F v Chan Tanny
[2003] SGHC 192

Case Number : Suit 1554/2001

Decision Date : 29 August 2003

Tribunal/Court : High Court

Coram : Lai Kew Chai J

Counsel Name(s) : Kwok-Chern Yew Tee and Mak Moo Theng (Lawrence Chua and Partners) for plaintiff; Edwin Tong and Karen Eu (Allen and Gledhill) for defendant

Parties : F — Chan Tanny

Tort – Negligence – Breach of duty – Whether gynaecologist on a balance of probabilities breached duty of care – Applicable standard of care

Tort – Negligence – Causation – Whether the injury suffered caused or materially contributed to by any breach of care

Tort – Negligence – Duty of care – Gynaecologist's duties to competently manage pregnancy of mother and delivery of baby

Tort – Negligence – Res ipsa loquitur maxim – Whether applicable

Introduction

1 The plaintiff, F, was born on 12 December 1998 and was diagnosed with subarachnoid haemorrhage (bleeding in the brain), intrauterine pneumonia, and a ventricular septa defect (hole in the heart). As a result, F is suffering from various other complications. The defendant ("Dr Chan") is a consultant obstetrician and gynaecologist, practising at Gleneagles Hospital. She managed the mother's pregnancy and delivered the baby. She worked closely with her partner and husband, Dr Sng Soo Pheow ("Dr Sng"), to whom she referred her patients for obstetrical and gynaecological ultrasound scanning.

2 F, suing by her mother ("Mdm C") and next friend, is claiming against Dr Chan for damages for pain and suffering, loss of amenities, and consequential losses arising from the alleged medical negligence of Dr Chan
in the monitoring and management of the gestation and the eventual delivery of F. There were numerous allegations directed to support the allegation that a Caesarean section should have been performed.

3 Dr Chan wholly disputes the allegations. She says there was no reasonable basis, at any stage throughout the pregnancy, for her to have intervened by ordering a Caesarean section or taken any other step.

Findings of Facts

Antenatal management

4 Mdm C, a teacher, first consulted Dr Chan on 15 April 1998 when she was five weeks pregnant. She was expecting her first child. Dr Chan took note of the medical history of Mdm C and of her husband, Mr D, a self-employed businessman. Mr D had a history of asthma. Apart from that, there was no other medical matter of note. Dr Chan's general and abdominal examination of Mdm C did not disclose any abnormality. Pelvic examination showed a normal cervix. Her expected date of delivery was assessed to be 13 December 1998. Mdm C was seen by Dr Chan regularly, with a total of
16 antenatal visits. I shall recite them as they were not in controversy, unless otherwise stated in

this judgment.

5 Dr Chan next saw Mdm C – on 9 May 1998. She was nine weeks pregnant. She was well and had no complaints. The foetal heart beats were heard and were regular. Mdm C's weight was 43.7kg.

6 On 13 May 1998, Mdm C telephoned Dr Chan complaining that she was experiencing lower abdominal pain and slight breathlessness. There was no bleeding *per* vagina. Dr Chan explained to her that in early pregnancies, slight pain in lower abdomen was usually of no significance in the absence of bleeding. She was advised to see Dr Chan for consultation if the symptoms persisted. She did not.

7 Mdm C next saw Dr Chan on her scheduled appointment on 30 May 1998. She was then 12 weeks pregnant. She was well and had no complaints. She had no further complaints of lower abdominal pain or breathlessness. Mdm C's weight was 44.7kg. The foetal heart rate was found to be 176 beats per minute, which was regular.

8 The ultrasound scan performed by Dr Sng showed a single foetus with a crown-rump length of 54mm, biparietal diameter of 21mm, head circumference of 70mm and femur length of 7mm. It was not in controversy that these readings were normal for her period of pregnancy. Dr Chan also noted that the nuchal fold was normal at 2.4mm thickness, which indicated a low risk of the foetus suffering from Down's syndrome. In addition, there was a normal amount of liquor (amniotic fluid). The placenta was in the posterior position, as shown by the ultrasound scan, which was exhibited.

9 Dr Chan again gave Mdm C supplementary iron, minerals, vitamins and calcium. She explained to Mdm C that, as a precaution, she should undergo an antenatal blood screening during her next consultation. Dr Chan further explained that she would further screen her for Toxoplasma infection, which could damage the foetal brain and the eyes, even though this was and is a rare condition. If detected, early treatment could prevent the damage.

10 Dr Chan next reviewed Mdm C on 27 June 1998. She was well with no complaints. She was 16 weeks pregnant and the uterine size was about 16 weeks. Her blood pressure was 120/70mm Hg. Dr Chan once again discerned that the foetal heart beats were regular. Her weight was 46.7kg. As the results of Mdm C's urine test showed a trace of albumin and 3+ glucose, Dr Chan arranged for Mdm C to, and she did, undergo a glucose tolerance test on 29 June 1998 to exclude diabetes. She was given supplementary iron, minerals, vitamins and calcium.

11 On 4 July 1998, Dr Chan telephoned Mdm C and informed her of the results of her blood test. Apart from being mildly anaemic, the blood test results were normal. Her Thalassaeimia screen was normal, on the basis of which Dr Chan concluded that her anaemia was due to nutritional deficiency (as opposed to genetic anaemia). In addition, she was screen negative for Toxoplasma IGM. Her screen for Syphillis and HIV (AIDS) were negative. Her random blood-sugar level was well within normal range and her glucose tolerance test showed she was not diabetic. Her urine test did not show any urine infection.

12 The blood test results also indicated that Mdm C was screen negative for Down's syndrome, Edward's Trisomy and neural tube defects. In this context, Dr Chan informed Mdm C that the blood test for Down's syndrome and Edward's Trisomy were not 100% accurate, and that she would have to do an amniocentesis in order to exclude completely these conditions.

13 However, Dr Chan advised Mdm C against undergoing amniocentesis for the following reasons. The test carries a risk of inducing a miscarriage. Secondly, Mdm C's age at 30 was not in the high risk group for Down's syndrome and Edward's Trisomy. Finally, Mdm C's blood test was screen negative. Dr Chan asked Mdm C to discuss with her husband and to let Dr Chan know whether she wished to undergo amniocentesis. As usual, Dr Chan advised her on a proper diet, which was mainly to consume

iron-rich food and reduce the intake of sugar food.

14 Mdm C attended her next medical review on 18 July 1998. She was well and had no complaints. At 19 weeks pregnancy, her uterine size corresponded to her period of pregnancy. Her blood pressure was 120/80mm Hg. The foetal heart beats were regular. Mdm C's weight was 47.2kg. Her urine test showed positive for albumin and negative for glucose. In the absence of hypertension, Dr Chan was of the view that the slight albumin in the urine was of no clinical significance. Dr Chan also gave Mdm C a copy of all her blood tests and explained to her again the interpretation of all the tests. Mdm C then informed Dr Chan of her decision to follow the latter's advice not to perform amniocentesis. Mdm C was given supplementary iron, minerals, vitamins and calcium and was told her next review was 3 weeks later on 8 August 1998.

15 On 8 August 1998, Dr Chan reviewed Mdm C, who was 22 weeks pregnant. She informed Dr Chan that she had recovered from a slight flu but was otherwise well. The uterine size was about 22 weeks. Her blood pressure was 120/80mm Hg. The foetal heart beats were heard and were regular. Her weight was 48.5kg.

16 Dr Chan ordered an ultrasound to be done on the same day as this was the appropriate time for a foetal anomaly scan. The ultrasound anomaly scan performed by Dr Sng showed a normal single foetus presenting by its breech. The biparietal diameter was 56mm, the head circumference was 208mm, the abdominal circumference was 175mm and the femur length was 38mm. Dr Chan was of the view that these measurements were normal for Mdm C's period of pregnancy. There was no abnormality detected in the foetal head, brain, spine, eyes, lips, mouth, nose, neck, heart, aorta, pulmonary artery, lungs, stomach, intestines, kidneys, bladder, umbilicus, liver, diaphragm, upper limbs and lower limbs. Head Circumference/Abdominal Circumference at 1.19 and Femur Length/Biparietal Diameter at 0.67 were both normal readings. The placenta was posterior in the normal position and the liquor was normal in amount. Dr Chan observed that the foetus was growing well, which indicated good placental function.

17 At her review on 29 August 1998, when she was 25 weeks pregnant, Mdm C was found to be well and had no complaints. The uterine size was about 25 weeks. The foetus was presenting by its breech and the foetal heart beats were heard and were regular. Her blood pressure was 120/80 mm Hg. Her weight was 49.8kg. She was given the usual nutritional supplements.

18 At her next consultation on 19 September 1998, Mdm C was 28 weeks pregnant. Her uterine size was about 28 weeks. The foetus was presenting by its breech. Mdm C's blood pressure was 120/80mm Hg and her weight was 50.7kg. She was well except for some mild and painless contractions.

19 Dr Chan conducted a vaginal examination, and noted that the cervical os was closed and that there was no bleeding. Dr Chan explained to Mdm C that she was experiencing Braxton-Hick's contractions and they were normally experienced in the later half of pregnancy. This was physiological, and in the absence of painful contractions and cervical dilation or show, she was not in labour. However, Dr Chan told Mdm C that if she experienced painful contractions, she should take a tablet, Ventolin (which Dr Chan prescribed to prevent premature labour), and to contact Dr Chan immediately. In addition, Mdm C was given the usual nutritional supplements and was told to return for consultations every fortnight instead of once every three weeks.

20 Her next follow-up consultation was on 3 October 1998 when Mdm C was 30 weeks pregnant. Dr Chan noted that the uterine size was about 30 weeks. Mdm C was well and had no complaints. She informed Dr Chan that the painless contractions had subsided. The foetus was presenting by its breech. Her blood pressure was 120/80mm Hg and her weight was 52.3kg. She was again given the usual nutritional supplements.

21 Mdm C was next reviewed on 17 October 1998. She was into her 32nd week of pregnancy. She was well with no complaints. The uterine size corresponded to her date and the foetus was presenting by its head. Her blood pressure was 120/80mm Hg and her weight was 52.0kg. She was given nutritional supplements.

22 At the follow-up review on 31 October 1998, when Mdm C was at 34 weeks of pregnancy, her uterine size was about 34 weeks. The foetus was presenting by its head and the head was engaged. The foetal heart beats, according to Dr Chan, were regular. She performed a vaginal examination. The cervical os was closed and the foetal head was engaged in the pelvis. The maternal weight was 52.7kg. Mdm C told Dr Chan that her right ear
was blocked and was under the treatment of Dr Lee Chon Sham. Upon examination, Mdm Chan did not have a fever and she had no complaints to suggest infections of any other systems. She was given the usual nutritional supplements.

23 Mdm C was reviewed again on 7 November 1998. She was well and had no complaints. The foetus was presenting by its head and the head was engaged. Her blood pressure was 120/80mm Hg, and her weight was 52.48kg. She was given supplementary iron, minerals, vitamins and calcium.

24 The next review was on 14 November 1998. She was 36 weeks into pregnancy. Dr Chan noted that the uterine size was 36 weeks and the foetus was presenting by its head. The head was engaged. Mdm C's blood pressure was 120/80mm Hg and her weight was 52.6kg. Although Dr Chan found a trace of albumin and more than usual sugar in her urine, she was of the view that these findings had no clinical significance in the absence of hypertension and diabetes. Mdm C told Dr Chan that she had pus discharging from her right ear, and that she was being treated by an ENT specialist, a Dr Chew, in Mt Elizabeth Hospital, who advised her to defer any surgical treatment (*ie* ear washout) till after her delivery.

25 In accordance with her usual practice, Dr Chan at this point ordered an ultrasound scan to be performed by Dr Sng. The ultrasound showed a single foetus presenting by its head in the left occipito-anterior position. That was a favourable position for vaginal delivery. The placenta was in the posterior position with calcification. As placental calcification is very common in the later part of pregnancy, Dr Chan was of the view that it was of no clinical significance in the light of the progress of the present case. The progress of the foetus, so far as could be observed, was normal. No foetal abnormality was detected. The biparietal diameter was 83mm, the head circumference was 314mm, the abdominal circumference was 297mm and the femur length was 68mm. Dr Chan also noted that the foetal heart rate was 146 beats per minute. The foetal weight was estimated to be 2,387g. The amniotic fluid index was normal, noted at 8.9, and the umbilical artery blood flow was normal. According to the notes of Dr Sng, who confirmed his findings in his evidence, the ultra sound scan showed no sign of placental insufficiency. It showed a normal foetus of 2,387g in weight.

26 A slight slowing down of foetal growth was detected at about 36 weeks of gestation. Having considered all the relevant clinical observations, Dr Chan was of the view that the foetus, though slightly small in size, was in the acceptable weight range given Mdm C's stature.

27 I should note at this point that there were different versions as to what Dr Chan and Dr Sng on the one hand had said to Mdm C and her husband, Mr D, on the first occasion on 14 November 1998 and to Mdm C alone on the second occasion during the review of her pregnancy on 28 November 1998.
I now summarise the evidence and set out my findings of what had transpired.

28 Mdm C in her affidavit evidence stated that on 14 and 28 November 1998, Dr Sng who did the ultrasounds told her that "there were some white spots on the placenta suggesting placenta ageing and that this might cause insufficient oxygen supply to the baby". She was worried that the placenta

ageing might cause problems to the foetus. Dr Chan told her that placental calcification was common in late pregnancy and therefore it was not necessary to deliver the baby earlier. Her advice was to wait and observe the foetus. My attention was drawn to a note made by Dr Sng in the Birth Defects/Congenital Malformations Notification Form submitted to the National Birth Defects Registry of the Ministry of Health almost a year after the birth of F. In it, Dr Sng stated that the suspected birth defect detected by ultrasound scans was "Intra-uterine growth retardation with Placenta Calcification". Dr Sng readily admitted in cross-examination that he had made a mistake in making this entry. He meant that there was slow growth of the foetus, that the baby was small and was below the 10 percentile. When asked whether he had told Mdm C that the calcification might affect oxygen supply to the foetus, he denied it and said that probably Mdm C had misunderstood him. Though he had noted there was calcification, he did not mean to suggest that it was of any clinical significance. Placenta calcification was not a birth defect.

29 Mr D said in his evidence that at the review on 14 November 1998, at which he was present, Dr Sng told them that the ultrasound scans showed placenta calcification. He asked Dr Sng what could be done about the calcification. He was told there was nothing he could do about the calcification, noting that "the baby was small". Mr D did not allege that Dr Sng had said that the calcification might affect oxygen supply to the foetus. I find that Dr Sng did not say that calcification might affect oxygen supply and I accept that his note in the form was an honest mistake.

30 According to Dr Chan, what transpired between her and Mdm C at the review on 14 November 1998 was this. Having being apprised of the clinical findings, Mdm C asked whether the foetus should be delivered at an earlier date. Dr Chan advised her that induction of labour carried a higher risk of Caesarean section which should only be performed with medical indications such as absence of foetal growth or if the pregnancy was post-matured. Those clinical signs were absent in her pregnancy. I accept Dr Chan's version of what had transpired.

31 A slightly sharper dispute on the facts arose in relation to the review of Mdm C on 28 November 1998. Dr Chan said in evidence that Mdm C continued to be well and without complaints. She noted that Mdm C's uterine size corresponded to her date, which was about 37 to 38 weeks of gestation. Her blood pressure was normal at 120/80mm Hg, and the maternal weight was 53kg. The foetus was presenting by its head in the left occipito-transverse position. The foetal head was engaged, which indicated that Mdm C's pelvis was adequate for vaginal delivery. The foetal heart beats were heard and were regular. In addition, Mdm C informed Dr Chan that she had felt active foetal movements. The urine test revealed a trace of albuminuria but in the absence of hypertension it was of no clinical significance.

32 An antenatal Cardiotocography ("CTG") was performed. The CTG trace showed a normal and regular baseline foetal heart rate of about 150 beats per minute. According to Dr Chan's reading, the CTG trace was not optimal as there was a narrow beat-to-beat variation. However, she noted that there were some accelerations and no deceleration. She also noted that there were numerous foetal movements, as marked by the patient (more than 30 foetal movements in around one hour of monitoring). In those circumstances, Dr Chan was of the view that there was no indication of foetal hypoxia or placenta insufficiency.

33 To confirm her views, Dr Chan ordered another ultrasound scan to be done on the same day. Mdm C asserted that the ultrasound scan was carried out before the CTG. In my view, she was mistaken. It was unlikely that she would have remembered. Dr Chan made the following observations from the ultrasound scan. The foetus was presenting by its head in the left occipito-transverse position. The placenta was posterior with calcification in evidence. The foetal skull, lips, stomach, liver, diaphragm, kidneys, bladder, umbilicus and umbilical cord were normal. The biparietal diameter was 87mm, the heard circumference was 327mm, the abdominal circumference was 338mm and the femur length was 68mm. The foetal heart rate was found to be 157 beats per minute. The amniotic

fluid index and the umbilical artery blood, which were two very important parameters, were both found normal. The foetal weight was estimated to be 2,774g. The foetus had grown by 387g in two weeks, which was again normal. The results gathered from the ultrasound and the clinical observations of both mother and foetus confirmed Dr Chan's views that there was no foetal hypoxia and that there was no indication to deliver the baby earlier.

34 The 15th antenatal visit was on 5 December 1998 when Mdm C was 38+ weeks into gestation. Her blood pressure was normal and her weight was 53.4kg. Her uterine size was found to correspond to date and the foetus had grown. The foetus presented itself as previously noted and the foetal head was engaged. The foetal heart beats were heard and were regular. Dr Chan conducted a pelvic examination which showed the cervical os to be closed and the foetal head was in the pelvis. These vaginal findings indicated that she was not in labour and the pelvis was adequate for vaginal delivery. She was again given the usual nutritional supplements.

35 On 11 December 1998 at about 8.30am Mdm C, who was 39+ weeks pregnant, had a slight show and had two bouts of pain between 3.00am to 4.00am and 7.00am to 8.00am. She consulted Dr Chan at 11.50am and told her of the episodes. Dr Chan examined her and noted that the uterine size corresponded to her date and the foetus had grown. The foetal heart beats were heard and were regular. Her blood pressure was normal and her weight was 53.3kg. Vaginal examination showed the cervix to be partially effaced. The cervical os was closed.

Intrapartum management

36 I turn first to the early labour management of Mdm C in the antenatal ward. Dr Chan told Mdm C that she was in early labour because she had painful uterine contractions and show. She ordered her to be admitted to the maternity unit of Gleneagles Hospital for monitoring of her labour progress. She was admitted to the antenatal ward at 12.30pm. It was noted by the nurses on duty that she had weak and irregular contractions with show. The foetal rate was normal, at 160 per minute. Her blood pressure was 110/70mm Hg, which was normal. The urine tests for glucose and albumin were found to be normal. A CTG monitoring was ordered and it commenced at 1.18pm.

37 Dr Chan studied the CTG trace after it was completed. She noted that it showed a similar pattern as that done on 28 November 1998, with abnormal baseline foetal heart rate of 145 per minute. There were no regular uterine contractions. The beat-to-beat variation was narrow but there were some slight acceleration and no deceleration. She considered these readings in the light of the total clinical picture and concluded that there was no indication of foetal hypoxia. She therefore felt that there was no reason to change the course of management by performing an immediate Caesarean section. By "the total clinical picture", she meant the normal pregnancy progress of Mdm C with no medical complications, normal placental function reflected by normal foetal growth, normal amniotic fluid index and normal umbilical artery blood flow.

38 At about 5.30pm later the same day Dr Chan examined Mdm C. She felt foetal movements. The foetus was presenting by its head. The foetal head was engaged in the left occipito-anterior position, which was favourable for vaginal delivery. However, Dr Chan noted that Mdm C's uterine contractions were weak and irregular. Vaginal examination showed that the cervix was partially effaced and thick and the cervical os was 0.5cm dilated, indicating a low cervical score of 2 (modified Bishop Score 2). The foetal weight was estimated to be around 3.0kg. In the circumstances, Dr Chan formed the view that Mdm C should be given the opportunity to try for vaginal delivery.

39 At this juncture, in light of the fact that Mdm C was experiencing a delayed latent phase of labour (*ie* the initial phase) of about 14 hours without progressing into the active phase of labour (the later stage of labour), Dr Chan felt that it was necessary to augment her labour by inserting a tablet of Prostin E2 vaginally. She noted in her case notes that Mdm C was undergoing a "trial of labour",

meaning that the progress of labour was slow and required augmentation.

40 Dr Chan explained that she chose vaginal Prostin E2 in order to augment the labour (as opposed to artificial rupture of the membranes and intravenous Syntocinon) due to its advantages when the cervical score is low and when the patient was in the latent phase of labour. The use of Prostin E2 has been proven effective in ripening the cervix and augmenting labour when the cervical score is low. Dr Chan explained that Prostin E2 served to dilate the cervical os and encourage uterine contractions as well. She also considered that Prostin E2 was a safe and simple method, and avoided the complications associated with augmentation of labour by early amniotomy and intravenous Syntocinon, such as increased incidence of chorio-amnionitis and cord compression.

41 Dr Chan said that prior to inserting the Prostin E2 she explained to Mdm C in accordance with her usual practice that she would insert a vaginal tablet (Prostin E2) into her vagina in order to ripen her cervix and to augment her labour. Her explanation was given in the presence of the assisting nursing staff. As Mdm C did not protest or express any objection to the procedure, Dr Chan proceeded to insert the tablet into her vagina. Mdm C alleged that Dr Chan had failed "to obtain (her) informed consent before administering the tablet, Prostin E2". In my view, this allegation is not made out by Mdm C. She knew that a tablet was inserted into her vagina. In the course of her cross-examination, she said it was her first pregnancy and she left matters in the hands of her gynaecologist. She gave the impression that she could not remember, but her failure to recollect is not inconsistent with her having been told about Prostin E2 and the reasons for its use. The simple truth was that she forgot. In my judgment, her informed consent could be implied in all the circumstances.

42 Apart from instructing the ward nurse to continue monitoring Mdm C, Dr Chan also ordered a second CTG to commence after Mdm C had her dinner. The cardiotocograph was therefore started at 8.15pm on 11 December 1998.

43 After the completion of the CTG tracing, which was at about 9.00pm, Dr Chan reviewed the CTG and the patient at 9.30pm. The CTG tracing showed a similar pattern to the two previous CTG tracings and did not indicate the existence of foetal hypoxia. The contractions were infrequent and weak indicating that Mdm C was not in an active phase of labour.

44 Dr Chan checked with Mdm C who confirmed to her that there were frequent foetal movements at that time. Dr Chan herself checked the foetal heart again with an ultrasound Doppler and noted that the foetal heart beats were normal and were regular. Dr Chan also felt foetal movements during the examination. She conducted a vaginal examination which showed the cervix to be partially effaced. The cervical os was 1.0mm dilated. The membranes were intact and the vertex was at station –1. She assessed that the pelvis was adequate for vaginal delivery. Dr Chan explained that as Mdm C was still not then in active phase of labour, she did not perform an amniotomy because she was aware that early amniotomy was associated with an increased incidence of chorio-amnioritis and cord compression.

45 After the examination, Dr Chan instructed the ward staff to send Mdm C to labour ward for close monitoring and continuous CTG monitoring when her contractions became regular. They were to keep Dr Chan informed. Dr Chan also instructed for an enema to be given to the patient to clear her bowel contents before she was sent down to the labour ward.

46 At 12.25am on 12 December 1998, Mdm C had moderate contractions. The vaginal examination performed by the ward staff showed that the cervix was about 2cm dilated. The foetus was presenting by its head and the vertex was at station –2. The foetal heart beats were regular at 160 beats per minute. The membranes were intact. Mdm C was transferred to labour ward at 12.40am.

47 I now turn to the intrapartum management of Mdm C in the labour ward. She was in labour ward

from 12.45am on 12 December 1998 until the time she delivered F at 10.08am the same day. Dr Chan was informed by the labour ward staff at 12.45am that the patient had painful contractions and she wanted epidural anaesthesia to be given to her to relieve the pain. Dr Chan then instructed the nurse to contact an anaesthetist, Dr Peter Wang, to give her the epidural anaesthesia.

48 After the administration of epidural anaesthesia by Dr Peter Wang, an intravenous drip with Dextrose 5% was set up to maintain Mdm C's hydration. She was on continuous cardiotocograph monitoring in the labour ward throughout labour. Dr Chan was kept informed of her progress. She had instructed the nurse to inform her if any abnormality developed.

49 At 2.45am Dr Chan called the labour ward staff to enquire about the condition of Mdm C. She asked about the base-line foetal heart-rate, the variability, and whether there were any accelerations or decelerations. She was informed by the nurses that apart from the CTG being "flatish" (narrow beat-to-beat variation), the other parameters of the CTG were normal. There was normal baseline foetal heart rate and an absence of decelerations. At that junction, Dr Chan's interpretation of the CTG in light of the other objective clinical signs was that there was no indication of hypoxia or need to perform an immediate Caesarean section.

50 Dr Chan noted that vaginal examination performed by the labour ward staff at 3.00am showed that the cervix was partially effacted. The cervical os was 1.5cm dilated and the membranes were intact. Mdm C's bladder was full and she was catheterized with 400ml of urine drained. Up to this stage, she was still not in the active phase of labour. Nothing abnormal was reported to Dr Chan from then onwards till she examined her again at 6.10am.

51 At her examination of Mdm C at 6.10am on 12 December 1998 Dr Chan found that the uterine contractions were not strong. The CTG showed a baseline heart rate of 150 beats per minute. There was narrow beat-to-beat variation, but till then, there were no decelerations throughout the time when she was in the labour ward. Dr Chan's vaginal examination of Mdm C showed the cervix to be effaced. The cervical os was 7cm dilated. The membranes were intact. As Mdm C was by that time in an active stage of labour, artificial rupture of the membranes was done and it showed light meconium-stained liquor.

52 Dr Chan stated that the presence of meconium in the amniotic fluid, without accompanying signs of foetal hypoxia, was not a sign of foetal distress. She therefore did not feel there was a need for active intervention. In her opinion, a Caesarean section without medical indication was not justified in this case. A Caesarean section carried a higher morbidity and mortality risk to the mother with no benefit to the foetus. Dr Chan noted that the diagnostic criteria of foetal hypoxia in a CTG – the presence of late decelerations and baseline bradycardia (slow rate of heart contraction) – were not present in this case. In addition, Dr Chan noted that the foetal head was at station –1, in the left occipito-transverse position. Since there was progress in labour, Dr Chan therefore decided to allow the labour to continue and to aim for a vaginal delivery.

53 As Mdm C's contractions were not strong at that time, a Sntocinon drip was ordered (3iu of syntocinon in 500ml of Dextrose 5% maintained at 10 drops per minute, equivalent to 3miu per minute) to improve the contractions. The drip was set up after Dr Chan had explained the rationale of the Syntocinon drip to Mdm C in the presence of the nursing staff. Dr Chan stayed with Mdm C and checked her condition soon after rupturing the membranes. The foetal head was engaged and the vertex had descended to station O. The pelvis was assessed to be adequate for vaginal delivery. The umbilical cord was not felt. Following the setting up of the Syntocinon drip, Mdm C's uterine contractions became stronger.

54 Dr Chan reviewed Mdm C again at 8.45am. She noted that there were some foetal heart accelerations from 7.40am. Subsequently, there were mild and uncomplicated variable decelerations

with quick recovery and shouldering from about 8.00am. Dr Chan stressed that throughout the CTG tracing, there were no late decelerations or baseline bradycardia which would have indicated hypoxia. There was, however, a narrow beat-to-beat variation. The baseline foetal heart rate was all the while at 140 to 150 beats per minute and it rose to 170 beats per minute from 7.40am. Dr Chan concluded that the foetal tachycardia (excessive rapid action of the heart) with mild and uncomplicated variable decelerations was likely to have been due to the mild maternal pyrexia of 37.3°C. She told me that in the presence of epidural analgesia, it was not a sign of foetal distress. She excluded the possibility of foetal hypoxia based on the CTG tracing and on her assessment of the objective clinical signs.

55 Those objective clinical signs repay reminder. First, the pregnancy was normal without medical complications such as hypertension, antepartum bleeding or any other medical condition that would affect the placental function. Secondly, the foetal growth was normal by clinical and ultrasonic assessments. Thirdly, the placental function was normal as indicated by normal foetal growth, normal amniotic index and normal umbilicial artery blood. Lastly, there were no sentinel events in labour, such as cord collapse, abruption placenta and uterine rupture which could cause foetal hypoxia to develop during labour.

56 In the circumstances, Dr Chan decided that she should proceed with a vaginal delivery, there being no reasonable basis to intervene or alter the course of her management of Mdm C's pregnancy.

Conduct of assisted delivery by vacuum extraction

57 At 9.30am on 12 December 1998 Dr Chan was informed by the labour ward staff that Mdm C was ready for delivery. The nurse had checked the patient and found that the cervical os was fully dilated. The nurse had also catheterized the patient and released 350ml of urine. The patient was positioned in the lithotomy position and was encouraged to bear down by the labour ward staff.

58 Dr Chan cleansed and draped Mdm C at 9.45am and did a vaginal examination which confirmed that the cervical os was fully dilated. The foetal head was in the left occipito-transverse position, at station +2. The broadest diameter of the foetal head had passed through the pelvic brim. The pelvic size was assessed to be adequate for vaginal delivery. The CTG showed that the baseline foetal heart rate was between 160 to 180 beats per minute. Dr Chan noted that there was some improvement in the baseline variability with some accelerations. There was no bradycardia or late decelerations. There were mill and uncomplicated V-shaped variable decelerations, with quick recovery to the baseline. Dr Chan was of the opinion that the CTG pattern did not indicate foetal hypoxia or any medical conditions that called for Caesarean section at that stage. The uterine contraction was 1 in one-and-a-half to
two-and-a-half minutes. She therefore encouraged the patient to bear down with each contraction.

59 Mr D was standing at the right side of Mdm C's bed. He was helping to support her head when she bore down. The attending midwives also helped by pushing the patient's uterine fundus towards the pelvis while she bore down. It was a common practice among midwives to apply fundal pressure in the second stage of labour to help in the delivery. In this case, the procedure of applying fundal pressure was carried out due to the fact that the patient was under epidural anaesthesia. She was therefore unable to feel the bear-down sensation. Despite the help provided by the midwives, Mdm C was still unable to bear down effectively. Dr Chan then decided to assist the second state of labour with vacuum extraction after informing Mdm C and her husband. They did not express any objection and I find that in the circumstances they gave their implied consent.

60 Dr Chan chose a Bird's medium-sized cup as this was an appropriate instrument to use. The cup was applied to the foetal head over the occiput, at the flexion point to give the best flexion to the foetal head. The indicator of the cup pointed to the occiput of the foetal head. Dr Chan checked to make sure that the vacuum cup did not catch the vaginal wall. After that, Dr Chan made a right

medio-lateral epsiotomy in order to allow the foetal head to be delivered. The automatic timer was started and the vacuum pressure was gradually increased, starting at 0.2kg/cm^2 and increasing at the rate of 0.1kg/cm^2 per minute to 0.5kg/cm^2.

61 Dr Chan stated in her affidavit evidence that during the uterine contractions, her right hand applied traction downward and posteriorly, and pulled in synchronisation with the uterine contractions. The thumb and index finger of her left hand pressed the cup and the foetal head backwards during traction. Just as she began applying traction downwards during the contraction, the vacuum cup slipped due to leakage in the system. The cause of the slip was identified and the cup was re-applied. I was told and it was not disputed that slippage of the vacuum cup during vacuum traction was not uncommon and in any event did not have any adverse effect on the baby when the cause of the slip was the leakage of the system. Traction was applied during the subsequent contractions.

Birth of the baby

62 The foetus was delivered at 10.08am on 12 December 1998. The total vacuum time taken was recorded as 9 minutes. The labour ward nurses also recorded that the maximum pressure used was 0.5kg/cm^2. Both the vacuum time taken and the pressure applied were well within the acceptable range. An intravenous syntocinon 5iu was given after the delivery of the baby to help contract the uterus.

63 After the baby was born, the umbilical cord was clamped and cut, and the baby was handed over to the midwife at delivery. The baby was noted to be pink but did not cry. The tone was poor with Apgar score 6 to 7 at 1 minute and 5 minutes respectively. Dr Chan called in the paediatrician, Dr Thomas Wong ("Dr Wong"), to attend to the baby. He examined F 12 minutes after her birth. There was some fresh meconium in the hind water. I will shortly set out the facts based on the evidence of Dr Thomas Wong.

64 I return to what Dr Chan did after the birth of the baby. After cutting the umbilical cord, she proceeded on to deliver the placenta by the standard method of controlled cord traction. The placenta could not be delivered. Dr Chan put her right hand into the patient's uterus to check and found that the placenta was accretic. Dr Chan then manually removed the placenta with her right hand in the uterus separating the uterus from the uterine wall and with her left hand on the lower abdomen guarding the uterine fundus. As Mdm C was under epidural anaesthesia, the entire procedure was done without causing her discomfort. Dr Chan checked the vagina and uterus and confirmed that there was no tear. The placenta was therefore removed at the third stage of labour without complication. Mdm C's uterus had contracted well and the blood loss during delivery was normal (200ml). The episiotomy was repaired in layers.

65 Mdm C was well after delivery and she was transferred to the post-delivery ward at 11.50am. On 13 December 1998 Dr Chan checked the patient at 8.00am in the ward. The patient's condition was found satisfactory. Dr Chan checked on the patient on 14 and 15 December 1998. Mdm C recovered well. She stayed in the ward till her discharge on 15 December 1998.

The baby

66 Dr Wong's first examination of F 12 minutes after her birth showed that the baby was breathing regularly. She was taking deep gasps every few breaths. This indicated that she was able to breathe on her own and did not require intubation. A vacuum mark was noted. This was, in Dr Wong's view, consistent with and usual in a vacuum delivery. There was no severe
vacuum trauma such as caput succedaneum, subgaleal haemorrhage, cephalhaematoma. At the same time, Dr Wong noted that the baby was hypotonic (limp and poor tone) and that her primitive reflexes

and tendon reflexes were absent. Also, the baby did not cry. These signs to Dr Wong meant that the baby was suffering from neurological problems.

67 Based on these neurological signs, Dr Wong made a provisional diagnosis that the baby was suffering from "birth asphyxia".

68 F was warded in the Neonatal Intensive Care Nursery. The heart rate was found to be 174 beats per minute. The respiration rate was 50/min and the blood pressure was 60/47mm/Hg (mean 51). According to Dr Wong, the baby's first vital signs were normal and there were no signs of cardiovascular compromise.

69 A blood sample was taken for culture and C-reactive protein to ascertain whether the baby had any infection. The baby was started on intravenous fluids (a 10% dextrose drip) as well as antibiotics (ampicillin, gentamycin) and phenobarbitone (to sedate the baby to prevent convulsions). The blood sample results showed negative for C-reactive protein (a protein produced by the liver in increased amounts in the presence of bacterial infections) and there was an absence of bacterial growth in the blood.

70 A chest X-ray of the baby was also performed to check the condition of her lungs. The report of the chest X-ray stated: "Heart is not enlarged. There is increased shadowing seen in both lungs. This is probably due to early consolidation. Pulmonary vasculature is within normal limits". This suggested that the baby was suffering from congenital pneumonia (a lung infection). In Dr Wong's view, the consolidation of the lungs was not due to meconium aspiration, as the latter generates a different picture.

71 When reviewing the baby at 12.30pm on 12 December 1998, Dr Wong was informed by the staff nurse that the baby had displayed convulsions, and was observed to suffer from tonic spasms of both the upper limbs and the left lower limb. Dr Wong said that those signs confirmed that the baby was suffering from neurological problems.

72 The baby's blood gases, taken on the same day, were found to be normal. The absence of acidosis (pH < 7) indicated that it was unlikely that the baby had intra-partum hypoxia.

73 In order to better ascertain the neurological status of the baby, Dr Wong ordered a CT scan (Brain) of the baby to be done. The CT Scan (Brain) was carried out by Dr Chang S K ("Dr Chang"), a consultant radiologist in private practice at Gleneagles Hospital. I shall return to his findings shortly. After the CT Scan (Brain) had been performed, the baby was sent back to the Neonatal Intensive Care Unit where Dr Wong performed a lumbar puncture in order to exclude meningitis.

74 From the lumbar puncture, Dr Wong extracted three bottles of uniformly blood-stained cerebro-spinal fluid, indicating the likelihood of the baby suffering from a subarachnoid haemorrhage.

75 After the lumbar puncture had been performed, Dr Wong observed that the baby suffered one attack of spinal arching with tonic upper limb extension, each lasting 5 to 40 seconds. A blood test was done immediately to ascertain whether the baby's seizure was possible due to low blood-glucose levels. However, Dr Wong noted that the blood-glucose at 4.1mmol/l was normal.

76 I now return to the CT scan performed by Dr Chang. After the CT scan, while interpreting the CT scan, Dr Chang commented verbally to Dr Chan that the scan was normal and there was no sign of fracture or brain oedema. Upon being told this by Dr Chang, Dr Chan conveyed this verbal report to the patient and her husband, Mr D. Dr Chan also told Dr Wong about Dr Chang's readings.

77 Dr Thomas Wong then informed Dr Chan that he had just done a lumber puncture and obtained blood-stained cerebro-spinal fluid. Dr Chan immediately informed Dr Chang of Dr Wong's finding of the blood-stained cerebro-spinal fluid.

78 Dr Chang re-examined the CT scan films and reported on 14 December 1998 in the following terms. "There is increased attenuation seen of the posterior interhemispheric fissure, suspicious of subarachnoid haemorrhage. Increased attenuation in the occipital horn of the right lateral ventricle is suggestive of blood. There is no intraparenchymal haemorrhage. The ventricles are normal in size. There is no midline shift". Dr Chang revised his earlier views. He explained and I accept that after his verbal provisional views were conveyed to Dr Chan he received the hard copy pictures of the CT scan (Brain) on 14 December 1998. From these hard copies he discerned that there was some slight shadowing, which was suspicious of subarachnoid haemorrhage.

79 I turn to Dr Wong's review of the baby on 13 December 1998. The baby's first urine was passed at 5.10am that morning. At the examination later that morning, Dr Wong noted that the baby continued to display symptoms of neurological abnormalities. She was still hypotonic, and tendon jerks were absent. The baby's pupils were small and her eyes tended to look up with divergent strabimus (squinting). Dr Wong noted that the baby's cries were sometimes aphonic and sometimes normal-toned.

80 The baby's first bowel movement was noted at 5.00pm. Thereafter the baby passed urine and stools regularly, indicating that she was not suffering from any renal or gastrointestinal abnormalities.

81 However, upon review at 6.45pm, the baby had a seizure, and a soft systolic murmur was heard at this point for the first time. Dr Wong thus recommended to the baby's parents that Dr Brenda Wong, a paediatric neurologist, should be consulted in respect of the baby's neurological abnormalities. The parents agreed and Dr Brenda commenced the management of the baby from 14 December 1998.

82 On 14 December 1998, Dr Wong received the CT Scan (Brain) report by Dr Chang, which stated that there was a suggestion of haemorrhage in the posterior inter-hemispheric fissure and right occipital horn.

83 Dr Brenda Wong assessed the baby as having a decreased and hypotonic neurological status. Further, at 5.05pm, the same day, a murmur (grade 3/6) was noted, and the baby was diagnosed as having a possible ventricular septal defect.

84 At 7.45pm, Dr Brenda Wong ordered another blood test to be conducted, which showed that the baby's blood counts, calcium and magnesium levels to be normal.

85 On 15 December 1998 Dr Wong observed that the baby was still quiet and depressed, but had normal movements. The blood tests conducted the previous day also showed coagulation screen was normal. It meant that the baby's blood clotted normally and that this was not the cause of the baby's haemorrhage.

86 As the baby's neurological status at this juncture was stable, Dr Wong ordered that she be started on milk feeds at 5cc three hourly. Thereafter, the feeds were increased steadily and upon discharge on 23 December 2002, the baby was able to suck 50cc per feed and her weight on discharge was 3130g.

87 On 22 December 1998, Dr Wong was able to elicit the baby's Moro reflect for the first time (meaning that the baby was coming out of her neurological depression). However, the baby remained hypotonic and was noted to turn her head preferentially to the left. Her cries were single and strained. On 23 December 1998, the baby was discharged.

88 Dr Wong summarised his diagnosis based on the consultations he had and the tests he had conducted. He stated that contrary to his provisional diagnosis of birth asphyxia, he concluded that the baby did not suffer from intrapartum hypoxia. His conclusions were based on his findings as

follows. First, the baby was pink at birth. Secondly, the baby was able to breathe on her own at birth and did not require intubation or assisted ventilation of her lungs. Thirdly, the baby's blood gases were normal and there was no profound metabolic acidosis (pH<7) at birth. Fourthly, the baby had Apgar scores of 6 and 7 at 1 minute and 5 minutes respectively. Fifthly, there was no evidence of multi-system dysfunction, such renal or liver failures which would happened if the brain, the most important organ, was deprived of oxygen. Lastly, there was no brain swelling on the CT scan (Brain).

89 The first MRI report was done on the baby on 24 March 1999, about three-and-a-half months after her birth. It showed that there was no significant abnormalities in the brain parenchyma. The report stated that the only abnormality was this: "The temporal horn of the right lateral ventricle is dilated. This is probably developmental in origin".

90 It is appropriate to summarise the recent medical condition of F who is about five years old. She unfortunately has multiple medical problems. She has hypotonic Cerebral Palsy with global developmental delay. She suffers from severe chronic lung diseases secondary to previous recurrent aspiration pneumonia related to gastroesophageal reflux. Her problems also include dysfunctional swallowing. She has a small peri-membranous ventricular septal defect.

91 One of her predominant problems, according to Dr Daniel Goh Yam Thiam, Consultant Paediatrician of the National University Hospital, lies in her chronic respiratory disease. She has poor respiratory reserves and has nocturnal hypoventilation. This is contributed by the chronic lung disease secondary to the previous multiple aspiration pneumonia as well as muscle weakness from her hypotonia. She is provided a mask interface with a machine to improve her breathing during the night hours. She is also on continuous supplemental oxygen *via* nasal prongs throughout the day.

92 According to Dr Goh, her neurological status remains much the same. She will be totally dependent for all activities of daily living.

Plaintiff's allegations

93 In this case, the broad allegation is that Dr Chan failed to consider properly the various signs of foetal distress at various stages of pregnancy with the result that she did not timeously intervene by a Caesarean section which, if done in time, would have prevented F from being inflicted with the neurological defects described in this judgment. In order to succeed in her claims for damages for negligence, the plaintiff has to prove (1) she was owed a duty of care by Dr Chan; (2) Dr Chan was in breach of that duty; and (3) the plaintiff suffered loss and damage in consequence of that breach. Those elements to constitute a cause of action in negligence may be described by way of abbreviation only as duty, breach and causation. So far as duties of care are concerned, which are set out below in the alleged breaches of duty, it was not in dispute that Dr Chan owed those duties of care. The two controversial aspects of the plaintiff's claims are in the form of two questions. First, did Dr Chan on a balance of probabilities breach any duty of care? Secondly, did Dr Chan's breaches, singly or in any combination, cause or materially contribute to the injury suffered?

94 According to the Amended Statement of Claim, the plaintiff alleges that Dr Chan:

(1) failed to consider the significance of placental calcification which was apparent in the 36th and 38th week;

(2) ignored or dismissed the placental calcification which progressed into placenta accreta, thereby complicating the 3rd stage of labour;

(3) failed to read or interpret correctly the CTG on 28 November 1998;

(4) failed to consider adequately the condition of the baby or to advise the mother to seek a further opinion (despite the mother's request to deliver the baby early);

(5) failed to deliver baby by Caesarean section due to failure to see meconium-stained liquor, thereby resulting in the baby having intranatal pneumonia; and

(6) failed to take any proper timely measures or revise method of delivery in light of abnormal CTG.

95 These allegations of the plaintiffs have to do with (a) Dr Chan's antenatal care; (b) Dr Chan Intrapartum care; (c) the vacuum delivery carried out by Dr Chan; and (d) the cause of F's neurological status.

Standard of care

96 To answer the questions whether Dr Chan had on a balance of probabilities breached any of the duties of care set out above, the test applicable was that laid down in *Bolam v Friern Hospital Management Committee* [1957] 2 All ER 118. A doctor would not be in breach of duty in attending to and treating his patient, if he had acted in accordance with a practice adopted as proper by a responsible body of medical men skilled in that particular field, notwithstanding that there was a body of opinion that might or would take the contrary view. This test has been modified by the decision in *Bolitho v City and Hackney Health Authority* [1997] 3 WLR 1151. It was held that "while assessment of medical risks was for medical experts to make, a judge could, in a rare case, disregard a body of opinion as not reasonable or responsible where it could not be logically supported": per LP Thean JA (as he then was) in *Yeo Peng Hock Henry v Pai Lily* [2001] 4 SLR 571 at 577I-A.

97 In relation to the law on causation, the plaintiff has to show that the breach of duty, or any combination of the breaches of duty, had caused or materially contributed to F's neurological defects. In *Bonnington Castings v Wardlaw* [1956] AC 613 at 620, Lord Reid said:

> It would seem obvious in principle that a pursuer or plaintiff must prove not only negligence or breach of duty but also that such fault caused or materially contributed to his injury, …

98 The following passage from *Clerk & Lindsell on Torts* (18th Ed), para 2-12 is useful when dealing with the evidence relating to causation:

> Proof that the defendant's fault caused the claimant's damage can become exceptionally problematic in two common forms of claim. These are (a) claims relating to liability for disease, particularly industrial disease and (b) claims where the scientific evidence suggests that the claimant's injury results from a combination of factors: "guilty" causes traceable to the defendant's fault, and "innocent" causes unrelated to any wrongdoing on the part of the defendants. Often these types of claims will overlap. Claims relating to disease are problematic because of the difficulties of establishing the aetiology of disease in any instance. … It may be contended that the condition has genetic origins, or results from environmental factors beyond the defendant's control, or is an inevitable result of the claimant's original disease. What is clear beyond doubt is that in action in tort, whether in negligence, breach of statutory duty or nuisance, the claimant must advance sufficient, convincing scientific evidence that the condition of which he complains was caused by the defendant's wrongdoing. The quality of the expert scientific evidence is all important.

Findings on the issues

99 Dr Lean Tye Hin, a Consultant Obstetrician and Gynaecologist, gave evidence for the plaintiff. He has been in private practice since 1977. For 18 years he was in service with the Ministry of Health. He is a Fellow of the Royal College of Obstetricians and Gynaecologists since 1965.

100 In his evidence, he referred to the affidavit evidence of Professor Philip Steer, Professor at the Academic Department of Obstetrics and Gynaecology, Imperial College Faculty of Medicine, Chelsea and Westminster Hospital. Professor Steer was of the view that the CTG trace done on 28 November 1998 showed "clear abnormalities and suggest(ed) that all was not well at that time". Professor Steer opined that the traces taken on 11 and 12 December 1998 showed "persistent uterine hyperactivity, almost certainly due to a combination of the use of prostalglandin and syntocinon to induce labour". According to Prof Steer, whose evidence was taken by video conferencing, the foetal heart rate pattern, according to the CTG tracings, became abnormal for long periods, showing evidence of umbilical cord compression and hypoxic stress.

101 Dr Lean noted that at the third ultra-sound scanning when the maturity was at 34 weeks, the baby was reported as small and there was noted "Placental Calcification" in the case notes. Thirty-five weeks into gestation, the foetus was reported as growing and that placental calcification was present with the comment attached to the notes "to observe". Quite out of context, Dr Lean noted that the ante-natal record were written in a dissimilar hand-writing and questioned if the findings were brought to the attention of or discussed with Dr Chan. Dr Lean did not give any basis for asides and, in my view, the less said about these most unneccesary innuendos, the better.

102 Dr Lean then went on to criticise the reporting of placental calcification seen at 34 weeks maturity. The report did not describe the extent, nor the diametic size of the calcifications. No reporting was made of the state of the placenta at the non-calcification site. This remark, to me, was completely gratuitous. He then speculated on the desirability of exploring the presence of placenta accreta. He further lamented that the placenta was not sent for histo-pathological assessment to obtain information in the search for "possible causes of the condition of this rather sick baby at birth".

103 Dr Lean said that his review of the first stage of labour showed what he described as "a few unusual manoeuvres". I wish that the text, tone and tenor of his expert evidence were more studied and restrained. I was unnecessarily irritated by the histrionics. By "unusual manoeuvres", he was referring *inter alia* to the insertion of Prostin E2, that Dr Chan had "ignored" the "flattish" CTG tracing without any basis for such a conclusion, meconium staining of the amniotic liquor, and the artificial rupture of the membrane which, he said, was too late. He opined that an amniotomy would have been a useful exercise at the outset of labour. It would have achieved early diagnosis of foetal distress and hastening the pace of labour. He said "[d]iscovering the presence of foetal distress early in labour could have prompted urgent delivery by a Caesarean section rather than the condition when labour was close to the commencement of the second stage". These speculations of what could have happened, unfortunately, denied the clinical findings of Dr Chan which in my judgment correctly recorded what she had diagnosed from time to time. Dr Lean further stated that Dr Chan had resorted to the use of the vacuum extractor and completed the second stage labour in 38 to 41 minutes and he volunteered the remark that "Dr Chan was in some hurry to deliver the baby". It would be idle to speculate on what he was suggesting; suffice it to say that such churlishness did not enhance his status as an independent expert giving evidence in a court of law.

104 Professor Philip James Steer gave evidence of his opinion on the foetal heart rate CTG traces of Mdm C taken on 28 November, 11 December and 12 December 1998. He is a professor at the Academic Department of Obstetrics and Gynaecology Imperial college Faculty of Medicine, Chelsea and Westminister Hospital. At the outset, Prof Steer made it clear that it was "unusual" for him to opine on the CTG tracings alone, "without reference to the clinical history". He conceded that the interpretation of the significance of the CTG tracings "may be dependent on the clinical events that they reflect[ed]". He said he would take care to limit his interpretation. In his affidavit, Prof Steer identified specific instances of "marked hypoxic stress" and "chronic foetal hypoxia and acidosis". However, when told in cross-examination of some of the findings of the ultrasound scan taken on the

same occasion as the CTG trace, he completely changed his views and said that he would have been "very reassured" by the findings and would not see a need to intervene in the pregnancy at that stage. There were a few matters which reduced the impact of the affidavit evidence of Prof Steer. First, it was pointed out to Professor Steer that he had reviewed a substantial portion of the CTG tracing twice over without realising it. As faxed copies were sent to him, and in the nature of CTG tracings which could be quite repetitive, the error may be understandable. The difficulty, however, was that Professor gave separate and at times different readings and interpretation to the same traces! In cross-examination, Prof Steer qualified his evidence by stating that his comments on the CTG tracings provide a description of what the tracings showed, and not necessarily an interpretation of what they mean. Moreover, in the context of his opinion, one would assume that Prof Steer, in using the word "hypoxia" he would be referring to hypoxia where an early delivery would have been necessary. In cross-examination, he claimed that by "hypoxia" he only meant less than ideal level of oxygen.

105 In contrast, the expert evidence of Dr Barry S Schifrin was far more impressive. He is a physician licensed to practice medicine in the State of California, USA. His analysis of the CTG tracings confirmed that there were "abnormalities of variability and a secondarily of baseline rate but no evidence of foetal hypoxia … As emphasised numerous times in the affidavits of others, there is simply no evidence of any systemic impairment of the oxygen available to this foetus during its entire labour". Dr Schifrin tendered his own analysis of the CTG tracings and these remained unassailable after his cross-examination.

106 Professor S Arulkumaran gave independent expert evidence for Dr Chan. He is Professor of Obstetrics and Gynaecology in the St George's Hospital Medical School, University of London. He had practiced Obstetrics and Gynaecology for approximately 15 years in Singapore up till August 1997. He was Chief of Services of Obstetrics and Gynaecology in the National University Hospital of Singapore and Head of the academic department of Obstetrics and Gynaecology in the National University of Singapore from August 1995 until August 1997. He enjoys an internationally-known status as a commanding authority on the management of high-risk pregnancy and labour. He is the author of "Fetal monitoring in Practice", and has many publications in established prestigious international journals. He totally disagrees with Dr Lean. In his opinion, "hypoxia is not the cause of the abnormal CTG and subsequent subarachnoid haemorrhage but rather subarachnoid harmorrhage was the cause of the suboptimal CTG throughout labour and the condition at birth". In addition, he believes that "subarachnoid haemorrhage was already present when the mother was admitted in labour without other etiological factors like hypoxia, chromosomal or major congenital malformation, medication or infection".

107 Professor Arulkumaran further stated that the subarachnoid haemorrhage was not due to vacuum delivery because "there was no external trauma of any degree to suggest the possibility of an internal trauma that is unusual to happen in isolation". This view must be correct, in my judgment. The vacuum delivery was performed by Dr Chan and it was not traumatic by any description.

108 Professor Arulkumaran expressed the unequivocal view that "the neurological outcome in the plaintiff was not due to hypoxia. Instead, it is likely to have been due to a pre-existing subarachnoid haemorrhage and the pathology that gave rise to that event, or due to some congenital malformation of the brain. My opinion is that such subtle pathology cannot be diagnosed antenatally and an early delivery or any other intervention by (Dr Chan) is unlikely to have resulted in a better outcome".

109 Professor Arulkumaran opined in conclusion that Dr Chan "managed Mdm C's pregnancy and the delivery of the plaintiff with care and due diligence".

110 Dr Ho Lai Yun also gave evidence for the defence. He is a Senior Consultant Paediatrican and Head of the Neonatology at the Singapore General Hospital. He dealt with the contention that

generalised hypoxia during the antenatal and/or intrapartum period was the cause of the plaintiff's injury. He further considered the plaintiff's further contention that subarachnoid haemorrhage was a cause of the injury. He said he had reviewed the documents. There was no evidence of continued foetal growth restriction and no signs of insufficient placental function as shown by the normal amniotic fluid index and umbilical blood flow.

111 Dr Ho further opined that the plaintiff was also unlikely to have suffered from intrapartum hypoxia resulting in hypoxic-ischaemic encephalopathy. He explained that during labour, if there was any significant hypoxia to the foetus, the other organs would be damaged before the brain. This was because the brain is a vital organ. It is protected from injury by drawing a larger share of oxygen supply from other organs. There was no brain damage by intrapartum hypoxia because there was no acidosis at birth, the baby did not have low Apgar scores at birth and there was no evidence of multi-system dysfunction (in particular, the renal function was normal after delivery).

112 To the question what had happened to the plaintiff, Dr Ho stated that the MRI reports obtained in this case suggested "multiple developmental abnormalities in the plaintiff's neurological system. These radiological findings, taken together with the other birth defects, such as dysmorphic features and a VSD, suggests that there was an event during the early development of the plaintiff which caused multiple abnormalities, leading to defects in the heart, facial features and the brain. This developmental abnormality is still evolving".

113 I now refer to the evidence of Dr Mary Rauff How Jing, who also gave evidence for Dr Chan. She is Associate Professor in the Department of Obstetrics and Gynaecology, Faculty of Medicine, in the National University of Singapore. She is also Senior Consultant in the Department of Obstetrics and Gynaecology at the National University Hospital. In Assoc Prof Rauff's opinion, the antenatal care that Dr Chan had provided for Mdm C was as good as any obstetrician could have provided. There was nothing in the antenatal period to give a hint that the foetus was going to have a spontaneous intracranial haemorrhage. When Mdm C went into labour, the earlier CTG was not abnormal but the subsequent trace when she was in established labour at 8.30pm onwards was very typical of a depressed foetus – one where there is evidence that the outcome after delivery was going to be poor. Dr Chan probably felt that the trace was non-reassuring but not abnormal.

114 Professor Mary Rauff said "fortunately for Dr Chan, she did not jump in and perform a Caesarean section. When we look at the case retrospectively with all the evidence before us, there was nothing we could have done to change the present state of the plaintiff. To have performed a Caesarean section may have pacified the plaintiff's mother as seeming to have done something positive, it would not have changed the present problems the plaintiff is having".

115 I accept the evidence of Dr Chan. Her evidence, which was supported by contemporaneous documents and tests, set out what had happened throughout the pregnancy and delivery. I have to say that I reject the evidence of both Dr Lean and Professor Steer. Dr Lean's blunderbuss approach, in which he hurled a number of allegations and innuendos which were all rebutted, did not commend his evidence to me. Prof Steer's opinion was so limited in scope and watered down in material respects that in the end his evidence did not assist the plaintiff's case at all. On the other hand, the probative force, analyses and logic of the expert opinions of the constellation of independent experts called by Dr Chan in her defence were impressive. I have no hesitation in accepting their evidence.

Conclusion

116 Being born is dangerous for the baby. An eminent professor told the trial judge in a case involving a baby born with brain damage this: "Throughout history, birth has been the most dangerous event in the life of an individual and medical science has not yet succeeded in eliminating that danger": see *Whitehouse v Jordan* [1980] 1 All ER 650 at 652J. In law, when a baby is still-born or

dies soon after birth or is born damaged or deformed, that fact in and by itself is no evidence of negligence on the part of the doctors or nurses attending the birth. It does not speak itself. The maxim *res ipsa loquitur* does not apply. In order to establish liability, and to obtain an award of compensation against a doctor or a hospital, it must be proved that on a balance of probabilities there has been negligence in law. In the field of professional negligence, there is no liability without proof of fault.

117 In my judgment, Dr Chan had competently managed the pregnancy of Mdm C and the delivery of F. What F has suffered and unfortunately will continue to suffer was due to developmental cause or causes which Dr Chan could do nothing about. Dr Chan had not breached any of the duties of care which have been alleged against her. Accordingly, the plaintiff's claims are dismissed with costs.

Plaintiff's claim dismissed with costs.