

<sup>2</sup> Fisher, E R, *et al*, *Laboratory Investigation*, 1968, 18, 689.

<sup>3</sup> Morris, J F, Ginn, H E, and Thompson, D D, *American Journal of Medicine*, 1963, 34, 867.

<sup>4</sup> Thomson, C, *et al*, *Quarterly Journal of Medicine*, 1974, 43, 399.

<sup>5</sup> Trygstad, C W, *et al*, *Journal of Pediatrics*, 1970, 76, 861.

# St Pierre University Hospital, 1000-Brussels, Belgium

A DE TROYER, MD, resident physician

P PADUART, MD, consultant nephrologist

J SHOCKERT, MD, consultant radiologist

R PARMENTIER, MD, professor of pathology

## Titrated prostaglandin E<sub>2</sub> tablets for induction of labour

We report here a study on the use of orally administered prostaglandin E<sub>2</sub> to induce labour in 100 women (41 primiparae and 59 multiparae).

### Patients, methods, and results

All the patients were at 38 to 41 weeks' gestation and had a recognised indication for induction. Their inducibility rating was assessed by Bishop's method<sup>1</sup>, 28 patients (13 multiparae and 15 primiparae) having a score below 6 and the remaining 72 patients (46 multiparae and 26 primiparae) having a score of 6-11. Patients who had had previous spontaneous onset of labour or rupture of membranes and patients of parity 5 or more were excluded from the trial.

Starting with a minimum dose of one tablet (0.5 mg), the tablets (prostin E<sub>2</sub>) were given 30 minutes after low amniotomy was performed and then at every hour, vaginal assessment being made every four hours. If the cervix was dilating and the uterus contracting satisfactorily on examination the original dose was continued. If not the dose was increased by one tablet (0.5 mg) up to a maximum of three tablets (1.5 mg) for a maximum of 12 hours, after which the treatment was changed to intravenous oxytocin. Doses were changed only after the four-hourly vaginal and abdominal examination. Intrauterine monitoring, using the Sonicaid FM2 with an intrauterine catheter, was undertaken on all patients receiving the maximum dose of 1.5 mg.

Labour was successfully induced and vaginal delivery achieved in 94 patients, including 13 who required forceps delivery. The mean amniotomy-delivery interval in multiparous patients was 6 h 56 min and in the primiparous patients 8 h 51 min. The doses used in each group are shown in the table.

Dose required to induce labour in 94 women in whom induction was successful

Maximum dose:	1.5 mg	1.0 mg	0.5 mg	Mean total dose
Multiparous (n = 57) ..	6	28	23	4.9
Primiparous (n = 37) ..	10	16	11	6.3

Six patients were excluded from the study, one because of persistent vomiting after the first dose of 1.5 mg prostaglandin E<sub>2</sub>. Two patients were delivered by caesarean section because of fetal distress (2 h 15 min and 8 h 10 min after amniotomy). Three primiparous patients did not progress satisfactorily after 12 hours of prostaglandin treatment: two required caesarean section, one after 8 and the other after 12 hours of oxytocin infusion, and the third had only achieved 3 cm dilatation after 12 hours but had a normal vaginal delivery after oxytocin infusion for a further 9 h 40 min. No serious maternal side effects could be attributed to oral prostaglandin. There was no uterine hypertonus or significant alteration in maternal blood pressure or heart rate. Eight patients developed mild fever, which subsided without treatment. Vomiting occurred in 18 patients and one patient developed diarrhoea. The fetal heart rate was not affected in the 94 successful cases, and the mean Apgar score after one minute was 8.6. Third stage complications included four retained placentae and five cases of post-partum haemorrhage of more than 500 ml.

### Discussion

The use of prostaglandin E<sub>2</sub> tablets is a further refinement of oral administration, which overcomes the bitter taste of the solution<sup>1</sup>

and the occasional nausea and vomiting it produces.<sup>2</sup> Oral administration also has advantages over intravenous infusions in ease of administration, convenience for medical and nursing staff, and increased acceptability to the patient.<sup>3</sup> The absence of overstimulation and adverse effects on fetomaternal wellbeing was noteworthy.

The incidence of maternal gastrointestinal side effects appears to be related to dose, as suggested by Craft,<sup>4</sup> who found an incidence of side effects 36% in women using oral prostaglandin E<sub>2</sub> solution. The low incidence of vomiting and diarrhoea in our series (19%) may therefore have been due to the low titrated dose administered or the use of prostaglandin E<sub>2</sub> tablets rather than solution.

The short induction-delivery interval, especially in multigravidae, together with the safety of prostaglandin E<sub>2</sub> tablets, the ease of administration, and acceptability to the patient with such a titrated dose commend this method of induction for routine clinical use in suitable patients.

We thank the patients who participated in this study, the consultant staff of St Mary's Hospital for permission to use their patients, the midwifery staff for their enthusiastic help, and the Upjohn Company for the supply of prostaglandin E<sub>2</sub> tablets.

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<sup>2</sup> Karim, S M M, and Sharma, S D, *British Medical Journal*, 1971, 1, 260.

<sup>3</sup> Kelly, J, Flynn, A M, and Bertraud, P V, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1973, 80, 923.

<sup>4</sup> Craft, I, *British Medical Journal*, 1972, 2, 191.

### Department of Obstetrics and Gynaecology, St Mary's Hospital, Manchester

A ALAILY, MB, MRCOG, registrar (now clinical tutor, Department of Obstetrics and Gynaecology, Wythenshawe Hospital, Manchester)

G A MOREWOOD, FRCS ED, MRCOG, lecturer (now consultant, Department of Obstetrics and Gynaecology, Wythenshawe Hospital, Manchester)

## Heerfordt's syndrome in two sisters

Two middle-aged sisters, living separately, developed sarcoidosis with bilateral uveoparotitis and cranial palsies within six months of each other.

### Case reports

A previously healthy 41-year-old housewife developed bilateral anterior uveitis in June 1972; there was no lacrimal gland enlargement or xerophthalmia. In August the parotid and submandibular glands were bilaterally enlarged and painful. There was no palpable lymphadenopathy or hepatosplenomegaly. Investigations showed Hb 12.9 g/dl; WBC  $7.4 \times 10^9/l$ ; ESR 4 mm h; protein electrophoresis normal; serum calcium 2.4 mmol/l (9.6 mg/100 ml); urinary calcium 8.75 mmol (350 mg)/24 h; Mantoux test negative at 1/1000 and 1/100 dilutions; diffusing capacity (DCO) marginally reduced at  $5.7 \text{ mmol min}^{-1} \text{ kPa}^{-1}$ ; chest radiographs and ECG within normal limits; Kveim test gave positive sarcoid histology. While being investigated the patient developed a left facial palsy. She was admitted to hospital and started on systemic corticosteroids. Her urinary calcium fell to 4.75 mmol/24 h, and she was discharged. By November 1972 chest radiographs showed bilateral hilar adenopathy. Reduction of the steroid dose resulted in a flare-up of the anterior uveitis and the development of peripheral lung mottling. She remained well on prednisolone 10 mg daily.

A previously healthy 35-year-old housewife, younger sister of the above patient, developed erythema nodosum in December 1972. Two months later her parotid and submandibular glands were bilaterally enlarged and painful, her mouth was dry, and her eyes felt gritty. She had enlarged lymph nodes in the right supraclavicular fossa and right epitrochlear region, bilateral anterior uveitis, and bilateral lacrimal gland enlargement with complete absence of tears. Investigations showed Hb 12 g/dl; WBC  $4.3 \times 10^9/l$ ; ESR 10 mm h; protein electrophoresis normal; serum calcium 1.9 mmol/l (7.6 mg/100 ml); urinary calcium 3.25 mmol (130 mg)/24 h; pulmonary function, ECG, and chest radiographs within normal limits. Biopsy of a supraclavicular node and parotid gland showed sarcoid tubercles and a Kveim test was positive.

In March 1973 she developed a complete bilateral facial nerve palsy. She was started on systemic corticosteroids. By the end of June her facial weakness had fully recovered and tear production was normal but she had bilateral