CASE REPORTS

ELECTROCARDIOGRAPHIC CHANGES IN ACCIDENTAL HYPOTHERMIA

BY

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Accidental hypothermia was considered to be a rare condition in adults (Rees, 1958). Within the past few years evidence has accumulated which shows that far from being a rare event, accidental hypothermia is a common condition in this country, especially during the winter months (Emslie-Smith, 1958; Duguid, Simpson, and Stowers, 1961; Prescott, Peard, and Wallace, 1962; and *Brit. med. J.*, 1963).

A patient with accidental hypothermia is reported below, and is of particular interest as serial electrocardiographic tracings were recorded at different body temperatures.

Case Report

The patient, a man aged 77, lived alone, but a home help called regularly three times a week, and he was visited occasionally on account of chronic bronchitis. His blood pressure was moderately raised (170/100 mm. Hg). He was a rather stubborn old man who prided himself on his independence, but took little care with his personal appearance, and was content to live with the minimum of home comfort.

On the evening of January 12, he was visited by a friend who noted nothing abnormal. A fire was burning in the grate, and before leaving at 7.0 p.m. his friend filled a bucket with coal and placed it by the hearth. At 10.30 a.m. the following day the patient was found unconscious lying fully clothed on the floor. The room was bitterly cold and it was evident that the fire had been extinguished for many hours. The full coal bucket had not been touched.

On examination there was extreme waxy pallor. The patient was deeply comatose, and failed to respond to painful stimuli. The skin felt cold to the touch and even the axillæ gave no impression of warmth. There was little subcutaneous fat, the muscles were quite stiff and it proved almost impossible to flex the limbs. On attempting to flex the neck the whole trunk was raised as though the spinal column had been replaced by a rigid structure. Although comatose, and in spite of extreme muscular rigidity, the patient made occasional slight spontaneous movements of his hands and feet. The respiratory rate was 12 a minute with a prolonged expiratory phase lasting about 4 seconds. Respiration appeared to be entirely diaphragmatic. Apart from the last two features the only visible sign of life was a fairly profuse lachrymation. The pulse was irregular and of poor volume; the rate was 35 a minute. The heart sounds were faint. The blood pressure was unrecordable by auscultation, but the brachial systolic pressure was 60 mm. Hg by palpitation. The pupils were dilated and failed to respond to light. The corneal reflex was absent, and the tendon reflexes could not be elicited. In spite of the extreme shock-like state, the superficial veins were not collapsed, and when the patient was admitted to hospital venepuncture presented no difficulties. The blood withdrawn felt cold but did not appear unduly viscous, and the clotting time was not prolonged.

The patient was admitted to hospital at 1.0 p.m. on January 13. The rectal and axillary temperature was 78° F. (25.5° C.).

Blood: The hæmoglobin was 12.7 g. per 100 ml. (86%), with a total white cell count of 10,000 per c.mm.; 9300 polymorphs per c.mm. (93%), 500 lymphocytes per c.mm. (5%), and 200 monocytes per c.mm. (2%). Eosinophils were absent. The polymorphs showed a shift to the left. The blood sedimentation rate was 3 mm. in one hour. The serum cholesterol was 225 mg. per 100 ml. (normal range 160–250 mg. per 100 ml.) and the total serum proteins were 7 g. per 100 ml., with albumin 4.1 g. per 100 ml. and globulin 2.9 g. per 100 ml. The serum amylase was 5 units per 100 ml. (normal range 3–10 units per 100 ml.).

FIG. 1.—Electrocardiogram 50 minutes after admission (1.50 p.m.). The J wave is prominent. Body temperature was 78° F. (25.5° C.).

Electrocardiograms were obtained on three occasions. The first (Fig. 1) was performed 50 minutes, the second (Fig. 2) 3½ hours, and the third (Fig. 3) 9 hours after admission. The corresponding temperatures recorded at these times were 78° F. (25.5° C.), 80° F. (26.5° C.), and 90° F. (32° C.) respectively.

The typical changes of hypothermia (Emslie-Smith, 1956) are clearly demonstrated in all leads. There is marked bradycardia and the first two tracings demonstrate probable atrial fibrillation. The first recording (Fig. 1) also shows a baseline irregularity due to fine muscle tremors. The QT interval is prolonged and the J waves are unusually prominent, particularly in the early tracings. It is clear that the amplitude of the J wave decreases as the body temperature rises, and at 90° F. (32° C.) it is only one-sixth of its original amplitude.

Treatment and Course. The patient was nursed in a warm room, and an electric blanket was used. One mega unit of benzylpenicillin was given intramuscularly. By 8.0 p.m., some seven hours after admission, there was no improvement and 200 mg. hydrocortisone hemisuccinate was injected intravenously. His colour appeared to improve slightly but this improvement was transient and death occurred at 2.0 a.m., about 13 hours after admission.

Necropsy was performed by Dr. R. Bishton 34 hours after death. There was almost complete absence of subcutaneous fat. The left lung showed bronchopneumonic consolidation in all lobes, and the lower lobe showed alternating emphysematous and bronchiectatic areas. There was moderate hypertrophy of the left ventricle, while the right ventricle was flabby and dilated. The anterior descending branch of the left coronary artery was partially occluded by organized thrombus and there were scattered areas of old ischaemic fibrosis in the myocardium of the left ventricle. The brain showed slight senile atrophy. The pancreas was macroscopically and microscopically normal.

Discussion

Serial electrocardiograms in accidental hypothermia have rarely been reported. Torresani, Nicolai, and Delboy (1960) report a case in which serial electrocardiograms show a progressive diminution in amplitude of the J wave and shortening of the QT interval. In their patient the rectal temperature was 95° F. (34.9° C.) on admission and 94° F. (34.4° C.) some nine hours later.

FIG. 2.—Electrocardiogram 3½ hours after admission (4.15 p.m.). The J wave is smaller. The body temperature was 80° F. (26.5° C.).

The patient was comatose on admission but after nine hours could answer simple questions, and reflexes previously absent had returned to normal.

It seems unlikely that the temperature readings accurately reflected the true body temperature for the following reasons: first, the temperature on admission was only a few degrees below normal, whereas the patient's clinical condition and electrocardiographic pattern suggested profound hypothermia; and second, although there was a marked improvement after nine hours in both the clinical state and the electrocardiogram, the patient's temperature was recorded as being lower at this time than on admission, in spite of active rewarming.

An intracavity temperature may be influenced by a pool of secretions or, for example, in the rectum by the presence of faeces (Drew, 1961).

There seems to be no correlation between the rectal temperature and the appearance of the J wave in cases of accidental hypothermia (Duguid *et al.*, 1961; Prescott *et al.*, 1962). On the other hand Emslie-Smith, Sladden, and Stirling (1959) found that this deflection could always be reproduced in experimental animals when direct epicardial and cavity electrodes were used. The amplitude of this deflection was inversely related to the oesophageal temperature.

FIG. 3.—Electrocardiogram 9 hours after admission (10 p.m.). The J wave is still present, but the amplitude is greatly reduced. The body temperature was 90° F. (32° C.).

The J deflection is usually of small amplitude and found particularly in leads related to the left ventricle. Changes of the magnitude recorded in the present case are rare. Emslie-Smith *et al.* (1959) noted that the J wave was most conspicuous in the thinnest patients.

If the magnitude of the J deflection indicates severe cooling of the heart muscle, for reasons previously given, the skin or rectal temperature may bear little relation to the myocardial temperature. If the patient has little subcutaneous fat there will be minimal insulation of the central organs of the body, and heat will be lost relatively quickly. This would mean that in a thin person the skin or rectal temperature would more accurately represent the true body temperature. In an obese subject, however, the central body temperature may be much higher than that recorded in the rectum or axillæ. This would explain the apparent discrepancy often found between the appearance of the J wave and the recorded temperature.

Hypothermia usually causes a respiratory and metabolic acidosis (Fairley, Waddell, and Bigelow, 1957). Some authorities consider that the hypothermic cardiographic changes are due primarily

to a low alkali reserve (Osborn, 1953; Altschule and Sulzbach, 1947); others consider there is no such relation (Corvino and Hegnauer, 1955; Emslie-Smith *et al.*, 1959). Although the alkali reserve is often low in accidental hypothermia there seems to be no correlation between the electrocardiographic changes and the alkali reserve in the series published by Duguid *et al.* (1961), or in those described by Prescott *et al.* (1962). The fact that in this patient the J wave became less evident in spite of a deterioration in the patient's clinical condition suggests that the body temperature is directly responsible for its appearance and magnitude.

Many theories have been proposed to explain the J deflection. Blades and Pierpont (1954) postulated anoxia and Osborn (1953) considered the hypothermic deflection was a 'current of injury' due to acidosis. The following theory is tentatively proposed since it explains some of the known facts concerning the electrocardiographic abnormalities.

Hollander and Webb (1955) found that the duration of the atrial potential in the rat was increased by a fall of temperature, and by using intracellular micro-electrodes they demonstrated that the duration of action potential is dependent upon the rate of membrane repolarization. Marshall (1957) found that the duration of action potential in rabbit atria was increased by lowering the temperature. The anterior septal region is the first part of the myocardium to be excited, the wave of depolarization then spreading to the right anterior and right posterior ventricle followed by invasion of the apex and left posterior ventricles; finally, the base of the heart is the last to be depolarized, the base of the left ventricle being excited a fraction of a second after the base of the right ventricle (Keele and Neil, 1961). If the rate of depolarization is slowed by hypothermia the QRS interval will be widened, but as the various parts of the myocardium are still being activated in normal sequence, the QRS interval will not be notched as in bundle-branch block. If the temperature is still further reduced it is possible that a left ventricular lead will show an initial Q wave due to flow of current in the interventricular septum away from the exploring electrode, followed by an R wave as the apex and mid-ventricular mass is excited. As this current diminishes the R wave tends to fall to the isoelectric line, but if the process of depolarization is slowed sufficiently the base of the left ventricle may be activated as the rest of the myocardium is tending towards zero potential. This will result in a second surge of current towards the exploring electrode which, by convention, will be registered as a second upstroke in the cardiogram. Only when the epicardial surface of the base of the left ventricle is depolarized will the potential fall to zero.

This may explain why the majority of authors have found J waves in the left ventricular leads. If potential changes are small they will be registered only in leads relatively near the area of depolarization (Goldberger, 1953). When J waves have been recorded in lead AVR they are invariably inverted, possibly due to the fact that this lead 'faces' the ventricular cavities, so the J wave current flows away from the electrode with a resulting downward deflection.

The diminution in voltage of the J wave may be due to a recovery in the rate of depolarization as the temperature rises. The prolonged QT segment suggests that the rate of repolarization (dependent upon intracellular energy-producing mechanisms) has not increased. This may indicate irreversible myocardial damage.

Summary

Serial electrocardiographic tracings were performed in a patient with accidental hypothermia, and the changes appeared to be directly related to the body temperature.

It is suggested that the electrocardiogram may give a better indication of the central body temperature than axillary or rectal temperatures.

A tentative theory is proposed in an attempt to account for the cardiographic pattern in hypothermia.

I thank Dr. J. Apley and Dr. J. Cosh for their help and encouragement, Dr. R. Bishton for the necropsy details, and Dr. R. Gibson and staff of the Bath Central Laboratory for the investigations.

References

- Altschule, M. D., and Sulzbach, W. M. (1947). Tolerance of the human heart to acidosis; reversible changes in RS-T interval during severe acidosis caused by administration of carbon dioxide. *Amer. Heart J.*, **33**, 458.
- Blades, B., and Pierpont, H. C. (1954). A simple method for inducing hypothermia. *Ann. Surg.*, **140**, 557.
- Brit. med. J.* (1963). Editorial. Deadly cold. **1**, 203.
- Corvino, B. G., and Hegnauer, A. H. (1955). Ventricular excitability cycle. Its modification by pH and hypothermia. *Amer. J. Physiol.*, **181**, 553.
- Drew, C. E. (1961). Profound hypothermia in cardiac surgery. *Brit. med. Bull.*, **17**, 37.
- Duguid, H., Simpson, R. G., and Stowers, J. M. (1961). Accidental hypothermia. *Lancet*, **2**, 1213.
- Emslie-Smith, D. (1956). Changes in the electrocardiogram during preoperative hypothermia in man. *Aust. Ann. Med.*, **5**, 62.
- (1958). Accidental hypothermia; A common condition with a pathognomonic electrocardiogram. *Lancet*, **2**, 492.
- , Sladden, G. E., and Stirling G. R. (1959). The significance of changes in the electrocardiogram in hypothermia. *Brit. Heart J.*, **21**, 343.
- Fairley, H. B., Waddell, W. G., and Bigelow, W. G. (1957). Hypothermia for cardiovascular surgery. Acidosis in the rewarming period. *Brit. J. Anaesth.*, **29**, 310.
- Goldberger, E. (1953). *Unipolar Lead Electrocardiography and Vectorcardiography*, 3rd ed., p. 72. Lea and Febiger, Philadelphia.
- Hollander, P. B., and Webb, J. L. (1955). Cellular membrane potentials and contractility of normal rat atrium and the effects of temperature, tension and stimulus frequency. *Circulat. Res.*, **3**, 604.
- Keele, C. A., and Neil, E. (1961). *Samson Wright's Applied Physiology*, 10th ed., p. 109. Oxford University Press, London.
- Marshall, J. M. (1957). Effects of low temperatures on transmembrane potentials of single fibers of the rabbit atrium. *Circulat. Res.*, **5**, 664.
- Osborn, J. J. (1953). Experimental hypothermia: respiratory and blood pH changes in relation to cardiac function. *Amer. J. Physiol.*, **175**, 389.
- Prescott, L. F., Peard, M. C., and Wallace, I. R. (1962). Accidental hypothermia. *Brit. med. J.*, **2**, 1367.
- Rees, J. R. (1958). Accidental hypothermia. *Lancet*, **1**, 556.
- Torresani, J., Nicolai, P., and Delboy, C. (1960). Modifications électrocardiographiques au cours d'un coma avec hypothermie. *Arch. Mal. Cœur*, **53**, 809.