TOXAEMIA OF PREGNANCY TREATED BY ABDOMINAL DECOMPRESSION

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Abdominal decompression given regularly to normal parturients has a definite and clearly observed role in the prevention of the onset of the toxaemias of pregnancy. Thus Heyns[1] pointed out that only 2 cases of pre-eclampsia occurred out of 300 White decompression patients—both were mild. Since the clinic incidence of pre-eclampsia at Queen Victoria Maternity Hospital is 7·6%, this represented 11·5 times the standard error of the difference, a clearly significant figure. This has led me to use abdominal decompression therapeutically in the toxaemias of pregnancy to establish whether those factors acting prophylactically, will benefit the patient with the established disease.

Patients with 'moderate' or 'severe' pre-eclampsia, essential hypertension or chronic nephritis, have been treated by abdominal decompression both at Queen Victoria Hospital, Johannesburg (to date 28 patients), and at Baragwanath Hospital, Johannesburg (to date 65 patients). 'Moderate' is defined as blood pressure in the range 140/100—160/110 mm.Hg, albuminuria + to ++, Esbach reading up to 2 G protein per 24 hours, with moderate oedema ++; 'severe', as blood pressure range over 160/110 mm.Hg, albuminuria ++ to solid, Esbach reading over 2 G/24 hours, with gross oedema +++. The same blood pressure and albuminuria levels have been applied in categorizing essential hypertension and chronic nephritis.

All 'mild' cases (i.e. less than these levels), have been excluded on the assumption that they would have settled spontaneously without any special treatment.

TREATMENT

This consisted of abdominal decompression, bed rest and a low sodium diet. No sedative, diuretic or antihypertensive drugs were used. A control series of 65 patients at Baragwanath Hospital have been treated on appropriate drug therapy, bed rest and low sodium diet. More recently a third series has been started, of patients treated on bed rest and low sodium diet only. Should one of these patients deteriorate or fail to improve within 48 hours, she is transferred to one of the other two series alternately, and treated appropriately. Inclusion of patients in the 3 different series is unselected, and performed as a routine by the ward sister on admission to the antenatal ward. Deterioration of their condition calls for medical or surgical induction of labour.

Abdominal decompression was given twice daily for a period of half an hour at a time each day. The pressures used were individually gauged according to tolerance. In the average case the patients were decompressed to from −50 to −80 mm.Hg for 15 seconds in every half-minute. If this produced dizziness, the decompression time was correspondingly reduced to 10 seconds per half-minute and so on. It was very rarely necessary to reduce it to 5 seconds per half-minute. On the whole this toxaemic group tolerated decompression better than a normal blood pressure control group, and resultant dizziness was rare. One patient fainted on one occasion while in decompression, but did not do so on subsequent occasions. Decompression was given twice for ten minutes on the first day of treatment, twenty minutes on the second, and thirty minutes on the third and subsequent days.

The ward sister sent off weekly midstream-urine specimens for bacterial culture, microscopy and chemical analysis, and charted blood pressure readings 4-hourly. Daily urine albumin

levels were charted (trace to ++++) and Esbach readings recorded. In the severe group all urine was tested at the time of passing. Strict intake-output charts of all fluids were kept. The patients were weighed daily and the weights recorded.

Once a week blood was sent to the laboratory for determination of the levels of blood urea, uric acid, electrolytes (Na, K, Cl, CO_2 content), serum proteins, haemoglobin and transaminases.

The blood pressure was taken and charted by me half an hour before and immediately before each session in decompression, again after 15 minutes decompression, after 30 minutes decompression, and after half an hour of bed rest following decompression. This amounted to 10 daily recordings by the same observer on each patient and this was repeated throughout their stay in hospital until delivered or discharged home. The decompression periods were usually mid-morning and mid-afternoon. These decompression readings in conjunction with the 4-hourly sister's chart gave a good assessment of the progress of the daily blood pressure.

Decompression was given in labour whenever possible for facilitation of labour and pain relief,[2,3] and was given throughout the contractions and at much stronger pressures according to individual preference. Routine sedation in labour was used when necessary. When pregnancy required termination by medical or surgical induction of labour, abdominal decompression was practised during labour contractions (or 'pitocin-contractions' if on an intravenous oxytocin infusion). The patient's blood pressure and the conditions prevailing at the time of delivery determined whether a spontaneous vaginal delivery was allowed or whether episiotomy, forceps or Gasyd outlet suction,[4] was used to assist delivery. Decompression was not used in labour at Baragwanath Hospital for technical reasons, and very few instrumental deliveries, with a view to shortening the second stage of labour, were performed.

THE RESPONSE TO THERAPY

This has been graded for convenience into 4 grades:

Grade I : *Very good response*. Pre-eclamptic toxaemia settled completely, and the blood pressure recordings were normal (less than 120/80 mm.Hg) thereafter. The albuminuria and oedema disappeared.

Grade II : *Good response*. The condition settled down almost completely. The blood pressure was normal with occasional raised levels or with traces of proteinuria persisting.

Grade III : *Moderately good response*. The condition showed some signs of improvement, such as a lessening from severe to moderate, or from the moderate to the mild category. The patients were kept in hospital and treated until the spontaneous onset of labour, or labour was prematurely induced by medical or surgical means.

Grade IV : *Indifferent or poor response*. The condition showed no signs of settling down. It either continued without obvious improvement, or deteriorated requiring termination of pregnancy by medical or surgical means. This was then done to avoid the risks of eclampsia, abruptio placentae or intra-uterine death from inefficiency of placental blood supply.

Analysis of Results

Analysis of the results of the patients treated to date shows that the results of decompression treatment on pre-eclamptic patients are good. Thus, 87% of White pre-eclamptics showed some benefit from treatment, and 13% a poor response. Of these 87%, 69% showed a good response to treatment.

In the White groups with essential hypertension and chronic nephritis, the results were not so good. Only 50% showed any benefit from treatment, 30% showing a good response. Twenty-seven of the 29 White infants born to this group were living, and 2 were macerated stillbirths; intra-uterine death resulting from placental insufficiency in essential hypertensive mothers.

In 45 non-White mothers treated with decompression between February and August 1964, the results were not nearly so good. The best results were in the decompression-treated pre-eclamptic group where 76% showed some benefit from treatment, 38% showing a good response, compared with 59% of the drug-treated group of patients showing some benefit and only 18% showing a good response. The results of treatment with decompression or with drugs in the groups of patients with underlying essential hypertension or chronic nephritis were similar. Here only 33% showed any benefit from treatment, while only 0·7% showed a good response. The over-all results of 45 decompression-treated mothers compared with 45 drug-treated mothers were as follows: 20% of the decompression-treated patients showed a good response, 53% showed some benefit from treatment, and 47% showed a poor response, as compared with 7% good response, 38% showing some benefit, and 62% poor response in the drug-treated cases.

The foetal mortality rate of the decompression-treated group was 15%, i.e. 85%, 41 out of 48 infants born—including twins—lived. In the drug-treated group the mortality rate was 22%, i.e. 78% lived (39 of 50 infants delivered). Once again; the lowest mortality was among the decompression-treated pre-eclamptics, 9% (21 of the 23 infants delivered in this group) lived, as compared with a 16% mortality in drug-treated pre-eclamptics (16 of 19 infants delivered in this group lived).

Those pre-eclamptics who responded very well to decompression treatment and were discharged home, were all subsequently readmitted from the antenatal clinic with recurrent pre-eclampsia. If a severe pre-eclamptic settles on the treatment, it obviously does not mean that the toxaemia is cured, and daily decompression must be continued, if not in hospital then at home or as an outpatient.

It is still possible that those patients not generally improved on this regime of twice-daily decompression might respond better if decompression were given more frequently, say five times daily, or if periodic intermittent decompression were given during the day and night in a special 'decompression bed'.

The patients feel well on decompression treatment. They are not drowsy because they are not taking sedatives. They are allowed lavatory facilities out of bed, but for the rest of the time they are kept in bed. In White patients the average total labour took 5 hours 54 minutes and the first-stage average time was 5 hours 36 minutes in a group which contained 64% primigravidae. In non-White patients the average total labour time of 8 hours and first-stage labour times of 7½ hours, were similar in decompression-treated and drug-treated patients.

The fact that the blood pressure almost always falls to normal levels after half an hour of decompression is interesting. The vascular haemodynamics are obviously entirely altered by abdominal decompression. The blood flows into the low-pressure zone in the abdominal compartment more freely, there being probably less resistance to inflow. The blood pressure taken at the arm drops, the

patient may get dizzy or even faint, possibly from temporarily reduced cerebral blood-flow. The placenta and kidneys, lying in the low-pressure abdominal compartment, probably receive a greater supply of blood during abdominal decompression, and a possible 'flushing effect' of the placental blood-vessels is envisaged. Where the vessels are chronically damaged as in the essential hypertensive and chronic nephritic group of patients, it may be that this 'vascular flushing' effect during decompression cannot occur, or is minimized, thus failing to improve the placental (and therefore foetal) blood flow, nutrition and oxygenation. Nevertheless, the attempt to improve the results in this group still presents a challenge.

Microscopically dilated and congested placental villous capillaries are very much in evidence where decompression has been used. Although possibly owing to hypoxia, this might equally well have been caused by improved capillary circulation with opening up of new capillaries. The postpartum haemorrhage rate was 14·3% in White patients (average for Queen Victoria Hospital 10 - 12%), and 11% in non-White patients treated by decompression, as compared with a 7% incidence in the drug-treated group. This higher incidence may be related to increased placental congestion induced by twice-daily decompression.

In the pre-eclamptic group, the babies and placentae were heavier and larger on an average than in the essential hypertensive and nephritic group of White and non-White patients. This indicates a better placental blood supply and oxygenation in this pre-eclamptic group. They were also a little heavier in non-White pre-eclamptics treated with decompression than in those treated with drugs, but were similar in patients with essential hypertension or nephritis, irrespective of the method of treatment. The prematurity rates were similar using either method of treatment. The uric acid levels were raised above 5 mg. in 81% of White pre-eclamptics treated by decompression, and continued to rise antenatally despite clinical improvement of the toxaemia. 60% of the non-White decompression-treated pre-eclamptics had a raised serum uric acid level, compared with 30% of the drug-treated pre-eclamptic group. The reason for this is not apparent. Raised serum transaminase levels were found in decompression and drug-treated pre-eclamptics, but raised blood urea levels as described by Riedel[5] and others were not found. There were reduced levels of serum proteins in 71 - 75%, and serum CO_2 content in 64 - 75% of White and non-White patients treated by either method. The other electrolytes showed no obvious changes.

CONCLUSION

A mechanical approach to the treatment of the toxaemias of pregnancy is being used with the object of improving the placental blood supply, with a possible 'flushing effect' of the placental blood-vessels. The treatment shows promise with regard to the treatment of pure pre-eclampsia, but is not so effective in essential hypertensive and chronic nephritic patients.

Much work remains to be done to assess the effect of the treatment on a large number of toxaemias, and to establish experimentally the best methods of using abdominal decompression in the toxaemias of pregnancy.

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