error the interocular latency for the ($-1/-1\times90°$) dioptre lens was within the normal range of 6 ms for all subjects. However, 31% of recordings with the ($+1/+1\times90°$) dioptre lens, 56% with the ($-2/-2\times90°$) dioptre lens, and 100% of the identifiable responses recorded with the ($+2/+2\times90°$) dioptre lens showed an abnormally increased interocular delay. The maximum relative delay observed was 32 ms.

Comment

Reduction in amplitude of the VER with refractive error has previously been reported.[4] Our study, using the pattern-reversal method, confirms that finding. More importantly, our study illustrates the significant changes in absolute and relative latency of the P2 component when refractive errors which approximate to those found in the population at large are introduced to defocus a small stimulus field and high spatial frequency pattern. This effect is greatest for refractive errors of ($+2/+2\times90°$) dioptres. Because a relative or absolute prolongation of P2 latency is often found in cases of suspected MS, and because of their similarity to the findings of our study, we would emphasise that refractive errors should be reduced or eliminated to minimise false-positive results. Furthermore, the VA must be considered in the interpretation of the VER.

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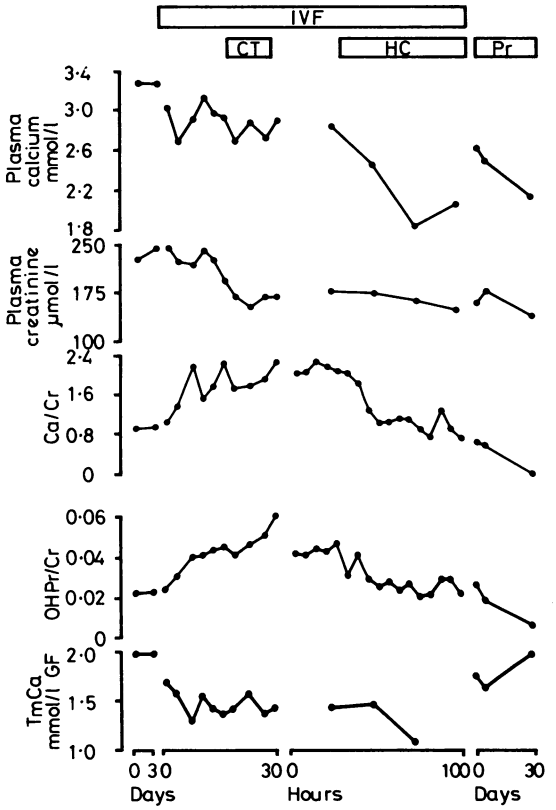
Acute effects of saline, calcitonin, and hydrocortisone on plasma calcium in vitamin D intoxication

Hydrocortisone reduces plasma calcium in hypercalcaemic states other than primary hyperparathyroidism.[1] The mechanisms, however, are not entirely clear. Calcium absorption and renal tubular reabsorption of calcium are reduced and a direct reduction in bone resorption has been suggested. On the other hand, chronic administration of cortisol increases bone resorption and predisposes to osteoporosis.[2 3] We report the case of a patient with vitamin D intoxication in whom acute administration of hydrocortisone resulted in a rapid reduction in bone resorption with consequent abolition of hypercalcaemia. All methods used have been described.[4]

Case report

A woman aged 36 developed hypoparathyroidism after a thyroidectomy in 1964. She was maintained on vitamin D_2 (200 000 U/day) and calcium supplements. Three years after the thyroidectomy she developed hypothyroidism and was maintained on thyroxine 0·3 mg/day. In 1978 she was noted to be hypercalcaemic. Vitamin D and calcium supplements were withdrawn, but her plasma calcium concentration remained high and she was referred for further management. Initial investigation (figure) suggested that the hypercalcaemia (plasma calcium concentration 3·28 mmol/l (13·1 mg/100 ml); normal range 2·22-2·60 mmol/l (8·9-10·4 mg/100 ml)) was due to a combination of increased bone resorption (fasting urinary OHPr:Cr 0·023; normal <0·017) and decreased glomerular filtration (creatinine concentration 223 μmol/l (2·5 mg/100 ml)). She had low absorption of radiocalcium (0·39 fraction of dose absorbed per hour; normal range 0·3-1·2) and plasma 25-hydroxy vitamin D concentration was raised at 650 ng/ml (normal range 5-72·5 ng/ml). The concentration of parathyroid hormone was unmeasurable and of alkaline phosphatase normal at 6·2 KA units/100 ml (normal range 3-13), as was that of plasma phosphate at 1·12 mmol/l (3·47 mg/100 ml); normal range 0·8-1·45 mmol/l (2·5-4·5 mg/100 ml)). The patient was clinically and biochemically hyperthyroid (T4 = 255 mmol/l (normal range 60-140)), and thyroxine was discontinued.



Changes in plasma and urinary biochemistry after saline 3 l/day (IVF), calcitonin (CT), hydrocortisone infusion (HC), and prednisone (Pr). Doses used and normal ranges described in the text.

Conversion: SI to traditional units—Calcium: 1 mmol/l ≈ 4 mg/100 ml. Creatinine: 1 μmol/l ≈ 0·0113 mg/100 ml. Ca:Cr: 1 ≈ 0·353. OHPr:Cr: 1 ≈ 1·16. TmCa: 1 mmol/l GF ≈ 4 mg/100 ml GF.

She was put on a low calcium diet and rehydrated with intravenous saline, which resulted in a fall in plasma calcium due to improved glomerular filtration and reduced renal tubular reabsorption of calcium. Bone resorption, however, remained high and the patient remained hypercalcaemic. Frusemide (two consecutive daily doses of 40 mg) and calcitonin in low doses (20 U/day for 12 days) did not affect the hypercalcaemia. Hydrocortisone sodium succinate 50 mg was added to each litre of saline given for a period of three days, the fluid regimen remaining otherwise unchanged. Within four hours of starting hydrocortisone there was a reduction in urinary hydroxyproline and urinary calcium concentrations accompanied by a reduction in plasma calcium concentration, which became subnormal by the second day. Renal tubular reabsorption of calcium fell further but glomerular filtration rate remained unchanged. The patient was later discharged taking prednisone 5 mg and thyroxine 0·1 mg daily, having become clinically and biochemically hypothyroid (T4 < 10 mmol/l). She has remained normocalcaemic with no evidence of increased bone resorption, but mild renal impairment has persisted.

Comment

The plasma calcium concentration was reduced by the established effects of saline infusion on glomerular filtration rate and renal tubular reabsorption of calcium. The low calcium absorption in the face of a high plasma 25-OHD_3 concentration and the increase in bone resorption during saline infusion, which was related to improvement in renal function, are unexplained. Neither frusemide nor calcitonin reduced renal tubular calcium reabsorption or bone resorption. The effect of hydrocortisone on the plasma calcium concentration was due mainly to a reduction in bone resorption, although it also decreased the tubular reabsorption of calcium. The rapid onset of action

suggests a direct action on bone, as has been shown in tissue culture.[5] The previous thyroidectomy and parathyroid removal, with the lack of response to calcitonin, indicate that the reduction in bone resorption could not have been mediated by a reduction in parathyroid hormone secretion or rise in endogenous calcitonin. It is unlikely that developing hypothyroidism played any major part in the action of hydrocortisone on bone resorption in this patient, since the response to hydrocortisone was rapid and the plasma half life of T4 is of the order of 6-7 days. Indeed, after the initial withdrawal of the thyroid supplements there was a steady increase in bone resorption until the administration of hydrocortisone.

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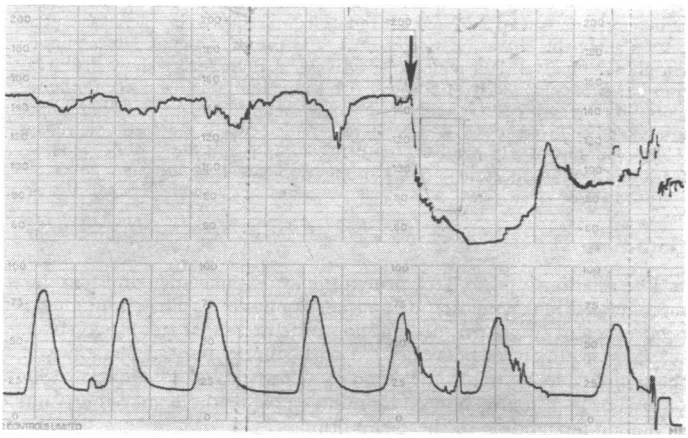
Hypoxic stillbirth due to entangled intrauterine catheter

Uterine pressure is recorded, using polyvinyl intrauterine catheters, in many obstetric units practising active management of labour. It carries only a small risk to mother or fetus.[1] There have been only sporadic reports of uterine perforation or disruption of fetal vessels.[2-4] A case has been described of umbilical cord entanglement. We report a similar experience.[5]

Case report

The patient, a 31-year-old primigravida, had conceived after 10 years of voluntary infertility. She was admitted at 38 weeks' gestation for rest and observation because of pre-eclampsia. The results of tests for fetal welfare were satisfactory. Labour was induced at term by artificial rupture of the membranes and intravenous oxytocin. A fetal scalp electrode was applied and an intrauterine pressure catheter introduced to 15 cm by the standard technique. The fetal heart rate at that time was normal. Initial progress in labour was slow but acceptable. A lumbar epidural block provided analgesia. After several hours of strong contractions variable decelerations of the fetal heart rate (FHR) were noted (figure) and the progress of labour was unsatisfactory. It was decided to perform lower-segment caesarean section. An attempt to remove the intrauterine catheter met with resistance and coincided with a sharp fall in the FHR, which recovered after several minutes. When the patient was anaesthetised a further attempt to withdraw the catheter was made but abandoned. A fresh stillborn infant weighing 3560 g was delivered. At operation the catheter was found to be acutely kinked and entangled in the umbilical cord, which was compressed.



Recording showing variable decelerations of fetal heart rate due to compression of umbilical cord. Attempted removal of intrauterine catheter produced sharp (arrow) and sustained fall in rate.

Comment

Usually intrauterine pressure monitoring using a polyvinyl intrauterine catheter is safe. In our case we did not appreciate that the catheter was compressing the cord during labour, producing variable deceleration of the FHR. Trying to remove it led to intrauterine asphyxia and fetal death. If variable decelerations in FHR occur with an intrauterine catheter in situ umbilical cord compression by the catheter should be considered. When a gentle attempt to remove the catheter meets with resistance or produces further deceleration of the FHR further attempts to remove it should not be made.

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Successful treatment of malignant testicular teratoma with brain metastases

The outlook for some patients with metastatic testicular teratoma has improved considerably with recent advances in chemotherapy.[1] The presence of brain metastases, however, is generally accepted as unfavourable and we have found no reports of successful treatment. We report a case in which extensive chemotherapy combined with surgery and radiotherapy seems to have been effective.

Case report

A 25-year-old postgraduate student presented in December 1974 with a swollen right testis. Orchidectomy and histological examination showed a malignant teratoma with trophoblastic features. Lymphangiography showed metastases in iliac nodes. He was treated by pelvic and para-aortic irradiation to a dose of 4000 rads. He remained well until January 1976. He then developed a right lower-lobe pneumonia as well as severe frontal headaches followed by a mild left hemiparesis. Chest *x*-ray examination confirmed the presence of metastatic tumour in the right lower lobe and also in the left mid-zone. Computerised tomography (CAT) of the brain showed deposits in the right parietal and right frontal regions. His serum gonadotrophin (HCG) concentration was 52 000 IU/l. Alpha fetoprotein (AFP) was indetectable. Lymphangiography showed no evidence of intra-abdominal metastases.

Treatment was started in January 1976. Over the next 12 months he received 19 courses of systemic chemotherapy, usually in conjunction with intrathecal methotrexate (figure). By January 1977 there had been a partial response, as judged by: (1) resolution of the hemiparesis after two months; (2) sequential CAT brain scans showing improvement but also a persistent abnormality in the right parietal area; (3) sequential chest radiographs showing partial resolution but isolated shadows in the right and left mid-zones; (4) serial serum HCG concentrations, initially falling to normal after 10 weeks' treatment but later rising, indicating persistent active tumour (figure). Consecutive right and left thoracotomies to remove residual lung metastases were performed in February and March 1977 (Mr A R Makey). A lesion from the right middle lobe contained only necrotic tissue