induced tachycardias (Katz and Zitnik, 1966 ; Greenspan *et al.*, 1966). It would therefore seem to be suitable for use in patients with a rapid arrhythmia in the face of an unclear history of digitalis administration. For this reason we wish to record a dangerous complication of the use of lignocaine—namely, the conversion of atrial flutter with 2 : 1 atrioventricular block to 1 : 1 conduction.

CASE REPORT

A 40-year-old housewife with chronic rheumatic heart disease was admitted to hospital on 5 October 1967 for treatment of a persistent tachycardia. In 1954 she had a closed mitral valvotomy for tight mitral stenosis. She was improved considerably by the operation and remained in sinus rhythm.

In April 1967 she became breathless and was started on digitalis. In August her symptoms increased and her treatment was changed to digoxin 0.25 mg. t.d.s. She was seen in the outpatient department on 28 September, when she was observed to have a regular tachycardia of 140 beats a minute. Her jugular venous pressure was markedly raised and the signs of mitral stenosis were present. An electrocardiogram showed atrial flutter with an atrial rate of 300 a minute and 2 : 1 atrioventricular block. The rhythm was unaffected by carotid sinus pressure. Digoxin was stopped and hydrochlorothiazide with potassium supplements begun. One week later the cardiac findings were unchanged though the jugular venous pressure had fallen to normal.

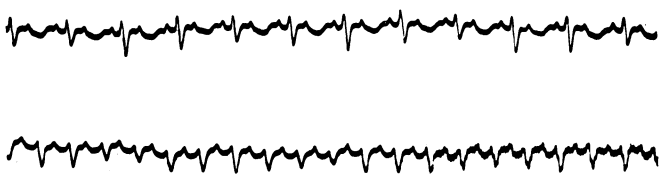

FIG. 1.—Atrial flutter with 2 : 1 block in upper tracing converted to 1 : 1 conduction (lower tracing) after intravenous lignocaine.

The patient was admitted to hospital. Her plasma potassium at this time was 4.6 mEq/l. It was thought that the arrhythmia could still be digitalis-induced, and 100 mg. of lignocaine was given intravenously. This resulted in slowing of the atrial rate to 250 a minute with 1 : 1 conduction (Fig. 1). With this the patient complained of dizziness, sweated profusely, and had an unrecordable blood pressure. The previous rhythm returned spontaneously after two minutes.

Two weeks later sinus rhythm was restored with direct-current shock. Immediately after cardioversion there were frequent atrial ectopic beats. These were readily abolished by intravenous lignocaine (100 mg.) (Fig. 2). Clinical examination showed tight mitral stenosis and mild aortic incompetence. Mitral revalvotomy was successfully carried out on 26 October 1967.

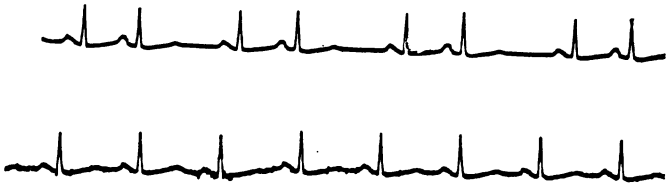

FIG. 2.—Atrial ectopic beats after cardioversion abolished by intravenous lignocaine.

COMMENT

Lignocaine is generally thought to be less effective in the treatment of atrial than ventricular arrhythmias. In our case the possibility of digitalis intoxication, though rarely presenting as atrial flutter, was seriously considered ; hence the decision to give intravenous lignocaine. The patient responded by developing 1 : 1 conduction with a ventricular rate of 250.

The mechanism of action of lignocaine in cardiac arrhythmias is not clear. Clinically it appears to have an action similar to that of phenytoin, quinidine, and antazoline. All these drugs decrease myocardial excitability and prolong conduction time. Lignocaine does not, however, seem to depress contractility of cardiac muscle in the doses recommended (Friedberg, 1966 ; Spracklen *et al.*, 1968). The production of 1 : 1 conduction in atrial flutter has been reported with quinidine, antazoline, and phenytoin (Dreifus *et al.*, 1964 ; Grissom *et al.*, 1967). In the above case lignocaine appears to have slowed the atrial rate without increasing the atrioventricular block. This resulted in 1 : 1 conduction.

We believe, therefore, that intravenous lignocaine should not be used in atrial flutter with block unless there is overwhelming evidence of digitalis intoxication.

We wish to thank Dr. T. A. Kemp for permission to publish the details of this case.

A. R. ADAMSON, M.B., M.R.C.P.
F. H. N. SPRACKLEN, M.D., M.R.C.P.
St. Mary's Hospital, London W.2.

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Ocular Damage Due to Paraquat and Diquat

Brit. med. J., 1968, 2, 224

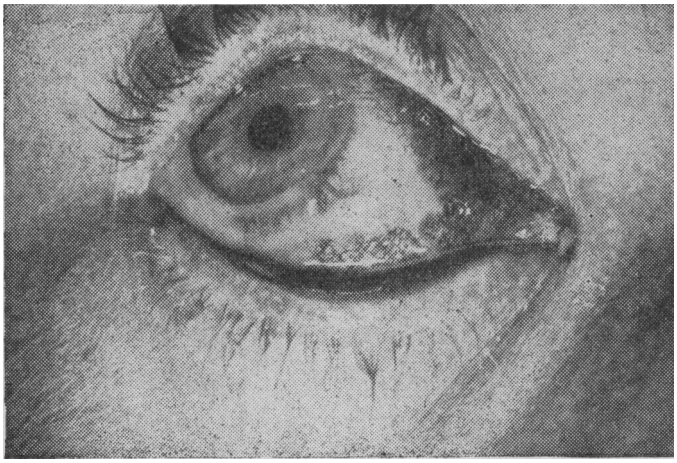
Paraquat and diquat are dipyridilium compounds which act as herbicides and desiccants, and are widely used in agriculture for the control of weeds in plantation crops and occasionally for defoliation before harvesting.

Dipyridilium compounds have been shown in experimental animals (Clark, McElligott, and Hurst, 1966) and in three fatal cases (Bullivant, 1966 ; *Brit. med. J.*, 1967) to produce specific changes of an irreversible nature in the lungs. While the systemic effects are documented and skin irritation has been noted (*Brit. med. J.*, 1967) it is less commonly recognized that they may cause serious damage if they come into contact with the eye. No case of ocular damage due to diquat or paraquat has been described, and as these substances are being used with increasing frequency attention should be drawn to their serious local ocular effects by reporting the following case.

CASE REPORT

The patient, a 36-year-old farmer, reported that while mixing Preeglone Extra fluid concentrate (a paraquat/diquat mixture) with water he splashed some solution on to his right eye and eyelids. He noticed slight irritation and washed the eye with water. Over the next three days mild irritation occurred, and he consulted his family doctor. One week later the eye became considerably more irritable and his admission to hospital was arranged.

On admission his eye had extensive loss of bulbar conjunctiva around all aspects of the globe with associated loss of more than 50% of tarsal conjunctiva of the lower eyelid and a smaller loss of that of the upper eyelid. The denuded areas were clean (see Fig.). The corneal epithelium beside the limbus was destroyed in all quadrants and the remainder of the corneal epithelium was oedematous. There was a low-grade reactive anterior uveitis. The skin of the eyelids and surrounding area was not affected.



Right eye, showing extensive loss of conjunctiva.

He was treated initially with chloramphenicol ointment and 1% atropine drops and the eye was covered. After four days, during which no change occurred and the possibility of a conjunctival graft was actively considered, healing of the denuded areas began and progressed steadily until his discharge from hospital 11 days later. The corneal epithelium healed and the uveitis subsided. During his period of treatment there was a tendency to adhesion between the opposing denuded surfaces, and these had to be repeatedly separated to prevent permanent adhesion with obliteration of the conjunctival fornices.

COMMENT

The proprietary preparation Preeglone Extra contains equal proportions of paraquat and diquat plus a surface-active agent. As these compounds are of similar chemical formulation they probably have a similar and equal effect on the eye. This effect is particularly dangerous, since the action is insidious, the full extent of the damage due to a single splash of the solution not being apparent for over one week.

It has been noted in experimental work on rabbits that after the instillation of an aqueous solution of paraquat dichloride into the conjunctival sac no initial irritation was evident, but mild inflammation of the conjunctiva and nictitating membrane followed within 12 hours and persisted for 48 to 96 hours though no corneal damage resulted (Clark *et al.*, 1966). This is in contrast to most chemicals causing ocular burns, which have an immediate action, and some continue to produce damage over a period of time after a single application.

It is remarkable that one relatively minor splash of the preparation, possibly in aqueous solution, which initially produced only transient ocular discomfort and no surrounding skin damage, caused a serious ocular burn. In this respect the ocular burn was similar to that produced by an alkali, which becomes bound to the tissues of the eye, but, unlike alkali burns, the effect was very slow and there was no immediate damage.

In view of the damage produced by an apparently trivial splash on the eye, paraquat and diquat should be handled with caution. The delayed effect of an ocular burn should be emphasized, and as irrigation of the eye is apparently rendered ineffective by the presence of a surface-active agent it must be understood that treatment is difficult.

J. STANLEY CANT, M.B., F.R.C.S.ED., F.R.C.S.GLASG., D.O., Consultant Ophthalmic Surgeon.
D. R. H. LEWIS, M.B., B.SC., M.R.C.P.GLASG., Registrar in Ophthalmology.
Tennent Institute of Ophthalmology, University of Glasgow.

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