Vasovagal Faint in the Supine Position

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Vasovagal syncope (Weissler and Warren, 1959) is common in the dental surgery, and it is now recognized that a patient may faint during the induction of general anaesthesia (Bourne, 1967). The reflex is rare in the supine position except in late pregnancy. This report describes a severe vasovagal faint in a patient lying flat and supine.

Case Report

The patient, a youth aged 17, suffered from asthma and had received steroid therapy for 11 years; he was currently having corticotrophin 20 units twice daily. He needed extensive conservative dentistry. Attempts at treatment under local anaesthesia alone had been unsuccessful, so it was decided to combine this with intravenous diazepam sedation. Neither premedication nor additional steroid therapy was given. He was positioned flat and supine and the heart rate was recorded by means of a beat-to-beat rate meter connected with an E.C.G. machine.

He looked pale and anxious, the pulse rate ranged between 60 and 80 beats per minute, and the systolic blood pressure was 120 mm. Hg. Diazepam 7·5 mg. was injected intravenously at the rate of 2·5 mg. every 30 seconds. During the injection he became extremely pale and unconscious. About 30 seconds after venepuncture the heart rate fell to about 30 beats per minute. Four beats after this the P waves disappeared from he E.C.G. During the next five minutes the heart rate fluctuated between 35 and 60 beats and P waves reappeared intermittently. Systolic blood pressure by auscultation was 70 mm. Hg. He was tilted head down, oxygen was administered, and atropine 0·5 mg. was given intravenously. Over the next two minutes the heart rate rose to 50 to 60 beats and P waves became regularly re-established. The systolic blood pressure was then 80 mm. Hg. with the pulse rate unchanged. Intavenous injection of 0·5 mg. of atropine was repeated and the heart rate rose to 70 to 80 beats with no change in blood pressure. His colour improved and he regained consciousness. Dental treatment was not started and he was admitted to hospital for observation; he was discharged after 24 hours.

Six months later he was readmitted to complete dental treatment. To investigate adrenal function Synacthen (tetracosactrin) 0·25 mg. was given intravenously; this produced a rise in plasma cortisol from a resting level of 17·4 μg./100 ml. to 24·9 μg./100 ml. after 30 minutes, indicating a satisfactory response. It was decided to carry out dental treatment under general anaesthesia, but to administer 7·5 mg. of diazepam beforehand.

The patient seemed calm and relaxed and was brought to the theatre without premedication. Electrocardiographic and heart rate monitoring were initiated. The resting heart rate was 60 to 70 beats per minute, and this fell to 50 to 60 beats after venepuncture before injection of 2 ml. of physiological saline. This was followed by 7·5 mg. of diazepam, which affected neither heart rate nor electrocardiogram. Atropine 0·5 mg. was then given with no effect on heart rate. Anaesthesia was induced with 50 mg. of methohexitone and 50 mg. of suxamethonium. Nasotracheal intubation was carried out and anaesthesia was then maintained with nitrous oxide: oxygen (5L.: 3L.) and halothane 1·5% and continued uneventfully.

Comment

There seemed to be three possible explanations of the patient's collapse: acute adrenal insufficiency, an unfavourable reaction to diazepam, or a vasovagal faint.

Acute adrenal insufficiency seemed unlikely, since this syndrome usually develops in the postoperative period and then hypotension is accompanied by a rise in heart rate (Salassa *et al.*, 1953).

Diazepam has been used extensively intravenously for sedation and induction of anaesthesia in doses up to 50 mg. with remarkable freedom from cardiovascular effects (Brown, 1968). In the present case the fall in heart rate occurred about 30 seconds after the insertion of the needle, by which time not more than 2·5 mg. of the drug could have entered the circulation; furthermore, little cardiovascular reaction was observed during the administration of 7·5 mg. of diazepam on the second occasion. The heart rate always decreases in fainting (Edholm, 1952). The electrocardiogram showed suppression of the sinus node with ventricular escape, and this suggests powerful vagal overactivity. During the attack atropine 0·5 mg. intravenously was followed by a small rise in heart rate and blood pressure. When the dose was repeated the heart rate rose by about 20 beats, but the systolic blood pressure remained unchanged. Barcroft *et al.* (1944) found in a study of post-haemorrhagic fainting that atropine administered after fainting produced an increase in cardiac output and rate, but the reduction in total peripheral resistance persisted owing to vasodilatation in the muscle arterioles which are not affected by atropine.

A vasovagal faint therefore seems to be the most likely explanation of the syndrome which developed in this fearful and apprehensive patient. Weissler and Warren (1959) stated that when fainting reactions occur in recumbent subjects they are particularly severe, often protracted, and associated with pronounced degrees of bradycardia.

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"Idiopathic" Lactic and βHydroxybutyric Acidosis

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In 1961 Huckabee reported the cases of nine patients suffering from a variety of disorders in whom an acute and rapidly progressive acidosis developed. This acidosis was due to the accumulation of lactic acid and was characterized by a rise in the blood lactate/pyruvate ratio, such as is seen in severe hypoxia or ischaemia, but without either of these conditions being present. Since then about 40 further cases have been recorded, most of which ended fatally. In many instances the acidosis was accounted for by the rise in blood lactate but not in others; the unknown acids were not identified.

We report here the case of an apparently non-diabetic patient with Gram-positive septicaemia in whom the level of blood β-hydroxybutyrate was nearly as high as that of lactate.

Case History

A 41-year-old man was admitted to hospital with a 72-hour history of malaise and fever. Twenty-four hours previously he had become unsteady on his feet and dysarthric. On admission he was febrile (102°F.; 38·9°C.), stuporous, and breathing rapidly (40/min.) and deeply. There was no cyanosis, the peripheral circulation was excellent, and the jugular venous pressure was not raised. There was a partial right hemiparesis which progressed

over the next six hours. The blood pressure was 170/70 and there were no abnormal findings in the chest. A few small petechiae were noted on the trunk; numerous new lesions appeared soon afterwards, together with Osler's nodes on several digits. At this time the chest *x*-ray picture, haemoglobin, and white cell and platelet counts were normal. The serum sodium was 140, potassium 3·7, chloride 100, and bicarbonate 8 mEq/l., giving an "anion gap" ($Na + K - Cl - HCO_3$) of 35·7 mEq/l. The plasma urea was 57 mg./100 ml., later 42 mg., total blood reducing substances were 130 mg./100 ml., salicylate was absent, and the urine was both free of reducing substances and negative to Acetest.

A diagnosis of septicaemia was made and ampicillin, cloxacillin, and sulphadimidine were given. *Staphylococcus aureus* was isolated from a blood culture.

One hour after admission arterial blood analysis gave the following results: pH 7·31; P_{O_2} 100 and P_{CO_2} 29 mm.Hg; standard bicarbonate 15·5 mEq/l.; base deficit 11 mEq/l. Pulse, blood pressure, and peripheral circulation were well maintained. After three further hours arterial blood was again analysed: pH 7·24; P_{O_2} 96 and P_{CO_2} 21 mm.Hg; standard bicarbonate 12 Emq/l.; base deficit 17 mEq/l. The urine pH was 5·2 and the urine was again negative to Acetest. The acidosis and large anion gap, in the absence of renal failure, ketonuria, or salicylate intoxication, suggested the diagnosis of "idiopathic" lactic acidosis. Gas chromatographic analysis (Barnett *et al.*, 1968) of venous blood taken 30 minutes later showed the following: lactate 5·3 μmole/ml. (*0·95 ± 0·33); pyruvate 0·2 μmole/ml. (0·099 ± 0·03); β-hydroxybutyrate 4·74 μmole/ml. (0·035 ± 0·01); succinate 0·003 μmole/ml. (<0·008); lactate/pyruvate ratio 26·5 (10·28 ± 2·54). Blood pressure, peripheral circulation, and urine output were well maintained until four hours after the sample of blood was taken for estimation of organic acids.

Over the next few hours a total of 500 mEq of bicarbonate was given intravenously raising the arterial pH to 7·30, but the patient became shocked and died 15 hours later. Necropsy showed widespread evidence of septicaemia, including haemorrhagic lesions in the right cerebellar and left cortical hemispheres, and infarcts of both spleen and kidneys. The left ventricle was moderately hypertrophied; the coronary arteries were patent. The liver was congested but microscopically showed nonspecific changes compatible with septicaemia, consisting of a mild infiltrate of polymorphs and plasma cells in the portal tracts; there was no evidence of liver cell necrosis. The right middle ear was moist and a staphylococcus of the same phage type as that in the blood was later grown.

Comment

Arterial oxygen saturation and blood pressure were normal and the peripheral circulation was excellent as judged clinically until some four hours after blood was taken for the measurement of blood organic acids. This case is therefore similar to those described by Huckabee (1961) and reviewed by Waters *et al.* (1963) and Tranquada *et al.* (1966). Waters *et al.* (1963) predicted that "disturbances characterized by the accumulation of an organic acid other than lactate may eventually be identified." In the present case blood lactate and β-hydroxybutyrate were found in almost equal concentrations; about 60% of the excess anion concentration can be accounted for by these acids. The absence of acetoacetate from the urine, as judged by a negative Acetest test, suggests that a gross rise of acetoacetate in the blood had not occurred; this implies a substantial increase in the hydroxybutyrate/acetoacetate ratio, which is in contrast to ordinary starvation or diabetic ketosis, when the concentration of both are raised together. It should be noted that the rise in blood hydroxybutyrate was much greater than is usually seen in subjects starved for two to three days. Blood succinate was within normal limits.

Idiopathic lactic acidosis has usually been treated by infusion of sodium bicarbonate, of which large amounts are generally required (Waters *et al.*, 1963), and occasionally survivals have been reported. The raised blood lactate/pyruvate ratio has been interpreted, in the absence of obvious lack of oxygen reaching the tissues, as indicating failure of proper tissue utilization of oxygen. On this assumption Tranquada (1964) and Tranquada *et al.* (1966) injected methylene blue as an electron acceptor, bypassing the normal electron transfer chain; the contribution of methylene blue therapy has, however, not been fully assessed. It should be pointed out that the reaction determining the lactate/pyruvate ratio is

$$\text{Lactate} + \text{NAD}^+ \rightleftharpoons \text{Pyruvate} + \text{NADH} + \text{H}^+.$$

Thus, as previously noted by Leppla *et al.* (1964), lactate/pyruvate may be partly determined by the cell pH as well as by the NADH/NAD ratio. The mildly raised lactate/pyruvate ratios reported in many cases could therefore be secondary to the acidosis, the primary defect being a failure of hepatic conversion of circulating lactate to glucose.

Finally, some comment is needed on the distinction between cases in which the acidosis appeared to be entirely accounted for by lactate accumulation (Huckabee, 1961) and those in which other acids must also have contributed (Waters *et al.*, 1963; Sproule *et al.*, 1966; case II, Coronato and Cohen, 1969). The lactate/pyruvate and hydroxybutyrate/acetoacetate ratios are determined by the factors outlined above, but the absolute levels of the individual acids—for example, lactate, pyruvate, hydroxybutyrate, and acetoacetate—are controlled by other mechanisms, and it is presumably these which determine whether or not lactic acid is entirely accountable for the acidosis.

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* Normal values, non-fasting, ± 1 S.D.

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