IMMINENT ECLAMPSIA*

THE CLINICAL STATE

C. J. T. CRAIG, M.D., M.R.C.O.G., *Senior Lecturer, Division of Obstetrics and Gynaecology, University of Cape Town and Cape Provincial Administration*

The term eclampsia is derived from the Greek *eklampein* meaning a flash, and its etymology suggests the acute onset of the convulsions. Superficially, therefore, it would appear paradoxical to define any state as being one of 'imminent eclampsia'. However, although the aetiology is unknown, sufficient knowledge of the preceding history and manifestations of eclampsia has accumulated to justify such a specific term.

Where the net of antenatal care is widespread and where such services are accepted and utilized by all who are pregnant in a community, the incidence of severe toxaemia and eclampsia is minimal. Few obstetricians in highly developed, civilized areas are afforded the opportunities for studying and treating any large numbers of cases of imminent eclampsia. In Cape Town, antenatal care is available for all sections of the population. Ignorance, lack of fares for transport and occasionally superstition, are some of the reasons why these facilities are not used, or only occasionally used, by many pregnant women. As a result, severe toxaemia of late pregnancy is relatively frequently seen.

A study of the Clinical Reports of the Department of Obstetrics of the University of Cape Town for the years 1953 - 1959 inclusive,[37-42] showed that in cases of eclampsia many patients have their first convulsion while in hospital (Table I).

TABLE I. ECLAMPSIA IN OBSTETRIC TEACHING UNITS, CAPE TOWN

Year	Cases of eclampsia	First convulsion in hospita	
		No.	%
1953	47	19	40
1954	54	23	43
1955	53	29	55
1956	41	16	39
1957	35	23	66
1958	37	19	51
1959	44	21	48
Total	311	150	48

*Extract from thesis accepted for the degree of Doctor of Medicine of the University of Cape Town.

Table I indicates that approximately half the cases of eclampsia seen over a 7-year period had a first convulsion while in hospital. The majority of these patients presented signs and symptoms of sufficient severity to forewarn of the impending convulsion. A logical conclusion, therefore, is that these manifestations were not correctly assessed or that, if correctly assessed, the treatment administered was inadequate, ineffectual or both.

From the aforegoing the impression may have been gained that the problem of imminent eclampsia as recorded by the number of first convulsions occurring in hospital, was greater in Cape Town than in other centres. Table II shows a similar picture from many other centres throughout the world. Table II was compiled from a random selection of clinical reports of different maternity units and from a personal communication.[7] On the figures as shown in Table II, which are all of fairly recent origin and taken from scattered areas on 3 continents, 73% of patients with eclampsia have their first convulsion while in hospital. The figures from Cape Town and those in Table II, provided a sufficient justification for a

TABLE II. ECLAMPSIA IN OTHER OBSTETRIC UNITS

Country	Hospital	Year	Cases of eclampsia	First convulsion in hospital	
				No.	%
England	Queen Charlotte's[7,29]	1949-1958	41	41	100
England	Princess Mary Maternity[28]	1957	6	2	33·3
New Zealand	Department of Obstetrics, University of Otago[43]	1955	3	1	33·3
England	Liverpool Maternity[17]	1954	2	1	50
Australia	The Royal Women's, Melbourne[35]	1955	26	16	61
Scotland	Royal Infirmary, Edinburgh[34]	1957	9	7	78
Uganda	Makerere College Medical School[18]	1956-1958	17	8	47
England	Kingston Hospital[14]	1958	5	2	40
Ireland	Rotunda[30]	1930	7	7	100
	Total		116	85	73

prospective survey into the clinical state of imminent eclampsia. The survey of 100 cases was conducted over a period of 17 months and lasted from April 1960 to August 1961. The majority of the patients were treated in the Peninsula Maternity Hospital, Cape Town.

THE CLINICAL STATE

It is often impossible on clinical grounds to place a patient into a specific category when hypertension and proteinuria are found at a first visit in the third trimester of pregnancy. Therefore the terms 'imminent eclampsia' and 'severe toxaemia of late pregnancy' will be used to include any one, or a combination of, the following conditions: pre-eclamptic toxaemia, essential hypertension or hypertensive vascular disease, essential hypertension with superimposed pre-eclamptic toxaemia, and chronic nephritis.

No definition watertight to exception is possible, since the convulsions of eclampsia may occur without any warning symptoms and with the minimum of warning signs. Such cases are, however, rare.[2-4,32] It is to be expected, therefore, that the majority, if not all, of the cases of eclampsia will have a common pre-convulsive clinical picture. It is this state which is known as 'imminent eclampsia' or 'severe toxaemia of late pregnancy'.

Symptoms

The symptoms include all or any of the following: Severe headache, usually frontal in type; visual disturbances, including blurred or double vision and spots or flashes before the eyes; vomiting; epigastric pain; oliguria; muscular twitching; irritability; and occasionally jaundice.

The majority of these symptoms are the sequelae of a generalized arteriolar hypertonus and the effects of the latter upon the kidneys, liver and other organs. The occurrence of generalized oedema may also cause some of the symptoms. Symptoms may or may not be present in every case; but when present they are universally accepted as specifically indicating imminent eclampsia.[1,5,6,13,16]

Signs

There is less uniformity concerning the signs that should be accepted as signifying imminent eclampsia or severe toxaemia.

Dieckmann[6] defined a case of severe pre-eclampsia as one in which (*a*) two or more of the following signs are present — repeated systolic blood pressures of 160 mm.Hg or more and diastolic pressures of 100 mm.Hg or more, proteinuria of more than 3·0 G. per 24 hours (3-plus), or marked oedema; (*b*) at least one of the following signs is present — repeated blood pressures of 180/115 mm.Hg or more, proteinuria of 5·0 G. per 24 hours (solid), or generalized oedema; or (*c*) one of the signs listed in (*a*) occurs together with the development of marked cerebral, visual, gastro-intestinal or renal symptoms. The above definition, except for (*c*), is exactly the same as that given by the American Committee on Maternal Welfare as quoted by Pankamaa *et al.*[25]

Kellar[12] considered eclampsia to be imminent where (*a*) the blood pressure is greater than 160/110 mm.Hg; (*b*) oedema is widespread; (*c*) albuminuria, often of a severe degree, is present; (*d*) symptoms of oliguria, headache, visual disturbance, vomiting and epigastric pain, perhaps jaundice, and occasionally muscular twitching, may also be present. The definition of Kellar is the one stated in the Clinical Reports of the Simpson Memorial Pavilion of the Royal Infirmary, Edinburgh.[34] Townsend of Melbourne,[36] like Dieckmann and Kellar, made a special study of hypertension in pregnancy. He believed eclampsia to be likely where the systolic blood pressure is greater than 160 mm.Hg and the urine, after standing for one hour, is half solid with protein.

De Soldenhoff[5] classified as having severe toxaemia those patients who have a maximum blood pressure of 160/100 mm. Hg or more, together with one or more of the following symptoms, if at all marked — headache, epigastric pain, oedema, albuminuria or vomiting. O'Sullivan[24] accepted the same blood pressure, together with an albuminuria of two parts, while Hamilton *et al.*,[10] also accepting the same blood pressure, added that the urine must contain 2 mg. or more of albumin per litre. Molumphy and Garcia[22] have a nearly similar definition of severe toxaemia with (*a*) blood pressure of 160/100 mm.Hg or above; *and* (*b*) albuminuria, 2-plus or more; *and* (*c*) oedema. Gibson[9] of Belfast set an even stricter grading for severe toxaemia, i.e. a blood pressure of 170/105 mm.Hg and/or urine containing 3-plus of albumin. The Helsinki University Women's Clinic[25] had the same definition as the Royal Infirmary, Edinburgh.

Employing the prognosis of the child as a means of defining severe pre-eclampsia, Pankamaa *et al.*[25] concluded that proteinuria of over 5 G. in 24 hours and a blood pressure of over 180 mm.Hg systolic, were significant. Morris[23] emphasized that it is not possible to assess (*a*) cerebral arteriolar vasospasm, (*b*) increased irritability of the motor cortex, or (*c*) cerebral oedema, which are the underlying causes of the convulsions of eclampsia. He felt that no true definition of imminent eclampsia could be stated, but for practical purposes believed that any patient whose diastolic pressure rises to 100 mm.Hg or above, should be treated as having imminent eclampsia. He showed that the higher the diastolic pressure (which reflects vasoconstriction) the greater the incidence of eclampsia.

The views of Morris give the lead to possible assessment of the factors he quotes as being beyond assessment. McCall[20] demonstrated that in all types of toxaemia of late pregnancy, the cerebral vascular resistance is elevated. He measured this resistance and found it to be elevated 38% above normal in pre-eclampsia, 56% in eclampsia and 63% in hypertension with superimposed pre-eclampsia. Despite the increased vascular resistance, the cerebral blood flow remains normal in all types of toxaemia. Although of great theoretical interest, these investigations play no part in the practical management or defining of cases of imminent eclampsia.

EEG studies. In attempting to give a practical guide concerning the patients who may develop eclampsia, many electroencephalogram (EEG) studies have been made on eclamptic patients. Maltby and Rosenbaum[19] were the first to attempt such a study and detected a cerebral dysrhythmia in 13 out of 20 patients with eclampsia compared to 2 out of 20 controls. Gibbs and Reid[8] took tracings from pre-eclamptic and normal pregnant women; 4 out of 8 of those with pre-eclampsia showed abnormal high-voltage fast activity, while this was absent in the normal women. Jost[12] was able to get records of 9 eclamptic patients during and after convulsions. He found that the EEG patterns may become normal when the blood pressure is lowered. In one of his cases, an EEG taken three days before the onset of the convulsions was normal. Parvianinen *et al.*[26] reported on two patients with severe pre-eclampsia and marked cerebral dysrhythmia, who showed normal EEG patterns a few days after treatment with ammonium chloride was begun. Roubillard and Villavicencia,[31] quoted by Kolstad,[15] believed that toxaemia of pregnancy precipitates convulsions in predisposed women, and that such a predisposition can be demonstrated by EEG tracings. McIntosh[21] observed a posteclamptic cerebral dysrhythmia in 5 of 6 patients and stated that persons with dysrhythmia are more prone to eclampsia. James[11] in 1955, and Kolstad[15] in 1961, reviewed most of the above and other relevant literature on the EEG in eclampsia and found that the idea that aclampsia occurs on the basis of an inherited cerebral dysrhythmia was generally accepted. This concept would mean that a definite place existed for the use of EEG studies in patients suffering from severe toxaemia of pregnancy, and that all those with a dysrhythmia 'should be dealt with radically and promptly'. Neither James nor Kolstad, however, could support this view from their own figures. Kolstad was unable to detect a correlation between blood-pressure elevation and EEG patterns, or between the degree of oedema, proteinuria or cerebral

symptoms and EEG tracings. In one of his patients an EEG study done 38 hours before the onset of eclamptic convulsions was within normal limits. It is possible that the previous conclusions on the value of the EEG in eclampsia were based falsely on the post-convulsive tracings which, if followed up, can revert to normal. The work of Jost, of Kolstad and of Poidevin[27] showed that the EEG has no practical value in the selection of patients likely to develop eclampsia from a group who are suffering from severe toxaemia of pregnancy.

IMMINENT ECLAMPSIA

Criteria for Defining Imminent Eclampsia — Cape Town 1960/61

Since I was unable to find a universally accepted standard for defining imminent eclampsia, and accepting that EEG studies would be of little clinical value in helping to arrive at such a standard, I decided to make my own standards, as follows:

1. A blood pressure of 160/110 mm.Hg or more, *and* urine containing 2-plus of albumin or more; or

2. Either of the above two signs with symptoms of severe headache, visual disturbances, vomiting or epigastric pain.

Of the 100 patients in this survey, 82 fall into the first category and 18 into the second.

Incidence of Imminent Eclampsia

The varying incidences reported are mainly the result of the different standards of definition used. Retrospective surveys culled from hospital clinical reports appear to give higher incidences than prospective surveys.

O'Sullivan [24] found the incidence of imminent eclampsia to be 6·3% of 2,062 cases of pre-eclamptic toxaemia. Kellar,[13] from Edinburgh, reported only 70 cases in 7 years. The medical and clinical report of the Simpson Memorial Pavilion, Royal Infirmary,[33] Edinburgh, for the year 1960 noted 12% of severe cases in 337 patients suffering from pre-eclampsia. Molumphy and Garcia[22] reported 61 cases in 2 years at the Baltimore City Hospital. De Soldenhoff[5] (Scotland) found 352 cases among 477 with toxaemia. Townsend[36] (Melbourne) made the incidence 40·1% of 425 cases of pre-eclamptic toxaemia. The incidence for the survey reported here, which lasted 17 months, was 13·6% of 711 patients with toxaemia of late pregnancy.

The Survey of Imminent Eclampsia

The present survey covers the first 100 patients with severe toxaemia treated with 'avertin' by the staff of the Peninsula Maternity Hospital. Three of these patients, after treatment with avertin by the Flying Squad, were admitted to other institutions. Since their further management did not differ from the routine at the Peninsula Maternity Hospital, they have been included in this survey so as to obtain a consecutive series. The criteria for deciding that a particular patient was in a state of imminent eclampsia were strictly adhered to at all times. In each patient the blood-pressure reading was confirmed at least once and usually by a second observer as well. In all antepartum and intrapartum cases the foetal heart was heard before the commencement of treatment. Very occasionally patients who fulfilled the criteria for a diagnosis of imminent eclampsia were already in the second stage of labour. Such patients were not given avertin, but were submitted to general anaesthesia, and labour was completed by means of a forceps delivery. As far as possible, I believe that no bias existed in the selection of these 100 cases of imminent eclampsia.

RESULTS

There were 10 White and 90 non-White patients. There were 55 non-booked and 45 booked admissions.

Age

The age distribution of the patients in this survey is shown in Table III. The mean age for all patients was 26·3 years,

TABLE III. AGE DISTRIBUTION IN IMMINENT ECLAMPSIA

Age (years)	*Primigravidae*		*Multigravidae*		*All patients*
	No.	*%*	*No.*	*%*	*No. and %*
15–19 ..	14	34	3	5	17
20–24 ..	18	44	9	15	27
25–29 ..	6	14·5	11	19·3	17
30–34 ..	2	5·0	16	27	18
35–39 ..	1	2·5	13	22	14
40–44 ..	—	—	6	10	6
45–49 ..	—	—	1	1·7	1
50–54 ..	—	—	—	—	—
Total ..	41	100	59	100	100

the mean age for primigravidae was 21·7 years, and the mean age for multigravidae was 31·0 years.

The youngest patient was aged 15 and the oldest 47.

Gravidity

Table IV classifies certain basic facts concerning primigravidae and multigravidae.

TABLE IV. GRAVIDITY IN IMMINENT ECLAMPSIA

	Primigravidae	*Multigravidae*
Number ..	41	59
Booked ..	27	18
Non-booked ..	14	41
Mean age (yrs.)	21·7	31·0
Infants delivered	41	60
Stillbirths ..	1	4
Neonatal deaths	5	7

TABLE V. NUMBER OF PREVIOUS PREGNANCIES IN MULTIGRAVIDAE WITH IMMINENT ECLAMPSIA

*Number of previous pregnancies**	*Number of patients*
1	15
2	7
3	4
4	7
5	6
6	6
7	1
8	2
9	3
10	6
11	1
12	1

* Average number of previous pregnancies = 4·7.

Table V shows the numbers of previous pregnancies among the multigravidae; the mean is 4·7 previous pregnancies.

Mode of Admission

There were 27 booked primigravidae. One of these patients was admitted *via* the Flying Squad. She developed a severe headache when 31 weeks pregnant and was seen by her general practitioner who found her in a state of imminent eclampsia.

There were 14 non-booked primigravidae of whom one was a Flying Squad admission. This patient was fetched 75 miles from a rural hospital.

There were 18 booked multigravidae of whom one was a Flying Squad admission. This patient had failed, despite letters, to attend the antenatal clinic for 2 months.

There were 41 non-booked multigravidae of whom 15 were admitted *via* the Flying Squad. Altogether there were 18 admissions *via* the Flying Squad.

Maturity

Since all the patients were given avertin as soon as they fulfilled the criteria for the diagnosis, Table VI shows that at least 95% of these patients were admitted with a diagnosis, or near diagnosis, of 'imminent eclampsia'. The period of

gestation at the time of the first avertin administration, therefore, probably reflects the duration of pregnancy at which imminent eclampsia is most likely to develop. It was not always possible to obtain an accurate date for the last menstrual period from each patient. By perusal of antenatal records where available, and by considering other factors such as quickening and foetal size, a reasonably true estimate of the period of gestation was obtained for all cases (Table VII). This Table shows that there is little difference in the stages

TABLE VI. NUMBER OF DAYS IN HOSPITAL BEFORE ADMINISTRATION OF AVERTIN (PRIMIGRAVIDAE AND MULTIGRAVIDAE)

Days in hospital	*Number of patients*
0	83
1	9
2	3
4	1
5	1
18	2
21	1
	100

TABLE VII. DURATION OF PREGNANCY AND THE DEVELOPMENT OF IMMINENT ECLAMPSIA

Duration of pregnancy (weeks)	*Primigravidae*	*Multigravidae*	*All patients*	*Booked cases*
26	—	1	1	—
27	—	—	—	—
28	—	—	—	—
29	1	2	3	—
30	1	4	5	—
31	2	1	3	1
32	3	2	5	1
33	2	7	9	2
34	4	4	8	2
35	1	2	3	2
36	3	5	8	3
37	6	11	17	9
38	5	8	13	6
39	6	2	8	7
40	4	5	9	8
41	2	4	6	3
42	1	1	2	1
Total	41	59	100	45

of pregnancy when imminent eclampsia develops in primigravidae and multigravidae. Altogether 34% of primigravidae and 39% of multigravidae were less than 36 weeks pregnant. Among the 45 booked patients, only 18% were less than 36 weeks pregnant, while 67% of the 55 non-booked patients were less than 36 weeks pregnant. It was felt that these facts, indicating prematurity, would affect the foetal loss. This is clearly indicated in Table VIII, which shows that 70·6% of the

TABLE VIII. FOETAL LOSS IN RELATION TO THE DURATION OF PREGNANCY AT WHICH IMMINENT ECLAMPSIA DEVELOPS

	Foetal loss	*Duration of pregnancy (weeks)*	*Booked or non-booked*
1	NND	26	NB
2	*SB	29	NB
3	SB	29	NB
4	NND	29	NB
5	NND	30	NB
6	*NND	31	B
7	*NND	32	NB
8	SB	33	NB
9	*NND	33	NB
10	NND	33	NB
11	NND	34	NB
12	SB	35	B
13	*NND	36	B
14	*NND	40	B
15	SB	40	B
16	NND	41	NB
17	NND	41	NB

* Primigravidae.
NND = neonatal death, SB = stillbirth.

total foetal loss occurred in cases where the diagnosis of imminent eclampsia was made before the 36th week of pregnancy. A 100% foetal loss occurred in the 4 cases diagnosed before the 30th week of gestation.

Tables VII and VIII show that there was a 33·4% foetal loss among 37 patients diagnosed before the 36th week of pregnancy, but only a 7·9% foetal loss among 63 patients where the diagnosis was made when the patient was 36 or more weeks pregnant. Prematurity is undoubtedly the major cause of foetal loss in patients with imminent eclampsia. The standard of antenatal care is an important factor in allowing the development of imminent eclampsia — especially the early development before the 36th week of pregnancy.

Symptoms

Symptoms were present in 86% of patients. Table IX indicates the variety and frequency of the persisting symptoms.

Severe persistent headache was the most frequent complaint and was present in 81 patients. Usually frontal in type, it was often not relieved by common household analgesics. Of the 19 patients who were headache-free, 14 were completely symptom-free. The remaining 5 patients complained respectively of (*i*) antepartum haemorrhage — 2; (*ii*) extreme irritability — 3, associated with fainting and

TABLE IX. THE NATURE AND FREQUENCY OF THE SYMPTOMS SEEN IN 100 CASES OF IMMINENT ECLAMPSIA

Symptom	*Number of patients*
Severe headache	81
Visual disturbance	18
Extreme irritability	15
Epigastric pain	11
Antepartum haemorrhage	10
Vomiting	8
Dyspnoea	5
Vertigo	2
Fainting	2
Congestive cardiac failure	2
Oliguria	1

TABLE X. THE NUMBER OF PRESENTING SYMPTOMS PER PATIENT

Number of symptoms	*Number of patients*
0	14
1	37
2	31
3	16
4	2
	100

epigastric pain in 1 case each. Many patients had more than 1 symptom (Table X). The maximum number of presenting symptoms in any one patient was 4, although no less than 11 different symptoms were recorded.

In discussing the selection of criteria for diagnosing imminent eclampsia, I expected that symptoms would be more prevalent among the primigravidae and so ensure their rightful inclusion for special treatment. I reasoned that primigravidae developing a raised blood pressure for the first time would be more sensitive to its effects than multigravidae, who, in many cases, would have underlying hypertensive vascular disease and so would be accustomed to the effects of hypertension. To determine whether this was in fact true, the 14 patients in whom there were no presenting symptoms of imminent eclampsia were carefully assessed (Table XI).

The findings in Table XI were totally unexpected to me. No less than 11 of the 41 primigravidae had asympto-

TABLE XI. DATA CONCERNING PATIENTS WITH ASYMPTOMATIC IMMINENT ECLAMPSIA

	Patient	*Age (years)*	*Blood pressure (mm.Hg)*	*Albuminuria*	*Oedema*
Primigravidae:					
	1	20	230/130	Solid	Gross
	2	19	180/140	++	Moderate
	3	20	170/120	++	Moderate
	4	17	170/130	++	Gross
	5	27	180/130	+++	Gross
	6	17	160/110	Solid	Gross
	7	18	160/115	+++	Nil
	8	21	160/110	Solid	Gross
	9	25	165/110	+++	Nil
	10	25	215/130	Solid	Slight
	11	25	185/135	Solid	Moderate
Multigravidae:					
	1	41	230/130	+++	Slight
	2	40	200/130	+++	Slight
	3	27	210/120	Solid	Moderate

Mean age = 24·4 years, primigravidae = 21·3 years, multigravidae = 36 years.
Mean blood pressure = 187/124 mm.Hg.

matic imminent eclampsia, whereas only 3 of the 59 multigravidae were asymptomatic. The mean age of these 14 patients was 24·4 years, compared with a mean age of 26·3 years for the whole series. The mean blood pressure of these 14 patients was 187/124 mm.Hg compared with a mean of 195/126 mm.Hg for the whole series. It is obvious that symptoms in cases of imminent eclampsia are much less frequently seen in primigravidae. Asymptomatic primigravidae do not differ from those with symptoms of imminent eclampsia with regard to age or presenting blood pressure. In this series of 100 cases of imminent eclampsia, 3 patients developed convulsions. All had presented with symptoms and 2 were primigravidae. Thus of the 30 primigravidae with presenting symptoms, 2, or 1 in 15, developed eclampsia. Although the numbers are few (it is difficult to obtain large numbers in this type of work) I believe that the appearance of symptoms in primigravidae who have the other criteria of imminent eclampsia, indicates an even greater likelihood of convulsions. Put in another way — to delay treatment in severe cases of toxaemia in primigravidae because they are asymptomatic is not justified, because the onset of symptoms increases the likelihood of convulsions.

Foetal loss was assessed against the presenting maternal symptoms (Table XII). Among the 41 primigravidae in this series there was a foetal loss of 6. Four of these 6 infants were born to mothers who were asymptomatic. From the foetal viewpoint it also appears unwise to delay delivery in an asymptomatic primigravida with imminent eclampsia. Symptoms were present in all multigravidae who suffered foetal loss. Since only 3 out of 59 were asymptomatic this is not an unexpected finding.

TABLE XII. FOETAL LOSS IN RELATION TO THE PRESENTING MATERNAL SYMPTOMS

	Foetal loss	*Maternal symptoms*
Primigravidae:		
	SB	Headache, visual disturbance, epigastric pain
	NND	Headache
	NND	Nil
	NND	Nil
	NND	Nil
	NND	Nil
Multigravidae:		
	SB	Headache, dyspnoea, antepartum haemorrhage, congestive cardiac failure
	SB	Headache, epigastric pain
	SB	Headache, visual disturbance, epigastric pain
	SB	Headache
	NND	Headache, small antepartum haemorrhage
	NND	Antepartum haemorrhage
	NND	Headache, epigastric pain, vomiting
	NND	Headache
	NND	Headache
	NND	Headache
	NND	Headache

The absence of symptoms in cases of imminent eclampsia, particularly in primigravidae (? young patients) does not justify a less immediate or a less intensive form of management.

Blood Pressure

The overall mean presenting blood pressure in this survey was 195/126 mm.Hg. For primigravidae the mean was 182/123 mm.Hg and for multigravidae the mean was 204/128 mm.Hg. Apart from Molumphy and Garcia,[22] who in their series found the mean blood pressure for primigravidae to be 170/115 mm.Hg and for multigravidae 190/120 mm.Hg, comparative blood pressures were not available. The Cape Town survey figures show that the minimal blood pressure for diagnosing imminent eclampsia as defined by me, was clearly surpassed in most cases. Only 2 patients had a blood pressure lower than the standard of 160/110 mm.Hg. The lowest blood pressure of any primigravida in this series was 150/105 mm.Hg, while the highest was either 230/130 or 205/155 mm.Hg. These latter 2 patients were aged 20 and 18 years respectively. The lowest blood pressure among multigravidae was 160/100 and the highest either 290/160 or 260/190 mm.Hg.

The foetal loss in relation to the presenting maternal blood pressure is shown in Table XIII. From among the 6 primigravidae with systolic blood pressures of 210 mm.Hg or more, 2 infants were lost. From among the 5 multigravidae with systolic blood pressures of 260 mm.Hg or more, 3 infants were lost. Six multigravidae had diastolic blood pressures of 160 mm.Hg or more, and there was a foetal loss of 3 from this group. Although the mean maternal blood pressure associated with infant loss differs little from the overall mean maternal blood pressure in this series, there is definitely an increased foetal loss in patients with exceptionally high systolic blood pressures and in multigravid patients with exceptionally high diastolic blood pressures.

TABLE XIII. FOETAL LOSS RELATED TO THE PRESENTING MATERNAL BLOOD PRESSURE

Foetal loss	*Maternal blood pressure (mm.Hg)*
Primigravidae:*	
SB	210/130
NND	185/135
NND	170/120
NND	220/110
NND	180/130
NND	160/110
* Mean = 187/122 mm.Hg	
Multigravidae:**	
SB	190/120
SB	175/105
SB	210/160
SB	180/130
NND	260/190
NND	260/130
NND	160/125
NND	180/120
NND	170/120
NND	260/160
NND	185/115
**Mean = 203/134 mm.Hg	

SB = stillbirth, NND = neonatal death.

The presenting blood pressures of the 3 patients who subsequently developed convulsions were 200/140 and 170/125 in the primigravidae, and 160/120 mm.Hg in the multigravidae.

Albuminuria

Table XIV indicates that only 17 of the 100 patients in the survey had less than a 2-plus albuminuria when first treated with avertin. At the opposite end of the scale, 44% of patients presented with a solid albuminuria. Since it is commonly believed that the duration of the albuminuria affects the foetal prognosis, Table XV was drawn up to show the foetal loss in relation to both the amount of and the known duration of the albuminuria. Of the total foetal loss, 82·3% occurred in patients with a 3-plus or solid albuminuria, whereas only 64% of the total patients had this degree of albuminuria. In primigravidae especially, the foetal prognosis bears a direct

TABLE XIV. THE PRESENTING ALBUMINURIA IN IMMINENT ECLAMPSIA

Albuminuria	*Primigravidae*		*Multigravidae*		*All patients*
	No.	*%*	*No.*	*%*	*No. and %*
Nil	2	4·9	—	—	2
Trace	1	2·5	2	3·4	3
+	4	9·8	8	13·6	12
++	8	19·6	11	18·7	19
+++	10	24·7	10	17·0	20
Solid	16	38·5	28	47·3	44
	41	100	59	100	100

TABLE XV. FOETAL LOSS IN RELATION TO THE PRESENTING MATERNAL ALBUMINURIA

Foetal loss	*Presenting albuminuria*	*Known duration of albuminuria*
	Primigravidae:	
SB	+++	2 weeks
NND	Solid	Nil before admission
NND	Solid	1 week
NND	+++	4 days
NND	Solid	Nil before admission
NND	Solid	2 weeks
	Multigravidae:	
SB	Solid	Unknown
SB	Solid	7 weeks
SB	Solid	Nil before admission
SB	+	2 weeks
NND	Solid	Unknown
NND	+++	Unknown
NND	Solid	Unknown
NND	Solid	Unknown
NND	Solid	1 week
NND	Trace	Unknown
NND	++	Unknown

SB = stillbirth, NND = neonatal death.

relationship to the degree of albuminuria present. The duration of the albuminuria also probably affects the foetal prognosis, although the available figures are too small to be significant. Albuminuria was known to have been present for 7 weeks in one patient delivered of a stillborn infant. Apart from the 7 in Table XV, there were another 16 patients with albuminuria present at an antenatal examination. In 4 of these 16 patients the albuminuria was present for more than 1 week, as was the case with 4 of the 7 patients in Table XV. Among these 8 patients with albuminuria of more than one week's standing there was a foetal loss of 4.

Table XIV shows that 17·2% of the primigravidae and 17·0% of the multigravidae had less than a 2-plus albuminuria. Table XVI shows the age groups of patients with solid albuminuria compared with the overall age groups, and that these age groups correspond. It was expected that some older multigravid patients with underlying hypertensive vascular disease and very mild superimposed pre-eclampsia would be included in this survey, but in fact such patients, if included, did not present as a group, an indication that the standards set for definition are satisfactory.

TABLE XVI. AGES OF PATIENTS WITH SOLID ALBUMINURIA COMPARED TO OVERALL AGES

Age (years)	*Solid albuminuria group*		*Total patients*
	No.	*%*	*No. and %*
15–19	5	11·3	17
20–24	13	29·7	27
25–29	10	22·7	17
30–34	8	18·2	18
35–39	5	11·3	14
40–44	2	4·5	6
45–49	1	2·3	1
Total	44	100·0	100

Oedema

The extent of the oedema detected in each patient is shown in Table XVII. Of the patients, 74% had moderate or gross oedema, while only 6% did not have any at all. There was no obvious difference in the extent of oedema detected in primigravidae and multigravidae. Table XVIII shows that the mothers whose infants died all had some degree of oedema — 88% having moderate or gross oedema.

TABLE XVII. DEGREE OF PRESENTING OEDEMA

Degree of oedema	*Primigravidae*		*Multigravidae*		*All patients*
	No.	*%*	*No.*	*%*	*No. and %*
Nil	3	7·4	3	5·1	6
Slight	9	22·2	11	18·8	20
Moderate	14	33·9	21	35·6	35
Gross	15	36·5	24	40·5	39
Total	41	100·0	59	100·0	100

TABLE XVIII. FOETAL LOSS IN RELATION TO THE PRESENTING MATERNAL OEDEMA

Foetal loss	*Presenting oedema*
Primigravidae:	
SB	Moderate
NND	Moderate
NND	Gross
NND	Gross
NND	Gross
NND	Gross
Multigravidae:	
SB	Moderate*
SB	Moderate
SB	Moderate
SB	Gross
NND	Slight
NND	Slight
NND	Moderate
NND	Gross
NND	Gross
NND	Gross
NND	Gross

* Received diuretics before admission.
SB = stillbirth, NND = neonatal death.

CONCLUSION

Imminent eclampsia is a clinical state in which the signs and symptoms of pre-eclamptic toxaemia develop to a degree sufficient to warrant special treatment to prevent the occurrence of convulsions. Retrospective diagnosis of such states are useful in providing criteria for the selection of patients, but add little to the assessment of the different drugs available for treatment. Since treatment is always urgent and is in the first instance taken to the patient, the diagnosis is made immediately on the presenting clinical symptoms and signs alone.

The incidence of imminent eclampsia is related to the standard of the available antenatal care in a given area. Where meticulous attention is paid throughout pregnancy to weight gain and changes in blood pressure, the number of cases of imminent eclampsia that develop is minimal. In Cape Town the relative frequency of the condition stems from the fact that many patients make no attempt to receive antenatal care, and secondly that the standard of antenatal care often falls short of basic, let alone superior requirements. A reassessment of the objectives of antenatal care with the accent on the maximum rather than the minimum that should be done for patients appears to be urgently needed in the services available in Cape Town. This applies equally to antenatal services administered by hospitals, municipal and other clinics, and to general practitioners and midwives.

Imminent eclampsia is a preventable state and thoughtful, thorough antenatal care would largely eliminate the need for a special scheme of management. Prevention is undoubtedly better than the most carefully planned and conducted 'cure'.

SUMMARY

1. The clinical state of imminent eclampsia is defined.

2. The need for recognizing and adequately treating this condition is stressed.

3. An analysis of the symptoms and physical findings in 100 cases is presented.

4. The influence of certain factors on the foetal prognosis is shown.

I am indebted to Prof. James T. Louw, Head of the Department of Obstetrics and Gynaecology, University of Cape Town, for helpful advice and criticism, and to Dr. A. W. Falconer, Superintendent, Peninsula Maternity Hospital, for permission to publish.

REFERENCES

1. Assali, N. S. (1958): *Clinical Obstetrics and Gynecology*, p. 381. New York: Hoeber and Harper.
2. Browne, F. J. and Browne, J. C. McC. (1955): *Antenatal and Postnatal Care*, p. 374. London: Churchill.
3. Clayton, S. G. and Oram, S. (1951): *Medical Disorders in Pregnancy*, p. 91. London: Churchill.
4. Craig, C. J. T. (1962): S.Afr. Med. J., **36,** 83.
5. de Soldenhoff, R. (1955): J. Obstet. Gynaec. Brit. Emp., **62,** 703.
6. Dieckmann, W. J. (1952): *The Toxaemias of Pregnancy*, p. 483. St. Louis: C. V. Mosby Co.
7. Friedman, S. (1961): Personal communication.
8. Gibbs, F. A. and Reid, D. A. (1942): Amer. J. Obstet. Gynec., **44,** 672.
9. Gibson, G. B. (1954): J. Obstet. Gynaec. Brit. Emp., **61,** 602.
10. Hamilton, J., Jeffcoate, T. N. A. and Lister, U. M. (1949): *Ibid.*, **56,** 413.
11. James, J. R. E (1955): *Ibid.*, **62,** 704.
12. Jost, H. (1948): Amer. J. Med. Sci., **216,** 57.
13. Kellar, R. J. (1955): J. Obstet. Gynaec. Brit. Emp., **62,** 683.
14. Kingston Hospital (1958): *Obstetric Report*.
15. Kolstad, P. (1961): Acta obstet. gynec. scand., **40,** 139.
16. Levit, M. F. and Altchek, A. (1960): *Medical, Surgical and Gynaecological Complications of Pregnancy*, p. 84. Baltimore: Williams and Wilkins.
17. Liverpool Maternity Hospital (1954): *Report*.
18. Makerere College (1956/57/58): *Obstetric Report*.
19. Maltby, G. L. and Rosenbaum, M. (1942): Proc. Soc. Exp. Biol. (N.Y.), **50,** 10.
20. McCall, M. L. (1949): Surg. Gynec. Obstet., **89,** 715.
21. McIntosh, R. R. (1952): J. Obstet. Gynaec. Brit. Emp., **59,** 197.
22. Molumphy, P. E. and Garcia, R. (1959): Obstet. and Gynec., **14,** 193.
23. Morris, N. (1955): J. Obstet. Gynaec. Brit. Emp., **62,** 696.
24. O'Sullivan, J. V. (1955): *Ibid.*, **62,** 704.
25. Pankamaa, P., Jarvinen, P. A. and Kinnunen, O. (1957): *Ibid.*, **64,** 857.
26. Parvianinen, S., Temmes, Y. and Soiva, K. (1950): *Ibid.*, **57,** 780.
27. Poidevin, L. O. S. (1955): *Ibid.*, **62,** 417.
28. Princess Mary Maternity Hospital, Newcastle upon Tyne (1957): *Report*.
29. Queen Charlotte's Hospital Staff (1960): *Queen Charlotte's Textbook of Midwifery*, 10th ed., p. 136. London: Churchill.
30. Rotunda Hospital (1959): *Report*.
31. Roubillard, G. J. and Villavicencio, C. (1950): Obstet. Ginec. lat.-amer., **8,** 422. (Op. cit.[16]).
32. Salerno, L. J. (1960): Clin. Obstet. Gynec., **3,** 53.
33. Simpson Memorial Maternity Pavilion, Edinburgh (1960): *Report*. p. 96.
34. *Idem* (1957): *Ibid.*
35. The Royal Women's Hospital, Melbourne (1955): *Report*.
36. Townsend, S. L. (1955): J. Obstet. Gynaec. Brit. Emp., **62,** 692.
37. University of Cape Town (1953): *Obstetric Report*.
38. *Idem* (1954): *Ibid.*
39. *Idem* (1955): *Ibid.*
40. *Idem* (1956): *Ibid.*
41. *Idem* (1957): *Ibid.*
42. *Idem* (1958/59): *Ibid.*
43. University of Otago (1955): *Obstetric Report*.