PROCAINE AMIDE THERAPY IN AURICULAR FLUTTER WITH 1 : 1 A-V CONDUCTION

BY

ROWLAND CALVERT AND ERIC SMITH

From the Medical Unit, Whipps Cross Hospital, Essex

Procaine amide hydrochloride was first introduced clinically by Mark *et al.* (1950). They drew attention to its effectiveness in the control of ventricular arrhythmias, especially paroxysmal ventricular tachycardia. This has since been amply confirmed. It is the drug of choice in the treatment of paroxysmal ventricular tachycardia, taking precedence over the more toxic and unpredictable quinidine. By contrast, reliance on procaine amide for the control of the supraventricular tachycardias is unwise, although it is frequently effective in the paroxysmal auricular and nodal tachycardias (Miller *et al.*, 1951; Berry *et al.*, 1951).

In auricular fibrillation and flutter the results obtained with procaine amide have been unimpressive: indeed, Berry *et al.* (1951) consider that it is not indicated in the treatment of these conditions. The grave arrhythmia, auricular flutter with 1 : 1 conduction, is a rare spontaneous occurrence, although it may appear when the commoner forms of auricular flutter with block are treated with quinidine or procaine amide (Berry *et al.*, 1951; Zapata-Diaz *et al.*, 1951). The influence of procaine amide on this arrhythmia as a presenting clinical problem has apparently not been described previously.

Case Report

History. An engineer, aged 50, had felt quite well until 7.30 a.m. on 16/1/52, when he suddenly felt faint and was sent home from work. While in the car he felt pain for the first time, and he vomited yellow fluid on arrival home. The pain was epigastric, severe and constricting, radiating to the chest but not down the arms. The pain became agonizing, especially in the sub-sternal region, and he "felt that his chest would burst." Continuous pain with intermittent exacerbations ensued until admission to hospital at 1.30 p.m. Morphine, 1/3 of a grain, was given with little relief.

Physical Examination. The patient was a well-built man, fully conscious, groaning with pain and greatly shocked with pallor, cold sweat, absent radial pulses, unrecordable blood pressure and temperature of 97.2° F. The heart sounds were faintly audible; the rate was 130 a minute, with regular rhythm. No other abnormalities were found apart from tenderness and guarding in the epigastrium. During examination he vomited 6 oz. of "coffee-ground" material with small blood clots.

Progress. At 3.30 p.m. the cardiac rate exactly doubled to 260 a minute and the electrocardiogram showed the pattern of auricular flutter with 1 : 1 A-V conduction (Fig. 1A). There was no response to carotid sinus pressure. Morphine, gr. 1/4, was given intravenously with only transient relief of pain.

At 6.45 p.m., with the signs unchanged, the intravenous injection of procaine amide was begun. 6 ml. (0.6 g.) were injected in three minutes, and at this point, with the needle still *in situ*, the heart rate dropped abruptly to 100 a minute; the electrocardiogram showed sinus rhythm and no evidence of coronary thrombosis (Fig. 1B). Simultaneously a facial flush replaced the ashen grey hue and the patient expressed immediate relief of distress. The radial pulses became palpable and the systolic blood pressure was 65. The procaine amide injection was continued up to a total of 10 ml. (1.0 g.) over ten minutes.

At 8 p.m. a further electrocardiogram was taken, but showed no change. The pain and abdominal tenderness had entirely gone. The heart rate was now 84 a minute, the rhythm was regular and the blood pressure 75/50. Proto-diastolic triple rhythm appeared, and was audible for the next ten days. The next morning (17/1/52) improvement was maintained, the blood pressure being 100/60, with a pulse rate of 84.

FIG. 1.—Serial electrocardiograms showing (A) 1:1 auricular flutter. (B) Replaced by sinus rhythm, after injection of 0.6 g. of procaine amide. A posterolateral cardiac impact later appeared (C), and subsequent records (D to G) showed gradual regression.

A small hæmatemesis at 6 a.m. caused no deterioration in his cardiac condition. A loud pericardial rub appeared, and a diagnosis of coronary thrombosis was made in spite of the normal electrocardiogram. The diagnosis was supported by a temperature of 99, E.S.R. of 25 mm. in one hour, and a white blood count of 13,300, with 90 per cent polymorphs, and was later confirmed by electrocardiogram, which showed a postero-lateral myocardial infarct (Fig. 1C). From this time clinical improvement was steadily maintained. The rub disappeared after two days; the blood pressure and pulse rate remained substantially unchanged, although occasional ventricular premature contractions were noted and persisted up to the time of discharge from hospital on 13/3/52. Serial electrocardiograms (Fig. 1D, E, F, G) showed the characteristic patterns of coronary thrombosis. By 26/2/52 the T wave was upright in lead CR7, but was still flat in lead III and remained so for a month after discharge. A functional bundle branch block is present in some of the serial tracings and is especially prominent in lead CR1.

The patient was treated with sedation, rest, and finally graduated exercises. Anti-coagulants were not given because of the hæmatemesis. On discharge he was fully ambulant without complaints and has since remained well.

Discussion

The exact diagnosis of the arrhythmia, shown in electrocardiogram A, is of importance, as the special indications for procaine amide in the rarer arrhythmias are at present under scrutiny. However, the extreme regularity of complexes leaves no doubt that this is a supraventricular tachycardia. Master *et al.* (1952) have pointed out that auricular flutter when complicated by a bundle branch block pattern may simulate ventricular tachycardia electrocardiographically, but the first cardiogram in this case shows that this same difficulty in differentiation may arise in the presence

of auricular flutter with 1 : 1 A-V conduction. Although the recent detailed work of Prinzmetal *et al.* (1951) has shown, in the experimental animal, by means of high-speed cinematography and direct lead electrocardiography, the unitary nature of auricular tachycardia and auricular flutter, we have preferred to label the present arrhythmia as auricular flutter with 1 : 1 A-V conduction.

The dramatic response, probably life-saving, we witnessed in the present case is worthy of further comment. East and Bain (1948) state "the human ventricle is not capable of maintaining rates of over 300, and speeds of over 200 are not well tolerated," while Gold (1950) states "a rate (ventricular) of 230 a minute fatigues and damages the circulation." In the present case with a ventricular rate of 260 a minute, maintained for three hours, and with absent peripheral pulses, the prognosis could rightly be regarded as grave. The abrupt response occurring during intravenous procaine amide therapy could scarcely have been fortuitous, or indeed the result of previous morphine administration, although the latter may at times control cardiac arrhythmias (Sabathie, 1947). Of additional interest was the relief of pain, following the procaine amide injection. This information has been volunteered by other patients treated with intravenous procaine amide and would be predicted from the known local anæsthetic properties of procaine derivatives.

Finally, it must be recognized that procaine amide is a potent drug, and its slow intravenous administration must be accompanied by electrocardiographic control, with immediate availability of appropriate antidotes to cover side-effects.

SUMMARY

A case of coronary thrombosis complicated by auricular flutter with 1 : 1 A-V conduction is described. The patient was in extremis at the time of starting intravenous procaine amide therapy. He regained sinus rhythm after 6 ml. (0.6 g.) had been injected over three minutes, and his further recovery was uneventful.

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REFERENCES

- Berry, K., Garlett, E. L., Bellet, S., and Geftter, W. I. (1951). *Amer. J. Med.*, **11**, 431.
 East, T., and Bain, C. (1948). *Recent Advances in Cardiology*. 4th ed., J. and A. Churchill, London.
 Gold, H. (1950). *Quinidine in Disorders of the Heart*. P. B. Hoeber, N.Y.
 Mark, L. C., Berlin, I., Kayden, H. J., Rovenstine, E. A., Steele, J. M., and Brodie, B. B. (1950). Abstract in *J. Pharmacol. and Exper. Therap.*, **98**, 21.
 Master, A. M., Moser, M., and Jaffe, H. L. (1952). *Cardiac Emergencies and Heart Failure*. H. Kimpton, London.
 Miller, H., Nathanson, M. H., and Griffith, G. C. (1951). *J. Amer. med. Assoc.*, **146**, 1004.
 Prinzmetal, M., Corday, E., Oblath, R. W., Kruger, H. E., Brill, I. C., Fields, J., Kennamer, S. R., Osborne, J. A., Smith, L. A., Sellers, A. L., Flieg, W., and Finston, E. (1951). *Amer. J. Med.*, **11**, 410.
 Sabathie, L. G. (1947). *Amer. Heart J.*, **33**, 719.
 Zapata-Diaz, J., Cabrera, E., and Mendez, R. (1952). *Amer. Heart J.*, **43**, 854.

POSTSCRIPT

Almost two years later (28/12/53) our patient was re-admitted with the same cardiac arrhythmia and circulatory collapse. When 7 ml. (0.7 g.) of procaine amide were injected over a period of seven minutes intravenously, the cardiac rate fell suddenly to 100 a minute. There was now electrocardiographic evidence of coronary thrombosis, from which he recovered uneventfully in six weeks.