Clinical and Endocrinological Effects of Intermittent Abdominal Decompression in Complications of Pregnancy *

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SUMMARY

Intermittent abdominal decompression was used in a series of patients whose pregnancies were complicated by pre-eclampsia, essential hypertension and suspected retardation of foetal growth. There was a statistically significant increase in serial urinary oestriol output in cases of pre-eclampsia and small-for-dates babies in which decompression therapy was used, but no significant increase occurred in cases of essential hypertension. The rise in oestriol levels with decompression was taken as evidence of improved foetal welfare, obviating the need to interrupt pregnancy in these cases and thereby reducing the prematurity rate. No significant increase in foetal or placental weight was observed in our treated cases. Persistently low serial oestriol levels call for induction of labour and a guarded prognosis due to the possible presence of congenital heart disease, while a sudden oestriol fall may signify impending foeto-placental catastrophe.

Further investigation into long-term results of abdominal decompression is indicated.


The clinical value of intermittent abdominal decompression in pregnancy using the Heyns suit has been described for cases of pre-eclampsia and chronic hypertension by Blecher and Heyns. More recently it has been shown that decompression therapy in patients with retardation of foetal growth and development is associated with improved clinical results and increase in output of urinary oestriol and pregnanediol.

The present investigation examines certain aspects of the clinical and hormonal effects of intermittent abdominal decompression in cases of pre-eclampsia, essential hypertension, and dysmaturity.

MATERIAL AND METHODS

The series consisted of 111 cases involving some 650 assays of both oestriol and pregnanediol. In the pre-eclampsia groups were 31 patients who had a blood pressure over 140/90 mmHg with, or without, proteinuria after the 20th week of pregnancy; 18 patients were treated by abdominal decompression and 13 untreated. Patients with blood pressure over 140/90 mmHg from the beginning of pregnancy were classified as essential hypertension and there were 15 cases in this category; 10 untreated and 5 given decompression therapy. Cases with superimposed toxaemia were excluded.

Fig. 1. A case of severe pre-eclampsia with fall in urinary hormone levels. The curved lines show the upper and lower limits of the normal range in pregnancy for oestriol, after Scommegna and Chattoraj; and for pregnanediol, a modification (MacRae et al.) of the graph by Russell et al.
There were 65 cases classified as small-for-dates, from clinical assessment of retarded intra-uterine foetal growth and 2 initial urinary oestriol levels below the normal range; in 35 cases decompression was used, and 30 were untreated. Some cases previously reported are included in this series.

Abdominal decompression was given with the Heyns suit in half-hourly sessions for 15 seconds in every minute, and a negative pressure of about 70 mmHg was aimed at. The sessions were given 3 times weekly or as often as twice daily, according to the urinary oestriol levels.

Estimations of oestriol were made from 24-hour specimens of urine collected, with few exceptions, on a day decompression was not used. Urinary oestriol assays were made by gas-liquid chromatography, after the method of Wotiz and Clark; and plasma oestriol was determined using the procedure described by Nachtigall et al. For urinary pregnanediol estimations the extraction technique of Klopper and associates and the gas chromatographic method of Brush and co-workers were employed.

RESULTS

Pre-eclampsia

In this group of 31 patients abdominal decompression was given to 18, of whom 13 had initial oestriol levels below the normal limit. Thirteen patients, 9 of whom had oestriol levels below the normal range, did not receive decompression treatment and were used as controls.

Fig. 1 shows falling urinary oestriol and pregnanediol levels in a case of toxaemia of pregnancy; the blood pressure at the 27th week was 150/90 mmHg and the urine contained a trace of albumin. The condition progressed to severe pre-eclampsia with a blood pressure of 170/120 mmHg and 6 g protein in the urine. Marked hormonal fall preceded the worsening of clinical changes. A live infant weighing 1500 g was delivered by caesarean section at the 32nd week of pregnancy.

Fig. 2 shows low urinary oestriol output at the 34th week of pregnancy in a patient with pre-eclampsia, whose blood pressure was 155/95 mmHg, without proteinuria. Abdominal decompression, given once daily for 2 weeks and then twice daily until term, was associated with a
rise in hormone levels and clinical improvement. Labour was induced at term and a live baby weighing 3,900 g was delivered.

Fig. 3 shows low hormone values in a patient who had had 2 previous pregnancies complicated by severe pre-eclampsia. The blood pressure was only moderately raised in the present pregnancy and there was no albuminuria. A severe accidental haemorrhage occurred at the 34th week, with intra-uterine foetal death. It is to be noted that the urinary oestriol level showed a marked fall 3 days before the antepartum haemorrhage.

**Essential Hypertension**

In a group of 15 patients classified as having essential hypertension, 5 were treated with abdominal decompression and 10 were left untreated. All 5 treated cases had initial oestriol levels below the normal range, and in 3 of the untreated group the initial oestriol levels were similarly below normal.

In Fig. 4 are shown serial urinary oestriol levels below normal limits and pregnanediol values in the lower range of normal, typical of the hormonal finding in cases of essential hypertension in this series. The pregnancy ended in spontaneous labour at the 37th week; a live child of 2,800 g was delivered.

Fig. 5 relates to a case of essential hypertension treated by abdominal decompression given once daily on alternate days from the 29th week of pregnancy. Plasma and urinary oestriol levels, which showed parallel values, failed to respond to decompression therapy, although there was a late rise in urinary pregnanediol output. Spontaneous labour 3 days before term resulted in the birth of a baby weighing 2,948 g.

From Fig. 6 it can be seen that abdominal decompression did not give a statistically significant increase in the urinary oestriol levels in cases of essential hypertension. In the treated group of the pre-eclamptic cases, however, the urinary oestriol showed a gradual rise which increased to statistically significant levels after the 38th week of pregnancy \((P>0.005)\) compared with the untreated group. The mean oestriol levels at 38 weeks in pre-eclamptic patients receiving decompression treatment was 20.4 mg, while in treated cases of essential hypertension the corresponding oestriol mean value was 13.79 mg. A clear increase, therefore, above the lower limits of normal range for urinary oestriol is seen in treated cases of pre-eclampsia, but in untreated pre-eclampsia, and in both treated and untreated essential hypertension cases of our series, the mean oestriol levels remained below the normal range.

As regards pregnanediol levels, the range recorded each week varied greatly in individual cases, allowing only a
mean value to be expressed. In cases of pre-eclampsia where decompression treatment was given, the mean urinary pregnanediol level at the 38th week of pregnancy was 47.7 mg and in the untreated group it was 47.32 mg; in cases of essential hypertension treated by decompression the mean urinary pregnanediol value at the same period of pregnancy was 47.8 mg, and in the untreated cases the mean value was 38.8 mg.

In the pre-eclamptic cases in the untreated group there was one perinatal death, a stillbirth due to an antepartum haemorrhage (see Fig. 3).

There were no perinatal deaths in the treated group of essential hypertension cases, but one neonatal death is recorded in the untreated group. The patient had a moderately severe hypertension throughout her pregnancy, with urinary oestriol levels below the normal range; premature labour occurred at the 38th week and a caesarean section was required due to foetal distress. The baby, weighing 2 920 g, died shortly after birth and postmortem findings showed a heart lesion with ventricular septal defect.

Small-for-Dates Babies

The comparative effects of intermittent abdominal decompression on serial urinary oestriol and pregnanediol levels in cases of suspected retardation of foetal growth are shown in Fig. 7. The untreated patient, with a history of previous birth of a dysmature infant, maintained a very low level of urinary oestriol and pregnanediol; the pregnancy was terminated at the 39th week and a 2 240 g baby was delivered. In the other patient, a primigravida treated by abdominal decompression, there was a rise in hormone levels, particularly when treatment sessions were increased from twice weekly to twice daily at the 35th week of pregnancy; the baby, born spontaneously at term, weighed 2 807 g.

Fig. 8 shows that the urinary output of oestriol and pregnanediol in a case of a suspected small-for-dates foetus can vary with the amount of decompression treatment given; the baby, born at term, weighed 3 100 g.

It is seen from Fig. 9 that low hormone output may accompany congenital abnormality of the foetal heart. There was also failure to respond to decompression therapy which was given thrice weekly for 2 weeks and then daily for 1 week. Intra-uterine foetal death occurred at the 37th week of pregnancy, and spontaneous labour resulted in a fresh, stillborn child weighing 1 309 g.

The chromatograph illustrated by Fig. 10 shows that there is a significant increase in mean urinary oestriol levels after the 37th week of pregnancy in a group of patients
Our investigation into the use of intermittent abdominal decompression in pregnancy shows that it can increase urinary oestriol and pregnanediol output in cases of pre-eclampsia and in suspected retardation of foetal growth. Whereas in both these complications of pregnancy urinary oestriol excretion shows a statistically significant increase after the 37th week, decompression therapy did not give rise to any improvement in the oestriol values in pregnancy complicated by essential hypertension (Figs. 6 and 10).

In the clinical care of our cases serial urinary oestriol values, which are accepted as an index of foetal viability, were used to decide whether to interrupt pregnancy or allow it to continue to term, thus, on the one hand, lessening the risk of intra-uterine foetal death and, on the other, helping to avoid the hazards associated with prematurity. It can be accepted from our results (Table 1) that the practice of permitting pregnancy to go to term, because of the increase in oestriol levels associated with abdominal decompression therapy, did not adversely affect the perinatal death rate; and that, particularly in cases of dysmaturity, the series shows a distinct fall in perinatal mortality. It is noted in the work of Blecher and Heyns that the foetal survival rate was significantly better in all groups of toxemic women treated by decompression, while in their cases of essential hypertension the clinical response was similar in treated and untreated groups.

This investigation also shows that serial oestriol assays can provide warning of impending foeto-placental mishap in the absence of other clinical evidence. In 2 cases (1 not in the present series) during urinary oestriol monitoring, a

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Fig. 8. The effect of decompression treatment is illustrated in a small-for-dates case. D = once daily sessions, D, = thrice daily sessions, S = cessation of treatment.

Fig. 9. Persistent low hormone levels unaffected by decompression treatment in a small-for-dates case in which there was a stillbirth from a congenital heart lesion.
TABLE I. COMPARATIVE REVIEW OF THE EFFECTS OF INTERMITTENT ABDOMINAL DECOMPRESSION ON DYSMATURITY, PRE-ECLAMPSIA AND ESSENTIAL HYPERTENSION

<table>
<thead>
<tr>
<th></th>
<th>Dysmaturation</th>
<th>Pre-eclampsia</th>
<th>Essential hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cases</td>
<td>Treated</td>
<td>Untreated</td>
<td>Treated</td>
</tr>
<tr>
<td>Premature deliveries</td>
<td>35</td>
<td>13</td>
<td>18</td>
</tr>
<tr>
<td>Mean weight of term babies</td>
<td>2956 g</td>
<td>2954 g</td>
<td>3021 g</td>
</tr>
<tr>
<td>Mean placental weight</td>
<td>514.1 g</td>
<td>578.2 g</td>
<td>522.6 g</td>
</tr>
<tr>
<td>Uncorrected perinatal deaths</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Corrected perinatal deaths</td>
<td>2</td>
<td>1</td>
<td>1</td>
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Perinatal deaths, corrected for cases incompatible with life

A guarded foetal prognosis must be given in cases of persistently low oestriol levels for additional reasons, such as encephalhy and congenital heart abnormalities. In our series there were 3 infants with congenital heart abnormalities and low oestriol levels; a stillbirth (Fig. 9) with intraventricular septal defect and anomalies in mitral and aortic valves; a neonatal death with a single ventricle and intra-atrial septal defect (both in the small-for-date group); and a neonatal death with ventricular septal defect in the hypertension group.

As regards the mode of action of abdominal decompression, it has been shown to enhance uteroplacental blood flow. This could have the effect of increasing the number of simultaneously functioning placental villi to give an action analogous to the physiological hyperventilation of pregnancy with its increase in tidal volume. By such means, therefore, foeto-placental metabolism could be improved and stimulated to greater production of foetal dehydro-epiandrosterone and placental oestriol and pregnanediol, an increase which may be related to the amount of decompression therapy given (Fig. 8). In cases of essential hypertension, however, it is possible that vascular pathology could reduce the dialysing effect of decompression and, as our series shows, limit hormone production.

It would be reasonable to assume that if by a certain procedure the secretion of an organ were increased for some substances, similar increases could extend to other vital processes, which can outlast the period of decompression treatment and benefit foetal health.

Although no statistical increase in the weight of the placenta and baby was found in our series, it is thought that a more extended investigation into the action of abdominal decompression in pregnancy, with special reference to long-term results for the infants, is indicated.

We wish to thank the North West Regional Hospital Board, London, for a research grant; and Miss V. White. Acknowledgment is made to the Proceedings of the Royal Society of Medicine for permission to reproduce Fig. 1, and to the Journal of Obstetrics and Gynaecology of the British Commonwealth for Figs. 7 and 10.

sudden fall in oestriol levels preceded concealed accidental haemorrhage which caused foetal death. In retrospect it may be reflected that, in the case illustrated in Fig. 5, immediate delivery could have resulted in a live foetus. Beischer and others report that in mild cases of accidental haemorrhage premonitory low oestriol values may be recorded, but in severe cases oestriol levels may be within normal range.

![Graph](image-url)
Malignant Hyperthermia in Anaesthesia

I. CASE REPORT*

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SUMMARY

Malignant hyperthermia is a rare but dreaded complication of general anaesthesia, the mortality rate being about 60-70%.

The first case of this condition to occur in Karl Bremer Hospital was diagnosed on 9 June 1971.


A coloured man of 38 years was admitted to hospital with the complaint of intermittent headaches since 1965. This symptom became much worse over the 3 months preceding his admission. No neurological signs were present except for a possible lesion in the right frontoparietal lobe, detected on radioactive scanning. The cerebrospinal fluid protein level was raised. A right-sided carotid angiogram was deemed necessary.

Pre-operative temperature varied from 35°C to 36.1°C and on the morning of the investigation it was 35.9°C. Pre-operative serum chemistry is reflected in Table I.

Preamedication (meperidine 50 mg, promethazine 25 mg, droperidol 5 mg and atropine 0.6 mg) was injected intramuscularly an hour before operation.

Anaesthesia was induced with thiopentone sodium 250 mg, and succinylcholine (Scoline) 40 mg. Endotracheal intubation was carried out and anaesthesia was maintained on nitrous oxide, oxygen, and halothane and spontaneous ventilation, using a modified Boyle Mark III circle with carbon dioxide absorption. No rigidity or other complication was observed during the course of the anaesthesia, which lasted an hour.

The carotid angiogram done, anaesthesia was discontinued, and this was followed by marked shivering and rigidity, which resembled the shivering often seen after halothane anaesthesia. This condition, however, was so marked and continued for so long, that it worried the anaesthetist.

About 15 minutes after the end of anaesthesia (11/2 hours after induction), nitrous oxide, oxygen and halothane were administered by mask, and the shivering and rigidity were controlled, only to recur on discontinuing the anaesthesia.

*Date received: 24 November 1971.