BRITISH MEDICAL JOURNAL

LONDON SATURDAY AUGUST 23 1947

CHRONIC HYPERTENSION AND PREGNANCY*

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Siward. Had he his hurts before?
Rosse. Ay, on the front.
Siward. Why then, God's soldier be he!
Had I as many sons as I have hairs,
I would not wish them to a fairer death.

MACBETH, Act 5, sc. 7.

Until comparatively few years ago discussion of the clinical features of the toxaemias of pregnancy and concerning their aetiology laid the chief emphasis on the state of the kidney and the presence or absence of albumin in the urine. Little or no attention was paid to the vascular changes which we now know to be one of the essential features and one of the earliest disturbances in pre-eclamptic toxaemia. Thanks to numerous investigators considerable progress has been made in elucidating its causes, though much still remains to be learnt.

I do not propose to discuss the causes of this hypertension, but rather to consider pregnancy as it occurs in the woman who before her pregnancy starts is the subject of chronic hypertensive vascular disease. Its importance will be apparent from the fact that these patients constitute about 25% of all cases of "toxaemia of pregnancy." Pre-eclamptic toxaemia is much more common, contributing about 70%, while chronic glomerular nephritis in pregnancy (sometimes badly named "nephritic toxaemia") is the rarest of all—about 5%. Notwithstanding the frequency of chronic hypertension in pregnancy it has received little attention in obstetric literature. This is, I think, due partly to the fact that obstetricians have confused the condition with "chronic nephritis"—with which, however, it has nothing in common—and partly because it is only in recent years that through antenatal care there have been opportunities for the study of women in the early weeks of pregnancy and that the recording of blood-pressure readings at these early antenatal visits has become routine.

The Standard of Normal Blood Pressure

A definition of chronic hypertension will depend on one's ideas as to what is the standard of normal blood pressure. Reid and Teel (1939) take the standard as 140/90. Chesley and Annitto (1947) also adopt this standard, but they ignore the reading at the first antenatal examination. On the other hand they include all cases in which this level is reached before the 24th week of pregnancy. Henry (1936) showed that the blood pressure in normal pregnancy tends to be lower than in the non-pregnant state. This is especially true of the diastolic pressure, and as a result the pulse pressure tends to be 10 mm. greater than in the non-pregnant. He regards this as a mechanism by which the heart is enabled to meet the increased demands made on it by the increase in blood volume and vascular area of normal pregnancy.

For many years we in the Obstetric Unit of University College Hospital adopted the standard 130/70 for the normal pregnant woman. Since 1940, influenced by the work of Robinson and Brucer (1939, 1940), we have used the standard 120/80. These workers showed that: (1) the normal range of blood pressure is 90 to 120 systolic and 60 to 80 diastolic; (2) blood pressure does not rise with age in normal persons but it does in hypertensive and pre-hypertensive persons; (3) a history of blood pressure above 120 systolic and 80 diastolic over a ten-year period is pathological, and an almost infallible sign of incipient hypertension; (4) transient rises of blood pressure should not be ignored and should lead one to suspect a further permanent rise; (5) slightly over 40% of the population (in the U.S.A.) is either actually or potentially hypertensive. It is usual for obstetricians to adopt 140/90 as the standard of normal blood pressure, but I believe that this is much too high and that if this standard is adopted many pregnant women with chronic hypertension will be overlooked and disaster may occur, or disaster that has previously occurred will remain unexplained. Some of my own most interesting and instructive cases of chronic hypertension in pregnancy had blood pressures under 140/90 when pregnancy began, and the diagnosis would have been missed had the usual standard been adopted. An example is Mrs. M., referred to later. It is customary, too, in antenatal clinics to ignore the first reading of blood pressure if it is found to be high. The patient is allowed to rest, and then a second reading is taken, and if it is lower, as it generally is, this is recorded as the true level. I believe that this is wrong and that the first reading is significant and should be recorded, though there is of course no harm in taking it again after rest and recording that also. Indeed, if the second reading is lower it may have a prognostic significance, as we shall see later. The blood pressure of the normal pregnant woman does not rise significantly with nervousness. If it does she is hypertensive or potentially so. I described this condition in 1933 as the "early warning rise of blood pressure" and showed that in 65% of such women the blood pressure later in pregnancy became permanently elevated.

* William Meredith Fletcher Shaw Memorial Lecture delivered at the Royal College of Obstetricians and Gynaecologists, London, on July 4, 1947.

Diagnosis

The patient may be known to have chronic hypertension before the pregnancy began. It may have followed on a previous eclampsia or pre-eclamptic toxaemia, though whether it is the *result* of these disorders is doubtful. There is evidence that such patients would have developed hypertension had they never been pregnant. Josephine Barnes and I (1945) in a study of the relatives of these women showed that in the patient who has chronic hypertension in pregnancy there is a familial tendency to the disease, as shown by its high incidence in blood relatives, while in pre-eclamptic toxaemia there is no such tendency, though I suspect that there is in subjects of pre-eclamptic toxaemia and eclampsia who after these disorders develop chronic hypertension. This view receives support from the work of G. W. Theobald (1933), who has shown that the death rate from chronic vascular hypertensive disease is not any higher among married than among single women. Whether the onset of the disease is hastened by eclampsia or pre-eclamptic toxaemia so that it appears at an earlier age than it would otherwise have done is a nice point for discussion.

The majority of patients, however, have not been under observation before they book at the antenatal clinic, and the possibility of diagnosis will then depend on the stage of pregnancy at which the hypertension is discovered. Chesley and Annitto (1947), as I have pointed out, take 24 weeks as the borderline. I have always placed it at 20 weeks. If the blood pressure is raised above the standard for the first time after that period the case is classed as " pre-eclamptic toxaemia " ; if before it, as " chronic hypertension." Sometimes the patient is seen for the first time after the 20th week, and then in the absence of a previous history classification is impossible between chronic hypertension and pre-eclamptic toxaemia.

Clinical Features

The pregnant woman with chronic hypertension is generally somewhat older than usual: in my last series the average age was 30.2 years as compared with 27.9 years for all pregnant women. She usually feels well, there is no oedema or albuminuria, and kidney function tests do not show any deficiency except in the most severe type of case. If there has been a previous pregnancy it has often ended in stillbirth or abortion. In my last series of 194 cases between 1942 and 1946, analysed for this lecture, there were 48 multiparae, who between them had 78 pregnancies but only 27 live infants—a foetal loss of 65.3%. It is of interest to note in passing that these same 48 women in their present pregnancy—that is, the one under observation and supervision—produced 45 live and surviving infants—93.7%, a foetal wastage of only 6.3%. This may be a tribute to the value of antenatal care in these cases.

Fall of Blood Pressure in the Second Trimester.—It is curious that in a certain proportion of cases of chronic hypertension in pregnancy the blood pressure falls in the second three months. This phenomenon was first noted by Reid and Teel (1939) and was confirmed by Chesley and Annitto (1947), who found that a decrease of 20 mm. Hg systolic occurred in 39.6%. In 11.3% the fall exceeded 40 mm. Hg, while in 50% there was no change. In my last series of 194 cases it fell in the second three months in 40%. The cause of the fall is not definitely known. Apparently it always occurs in pregnant hypertensive dogs, and Rodbard and Katz (1944) suggest that it is related to the low-resistance circuit that develops during pregnancy. As Chesley and Annitto (1947) point out, this mid-pregnancy fall is important in relation to diagnosis, for if the patient has not been seen early in pregnancy pre-eclamptic toxaemia is apt to be diagnosed. We shall see later that it has also some bearing on prognosis.

Exacerbation of Hypertension in Later Months.—Whether or not this fall in blood pressure occurs in mid-pregnancy the tendency is for a rise to take place in the later weeks. This may happen in the middle three months, but as a rule it does not set in till the 25th week or even much later, and it may not occur at all. In my last series of 194 cases it occurred in 54%. In the first series reported by Browne and Dodds (1942) it happened in 61%.

Relation of Blood Pressure to Albuminuria and Foetal Death

If and when the blood pressure reaches 160 mm. Hg systolic, albuminuria is apt to appear, and this may or may not be accompanied or followed by oedema. In other words, on the chronic hypertension is superimposed pre-eclamptic toxaemia. The level of blood pressure at which albuminuria appears varies considerably. The average in my last series was 160 mm. Hg systolic, but in one case the blood pressure reached 185/110 without albumin appearing. In another there was no albuminuria though the blood pressure at three antenatal visits was 160/80, 180/90, and 182/116, while in two others it was present when pressures of 134/75 and 140/90 were recorded. These figures are only rough approximations, as the blood pressure in chronic hypertension is exceedingly labile. Thus it may be recorded as 140/90 on the first occasion on which albumin is found at the weekly or fortnightly visit, but that does not necessarily mean that it was at that level when the albumin first appeared in the urine. The experiments of Chesley and Markowitz (1939) suggest that the albuminuria is caused by spasm of the afferent arterioles of the glomeruli. On immersing the hand in cold water for two minutes the blood pressure rises suddenly. If the systolic and diastolic blood pressures rose more than 16 mm. Hg albumin appeared in the urine and its onset coincided with release of the spasm. It is evidently due to the anoxia caused by the spasm injuring the glomerular capillaries. Clinical experience shows that when the blood pressure reaches 160 mm. Hg systolic, albumin usually appears in the urine. At the same time the foetus is apt to die *in utero* from concealed accidental haemorrhage. This is believed to be due to spasm of the spiral arteries of the decidua, causing anoxia and injury of the walls of the vessels distal to the constriction. When the spasm passes off and blood again floods the capillaries their walls give way. It will be recalled that Markee's (1940) observations on endometrial transplants in monkeys show that this spasm and consequent haemorrhage in the mucosa is the immediate cause and precursor of menstruation. That a similar spasm is present in women with pre-eclamptic toxaemia is well known, for it is visible in the retinal arterioles when the systolic blood pressure exceeds 150 mm.

The appearance of albumin in the urine in the patient with chronic hypertension is of very serious significance for the foetus. In my last series it developed in 14.9%. Among these the foetal and neonatal mortality was 17.8%. If the above conception of the cause of the albuminuria is correct it will be evident that the albuminuria is not itself the cause of the foetal death, nor is the death due to any toxic substance retained by the injured kidney. The albuminuria is associated with the foetal death because both are due to the same cause—that is, vascular spasm which in the kidney causes albuminuria and in the uterus causes concealed haemorrhage.

In normal pregnancy pre-eclamptic toxaemia with hypertension and albuminuria develop in only about 1% of cases. In my last series of cases of chronic hypertension it developed in 14.9%, and in the first series reported with

Miss Dodds in 1942 in 17.9%. If the view is correct that albuminuria is the result of raised blood pressure and vascular spasm, the question that has to be answered is: Why does the blood pressure tend to rise still further in such a large percentage of patients who start their pregnancy with chronic hypertension? The solution of this question would throw much light on the cause of the rise of blood pressure in pre-eclamptic toxaemia, but at present we do not know the answer.

Treatment

The Question of Terminating Pregnancy

In the more severe cases the question of terminating the pregnancy will arise. This should be done if at the beginning of pregnancy the tests of kidney function show decided deficiency or if there is well-marked retinal arteriosclerosis, exudates, or papilloedema, or persistent albuminuria. My experience shows that, even though renal function tests fail to show any definite failure to excrete or concentrate, and even though there are not any fundal changes, if before the 20th week there is persistent albuminuria it is not worth while continuing, for intrauterine death will almost certainly occur.

When in the more severe degrees of hypertension in early pregnancy there is difficulty in deciding whether the pregnancy should be continued the reaction to rest will be a valuable guide. This involves admission to hospital for a week or more, with daily records of blood pressure. If the blood pressure falls to normal or near normal levels the outlook with proper supervision is good. If, on the other hand, it remains at 150/100 or over the outlook is doubtful, and the decision as to whether the pregnancy should be allowed to continue may well rest with the woman and her husband. If it is allowed to continue very strict supervision throughout the entire pregnancy, preferably in hospital, will be necessary.

In patients in whom the results of these various tests are satisfactory the pregnancy may be allowed to continue. From the start of the pregnancy periods of rest should be enjoined, and the duration of these should increase as pregnancy progresses. Exacerbation of the hypertension is as a rule not likely to occur before the 25th week, but after that time the patient should be watched very carefully. The usual monthly visit is not enough. Even though all is going well she should be seen at least once a fortnight, and in the more severe cases once a week. Should the blood pressure rise to 150 mm. systolic she should be put to bed, preferably in hospital, and kept there for at least a week. When this is done the blood pressure usually falls to a safe level, and she may be allowed up about the ward and even out of doors. But it is rarely, if ever, safe to allow her home once this dangerous exacerbation has occurred. It is of the utmost importance to remember that the "critical level" of blood pressure is 160 mm. systolic, and that above that level albuminuria is apt to occur and the foetus to perish *in utero*. All our efforts must therefore be directed to keeping the blood pressure below this critical level, and for this purpose we have at present no better remedy than rest in bed, with a mild sedative at night such as 5–10 gr. (0.32–0.65 g.) of "carbromal." There is no need for any special diet restrictions, and a low-protein diet is unnecessary. Should pre-eclamptic toxaemia supervene with albuminuria and oedema, restriction of salt and fluid will be advisable.

In patients whose blood pressure is at a dangerously high level and does not respond to these simple measures potassium thiocyanate has been advised. I have used it in only two cases, and in neither did it seem to be of much value. In one the foetus died *in utero* at 23 weeks. The details of the other case are as follows:

Mrs. A., aged 32, had had one previous pregnancy that ended in abortion at four months. She first came under observation at 16 weeks, when her blood pressure was 198/120. Her father had died of heart disease and her mother of a stroke. Her highest blood urea was 30 mg. per 100 ml.; urea clearance, 56.45%; urea concentration, 1.25%; there was no albuminuria or any evident fundal changes. She was seen by Dr. Kenneth Harris, and under his supervision in the medical wards was treated with potassium thiocyanate. In due course, and when her pregnancy had reached 33 weeks, he wrote as follows: "Initially the B.P. showed improvement with potassium thiocyanate treatment (relief of headaches and lowering of B.P.), but now her B.P. has gone up in spite of continued drug treatment. I would recommend termination unless (*a*) her kidney function tests are all normal; (*b*) her B.P. falls appreciably in the next week." At 34 weeks retinal exudates appeared, and as the foetus was still alive caesarean section was done and a viable child obtained, which survived and is now well. It is of interest to note in passing that six months after delivery this patient's blood pressure is 210/120; there is no albumin in the urine and her fundus oculi has cleared up, so that she does not seem to be any worse.

With such simple measures as I have outlined the great majority of women with chronic hypertension go to term without any trouble and are delivered spontaneously of living healthy children. This is especially true of those whose blood pressure at the start of pregnancy is below 150/100. If, however, at any time papilloedema, with or without retinal exudates, develops, the pregnancy must be terminated at once; but if these develop late in pregnancy there is no need to sterilize unless the patient has all the children she wants or the hypertension is so pronounced that it seems unlikely that she will ever be able to bear a living child.

Method of Delivery

If there is no serious exacerbation and pre-eclamptic toxaemia does not supervene a natural delivery may be allowed at term. If a "breakdown" occurs, however, the case is different. Once albuminuria develops there is great danger of intrauterine death, and it may be advisable to terminate the pregnancy as soon as the foetus is viable. This should, if possible, be delayed until the end of the 37th week, counted from the first day of the last menstrual period. Even then it should be remembered that the foetus is often small, so that at the end of the 37th week it may not weigh more than 4 lb. (1.8 kg.), though the chances of survival seem to depend more on the degree of maturity than on weight. If therefore the pregnancy has reached the end of the 37th week there need be no hesitation in terminating it, though it is of course better to wait longer if it is possible to do so without undue risk of death *in utero*.

The really difficult cases for decision are those in which the pregnancy has reached only the 32nd or 33rd week, and where on account of uncontrollable hypertension with or without albuminuria the foetus is in daily danger of perishing. In such cases we have occasionally tried to gain a few more days by the use of "veratrone" in small repeated doses. This drug certainly lowers the blood pressure, but its action is very transient and the injections need to be repeated every three or four hours day and night, so that it hardly seems worth while, especially as supplies are limited in this country at the present time.

For the delivery of the premature infant caesarean section suggests itself as the easiest and safest method. It saves the infant from all trauma and therefore should in theory be the best method. Local anaesthesia is best, and as soon as the child is delivered its air passages should be cleared of all mucus and foreign material by a mucus extractor, if possible before it takes its first breath, for many of these infants die of inhalational pneumonia. Caesarean

section, however, is not the whole answer to the problem of delivery, and the results are not infrequently disappointing. The infant born by the natural passages seems to gain something that it loses by caesarean section. This is probably because, while respiration is inhibited by the higher centres during the last months of intrauterine life, the inhibition is slowly released by the slight asphyxia that occurs during natural labour, with the result that after birth the child breathes better than one born by caesarean section, in whom this stimulus is lacking. For this reason there is an increasing tendency at the present time to deliver more of these infants by induction of premature labour, using the rubber bougie or stomach tube so that the membranes may be intact as long as possible. It must be remembered that on account of the softness of the cranial vault and the delicacy of the cerebral vessels the premature infant is particularly apt to sustain a fatal cerebral haemorrhage during delivery. The slightest asphyxia, with resultant congestion of the cerebral veins, may lead to a haemorrhage into the lateral ventricles from the choroid plexus. A wide episiotomy so as to prevent delay of the head on the perineum and compression by the vulval ring may be a life-saving measure, and if there is delay on the perineum the application of forceps is advisable, but they must be applied laterally with the blades over the ears.

Prognosis

The Child

In our combined series the foetal loss was 9.2%. In the series recently reported by Chesley and Annitto (1947) it was 38.2%. The difference is certainly accounted for by the less rigid standard of hypertension adopted by them. The outlook for the child bears a definite relation to the height of the blood pressure at the start of pregnancy. Of the patients whose blood pressure at the start of pregnancy was 150/100 or over the foetal mortality was 63.6%. One may therefore say that, speaking generally, if the patient starts her pregnancy with a blood pressure of 150/100 or over there is only a 30% chance of her bearing a viable child. There are, however, not infrequent exceptions to this rule, and cases occasionally occur in which the blood pressure is 180 or over at the beginning of pregnancy or before it and yet the pregnancy is uneventful and successful. An example of this is Mrs. D., referred to later. One of the most puzzling questions in these cases is to distinguish at the beginning of pregnancy which patient will do badly and which well. I am not here speaking of the cases of malignant hypertension with badly involved kidney, papill-oedema, and retinal exudates, or persistent albuminuria, which always do badly and should be terminated as soon as possible, but of the case of mild or moderate degree in which there are no other abnormal signs except the hypertension itself. Why does one of these cases do badly and another do well? Why in one such case does the blood pressure rise dangerously, albumin appear in the urine, and the foetus die *in utero*, while in another apparently equally severe at the beginning of the pregnancy all goes well, the blood pressure never rises, there is no albuminuria, and a living child is born at term? It might be supposed that the two are different in nature, that the case that does badly is really an example of occult or latent chronic nephritis that lights up during the pregnancy. I do not believe that this is the explanation, for I have followed some of these patients for years. If they were examples of occult nephritis surely in the course of ten or twelve years the nephritis would have advanced sufficiently to become clinically manifest, with persistent albuminuria, yet it does not seem to do so. One example of a patient whose course I have followed for 14 years will illustrate this point.

Mrs. M. first came under our observation in 1933 when she aborted at 20 weeks. She was then aged 33 and there had been three previous pregnancies. The first had ended in eclampsia in 1927, the second and third had ended at seven months, and of these three pregnancies there had been no living child. In her fifth pregnancy she was first seen at the 12th week. Her blood pressure was 142/84; there was no albuminuria; the haemoglobin was 75.1%, the blood urea 22 mg. per 100 ml., and the urea clearance 67%. She was admitted to hospital with a diagnosis of "recurrent toxaemia," as it was believed that the underlying cause of the recurrence was chronic hypertension. She was kept in hospital until delivery. At 23 weeks the blood pressure fell to 110/75 and remained normal till the 30th week, when in spite of rest and care it began to rise until at 33 weeks it reached 168/118. Three days later albumin appeared in the urine and persisted till the end of the 36th week, when the membranes ruptured spontaneously. Caesarean section was done one week later and a living child weighing 4 lb. 4 oz. (1.9 kg.) was born; it survived, and is now 9 years old and well. After delivery the mother's blood pressure fell, and on discharge from hospital it was 126/72, but there was still a trace of albumin in the urine. Nine months later the blood pressure was 150/90 and the albumin had disappeared. In November, 1946—eight years after delivery—her blood pressure is 162/98 and *there is still no albuminuria.* The absence of albumin after all these years shows, I think, that hers is not a case of latent chronic glomerular nephritis (occult nephritis). The case of Mrs. D. (see below) lends support to this view.

In every case in which the foetus died *in utero* the blood pressure had not fallen in the second three months. On the other hand all patients in whom the blood pressure fell in the second three months gave birth to living children. This point may therefore have some prognostic value. Chesley and Annitto state that if a rise of blood pressure occurs in the second three months it is ominous for the foetus.

The Mother

Immediate Prognosis.—In the first series of 222 cases reported in 1942 by Miss Dodds and myself there were only two deaths. In the new series of 194 cases there were also two deaths, and neither could be attributed to the hypertension. One was due to ileus following caesarean section for fibroids, and another to pulmonary embolism. With exacerbation of blood pressure there is a special danger of ante-partum haemorrhage, eclampsia, and cerebral haemorrhage. In Chesley and Annitto's (1947) series of 218 patients the incidence of eclampsia was exactly ten times that in all patients, and the incidence of pre-eclamptic toxaemia was increased seven times.

Remote Prognosis.—The question at once arises: Is the woman who has chronic hypertension made any worse by pregnancy? Is her expectation of life shortened? In 1939 Gladys H. Dodds and I published the late results in 65 chronic hypertensive women who had gone through 86 pregnancies and had been followed up for periods up to twelve years. We wrote as follows: "Judged by the general condition, height of blood pressure, and cardiac changes the pregnancy did not seem to have any ill effect in 52 out of the 65 patients. In seven the effect of the pregnancy was unknown: six are dead (9.2%). . . . In spite of these six fatal cases we believe that the large majority of women with simple hypertension may pass through several pregnancies, go to term, and give birth to live infants without suffering any demonstrable deterioration in their condition." One example in illustration may be given.

Mrs. D.—This patient's first pregnancy had been terminated at 22 weeks at another hospital on account of albuminuria. She first came under our care in 1931 in her second pregnancy, when chronic hypertension was diagnosed. The highest recorded blood pressure was 164/80 and she was delivered of a macerated foetus. In the follow-up her blood pressure

four weeks after delivery was 180/110, and at five months post partum it was still 180/110, with no albuminuria. She then became pregnant for the third time, and at eight weeks her blood pressure was 164/106. Now this patient has been observed by us in five subsequent pregnancies. She has produced five living children, the last, which weighed 8½ lb. (3.85 kg.), in 1945. Eight weeks after the birth of this last child her blood pressure was 132/90 and in July, 1946—that is, one and a half years after the last child—it was 126/70. There is no albumin in the urine, the fundus oculi is normal, and kidney function tests are constantly within normal range. It is remarkable that in the first two pregnancies under our care the blood pressure remained elevated during the whole of the time. In the remaining three it fell to normal or subnormal levels in the middle months, usually but not always with a tendency to rise again towards the end of pregnancy.

I do not wish to claim that the hypertensive patient is actually benefited by pregnancy, though this case suggests that this may sometimes happen, but at least I can end the survey on a cheerful note. The experience gained by a further follow-up during and since the war confirms me in my opinion that pregnancy is not injurious provided the patient survives the immediate risks of the pregnancy itself, which are certainly somewhat greater than in the normal patient. Pregnancy does not seem to cause any permanent aggravation of the hypertension.

In 1945 Josephine Barnes and I approached the problem from another angle. We examined the blood pressures of approximately 2,000 women, half of whom were nulliparae and half had borne children. It is obvious that if pregnancy aggravated hypertension the mean blood pressures in the parous women should be significantly higher than in the nulliparae. The results classified in age groups are shown in Table I.

TABLE I.—*Showing Mean Blood Pressure in Nulliparous and Parous Women classified in Age Groups*

Age Group		Total No. of Cases	Systolic B.P.		Diastolic B.P.	
			Mean	S.D.	Mean	S.D.
10–19	N.	82	119·4	11·0	76·3	9·6
	P.	3	109·0	49·6	69·0	4·9
20–29	N.	401	119·3	15·7	76·2	11·3
	P.	208	121·8	14·1	77·4	10·8
30–39	N.	274	126·9	15·5	78·5	12·3
	P.	364	126·0	15·0	70·0	13·2
40–49	N.	105	134·9	19·3	85·0	12·1
	P.	275	134·7	22·3	85·5	13·0
Over 50	N.	53	152·7	30·9	91·6	17·0
	P.	191	155·1	30·7	93·2	16·0

N. = nulliparous. P. = parous. S.D. = standard deviation.

Table I shows that there is no significant difference between the mean blood pressures in nulliparous and parous women in any age group. It shows also that the mean blood pressure rises with age, but this applies equally to the parous and the nulliparous woman. Table II shows the same thing in a different way. Here two different standards of blood pressure are taken—120/80 and 140/90—but there is no significant difference between the numbers of nulliparous and parous women in any of the age groups. It may be remarked that these figures suggest that pre-eclamptic toxaemia and eclampsia do not of themselves *cause* chronic hypertension, or even cause a latent hypertension to appear at an earlier age than it otherwise would have done. On this optimistic note I may conclude.

TABLE II.—*Showing Percentages of Nulliparous and Parous Women with Blood Pressures over* 120/80 *and* 140/90

Age Group		Total No. of Cases	B.P. over 120/80		B.P. over 140/90	
			No.	%	No.	%
10–19	N.	82	23	28·0	0	
	P.	3	1	33·3	0	
20–29	N.	401	101	25·2	16	4·0
	P.	208	64	30·7	9	4·3
30–39	N.	274	117	42·7	23	8·4
	P.	364	133	36·6	27	7·4
40–49	N.	105	61	58·0	20	19·0
	P.	275	162	59·0	58	21·1
Over 50	N.	53	38	71·7	21	39·6
	P.	191	151	79·0	91	47·7

N. = nulliparous. P. = parous.

Summary

Chronic hypertension constitutes about 25% of all cases of toxaemia of pregnancy.

It must be differentiated from chronic nephritis and pre-eclamptic toxaemia.

In the chronic hypertensive the blood pressure tends to fall to a normal level in the second three months of pregnancy. It may or may not rise again in the later weeks.

If the systolic blood pressure rises above 160 mm. Hg albumin is likely to appear in the urine and the foetus to die *in utero*. The reasons for these occurrences and the relation between them are discussed.

Except in the more severe cases, with signs of renal involvement, retinal arteriosclerosis, exudates, or papilloedema, there is no need to terminate pregnancy in the early weeks.

If the pregnancy is allowed to continue treatment should aim at keeping the blood pressure below the danger level of 160 mm. Hg. For this the best agent is rest—if necessary in bed.

With careful supervision most patients with chronic hypertension go to term and deliver themselves spontaneously of living infants.

In those patients in whom the pregnancy is allowed to continue the outlook for a successful pregnancy depends chiefly on the height of the blood pressure at the beginning of pregnancy. If it is over 150/100 only about 33% give birth to viable infants.

The incidence of pre-eclamptic toxaemia is about seven times, and of eclampsia ten times, that in women who are normal at the start of pregnancy.

There is no reason to believe that the hypertension is permanently aggravated by the pregnancy.

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Medical men thinking of starting a practice in the more remote areas of the British Empire will be interested in an article (in French) by Dr. Kalbermatten entitled "The Bush Doctor" which appeared in *Acta Tropica*, 1946, **3**, 130. The author worked for six years as a "company doctor" in the Cameroons and Belgian Congo. He precedes a general account of living conditions by a plea that employers should acquaint prospective medical officers with them more accurately than at present. He believes that, though many companies prefer to employ an unmarried man, a wife and home form a very desirable background for the medical man and help to overcome the solitude that is often his lot. He stresses the need for a good all-round education in medicine, surgery, and obstetrics as well as a knowledge of tropical diseases, since the daily rounds leave little time for reading.