Electrocardiographic Abnormalities Simulating Myocardial Infarction in Intracerebral Haemorrhage and Cerebral Thrombosis

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During the past 15 years there have been a number of accounts of abnormal electrocardiograms recorded from patients who have cerebrovascular disturbance. Earlier reports dealt principally with subarachnoid haemorrhage. Levine (1953) referred to cascading T waves which became replaced by RS-T segment elevation in a patient who had a ruptured aneurysm of the circle of Willis. Burch, Meyers, and Abildskov (1954) found 10 abnormal electrocardiograms among the cerebrovascular accident admissions to a Louisiana hospital during the year 1950. Commoner abnormalities were a prolonged Q-T interval, T waves of increased amplitude and duration which were often negative, and sometimes large U waves. Later writers on the subject, such as Wasserman *et al.* (1956), described prolongation of the S-T interval and deep wide T waves which probably contained inverted U waves, but prolongation of the Q-T interval and wide upright T waves were also seen. Still more recent reviews of the subject have been those of Cropp and Manning (1960) and Hugenholtz (1962). The American authors mainly stress the absence of any clinical evidence of coronary infarction and the normality of the heart and coronary arteries at necropsy. Finnish authors (Koskelo, Punsar, and Sipilä, 1964), however, suggest that subendocardial haemorrhages may occur more frequently than is generally believed. The series of Burch, Wasserman, and others contained patients with subarachnoid haemorrhage together with some cases of intracerebral haemorrhage. The purpose of this report is to describe an instance of intracerebral haemorrhage complicating thrombosis with abnormal electrocardiogram but normal heart and coronary arteries at necropsy.

Case Report

A woman of 31 was admitted to hospital in September, 1966, with a history that she had fallen in her home three

days previously and on being picked up she had paralysis of the left arm and leg and a severe right-sided headache. Her doctor was unable to persuade her to go into hospital until she became stuporose on the third day. On admission she was conscious but in stupor, there were no signs of external injury, but she had conjugate deviation of the head and eyes to the right. Pupils were equal and there was no papilloedema. There was a flaccid hemiplegia involving face, arm, and leg on the left. The pulse was regular, 84/minute, blood pressure 120/70 mm. Hg. The heart was of normal size and there were no murmurs. Lumbar puncture was performed and this revealed a clear fluid under normal pressure with 35 mg. protein per 100 ml. and 1 lymphocyte per cu. mm. There was no xanthochromia. SGOT on the day of admission was 12 Fränkel units, white blood cells 11,000 per cu. mm., and erythrocyte sedimentation rate 12 mm. in 1 hour (Westergren). Chest and skull x-rays were normal. The electrocardiogram on the day of admission (three days after the stroke) resembled that of anterior myocardial infarction (see Fig. A) with T wave inversion in leads I, aVL, and V1 to V6, most marked in septal leads V2 to V4; Q-T_a was prolonged to 0.47 sec. Two days later a further electrocardiogram (Fig. B) showed that the previously inverted T waves were more deeply and symmetrically inverted in the same leads and even more suggestive of acute anterior myocardial infarction. Her general condition deteriorated and she died in coma with terminal bronchopneumonia on the fifth day after the onset of the paralysis.

Necropsy. There was no injury of the scalp or of the skull. The cerebral convolutions were flattened and the right cerebral hemisphere was swollen. Section of the brain revealed a massive right internal capsular haemorrhage with gross destruction of adjacent brain tissue. Examination of the cerebral vessels disclosed antemortem thrombus occluding the right middle cerebral artery; the point of rupture was not demonstrable. There were two small areas of intrapontine haemorrhage measuring less than 5 mm. across. The heart was of normal size, weighing 270 g. The pericardium, myocardium, and endocardium appeared healthy, and the coronary arteries showed minimal atheromatous changes

FIG.—Electrocardiograms (A) on admission, and (B) two days later. The serial changes simulate anterior myocardial infarction.

without narrowing or occlusion. Both lungs were bulky due to congestion and there was patchy bronchopneumonia at the bases. Histology of the heart revealed no evidence of subendocardial haemorrhage or myocardial ischaemia.

Discussion

It is now well established that a large number of intracerebral conditions including subarachnoid haemorrhage, intracerebral haemorrhage, cerebral thrombosis, subdural haematoma, and cerebral tumour can be associated with abnormal electrocardiograms (Levine, 1953; Burch *et al.*, 1954; Wasserman *et al.*, 1956; Hersch, 1964). The most frequently described abnormalities have been prolonged Q-T interval and large inverted T waves

sometimes associated with U waves. There is, however, one instance in which the abnormal record mimicked acute myocardial infarction even to the extent of showing Q waves, decreased amplitude of R waves, and elevation of S-T segments. This was the patient of Pfister and de Pando (1962), who died of subarachnoid haemorrhage from a berry aneurysm of the right anterior cerebral artery. At the necropsy there was no evidence of myocardial infarction, and the coronary arteries showed minimal intimal atheroma without narrowing or thrombosis. The present case confirms that such records can be obtained in cerebrovascular accident and that serial electrocardiograms can simulate the serial changes of acute myocardial infarction.

It is evident in this case that there is no associated

myocardial infarction or coronary artery disease. Moreover, subendocardial haemorrhages, considered by some authors (Koskelo *et al.*, 1964) to play an important part in the production of electrocardiographic abnormality in cerebrovascular disease, were not present. Our patient did not show hypotassaemia or hypothermia (cf. Wasserman *et al.*, 1956) and was normotensive at the time of the electrocardiographic records. Having excluded myocardial infarction, subendocardial haemorrhages, myocardial ischaemia due to shock or hypotension, hypokalaemia, and hypothermia, it is necessary to consider disturbances of the autonomic nervous system in producing the abnormal electrocardiogram.

Both sympathetic and parasympathetic autonomic disturbances have been blamed. Burch, DePasquale, and Malaret (1960) suggested that the cerebral lesions caused sympathetic storms which gave rise to intense sympathetic hypertonia. Injection of adrenaline in man can cause a prolonged Q-T interval (Hecht and Anderson, 1947). Cropp and Manning (1960), however, hold the view that the cardiographic changes may be due to vagal stimulation, since the vagus has a cortical representation in area 13 of the orbital surface of the frontal lobe which is often involved by the subarachnoid haemorrhage. Náva, Marchetti, and Tartara (1957) were able to produce prolonged Q-T interval, deeply inverted T wave, and bradycardia after injection of paraffin oil into carotid arteries in animal experiments. These changes were prevented by prior vagotomy. Prolonged stimulation of the vagus nerve leads to negative T waves (Manning, Hall, and Banting, 1937). The abnormal electrocardiograms show bradycardia more frequently than tachycardia (Cropp and Manning, 1960; Shuster, 1960). Shuster (1960) mentions that he was able to abolish depressed S-T segments in the electrocardiogram of a patient with subarachnoid haemorrhage by the use of atropine. Accordingly, there is a greater volume of evidence to support the theory of vagal stimulation. In our case we are dealing with right middle cerebral artery thrombosis and a massive right internal capsular haemorrhage and gross destruction of adjacent brain tissue. There were also small intrapontine haemorrhages. Although there was no haemorrhage in the orbital region of the right frontal lobe cortex, this was involved in gross cerebral oedema, and the extensive capsular lesion could well have involved the connexions between area 13 and the hypothalamus.

Summary

A case is described of a young woman who died of cerebral haemorrhage complicating right middle cerebral artery thrombosis whose serial electrocardiograms resembled those of anterior myocardial infarction. The heart at necropsy showed only minimal atheroma of the coronary vessels without narrowing or occlusion, and there was no myocardial infarction or subendocardial haemorrhage. Previously reported cases are reviewed and possible mechanisms of the electrocardiographic changes are discussed.

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References

- Burch, G. E., DePasquale, N., and Malaret, G. (1960). Selected problems in electrocardiography. *Ann. intern. Med.*, **52**, 587.
- , Meyers, R., and Abildskov, J. A. (1954). A new electrocardiographic pattern observed in cerebrovascular accidents. *Circulation*, **9**, 719.
- Cropp, G. J., and Manning, G. W. (1960). Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. *Circulation*, **22**, 25.
- Hecht, H. H., and Anderson, R. B. (1947). The influence of dibenamine (N,N-dibenzyl-β-chloroethyl-amine) on certain functions of the sympathetic nervous system in man. *Amer. J. Med.*, **3**, 3.
- Hersch, C. (1964). Electrocardiographic changes in subarachnoid haemorrhage, meningitis, and intracranial space-occupying lesions. *Brit. Heart J.*, **26**, 785.
- Hugenholtz, P. G. (1962). Electrocardiographic abnormalities in cerebral disorders. Report of six cases and review of the literature. *Amer. Heart J.*, **63**, 451.
- Koskelo, P., Punsar, S., and Sipilä, W. (1964). Subendocardial haemorrhage and E.C.G. changes in intracranial bleeding. *Brit. med. J.*, **1**, 1479.
- Levine, H. D. (1953). Non-specificity of the electrocardiogram associated with coronary artery disease. *Amer. J. Med.*, **15**, 344.
- Manning, G. W., Hall, G. E., and Banting, F. G. (1937). Vagus stimulation and the production of myocardial damage. *Canad. med. Ass. J.*, **37**, 314.
- Náva, S., Marchetti, G., and Tartara, A. (1957). Rilevi emodinamici ed elettrocardiografici nella embolia cerebrale sperimentale. *Minerva med.*, **48**, 3289.
- Pfister, C. W., and de Pando, B. (1962). Cerebral hemorrhage simulating acute myocardial infarction. *Dis. Chest*, **42**, 206.
- Shuster, S. (1960). The electrocardiogram in subarachnoid haemorrhage. *Brit. Heart J.*, **22**, 316.
- Wasserman, F., Choquette, G., Cassinelli, R., and Bellet, S. (1956). Electrocardiographic observations in patients with cerebrovascular accidents. *Amer. J. med. Sci.*, **231**, 502.