Giant peaked upright T waves in cerebrovascular accident

Peter J. Runge and George Bousvaros

From the Department of Medicine, Albany Medical College, and the Medical Service, Veterans Administration Hospital, Albany, N.Y., U.S.A.

Tall upright T waves, with a maximal T/QRS ratio (in lead V₄) of 1.32, appeared in a 52-year-old patient after a cerebral haemorrhage which was confirmed at necropsy. Hyperkalaemia during life and endocardial or myocardial damage were not found.

Electrocardiographic abnormalities often appear in cerebrovascular accidents and other central nervous system lesions (Byer, Ashman, and Toth, 1947; Burch, Meyers, and Abildskov, 1954; Shuster, 1960; Koskelo, Punsar, and Sipilä, 1964; Kreus, Kemilä, and Takala, 1969). The changes most commonly seen are large inverted T waves, which simulate myocardial ischaemia, and prolonged QT intervals. Rarely, development of large or tall peaked upright T waves has been described (Byer *et al.*, 1947; Burch *et al.*, 1954; Kreus *et al.*, 1969), but the amplitude of the T waves illustrated did not exceed 1.2 mV or 60 per cent of the voltage of the QRS, which is considered the upper range of normal (Braun, Surawicz, and Bellet, 1955). Furthermore, in none of the cases with large upright T waves were there necropsy data to rule out acute ischaemic heart injury. In the case to be reported, abnormally tall upright T waves developed after intracranial haemorrhage proven by necropsy, while other causes, such as hyperkalaemia or myocardial injury, could be excluded.

Case report

A 51-year-old man was admitted with a two-day history of dyspnoea, hoarseness, and malaise for which he took 'one or two' tablets of a proprietary acetylsalicylate preparation. One year before admission he underwent cholecystectomy for acute cholecystitis with perforated gall-bladder, and during the operation nodularity of the liver was observed. Liver biopsy showed micronodular cirrhosis.

On examination the patient appeared mildly confused and tachypnoeic. Temperature was

37° C., respirations 30 a minute, pulse rate 86 a minute, and blood pressure 110/80 mm. Hg. The lips were cyanotic and the pharynx was hyperaemic and covered with yellow exudate. Results of examination of the heart and lungs were normal. The liver edge was palpable 3 cm. below the right costal margin. Neurological examination was within normal limits. The admission diagnosis was pharyngitis with bronchitis. Prothrombin time was 22/13. Serum electrolytes were sodium 127, potassium 4.0, chloride 95, and bicarbonate 14 mEq/l. Arterial blood pH was 7.5, PCO₂ 15.5 mm.Hg, PO₂ 70 mm. Hg, and bicarbonate 12 mEq/l. Electrocardiogram (Fig., upper row) and chest x-ray were unremarkable. The patient was given nasal oxygen and seemed to improve. Eighteen hours after admission he became comatose with Cheyne-Stokes respiration. The gaze was fixed in the midline and the extremities were flaccid and areflexic. There was no response to painful stimuli. Cerebrospinal fluid was clear under normal pressure, and contained 40 red blood cells per cubic mm. Arterial blood pH was 7.28, PCO₂ 21.5, PO₂ 72 (with nasal O₂), and bicarbonate 10. These values were thought to show metabolic acidosis complicating the previous mild respiratory alkalosis. Prothrombin time was 34/14 and serum electrolytes were sodium 129, potassium 5.0, chloride 94, and bicarbonate 13 mEq/l. Central venous pressure was 2 cm. H₂O and arterial pressure (cuff) 100/70 mm. Hg. Chest x-ray was still normal. Sodium bicarbonate, 223 mEq, was administered slowly intravenously, and two hours later arterial pH was 7.49, PCO₂ 20.5, PO₂ 59 (still with nasal oxygen), and bicarbonate 15. Serum electrolytes were sodium 124, potassium 3.5, chloride 94, and bicarbonate 15 mEq/l. Salicylate levels in a blood sample obtained at this time were 44 mg./100 ml. The patient expired shortly thereafter.

The electrocardiogram of the patient was

virtually normal on admission (Fig., upper row) and not significantly different from that of his previous admission. The tracing obtained soon after the onset of the neurological disorder (Fig., middle row) showed giant peaked T waves. The largest amplitude was 2.6 mV in lead V₄ with a ratio of T/QRS of 1.32. Serum potassium at that time was 5.0 mEq/l. The T waves were almost as tall two hours later when the serum potassium had dropped to 3.5 mEq/l. (Fig., lower row). In both tracings the U waves were superimposed on the downslope of the T and the QU interval was prolonged.

At necropsy there were multiple diffuse small bilateral cerebral and subarachnoid haemorrhages, as seen in patients with bleeding diathesis. There was haemorrhagic enteritis but the mucosa of the stomach was intact. Significant coronary atherosclerosis was present involving all three major vessels without recent or old occlusion. The myocardium was free of hypertrophy, fibrosis, old or recent myocardial infarction, and there were no subendocardial haemorrhages or cellular infiltration. The liver showed moderate cirrhotic changes.

The necropsy findings adequately explain the fatal neurological episode but the nature of the preceding events is, even retrospectively, not clear. The initial respiratory alkalosis and the subsequent development of metabolic acidosis would be consistent with salicylate intoxication. However, the family denied excessive intake, and the level of 44 mg./100 ml. 24 hours after admission, unless a laboratory error, suggests very high initial levels inconsistent with the originally unimpaired consciousness and with the absence of gastric lesions at necropsy. If the salicylate aetiology of the initial events is accepted the intracranial haemorrhage may be explained as due to haemorrhagic diathesis secondary to acetylsalicylate intoxication. Acetylsalicylates are known to affect coagulability by a possible effect on the vessel wall and by interference with the second wave of platelet aggregation (O'Brien, 1968), the prolongation of the prothrombin time serving as a contributory factor.

Discussion

Tall upright symmetrical peaked T waves may be seen in several abnormal situations besides hyperkalaemia, and the diagnosis of the underlying condition from the morphology of the abnormal T waves is difficult or impossible (Braun *et al.*, 1955). Salicylate overdose has no known effect on the electrocardiogram except for abnormalities produced by associated changes in serum potassium (Woodbury, 1965). In adults hypokalaemia usually occurs (Robin, Davis, and Rees, 1959) which produces flattening of T waves, the opposite of what was seen in this patient. Shifts in pH as modest as those observed do not affect the electrocardiogram (Reid *et al.*, 1965). Myocardial infarction, subendocardial haemorrhages, or coronary thrombosis were

FIG. Upper row: leads II, V₃, V₄, V₅ on admission. Middle row: 19 hours later, shortly after the onset of the neurological episode. Giant upright T waves have developed. Lower row: 2 hours later. Giant peaked T waves persist.

not seen at necropsy. The T waves were normal on admission and were found increased after the onset of the cerebrovascular catastrophe, strongly suggesting a causal relation.

Though, typically, T waves become inverted after cerebrovascular accidents, several authors have mentioned the appearance of large upright T waves (Byer *et al.*, 1947; Burch *et al.*, 1954; Kreuz *et al.*, 1969). Examination of the electrocardiograms in these publications indicates that the ratio of the T/QRS was not above the range of 0.05 to 0.85 observed in 100 normal subjects by Braun *et al.* (1955). The remarkable amplitude seen in the present case with a T/QRS ratio of 1.32 justifies the use of the term 'giant' upright T waves.

Production of electrocardiographic abnormalities by central nervous system lesions has been ascribed to either subendocardial haemorrhages (Koskelo *et al.*, 1964; Kreuz *et al.*, 1969), or to an effect mediated through the nervous system, central or peripheral (Hugenholtz, 1967). In animal experiments, Yanowitz, Preston, and Abildskov (1966) have produced inverted T waves by stimulation of the right stellate ganglion or left stellate ganglionectomy, and large upright T waves by left stellate stimulation or right stellate ganglionectomy. Their work provides evidence for one peripheral nervous pathway through which upright and inverted T waves may be produced, and this may be pertinent to development of both types of abnormal T waves following central nervous system lesions in man.

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