(b) Various, including tuberculous cystitis (fatal) and acute rheumatism.

Maternal morbidity—that is, any case which shows a temperature of over 100° F.:

Septicaemia, 2.	Emotional, 6.
Sapraemia, 19.	Urinary, 3.
Gastro-intestinal, 19.	Pulmonary, 3.
Mammary, 22.	Various, 8.
Reactionary, 22.	

There were 7 maternal deaths during the year. Of these, 3 were in cases of eclampsia (see table); 1 in a case of placenta praevia from septicaemia (see table); 1 in a case of tuberculous cystitis and pyelitis; 1 in a case of mitral stenosis, already referred to; 1 in a case of acute pneumonia. Labour was induced, but the patient died shortly afterwards.

THE SIGNIFICANCE OF ALBUMINURIA IN PREGNANCY.*

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AFTER reading the paper by Dr. Davies-Colley, published in the BRITISH MEDICAL JOURNAL of June 17th, 1911, on the association of toxæmia of pregnancy with hæmorrhage, I thought that he and other readers of the JOURNAL might be interested in the following statistics of a somewhat similar nature which I have collected from the records of the above hospital for five years. Unlike Dr. Davies-Colley, I find a marked increase in the cases of *post-partum* hæmorrhage, not necessarily in eclampsia, but in cases of albuminuria.

When I was making up my first annual report on St. Helen's Hospital in 1906, my attention was directed to the fact that a large proportion of the various abnormalities and complications met with occurred in women who also showed albumen in the urine—for example, hæmorrhages, adherent placenta, placenta praevia, and stillbirths, and in publishing the report in the *New Zealand Medical Journal* (October, 1906) I commented on this fact. Since then I have given particular attention to the subject, and now that I have statistics of five years I thought it might be of interest to bring before you the facts which I have collected.

The one possible danger which the obstetrician associates with albuminuria is eclampsia. Well, out of 289 cases of albuminuria which have passed through St. Helen's Hospital, we have had only 5 cases of eclampsia, and yet in many the albuminuria was accompanied by severe toxic symptoms and marked oedema.

Eclampsia is, therefore, not a frequent accompaniment of albuminuria, although I am inclined to attribute our escape from it in many cases to the prophylactic treatment extended to them beforehand. As soon as the patient comes to the hospital to book, which may be in the early months, or not till the seventh or eighth month, a specimen of urine is examined, and if it contains albumen, or if she suffers from toxic symptoms, she is given directions with regard to her diet and the care of the bowels and skin. She is told to report herself from time to time at the hospital, the urine being examined on each occasion, and toxic symptoms inquired for. If these supervene to any extent, she is admitted for perhaps a week's treatment, during which improvement usually occurs. I have mentioned eclampsia merely to show that it is not one of the frequent accompaniments of albuminuria.

I have purposely refrained from calling these albumen cases nephritis. Many of them were so, the kidneys being organically diseased, but in many the albumen was transient, passing away during the puerperium. We examine the urine in all the albumen cases on the day the patient leaves the hospital. We can thus note which are the transient cases, and in which cases the albumen is still present.

The proportion of pregnancies in which albumen is present is a very large one. Out of 1,127 cases which have been under our care, indoor and outdoor, there were 289 albumens—that is, 25.6 per cent.

I have worked out a comparison between the normal

*Read at a meeting of the Otago Branch of the British Medical Association.

cases (that is, no albumen) and the albumen cases on the following points, and I find all the complications more numerous in the albumen cases:

1. History of previous labours.
2. Placenta praevia or low implantation.
3. *Ante-partum* hæmorrhage.
4. *Post-partum* hæmorrhage.
5. Mental symptoms.
6. Phlegmasia alba dolens.
7. Adherent membranes or placenta.
8. Morbidity.
9. Premature births.
10. Stillbirths and deaths of infants.
11. Inability to nurse, complete or partial.

History of Previous Labours.

I may say that I cannot guarantee perfect accuracy in numbers with regard to these previous histories, as some nurses, in writing up the charts, would get a much more accurate history of previous labours than others who were less thorough in questioning the patients.

Miscarriages.

		Normal (838).		Albumen (289).	
		No.	Per Cent.	No.	Per Cent.
1 Miscarriage	...	57	6.7	37	12.1
2 Miscarriages	...	12	1.4	13	4.8
3	...	3	0.35	3	1.04
4	...	1	0.12	1	0.34
5	...	0	0	4	1.36
6	...	1	0.12	1	0.34

Stillbirths.

In one of the albumen cases the whole five pregnancies resulted in premature stillbirths, and in one there were six miscarriages, one stillbirth at full term, and two which died in a few hours, representing a family of nine children lost.

		Normal.		Albumen.	
		No.	Per Cent.	No.	Per Cent.
Stillbirths	...	3	0.35	14	4.9
Died within a month	...	3	0.35	7	2.45
Phlegmasia	...	2	0.24	4	1.36
Eclampsia	...	0	0	11	3.8

It will thus be seen that, so far as previous histories go, the number of miscarriages in albumen cases is greatly in advance of those in normal cases, being nearly double for those with one miscarriage, treble for two, three, and four miscarriages, also increased for five and six miscarriages. In one of the normal cases there were six previous miscarriages and one stillbirth out of nine, but this case was distinctly syphilitic. There is a marked difference in the stillbirths at full time; the percentage was 0.35 for normal and 4.8 for albumen cases. Similarly, in those which died within a month there is a preponderance among the albumen cases.

Complications.

Among the complications of previous labours not many were inquired for, but phlegmasia was noted in two of the normal and four of the albumen cases; eclampsia in none of the normal and in eleven of the albumen cases.

Histories of Present Labours.

Coming now to present labours—in these the histories are quite accurate.

"Adherent Placenta or Membranes" including Total or Partial Adhesion of Placenta or Membranes.

		Normal (838).		Albumen (289).	
		No.	Per Cent.	No.	Per Cent.
Adherent placenta	...	28	3.3	44	15.2
"Placenta praevia or low implantation"	...	11	1.3	23	8

In many of the albumen cases the placenta was degenerate in parts. Some of these cases had free *ante-partum* hæmorrhage, some no hæmorrhage, the condition being only diagnosed after the birth of the placenta, when the latter was found

attached to the margin of the opening in the membranes through which the child had passed, the membranes being complete.

Ante-partum Haemorrhage—either accidental or unavoidable:

	No.	Per Cent.
Normal ...	11	1.3
Albumen ...	20	6.9

Many of these were only slight, but the preponderance in the albumen cases is marked.

Threatened Post-partum Haemorrhage.—Threatening post-partum haemorrhage—that is, cases in which the discharge was so profuse that special measures were required to overcome it and a nurse told off to watch for some hours for a possible return of it; in these I have not included cases due to adherent placenta:

	No.	Per Cent.
Normal ...	25	2.9
Albumen ...	26	9

Mental Symptoms, including Violent Hysteria, Melancholia, and Mania:

	No.	Per Cent.
Normal ...	1	0.12
Albumen ...	9	3.1

Morbidity (taking a temperature of 100° and over lasting for at least two consecutive days):

	No.	Per Cent.
Normal ...	53	6.2
Albumen ...	55	19

The total number of cases in which there was inability to nurse was 225. Of these, 83, or 36.8 per cent., had albumen. Of the rare complications, such as phlegmasia, anencephalus, and vesicular mole, we had one of each, and in each case there was albuminuria. Of the complications, which are usually associated with albumen—namely, toxæmia, oedema, and eclampsia—we had 29 with toxæmia, 56 oedema, and 5 eclampsia; all had albumen.

	Normal.		Albumen.	
	No.	Per Cent.	No.	Per Cent.
Premature births ...	4	0.47	30	10.3
Stillbirths ...	15	1.79	21	7.2
Died before leaving hospital	5	0.39	9	3.1

The history of the stillbirths, and the deaths shortly after birth, are particularly instructive. I found that in the normal cases there was a much larger proportion of what one might call unavoidable deaths—for example, due to impacted breech presentations in primiparae, a large child in a small pelvis, accidents to the mother shortly beforehand, and cases of prolapsed cord; whereas in the albumen cases these causes were less numerous, and the purely toxic causes more numerous.

Normal Cases (that is, those with no albumen).

1. Thirteenth pregnancy; previous 12 normal. Had worked sewing machine up to the last, and after one particularly heavy day she felt no more movements. The child was born dead, skin peeling.
 2. Fifth pregnancy. Had a fright and heavy fall a week previously, after which no more movements were felt; child macerated.
 3. Ninth pregnancy; induction at eighth month for contracted pelvis; had had eight previous craniotomies. This time the cord was prolapsed and the child was stillborn.
 4. Induction at eighth month for contracted pelvis; breech presentation; difficulty with birth of head; child stillborn.
 5. Primipara; contracted pelvis, head would not engage, high forceps required; child stillborn.
 - 6, 7. Presentation of cord.
 8. Second pregnancy; premature at six and a half months, the first pregnancy ending at five months; syphilis.
 - 9, 10. Incomplete closure of foramen ovale; died on the third and sixteenth days respectively.
 11. Her sixth pregnancy; premature at seventh month, no definite cause; mother healthy.
 12. A very large child, born asphyxiated; required long artificial respiration, but died in convulsions in twenty-eight hours.
 13. Sixth pregnancy; had severe ante-partum accidental haemorrhage; child was weakly, and died on the second day.
 - 14, 15, 16. These were admitted with haemorrhage from placenta praevia, one at sixth month; the other two became collapsed; internal version was rapidly performed, but the child was dead in each case.
- In the remaining four the mothers were weakly and anaemic, evidently preventing full nourishment of the child; in two they were macerated, in one the placenta was degenerate; and in the fourth it was her fifteenth pregnancy; the baby was

pinched and ill-developed, and died in two days with haemorrhage from mouth and rectum.

The last four, therefore, are the only ones in which the death of the child could be attributed to the poor health of the mother, unless we include the three cases of placenta praevia, which may indirectly have been due to the mother's health.

Albumen Cases.

A total of 30 cases of stillbirths or early death, 24 of these being due to ill health in the mother, and only 6 due to such causes as placenta praevia, prolapsed cord, or difficult birth (in a small pelvis):

- 1 and 2. Prolapsed cord.
3. Primipara, breech presentation, with impacted head and arms.
4. Large child in small pelvis.
5. Large child; required two and a quarter hours artificial respiration, but died in fourteen hours.
6. Central placenta praevia, severe haemorrhage; internal version performed, but the child died.

The remaining twenty-four had had histories from a health point of view; many showed symptoms of a severe toxæmia; several were cases of twins born prematurely, and one of them dead, sometimes macerated.

I may here say that in the healthy mothers, where twins were born, they both lived and were healthy, usually going to full term.

It is interesting to observe the different ways in which the toxæmia showed itself in the symptoms both of the mother and child.

Symptoms of Toxæmia.

1. Thirteenth pregnancy; heavy albumen and oedema; premature at eighth month. Her eleventh had died soon after birth, and her twelfth was stillborn at the seventh month; the previous ones healthy.
2. First pregnancy; heavy albumen. Sent in as urgent case from Middlemarch for excessive vomiting and jaundice; premature at eighth month; macerated.
3. First pregnancy, premature, eight and a half months, macerated; slight eclampsia on the following day with twitchings of face muscles, severe headaches, and swelling, with purple discoloration of face.
4. Tenth pregnancy; heavy albumen and oedema, stillborn; the second pregnancy was the only child living; there had been four premature births—two stillbirths at full term, one died after three hours and one after twelve hours.
- 5, 6. First pregnancies; heavy albumen, severe toxic symptoms, one full term, one premature.
7. Third pregnancy; premature; baby collapsed and died in two days from haemorrhage from the stomach.
8. Second pregnancy; severe vomiting and headaches; history of convulsive movements felt two days previously, then cessation; the first child had died, aged 7 months.
9. Fifth pregnancy; first and fourth stillborn; severe ante-partum accidental haemorrhage with collapse and inertia; child delivered rapidly by forceps, but dead.
10. Fifth pregnancy; under treatment in hospital for three weeks previously for toxæmia; child had spasmodic breathing from birth and poor circulation, frequently going blue and cold; died third day.
11. First pregnancy; baby had asphyxia at birth, heavy rapid breathing, spasms of larynx, and inability to swallow; died fifth day.
12. First pregnancy; heavy albumen; a twin premature at seven months; lived thirty-six hours.
13. First pregnancy; premature at seven months; born asphyxiated; died in a month.
14. Fifth pregnancy; brought to the hospital in a state of collapse, weak and cold from haemorrhage; child prematurely born rigid and dead. In her first pregnancy there had been haemorrhages, and the fetus died at the seventh month; her third and fourth were abortions.
15. Seventh pregnancy; vesicular mole.
16. First pregnancy; anencephalic fetus.

The remaining eight had the usual toxic symptoms; some of the children were premature, some macerated, some died in two or three days from convulsions or general weakness. Several of the mothers had a history of previous stillbirths or early deaths in the children.

Many of the mothers with heavy albumen collapsed to an alarming extent after labour, causing grave anxiety. The only death was one in which post-mortem examination revealed extensive changes in the kidneys.

In a few cases the baby had oedema; in some there was collapse of the child, requiring stimulants, and sometimes a saline per rectum, but afterwards recovering. Many of those who lived were below the average weight. In one case there were deformed feet. Two cases occurred of the child dying from haemorrhage of the stomach or bowels; one child had subsequent haemorrhage of the umbilicus, where the mother had heart disease, and was collapsed for some time after labour.

Return Cases.

The return cases to the hospital are also particularly instructive and interesting. I have details of fourteen return cases in which there was albuminuria for at least one of them. They distinctly show that in the pregnancy in which albuminuria was present, other complications were present; when the same woman had no albumen the labour was normal:

1. She first came to St. Helen's for her third pregnancy, which was normal in every way. Fourth pregnancy also normal. In both cases she nursed the baby entirely. Fifth pregnancy, albuminuria, complicated with uterine inertia, adherent marginal placenta, *post-partum* haemorrhage; milk had to be supplemented with artificial food.

2. Primipara; albuminuria; child born prematurely at eighth month. Second pregnancy, no albumen, and normal in other ways.

3. Her third pregnancy, heavy albumen, fetus macerated, premature at eighth month, excessive vomiting, and low condition of health. Fourth pregnancy, had treatment throughout whole period; the baby thrived, no complications, except a poor milk supply and a tendency to run a temperature.

4. First pregnancy, trace of albumen, subinvolution with retention of lochia; ran a temperature. Second pregnancy, no albumen, normal in every way.

5. First pregnancy, albuminuria, twins, one dead, marginal placenta, *ante-partum* haemorrhage, oedema and toxic symptoms requiring treatment in hospital beforehand. Second pregnancy, no albumen, normal in every way.

6. Had one before she came to St. Helen's with albuminuria and eclampsia. The first at St. Helen's, albuminuria, toxic symptoms treated in the hospital beforehand; had *post-partum* haemorrhage and collapse. Second, in hospital, albumen only slight, no complications, except very free discharge. Third, trace of albumen with slight oedema, no complications, no haemorrhage. Fourth, trace of albumen with oedema, faintness beforehand, but no complications. This one continued to improve in each successive pregnancy, as she understood better how to manage her health.

7. First pregnancy, no albumen, normal. Second pregnancy, very slight albumen, no complications. Third pregnancy, heavier albumen; had *post-partum* haemorrhage.

8. Fourth pregnancy, trace of albumen, milk scanty; baby lost weight. Fifth pregnancy, normal, no albumen; baby thrived. Sixth pregnancy, slight albumen; had frequent headaches and epistaxis; baby not very healthy, and losing weight.

9. First pregnancy, no albumen; normal in every way. Second pregnancy, trace of albumen, headaches; baby premature at eight and a quarter months.

10. First pregnancy, distinct albumen, threatening *post-partum* haemorrhage. Second pregnancy, no albumen; normal.

11. This one is particularly interesting. She came to book in the early months for her seventh pregnancy, and gave the history that at her second pregnancy she had had eclampsia, and in all since she had had oedema, headaches, and giddiness. She was given advice and treatment; no albumen appeared; baby 9½ lb., and thrived well; no complications. Eighth pregnancy; possibly she thought she was all right now, and took no precautions; there was albumen, oedema, and toxic symptoms; the babies, twins, were born prematurely at seven and a half months, both very weakly, requiring stimulants; fingers of both children pustular.

12. First pregnancy, slight albumen; child premature at seven months; born asphyxiated and weakly; milk scanty; child died in one month. Second pregnancy, trace of albumen, which did appear in a few days; milk good; normal confinement. Third pregnancy, no albumen; normal. Fourth pregnancy, slight albumen; threatened miscarriage at sixth month, with haemorrhage; milk poor.

13. First pregnancy, albumen, symptoms of eclampsia, during first day *post partum*; face swollen and purple, could not see; pulse rapid, twitching of facemuscles; child macerated; placenta degenerate. Second pregnancy, distinct albumen; patient under treatment for last two months for toxic symptoms; had *post-partum* haemorrhage and adherent placenta; baby thrived.

14. First pregnancy, heavy albumen and oedema; under treatment for last month; baby two weeks premature; membranes ragged and part retained. Second pregnancy, had advice and treatment throughout; very slight albumen, no oedema; placenta and membranes intact; went to full term; baby thrived.

When we come, then, to examine these cases carefully, we see a remarkable preponderance of complications in those showing albuminuria. It is evident that the albumen is only the visible sign of profound metabolic changes which are taking place in the body, and if the excretory organs fail to respond to the extra demand made upon them, the waste products of cell metabolism, probably from the disintegration of protein, circulate in the blood, act as an irritant, and lead to the development of inflammatory changes in the renal tissue, causing degenerative disease, and resulting in that type of nephritis of a transient nature which we usually separate from true inflammatory nephritis. If the condition is not recognized early, the waste products accumulate, and will lead to

more serious disease, ultimately resulting in the death of the mother from eclampsia. It is fortunate that we have this one sign—the presence of albumen—as an indication of the slow toxæmia which is going on, as in many cases there is absolutely no other symptom of the mischief which is brewing. Indeed, I remember one case in my private practice where the woman was in excellent health—always had been—but as I made a rule of examining a specimen of urine at the seventh month I examined hers, and was surprised to find it heavy with albumen. I immediately explained her condition to her, and put her on treatment. She got safely through the confinement in a normal way, but I had just returned home again at three in the morning when a message came from the nurse to say that she had eclampsia. Fortunately it was mild; she had only three fits, and made a good recovery; but until that first fit appeared, although I had been on the look-out for symptoms, there was no indication of anything being wrong.

The fact that haemorrhages are more frequent in cases of albuminuria is only what we would expect, as we know that renal inefficiency is associated with serious disturbances of the vascular system, manifested by an increase in the general blood pressure, by cerebral and retinal haemorrhages and epistaxis, and finally leading to changes in every organ of the body, the uterus included. If the condition recurs through two or three pregnancies, no doubt actual arterio-sclerosis would arise, and from the histories of previous pregnancies which I have collected, it is evident that the same cause has been acting in several, producing permanent changes. This would account for such complications as placenta praevia, haemorrhages, and adherent placenta or membranes, possibly also for phlegmasia alba dolens. The increase of cases showing mental symptoms is quite easily accounted for, as a toxæmia from any cause will affect the brain and nervous system. The increased morbidity is, I think, due not so much to the direct effect of the poison in the body, as there is usually no temperature before labour, but rather to a want of resisting power in the body generally, to the attack of those organisms which would be present in the vagina, and which would quickly get the upper hand.

The stillbirths and early deaths which so markedly preponderate in the albumen cases would be accounted for: (1) By the direct effect of the toxæmic blood circulating through the placenta, degeneration of which frequently occurs, and thereby gaining access to the child; for there can be no doubt that this poison does find its way to the child, as is shown directly by the occasional occurrence of oedema in the children. Might not the various haemorrhages in the newborn—for example, in the skin, from the mouth, the navel, the nose, and the gastro-intestinal tract—be also produced by the circulation of this toxin in the fetal blood? (2) Once degeneration of the placenta sets in, a certain amount of nourishment to the child is cut off and the child dies.

Exactly what causes premature birth it would be difficult to say, but possibly the toxins circulating in the blood act as an oxytoxic, setting up uterine contractions. Every patient is not similarly affected, as many show a slight albuminuria with no other complications than that of anaemia, whilst a few, but a very few, have heavy albumen and no complications other than anaemia, bad varicose veins, poor milk supply, and occasionally degenerate placenta.

There is no doubt, however, that the presence of albumen during pregnancy is a serious indication, and is likely to be the forerunner of some complication in labour. It is a strong plea for the more frequent examination of the urine during pregnancy and the more careful supervision of the general health, in the latter months, by the obstetrician engaged to attend the woman.

In the subsequent discussion on the paper one of the chief points raised by several members was the difficulty of understanding why placenta praevia should occur more frequently in cases of albuminuria. The reply to this was that these were probably not cases of transient albuminuria produced by a toxin, but cases of true nephritis in which endometritis had been set up, thus allowing the ovum to fall lower in the uterine cavity before it found a resting place.

As a cause of the oedema, albuminuria, and haemor-

rhages, Professor Malcolm suggested that the circulation of the toxix in the blood damaged the endothelium of the vessels, thus producing greater permeability in the walls of the vessels, and a greater liability of the capillaries to rupture.

It is also possible that the contractile power of the elastic and muscular walls is diminished; this would account for the *post-partum* haemorrhages.

A PERSISTENT OCCIPITO-TRANSVERSE PRESENTATION: LABOUR OBSTRUCTED AT THE PELVIC OUTLET.

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In the conduct of a case of difficult labour it is generally the practice to attach so much importance to the determination of the lengths of the diameters of the pelvic brim that the consideration extended to the pelvic outlet is apt to be rather meagre.

A moment's reflection on the normal mechanism of labour will show this to be irrational, as it is clear that any marked diminution in the size of the pelvic outlet will provide a most definite barrier to the passage of the child. For scientific delivery as accurate a knowledge of the pelvic brim as it is possible to obtain is, of course, essential. But our estimate of the size and shape of the pelvic outlet is of equal importance.

As a result of a number of observations that I have made on patients who had previously suffered from somewhat extensive perineal lacerations, I am convinced that a diminished pelvic outlet is a much commoner condition than is apparently generally supposed, and that the omission to recognize its presence has been responsible for a very large proportion of the difficulties experienced in delivery when once the fetal head has passed the pelvic brim.

In the estimation of the size of the pelvic outlet it is necessary to measure the distance between the ischial tuberosities, this being its narrowest diameter. This is of great value as far as it goes, but if we consider the process of extension by which the head is delivered it is obvious that it is not enough. The distance between the tuberosities is dependent on two factors—namely, the length of the pubic rami and the angle included by them—the subpubic angle. The ease with which the fetal head will escape from the vaginal outlet will depend very largely on that angle. Of two pelvis with an equal distance between the tuberosities, the one with the wider subpubic angle and shorter pubic rami will clearly offer the less obstruction to the passage of the head during its delivery by extension. To work at the best advantage the suboccipital region of the fetal head should lie in contact with the under border of the symphysis pubis during this process and form, as it were, the fixed point of a lever, the length of which is determined at any instant by the particular diameter of the fetal head escaping at the moment, and which consequently varies continually during the extension. If the subpubic angle be so small that contact between these parts is rendered impossible, it follows that the length of the lever must be increased by the distance between the suboccipital region and the under border of the symphysis. This increase must of necessity put an additional strain on the perineal tissues, and so increase the difficulty of delivery and render a laceration more likely. This condition consequently calls for additional care in extending the head over the perineum.

As the shoulders escape the same thing is true. Their escape is a much more fruitful source of perineal laceration than one is often led to believe, and, in my opinion, they are in the majority of cases the determining factor. The process of their delivery is frequently apt to be more hurried than scientific.

The case on which this note is based forms an excellent instance of a presentation at the pelvic outlet, which I have here described as an occipito-transverse. By this I mean that in these cases the sagittal suture lies deep on the

pelvic floor and in the transverse diameter of the pelvis, the posterior fontanelle being in the right and the anterior fontanelle in the left lateral aspect of the pelvis, or vice versa.

I am unable to find any description of this condition in the standard English textbooks, though it is dealt with by the German authorities, the condition being known as a *Tiefer Querstand*. It is not very rare, and the case under consideration is one in point. It is not necessarily dependent on obstruction of any kind, but is probably a variation of the mechanism of an occipito-posterior presentation. Its recognition is very important from a point of view of treatment, as delivery is greatly facilitated by the knowledge of a suitable method of applying forceps should the occiput not rotate forward under the symphysis. The general rule that their application should be made in the transverse diameter of the pelvis obviously does not hold good in these cases, as the result would be to apply them with one blade over the face and the other over the occiput of the child, and an attempt at delivery would end in disaster. Under these circumstances the forceps therefore should be applied in one or other oblique diameter of the pelvic outlet with their pelvic curve towards the occiput of the child. Then during traction the forceps are rotated gradually into the opposite oblique, with the result that the occiput is swung round under the symphysis, and delivery is then effected in the usual way. To any one with a knowledge of the application of forceps this method of delivery is quite simple, and in the case under consideration was attended with an excellent result.

M. B., a primipara, aged 20, came into labour on August 14th; she had had slight pains throughout the day. The membranes were said to have ruptured earlier in the day, but strong labour pains did not commence until 6 p.m. She was examined at 7 p.m., and the os was stated to admit three fingers. It was fully dilated by 10 p.m., and before 11 p.m. the head was well down on the pelvic floor, and could be seen just inside the vulva. The pains now became more frequent, and although they were occurring every four minutes and were very strong, the head did not advance. At 12 midnight meconium began to issue from the vagina and the pains became still more severe, and the pulse-rate rose steadily. She was admitted into Guy's at 2.30 a.m., and when I saw her she was beginning to become very distressed, the pains were frequent and severe. The pulse-rate was now 110, and the interval between the pains was short. The back of the child was made out to be to the right. Meconium was issuing from the vagina, and the fetal head was visible on separating the labia. The fetal heart was counted at 152.

The patient was anaesthetized, and on examination there was a large caput extending over both parietal bones, and the sagittal suture was found to be lying in the transverse diameter, with the posterior fontanelle to the right and the anterior fontanelle to the left. The distance between the ischial tuberosities was 2½ in., and the subpubic angle was noticed to be small. Obviously the further progress of the head was completely obstructed by the narrowness of the outlet. By applying forceps in the left oblique diameter, and by rotating them into the right oblique during traction, I was able to swing the occiput under the symphysis. Realizing the smallness of the subpubic angle, great care was used in extending the head and the living child delivered with a small amount of laceration. This was immediately closed with sutures, and primary union has taken place.

The child was not large, and weighed 6 lb. 12 oz. Its head was 14 in. in circumference, its biparietal diameter was 3½ in., bitemporal 3½ in., mento-vertical 5½ in., occipito-frontal 4½ in., and suboccipito-bregmatic 3½ in. The obstruction was, therefore, not due to any fetal abnormality. Under the anaesthetic the diagonal conjugate was measured and found to be 5 in., and an estimate of the pelvic brim showed that no source of obstruction was present. This agreed with the clinical history of the labour.

This case, then, serves to illustrate the following points:

1. The necessity of always determining in a case of labour the size and shape of the pelvic outlet.
2. In forming an estimate of the amount of obstruction likely to occur at the pelvic outlet we should be guided both by the distance between the ischial tuberosities and by the subpubic angle.
3. The existence at the pelvic outlet of a presentation, here described as an occipito-transverse.
4. The rational treatment of such a presentation, should it persist, consists in an application of forceps in an oblique diameter of the outlet, and while traction is being made they should be rotated into the opposite oblique, with the result that the occiput is swung under the symphysis and delivery effected in the usual way.