STOKES-ADAMS ATTACKS FOLLOWING SODIUM ANTIMONYLGLUCONATE (TRIOSTAM)

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All therapeutic trivalent antimony preparations can cause myocardial damage. Stokes-Adams attacks, and apparent myocardial infarction have followed sodium antimony tartrate administration, and electrocardiographic changes are the rule.[1]

Antimony dimercaptosuccinate (Astiban) also causes cardiographic changes which are frequent although it may be less toxic than the tartrate.[2] Myocardial toxicity from sodium antimonylgluconate (Triostam) seems to be largely ignored. The rarity of complications reported has given rise to complacency. Literature accompanying the ampoules makes no reference to possible cardiac side-effects, and the manufacturers do not consider bed rest is necessary during administration.[3] Most patients are treated out of hospital, frequently without advice to rest at all.

CASE REPORT

The patient was a healthy 26-year-old housewife. Before the injection she had no symptoms, and there was no evidence of

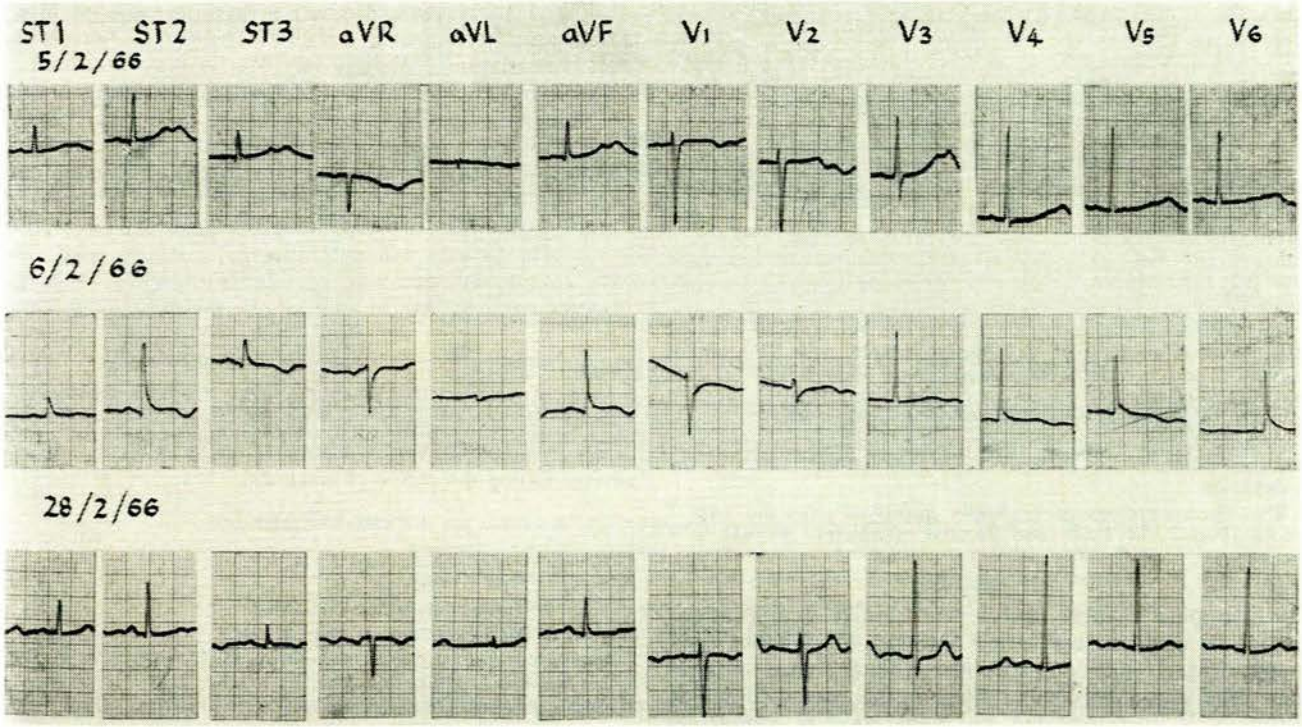

Fig. 1. See text.

any cardiac disease. She had received 190 mg. of Triostam IV daily, on 4 successive days for urinary bilharziasis. During this time she was ambulant. Her main exertion was carrying her year-old baby occasionally.

She complained of a mild frontal headache about half an hour after each injection, lasting 1 - 2 hours. She noticed that the tips of all her fingers were red, and itched on the second day. The skin in this area subsequently desquamated. A papular abdominal rash developed on the third day, and persisted for 10 days. It was while she was at her doctor's consulting room awaiting her fifth injection that she suddenly lost consciousness. This was 24 hours after her fourth injection (total of 760 mg. of Triostam). A pulse could not be detected, her blood pressure could not be recorded, and she stopped breathing temporarily and became cyanosed. She recovered after external cardiac massage, and was admitted to hospital.

During the next 24 hours she had 6 further attacks of loss of consciousness. These were preceded by a ringing in her ears, and a blurring of objects. While she was unconscious her pulse was impalpable and no heart sounds could be heard on auscultation. She breathed rapidly at a rate of 40 respirations a minute. Following external cardiac massage she recovered consciousness after about 90 seconds in each case, and at the same time her face became very flushed for a few seconds. She felt extremely weak and exhausted for about half an hour after each attack. An electrocardiogram (ECG) (Fig. 1, 5/2/66) showed flattening of the T waves, especially over the left precordial leads, with prominent U waves. An ECG (Fig. 2)

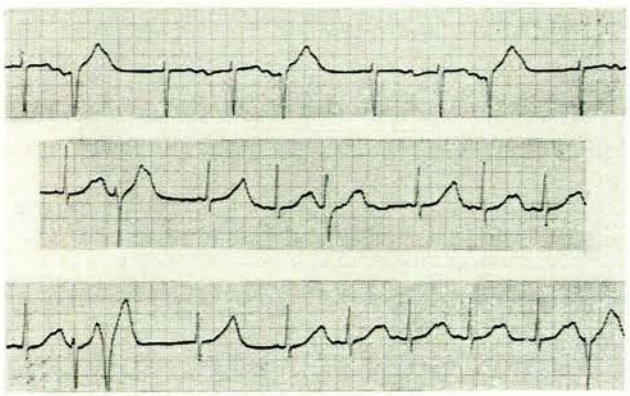

Fig. 2. See text.

taken shortly after an attack showed multiple ventricular extrasystoles.

Her serum potassium was 3·8 mEq./l., and her serum calcium 10·6 mg./100 ml. Her serum glutamic transaminase was 12 mu. and her ESR and white cell count remained normal.

The following day the ECG (Fig. 1, 6/2/66) showed changes suggestive of an inferior myocardial infarct, with ST-segment elevation and inverted T waves in leads 2, 3, and aVF. Her last attack of unconsciousness occurred 20 hours after the first attack, and 44 hours after the last injection.

Apart from sedation, she was given a dimercaprol injection, 100 mg. 4-hourly for 24 hours, and then 50 mg. 4-hourly for 2 days.

The electrocardiogram gradually improved over the next 3 weeks (Fig. 1, 28/2/66) and became completely normal in 6 weeks.

DISCUSSION

Cardiographic changes and severe cardiac complications can occur after widely varied amounts of different antimony drugs. Some preparations produce more changes than others, probably depending on the ease with which antimony is liberated,[4] but no preparation is completely safe.

In this patient there was no warning of any myocardial toxicity until 24 hours after her fourth injection. The skin rash which she developed suggested possible sensitivity to the drug, but the delay of 24 hours between the last administration and unconsciousness makes idiosyncrasy to the drug unlikely.

Various mechanisms whereby antimony exerts its toxic effects have been suggested. These include a direct toxic action on the heart muscle, suppression of intracellular enzyme activity, disturbance in nervous mechanisms via the vagus or sympathetic nerves, and disturbance in myocardial electrolyte balance. In this patient the first ECG (Fig. 1, 5/2/66) showed changes compatible with hypokalaemia, with flattening of the T waves, and a prominent U wave. The serum potassium was 3·8 mEq./l. An ECG taken 24 hours later (Fig. 1, 6/2/66) showed changes suggestive of an inferior myocardial infarction. The presence of some myocardial injury, possibly with patchy necrosis or infarction, could be due to a direct effect of the antimony on the myocardium, or indirectly, as a result of coronary spasm. A similar case was recently described by Honey[1] in a 23-year-old West African girl who developed a serious ventricular arrhythmia with ECG changes suggestive of anterior infarction following treatment with sodium antimony tartrate.

Exercise is a known precipitating factor in causing myocardial complications.[5] It has been suggested that patients should be confined to bed for 2 hours after an injection.[2] This patient exerted herself quite considerably during treatment, but it is unlikely that bed rest for 2 hours after an injection would have prevented her attack of unconsciousness which only occurred the following day.

Bilharziasis is very common in certain parts of South Africa and appears to be on the increase elsewhere in the world.[6] Antimonial therapy has been the corner-stone of therapy. Initial trials with widely publicized Ambilhar suggest that it is less effective than certain antimony preparations.[7] However, recent preliminary reports with Ambilhar are more encouraging and indicate an extremely high cure rate.[8,9] Nilodin is the only other non-antimony preparation which has established itself. It appears, however, that antimony preparations will continue to be widely used. The only completely safe way of administration is with the patient at rest in bed, in spite of the practical difficulties.

SUMMARY

Severe myocardial toxic effects resulted from the administration of 760 mg. of Triostam to a previously healthy 26-year-old woman who was ambulant during therapy. In spite of the rarity of myocardial complications with this drug, bed rest is advised during the period of treatment.

REFERENCES

1. Honey, M. (1960): Brit. Heart J., **22**, 601.
2. Davis, A. (1961): *Ibid.*, **23**, 291.
3. Berry, N. (1966): Personal communication.
4. Tarr, L. (1947): Ann. Intern. Med., **27**, 970.
5. Khalil, M. (1928): J. Egypt. Med. Assoc., **11**, 97.
6. Leading Article (1966): Brit. Med. J., **1**, 249.
7. Jordan, P. (1966): *Ibid.*, **1**, 276.
8. Powell, S. J. and MacLeod, I. N. (1966): S. Afr. Med. J., **40**, 881.
9. Clarke, V. de V. and Blair, D. M. (1966): Cent. Afr. J. Med., **12**, 64.