

**General Practitioner, Dr B**

**A Report by the  
Health and Disability Commissioner**

**(Case 00HDC12982)**



Health and Disability Commissioner  
*Te Toihau Hauora, Hauātanga*



## Parties involved

Mrs A	Consumer / Complainant
Dr B	Provider / General Practitioner
Dr C	Obstetric Specialist
Dr D	Obstetric Specialist
Dr E	Neonatal Paediatrician
Dr F	Obstetrician
Dr G	Obstetrician

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## Complaint

In December 2000, the Commissioner was notified by the Accident Compensation Corporation's Medical Misadventure Unit that it had accepted a claim by Mrs A on the basis that her son had suffered cerebral palsy as a result of 'medical error' by general practitioner Dr B. In June 2001, Mrs A confirmed that she supported an investigation of the following allegations:

- *Dr B did not exercise reasonable care and skill in his management of Mrs A's pre-natal care or labour.*
- *Due to Dr B's failure to refer on to a specialist during labour there was a delay in delivery and the baby sustained cerebral palsy and spastic quadriplegia.*

An investigation was commenced on 22 June 2001.

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## Information reviewed

- Relevant medical records from Dr B and the public hospital
  - ACC's file on Mrs A's claim for compensation as a result of medical error
  - Independent expert advice from general practitioner Dr William Ferguson
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## Information gathered during investigation

### *Summary*

Mrs A's second baby was born on 15 March 1998, a week after his due date. The baby was found to have suffered intrauterine growth retardation ("IUGR"). His weight at birth was

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2290 gms. The baby suffers from cerebral palsy and spastic quadriplegia, a form of spastic cerebral palsy which affects both his arms and his legs.

Mrs A applied to the Accident Compensation Corporation (“ACC”) for compensation, alleging that the baby’s condition was the result of medical error on the part of her lead maternity carer (“LMC”), Dr B. The Medical Misadventure Advisory Committee (“the Committee”) initially decided to recommend to ACC that the claim be declined. The Committee then considered further information from Mrs A, Dr B, and a number of obstetric specialists, and Mrs A’s claim was ultimately successful.

In accordance with section 5(1) of the Accident Rehabilitation & Compensation Insurance Act 1992 (“the Act”), ACC found that:

“[The baby’s] cerebral palsy, as a result of a failure to refer on to a specialist in the antenatal period and delay in delivery, is considered to have been due to a failure by the registered health professional to observe a standard of care and skill that was reasonable in the circumstances.”

Dr B applied to ACC for a review of its decision. His application for review was ruled invalid on the grounds that under the Act he had no review rights.

#### *Antenatal care*

In 1997, when Mrs A discovered she was pregnant with the baby, she chose general practitioner Dr B as her LMC. Dr B had delivered Mrs A’s first baby in 1995. Although her first baby was born weighing 2970 grams, Dr B advised the Committee that he did not consider the first baby’s small size a risk factor in Mrs A’s second pregnancy, as he had been a healthy baby and above the 10<sup>th</sup> percentile.

Mrs A had a positive pregnancy test on 7 July 1997. Dr B noted that her menstrual cycle was regular and her last menstrual period was on 31 May 1997, and he calculated her as being five weeks pregnant.

Mrs A had her next consultation with Dr B on 8 August 1997, at 10 weeks’ gestation. On that occasion Dr B performed a speculum vaginal examination and took swabs. At 14 weeks, on 5 September 1997, the foetal heart was heard for the first time and, at 18 weeks, on 2 October 1997, Dr B organised an ultrasound scan. According to the scan, which Mrs A had performed the following day, all foetal measurements were average for just under 18 weeks’ gestation, plus or minus 10 days. The measurements, therefore, were in agreement with the gestational age as calculated by Mrs A’s last menstrual period.

The findings of the scan were recorded as follows:

“There is a single viable intrauterine foetus with normal foetal heart activity and movements. Foetal anatomy is unremarkable. The placenta is posterior, fundal and is not lowlying. Liquor [amniotic fluid] appears normal. Foetal measurements are as follows:

BPD [head diameter]	39mm
Femur length	26mm
Abdominal circumference	124mm
Head circumference	149mm

All being average for just under 18 weeks gestation +/- 10 days.”

At 30 weeks, on 20 December 1997, Dr B recorded that Mrs A’s fundal height was 30cm. The fundus is the top of the uterus, and the fundal height is the measurement in centimetres from the pubic bone to the fundus. As a general rule, the fundal height (in centimetres) correlates with the gestational age of the pregnancy in weeks.

Dr B was away on holiday for Mrs A’s following two visits and she was seen by his locum. On 23 January 1998 the locum recorded that Mrs A’s fundal height was 34cm, which was consistent with the estimated gestational age of 33 weeks plus five days. He arranged for Mrs A to return to Dr B two weeks later.

At Mrs A’s consultation with Dr B on 9 February 1998, at 36 weeks’ gestation, Dr B recorded a fundal height of 34cm. He performed a scan of the baby’s head using an ultrasound machine in his rooms, and measured the baby’s biparietal diameter (the diameter of the head, or “BPD”) as 90mm.

Dr B advised the Committee that he routinely uses his own ultrasound machine to scan the foetal head at approximately 36 weeks, mainly to avoid the possibility of an undiagnosed breech presentation. He also uses his ultrasound machine to check for foetal movement, a heartbeat or twins. He advised:

“I am aware of my limitations but have made a number of useful diagnoses including ovarian tumour, enlarging uterine fibroid in pregnancy, several sets of twins and confirmation of early foetal deaths. Ultrasound scanning on referral is hardly ever available on a same day basis and some positive findings (for instance the detection of a foetal heart where one has previously been heard but is now absent) can be very reassuring to the patient.”

Mrs A advised the Committee that from about 36 weeks she was concerned about the size of her baby. She said she asked Dr B to refer her for a growth scan but Dr B assured her it was not necessary. Dr B could not recollect Mrs A requesting a scan. However, he advised the Committee that a fundal height of 34cm at 36 weeks was typical for a person of Mrs A’s build, “well within the normal range”, and did not indicate that a growth scan should be arranged.

Mrs A also advised the Committee that she mentioned to Dr B that midwives who saw her first baby after he was born wondered if he had suffered IUGR because he looked “overcooked”. Mrs A advised:

“Because of [those comments] I was worried it may have been happening in this pregnancy because I was not as big as last time – I knew something wasn’t right.”

According to Mrs A, Dr B responded to the effect that midwives forget what they learn in school and are “full of crap”. Dr B had no recollection of any such discussion and advised the Committee:

“I know that I would not have made the comment about midwives which [Mrs A] attributes to me. I do not believe that midwives forget what they learn and I would not say to a patient that anyone was ‘full of crap’.”

The following week, on 16 February 1998 at 37 weeks’ gestation, Mrs A’s fundal height was 33cm. According to Dr B, although this was 1cm less than the previous week, the baby’s head had descended and in his experience it was common for a fundal height to remain the same or diminish between visits if the head had descended.

At that consultation at 37 weeks, Dr B also re-measured the baby’s BPD. It was 93mm. Dr B was reassured because 93mm is the mean BPD measurement for 39 weeks, which indicated to him that the baby’s size was in advance of the gestation.

On 23 February 1998, at 38 weeks, the fundal height had increased to 34cm and the BPD was the same, at 93mm. At 39 weeks, on 2 March 1998, the fundal height was not recorded and the BPD was recorded at 94mm, which Dr B advised was larger than the mean for term. On 9 March 1998, at term plus two days, the fundal height was 35cm, and the BPD was not recorded. Therefore, between 37 weeks and term there was an increase of 2cm in fundal height, which according to Dr B was “an average increase in fundal height near term”. The Committee, however, noted that from 30 weeks’ to 40 weeks’ gestation, the fundal height increased by only 5cm.

Dr B advised the Committee:

“In my practice there is a wide variation in the absolute level of fundal height between patients at any given gestation. I take into account the size and race of the parents. I regard the rate of growth as a more important sign than the absolute level.”

Dr B advised that he did not measure the baby’s abdominal circumference or femur length because, like most general practitioners, he does not have the necessary training or skill to make reliable measurements. He did not feel that specialist review was indicated because he did not consider that his clinical assessment suggested abnormal foetal growth. Dr B was aware that with asymmetrical IUGR (where the baby’s brain is abnormally large compared to its liver), head growth was less affected than abdominal circumference. However, he did not believe that a baby with a term-sized head and an increasing fundal height would be significantly small.

#### *Baby overdue*

Mrs A went into spontaneous labour 10 days after the baby’s due date. She advised that she had asked Dr B whether she should be induced and had also questioned him about the possibility that the baby was losing weight in the last week, because that is how she had felt in her previous pregnancy. She advised:

“[H]e told me to go home and not to fret and gave me his home telephone number if I was worried about anything else. I had been worried for the past 4 weeks and he had had an answer, of sorts, for all of my concerns. This was the last time I saw him in his rooms and looking back I find his attitude towards my questions and concerns condescending and dismissive.”

In a letter he wrote to Mrs A, Dr B stated:

“I am painfully aware that if I had referred you for a specialist opinion in pregnancy that your baby may have been delivered early and may not now have cerebral palsy.”

#### *Labour*

On 15 March 1998 at around 4pm, Mrs A started having contractions. By 8.20pm, when she was admitted into the delivery suite at a public hospital, Mrs A was in early established labour. There had been a history of reduced foetal movements that day, although Mrs A had still noted ten “kicks”.

On admission into the delivery suite, a staff midwife performed an abdominal palpitation which was normal. She commenced a CTG (a cardiotocograph, used to record the foetal heart rate to monitor the progress of labour and, in particular, the wellbeing of the foetus), and the baseline recordings were initially normal. However, as Mrs A’s labour progressed the staff midwife noted that the foetal heart rate baseline was 165 beats per minute and there were early decelerations (periodic decreases in the foetal heart rate). She notified the acting charge midwife, who administered oxygen via a face mask and contacted Dr B.

Dr B arrived in the delivery suite at approximately 8.50pm. He advised the Committee that the CTG showed a borderline tachycardia (rapid heart rate) and deep Type I dips which mirrored the contraction intensity. Dr B did not consider that the CTG indicated a need for urgent action. He decided to continue monitoring the CTG to see whether the early decelerations and borderline tachycardia resolved or whether more serious signs developed. He also considered it appropriate to observe some further contractions before performing an internal examination.

Dr B advised that although there were four more early decelerations, there were also several occasions when the depth of descent was not so great. At that stage, 9.20pm, the monitor was disconnected so that Mrs A could go to the toilet.

At approximately 9.30pm Dr B performed a vaginal examination and found that Mrs A was 2cm dilated with an effaced posterior cervix (the baby’s head was facing down) and the head was at station –3 (the station refers to the degree of engagement of the baby’s head). He continued to monitor the contractions. He advised:

“A period followed where there was no deceleration with a contraction. I hoped this indicated that the trace might improve. However there followed several more deep early decelerations with prompt recovery to a rate exceeding the usual baseline. I was becoming increasingly concerned that there had been no improvement in the trace and decided there was a need for specialist review. I did not believe that the foetus was in

imminent danger but it seemed increasingly unlikely that the situation would spontaneously improve.”

At approximately 9.50pm Dr B ascertained that Dr C was the obstetric specialist on call, and telephoned Dr C at his home. Dr B detailed Mrs A’s relevant history, and described the status of the CTG and his examination findings. It was agreed that Mrs A be transferred to the care of the hospital team. Dr C asked Dr B to phone the on-call registrar and ask her to see Mrs A. Dr B then paged the on-call registrar on her locator. When she phoned Dr B back, he gave her the same information he had given Dr C and she advised that she would come to see Mrs A. The registrar subsequently telephoned Dr C at home to say she was busy resuscitating a patient in the Emergency Department and therefore could not attend to Mrs A. Dr C then drove to the public hospital.

At 10pm Dr B returned to Mrs A, recorded what he had done in the notes, and continued to monitor the CTG trace. Initially it showed an early deceleration down to 60 beats per minute for more than 30 seconds, but this was followed by several contractions without deep dips. Dr C arrived in the delivery suite at approximately 10.20pm, reviewed the CTG trace and examined Mrs A. He noted that the CTG was abnormal and had been since 8.30pm. He advised Mrs A that a Caesarean section was required, and recorded this in the notes at 10.30pm. Dr B assisted with the Caesarean preparations by taking blood, placing an IV line and writing up a consent form. The CTG monitoring continued until 10.53pm when Mrs A was taken to theatre. Dr B did not attend the delivery.

The baby was delivered at 11.14pm, weighing 2290gms. The baby’s Apgar scores were 3 at 1 minute and 9 at 5 minutes. The Apgar score is a measurement of a newborn’s response to birth and life outside the uterus, and is based on appearance (colour), pulse (heart beat), grimace (reflex), activity (muscle tone), and respiration (breathing). A baby whose total score is under 4 is in serious danger and needs resuscitation. A score of 4 to 6 means that the baby may need supplementary oxygen and his or her airway cleared. A score of 7 to 10 is considered good.

The baby was intubated twice and thick meconium (the first bowel motion) was suctioned from below his vocal cords. He was administered oxygen with positive pressure ventilation, as well as naloxone, a drug used to stimulate breathing. According to his hospital records, the baby showed signs of respiratory distress secondary to meconium aspiration. He also showed signs of IUGR, hypoxic ischaemic encephalopathy (an abnormal neurological state resulting from the brain being starved of oxygen) and convulsions, metabolic acidosis secondary to birth asphyxia (excess body acid due to oxygen starvation), and hypoglycaemia (low blood sugar).

The baby suffers from cerebral palsy and spastic quadriplegia, which affects every aspect of the family’s daily life.

*The Committee’s initial recommendation to ACC*

Mrs A filed a claim for compensation on the basis of medical misadventure, alleging that the baby’s cerebral palsy and spastic quadriplegia were the result of a delay in delivery attributable to Dr B.



The Committee's initial decision was to recommend to ACC that there was no medical evidence to establish a causal link between the alleged delay in delivery and the baby's cerebral palsy. In the Committee's unanimous view, there was no evidence to establish that an acute intrapartum event (an event occurring during delivery) had led to the baby's poor outcome.

In forming its initial view, the Committee had regard to the International Cerebral Palsy Task Force consensus statement regarding causal relationships between acute intrapartum events and cerebral palsy, developed under the auspices of the Perinatal Society of Australia and New Zealand.

The Committee noted:

"The [statement] records that contrary to previous beliefs and assumptions, clinical epidemiological studies indicate that in the majority of cases the events leading to cerebral palsy occur in the foetus prior to the onset of labour or in the newborn following delivery."

The International Cerebral Palsy Task Force statement sets out three essential criteria and five further criteria that together suggest an intrapartum timing but by themselves are non-specific. The three essential criteria are:

1. Evidence of a metabolic acidosis in intrapartum foetal, umbilical arterial cord or very early neonatal blood samples. pH < 7.00 and base deficit greater than or equal to 12mMol/L.
2. Early onset of severe or moderate neonatal encephalopathy in infants equal to or greater than 34 weeks' gestation.
3. Cerebral palsy is of the spastic quadriplegic or dyskinetic type.

The five non-essential criteria are:

1. A sentinel hypoxic event occurring immediately before or during labour.
2. A sudden rapid and sustained deterioration of the foetal heart rate pattern, usually following the hypoxic event where the pattern was previously normal.
3. Apgar scores of 0-6 for longer than 5 minutes.
4. Early evidence of multi-system involvement.
5. Early imaging evidence of acute cerebral abnormality.

The Committee noted that the baby's case did not meet the first of the essential criteria, since the cord arterial blood sample taken from the baby after birth did not meet the required pH of less than 7.0 and base deficit greater than or equal to 12, and a capillary blood sample taken when the baby was 40 minutes old met only one of those two index points (ie, the base deficit was 13). The Committee further noted that the baby's case did not meet the first three of the five non-essential criteria. It noted that the fourth of the non-essential criteria had "probably occurred", and that in relation to the fifth of the non-essential criteria, early imaging had not been obtained. The Committee therefore decided to recommend to ACC that Mrs A's claim for medical misadventure be declined.

*Responses to initial recommendation*

Before finalising its decision, the Committee invited Mrs A and Dr B to comment on its initial recommendation. Mrs A provided the Committee with reports from Dr C, the obstetrician who delivered the baby, and Dr D. Dr B did not provide comment. The Committee then asked two of its members – neonatal paediatrician Dr E and obstetrician Dr F – to comment on the reports of Dr C and Dr D.

In Dr C's opinion, there had been a "definite delay in seeking specialist help when the CTG was noted to be abnormal". According to Dr C, on arrival at the hospital Dr B "clearly misinterpreted the CTG, thought this was normal and decided to wait and monitor". Dr C also advised the Committee that when Dr B called him more than an hour later, Dr B told him he had seen CTG changes such as the baby's at other labours, and the babies had all been well at delivery. In Dr C's opinion, the baby suffered hypoxemia long and severe enough to cause brain damage.

Dr C also provided the Committee with his opinion on Dr B's antenatal care, and whether it would have been possible to detect that Mrs A's baby was small and therefore at risk. Dr C advised the Committee:

"It is well documented that caregivers miss up to 40% of small for dates babies. The use of fundal height measurements improves detection. I usually order a growth scan if the discrepancy between the fundal height and gestational age was 4 or more centimetres before 36 weeks' gestation. Other parameters in the past obstetric and medical history and current pregnancy need to be considered in the equation. There was a discrepancy of four centimetres at 38 weeks' gestation in [Mrs A's] records, but [Dr B] had explained that this was due to foetal descent. At 40 weeks' gestation fundal height was only 35cm, and would probably have raised suspicion of growth restriction, but I would not be too critical of [Dr B's] judgement not to perform a growth scan. However I am critical about the fact that he was measuring biparietal diameters in his rooms, and using his measurements to reassure himself and [Mrs A] that the baby was growing well. He had obviously suspected that the baby was not growing well, otherwise he would not have performed BPD scans. In my opinion, he should have ordered a growth scan to be performed by a radiologist, or someone qualified in performing growth scans."

In his report to the Committee, Dr D acknowledged that the interpretation of CTGs was "notoriously difficult even for experts". However, he advised that it was "prudent when any abnormality is evident to seek advice". In his opinion, Dr B was "at fault" by waiting until 9.50pm to refer Mrs A to Dr C. According to Dr D, Dr B should have referred Mrs A when he arrived in the delivery suite at 8.50pm.

Dr D did not agree with the Committee's finding that there was "no evidence" of an acute intrapartum event leading to the baby's poor outcome. In his opinion, it was a finding that "the evidence undermine[d]". Dr D agreed that only two of the three essential criteria in the consensus statement had been met. However, he noted that the capillary blood sample taken when the baby was 40 minutes old met one of the two index points, in that it showed a base deficit of 13mMol/L. According to Dr D, the result was obtained at a time that met the statement's definition of "very early".

In relation to Dr B's antenatal care, Dr D considered that Dr B was "at fault" in not obtaining a specialist opinion at or about 38 weeks' gestation. In Dr D's opinion, the antenatal record showed "clear evidence of intrauterine growth restriction from 37 weeks of pregnancy". Dr D stated:

"[Dr B] was concerned enough to carry out several BPD measurements, but did not seek further advice. This baby should have been delivered at term, under specialist supervision. (Such action would not however, have guaranteed that cerebral palsy would not be evident soon after birth.)"

After considering the reports of Dr C and Dr D, Committee member Dr E considered that there was medical error on the part of Dr B. He reached that view on the basis of Dr C's opinion that there had been a "definite delay in seeking specialist help when the CTG was noted to be abnormal", and Dr C's criticism of Dr B measuring the baby's BPD in his own rooms.

Committee member Dr F considered the reports of Dr C and Dr D and concluded that there had not been medical error. With regard to Dr B's antenatal care, Dr F stated:

"[Dr B] was clearly concerned about fetal growth and he obviously failed to appreciate the degree of growth restriction in view of [the baby's] extremely low birth weight. However, there was no history of antepartum bleeding or hypertension, fetal movements were satisfactory and [Dr B] did take steps to measure fetal growth although he did not order a growth scan to be performed by a specialist radiologist. A significant number of small for dates babies are not diagnosed prenatally and this cannot be considered to be medical error on the part of [Dr B]. [Dr D] comments that a specialist opinion should have been obtained at 38 weeks gestation and that the baby should have been delivered at term but concedes that such action would not have guaranteed that cerebral palsy would not be evident soon after birth. While in hindsight, specialist intervention may have resulted in a different outcome, I cannot find that this amounts to medical error on the part of [Dr B]."

In relation to Dr B's intrapartum care, Dr F continued to be of the view that the consensus statement criteria had not been met and that there was no evidence established of an acute intrapartum event leading to the baby's poor outcome. He stated:

"While there was a delay of approximately one hour before referral [to Dr C], it cannot be held that this delay was, on that balance of probabilities, the causal factor in [the baby's] poor outcome."

*The Committee's revised recommendation to ACC*

The Committee considered the additional reports from Dr D, Dr C, Dr E and Dr F, as well as Mrs A's antenatal records. I note that Dr F did not sit on the Committee that considered those reports, but had been replaced by another obstetrician.

In relation to Dr B's antenatal care, the Committee found:

“In examining the antenatal records, the Committee is of the view that they show that there was a significant fall off in foetal growth as evidenced by the failure of the fundal height to grow appropriately on measurement. In particular the Committee would note that from 30 weeks gestation to 40 weeks, there was only 5cm growth in fundal height. The Committee would further note that [Dr B] used antenatal ultrasound measurements of the head and specifically of the biparietal diameter as reassurance concerning foetal growth. It is well known that head growth is spared by comparison with abdominal circumference, which is the appropriate measurement for growth, or in the assessment of growth retardation. In addition, adequate assessment of the liquor volume was not undertaken. This false reassurance from the biparietal diameter measurements misled [Dr B] into accepting that foetal growth was satisfactory, when it was not and appropriate ultrasound and specialist consultation was not undertaken. The Committee is of the view that [Dr B's] antenatal care fell below the standard of care and skill reasonably to be expected in the circumstances.”

The Committee's revised decision was to recommend to ACC that there was medical error on the part of Dr B, both in relation to his antenatal care and the events surrounding the birth.

*Dr B's response to the Committee*

Before finalising its decision, the Committee again invited Mrs A and Dr B to comment on its revised recommendation. Dr B responded.

Dr B disputed Dr C's assertion that he had “clearly misinterpreted the CTG, thought this was normal and decided to wait and monitor”. Dr B reiterated the description he had given of the CTG in initial correspondence to the Committee, as follows:

“The CTG showed deep type 1 dips which mirrored the contraction intensity. I did not believe that the CTG indicated a need for urgent action but that recording should continue to see whether the early decelerations and borderline tachycardia resolved or more serious signs developed.”

In relation to Dr C's advice to the Committee that Dr B had told him he had seen CTG changes such as the baby's at other labours, and the babies had all been well at delivery, Dr B responded:

“I was shocked when I read this. I did not say this nor anything resembling it.”

With regard to Dr C's and Dr D's comments on his use of BPD measurements, Dr B denied that he had been using the measurements as a substitute for a radiologist's assessment. He further advised:

"I did not do the BPD measurements specifically to exclude IUGR. If I had [had] serious concern regarding the growth rate I would have ordered a scan or arranged referral to [the public hospital's] Fetal Assessment Unit. The totality of the evidence of clinical assessment, SFH [fundal height] measurements and BPD measurement were sufficient for me to believe that no further action was required."

In relation to Dr D's view that the antenatal record showed "clear evidence" of IUGR from 37 weeks, Dr B stated:

"I do not believe that [Dr D] is able to take a series of fundal height measurements and be any more certain of a diagnosis of growth restriction than the researchers who have applied themselves diligently to the refinement of this method and have been able to show only poor sensitivity and specificity. To my knowledge none of them have developed rules which can 'make a diagnosis'. At the very best careful application of fundal height charts will still miss more than 25% of small babies and more than half of those referred will be later found to be of normal size.

I am told by local specialists that serial fundal height measurements would be used by approx[imately] 50% of antenatal attendants. This variation in practice may be because the proper use and interpretation of fundal height measurements is controversial and the benefit of the practice is unproven."

Dr B provided the Committee with a number of articles on the use of clinical assessment, fundal heights and BPD to screen for babies small for their gestational age ("SGA"), which he had obtained through a search of the medical literature. Based on those articles, Dr B stated that clinical assessment alone was an insensitive means of detecting SGA, and the effectiveness of fundal height measurements was unproven, but probably better than clinical assessment alone. He stated: "Even using both methods 50% of SGA babies may be missed." Dr B concluded from his literature review that the evidence suggested that BPD measurements alone were at least as sensitive as fundal height measurements and clinical assessment in detecting SGA.

In relation to the Committee's conclusion that "false reassurance from the biparietal diameter measurements misled [Dr B] into accepting that foetal growth was satisfactory, when it was not", Dr B responded:

"Episodes of 'false reassurance' (false negative results) occur from time to time in screening and in medicine and do not imply error on the part of the clinician. The committee's statement is accurate but it has no bearing on the quality of the clinical decision. I believe to conclude otherwise is to connect the quality of the decision with the outcome. It is an unfortunate reality in medicine that doctors can be occasionally reassured by factors which in hindsight led them away from the correct diagnosis. The normal ECG in chest pain. The absence of fever in an unwell child. The key issue

seems to me to be whether my interpretation of the fundal height measurements was reasonable and whether it was reasonable to take reassurance from the BPD measurements.”

*Further responses to the Committee*

The Committee considered Dr B’s response. Mrs A provided the Committee with her own comments on Dr B’s response, as well as further reports from Dr C and Dr D.

According to Dr D, Dr B’s response to the Committee had expressed the view that measuring fundal height was a “flawed” method of assessing fetal growth. While Dr D accepted that the accuracy of the method was “not absolute”, he stated that the value of measuring fundal height was that abnormal growth would direct the doctor’s attention to the need for further, more accurate, investigation. Dr D stated:

“If [Dr B] has so little faith in this method of fetal growth assessment then why did he fill [in] the column [for recording fundal height] on five occasions (including the 36 weeks measurement) on his antenatal record card?”

In Dr D’s view, Dr B had “missed the point” by criticising individual fundal height measurements as being of little value. Dr D stated that the serial measurement of fundal height enabled a practitioner to detect insufficient growth over a period of time. Dr D considered that the increase in fundal height of only 3cm from 30 to 37 weeks should have led Dr B to seek specialist opinion by 38 weeks at the latest.

In relation to Dr B’s submissions to the Committee regarding fundal height measurements, Mrs A responded:

“[Dr B’s] latest report technically rules out the safe use of fundal height measurements in checking on the growth/health of a baby – what then, was he using to determine satisfactory growth? Measurements of the head? [the baby] was born with a head size [circumference] of [33cm] (at the very bottom end of the scale) – 2 whole centimetres smaller than my first baby – which is a lot.”

Dr C responded that Dr B did not have the training to perform fetal ultrasound measurements. Dr C also emphasised that Dr B had not recorded in Mrs A’s notes any concern about the CTG trace, and there was evidence of a temporary cessation of fetal heart monitoring. He stated: “Further, the wait of more than an hour is a concern.”

*Dr B’s further comments to the Committee*

In response to Dr D’s submission, Dr B disputed that he had suggested that fundal height measurements were of no value. He stated:

“I believe that the review of the literature demonstrated there are no universally accepted rules for identifying abnormal growth and that the measurements recorded by myself fell within the accepted range for most [clinical] trials where parameters for referral had been established.”

Dr B asked an obstetrician, Dr G, to review the Committee's decision to recommend that Mrs A's claim be accepted as medical error. In relation to Dr B's antenatal care, Dr G submitted that the Committee should accept Dr B's view that the baby was growing normally, and that Dr B had some years' experience to be competent to make that clinical decision. In relation to the delay seeking consultation in labour, Dr G noted that the Committee's decision hinged on the advice given by Dr C and Dr D. Dr G stated:

"My own view is similar to that of [Dr F] who gave independent advice to the Committee. The CTG recording is abnormal but the features are not diagnostic. The lack of baseline variability at the onset of the CTG may well suggest a pre-existing insult to the baby. There is substantial doubt that earlier referral and delivery would have altered the outcome and again that makes sustaining a finding of medical error rather difficult. To hold that view requires evidence that the delay was causative to [the baby's] poor outcome. It is not sufficient to appreciate retrospectively that there was delay.

There is circumstantial evidence that there was no feeling of an acute crisis at the time that [Dr C] was called to see Mrs A. Although he recognised the CTG as abnormal and decided for caesarean section, the baby was not delivered until 45 minutes later.

Incidentally, no comment was made by [Dr C] of the baby's size, which might be construed to reinforce the difficulty of recognition of growth retardation in pregnancy. The paediatric attendant at delivery was a neonatal nurse practitioner and I would have expected, if there was serious concern for fetal wellbeing, that a more senior paediatrician would have been asked to attend. It appears to me that the degree of compromise that was evident when [the baby] was born surprised all of the birth attendants.

I must also comment about the acid-base status of [the baby]. The only measure that can be used to give any indication of intrapartum events is the arterial cord blood sample, which shows a moderate metabolic acidosis but not sufficient to meet the criteria to define an acute intrapartum hypoxic event. It is also evident from the paediatric notes that the early neonatal course was not straightforward and that [the baby] had problems with meconium aspiration and ongoing metabolic acidosis. The pH in arterial blood at 3.50am on the morning following birth is still 7.15 with a base excess of -11, which raises the possibility that postnatal events may well have contributed to his outcome.

I believe it would be quite wrong for the Committee to use the capillary sample at 40 minutes of age as evidence of acute intrapartum hypoxia when an umbilical cord arterial sample is available."

#### *ACC final decision*

ACC accepted the Committee's final recommendation and accepted Mrs A's claim for compensation on the grounds of medical error. In its final recommendation to ACC, the Committee stated:

“The Committee remains of the view that there was sufficient evidence in the antenatal record as far as fundal height measurements were concerned, that it would have been appropriate for [Dr B] to have undertaken formal assessment of foetal growth in the late antenatal stage, and to have considered specialist referral had this confirmed growth retardation.

In the event, [Mrs A] was allowed to come into spontaneous labour, significantly post term; and [Dr B] failed to appreciate the significance of the cardiotocographic tracing at the time of [Mrs A’s] admission. The Committee has noted previously that the CTG trace on commencement showed a baseline tachycardia with a reduced variability and relatively deep, early decelerations. This was clearly an abnormal trace right from the time of [Mrs A’s] admission.

As stated previously, having considered the additional information before it, the Committee remains of the view that in his failure to diagnose foetal growth retardation and in the delay in seeking specialist consultation, [Dr B] failed to exercise a standard of care and skill reasonably to be expected in the circumstances. Our recommendation remains that this claim be accepted as error on the part of [Dr B].”

#### *Events subsequent to ACC’s final decision*

The Committee received a further submission from Dr B, who reiterated his view that the baby’s case did not meet the standards for intrapartum injury set out in the International Cerebral Palsy Task Force’s consensus statement, and that his interpretation of fundal height measurements by reference to published standards was reasonable.

The Committee’s Chairperson reviewed the submission. In her opinion, it did not contain any further information that would alter the recommendation to accept the claim.

Dr B subsequently applied to ACC for a review of its decision. Section 89(3) of the Accident Rehabilitation and Compensation Insurance Act 1992 (“the Act”) provides that a registered health professional who is dissatisfied with a decision of ACC made under subsection 6 or subsection 7 of section 5 of the Act may apply for a review of the decision.

ACC’s decision in relation to Mrs A’s claim was made pursuant to section 5(1) of the Act. ACC ruled, therefore, that Dr B’s application for review was invalid. Dr B disputed that decision and the issue of Dr B’s eligibility for review was the subject of a hearing before the Dispute Resolution Services (“DRS”). After considering submissions from Dr B and ACC and reviewing the relevant law, the DRS reviewer found that Dr B was not entitled to a review, and confirmed ACC’s decision. The DRS reviewer noted that he had “reservations over Dr B’s lack of ongoing redress under the Act”.

#### *Dr B’s response to current investigation*

In response to notice of this investigation, Dr B summarised his position as follows:

“I believe that my care in the antenatal period was reasonable. The diagnosis of growth retardation is often not made and cannot reasonably be expected to always be made. I believe my interpretation of the clinical findings in the antenatal period was reasonable.



...

Reference to the consensus statement of the International Cerebral Palsy Task Force referred to by the first MMAC [Medical Misadventure Advisory Committee] clearly shows that this case does not even meet the standard for consideration of intrapartum injury. The 2nd MMAC did not rebut this finding but have still concluded that cerebral palsy was caused by intrapartum injury.”

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## **Independent advice to Commissioner**

General practitioner Dr William Ferguson was asked to provide independent advice on the care Dr B provided to Mrs A. I note that the medical practitioners who advised ACC’s Medical Misadventure Committee were all obstetric specialists. I have sought the advice of a general practitioner because, in forming my opinion on whether Dr B provided services with reasonable skill and care, I am required to consider the care he provided in relation to the standards required of general practitioners, not specialists. Therefore, I considered it appropriate to seek independent advice from one of Dr B’s peers.

Dr Ferguson provided the following independent advice:

### **“Antenatal Care**

[Mrs A’s] baby had severe intrauterine growth retardation and the clinical observations recorded by [Dr B] reflected this reality. It has already been correctly pointed out by [Dr C] that IUGR is commonly missed with routine antenatal care. However in this case the concerns raised by [Mrs A] about the baby’s growth, and the false reassurances provided by [Dr B] as a consequence of his incomplete training in the use of ultrasound, amounts to an inadequate standard of care.

Unfortunately [Dr B] had insufficient skills with ultrasound to convert his concerns about the baby’s growth into a reliable interpretation of the ultrasound findings. It shows a very deficient understanding of abnormalities of foetal growth to assume that normal growth of head circumference parameters provides any assurance at all of satisfactory foetal growth and uteroplacental perfusion [the blood flow between mother and baby].

The appropriate response to any clinical concerns about foetal growth would be referral to a radiologist qualified to assess foetal growth. Detection of IUGR would then have prompted specialist referral, and most probably induction of labour prior to term. Early antenatal intervention may have provided the possibility of a better outcome.

### **Intrapartum care**

In retrospect of the delivery of a baby with obvious brain damage it would be very easy to emphasise the abnormal features of this CTG trace, and assume that more rapid intervention would have improved the outcome.

However I believe [Dr B's] management of [Mrs A's] labour was reasonable. I do not believe that any alterations in the management of this labour would have significantly changed the outcome. The CTG was abnormal, but does not in fact reach criteria outlined in the Royal College of Obstetricians and Gynaecologists guidelines [Appendix 1], or in the [women's public hospital's] guidelines [Appendix 2], that would trigger immediate delivery. When these standards of interpretation are applied the recommended advice is 'further evaluation, consider delivery, perform fetal blood sample (if reassuring, repeat in one hour)' or words to that effect.

In reaching this conclusion about the CTG I feel the following points are relevant:

1. There is no obvious deterioration in the trace over the one hour leading up to the time of delivery. This would be the sine qua non of an acute hypoxic insult in progress.
2. The decelerations are neither 'late' nor variable and thus do not signify acute hypoxia. They are classical type 1 or 'early' decelerations, suggesting vagal stimulation caused by foetal head compression, associated with the contractions.
3. The trace is abnormal because the baseline appears slightly high, rather fixed and the variability is reduced. These changes have been described in the literature in babies with pre-existing neurological damage, although in this regard I don't believe they could in any way be interpreted as conclusive. The 'abnormalities' are not sufficiently striking that immediate, urgent action was necessitated. The trace could be technically described as 'reactive' (and therefore healthy) as there are transient elevations of FHR of >15bpm for longer than 15 seconds. This interpretation would have provided some understandable (but false) reassurance that all was well. In a multiparous woman who may well have been progressing very rapidly in labour, this CTG could reasonably have been observed expectantly for a short period of time. Appropriate management at this point would have been vaginal examination and assessment of the amniotic fluid by rupturing the membranes.

The fact that delivery took just under one hour from the arrival of the specialist suggests even the specialist did not think in the first instance that this was an immediately life-threatening abnormality. CTG appearances commonly associated with acute severe hypoxic brain damage would have prompted a 'crash' LSCS that would have seen the baby delivered in less than half this time. A true test of specialist opinion of this CTG would have been if [Mrs A] was assessed and found to be 8–9 centimetres dilated. I believe responsible management would have been to await vaginal delivery, and perform foetal scalp blood sampling if the facilities were available.

Subsequent developments confirm that this baby suffered from a chronic hypoxic/ischaemic encephalopathy, and that the damage was existent prior to the onset of labour. I believe the evidence for this is as follows:

1. Severe intrauterine growth retardation and reduced amniotic fluid volume (evidenced by the thick meconium) is proof of severely reduced uteroplacental perfusion over at least several weeks prior to the onset of labour. This provides a more likely scenario for brain injury than the brief events of this labour.
2. An apgar score of 9/10 at 5 minutes, an umbilical artery blood pH 7.16 and base excess of -11 is unlikely to be compatible with an acute hypoxic cerebral injury occurring over the hour before birth with sufficient severity to cause the baby's brain damage.
3. The neonatal CT head scan showed evidence of cerebral atrophy and this is absolute confirmation of pre-existing neurological damage, unrelated to the events of the birth.

**Answers to the Specific Questions:**

[What are the specific standards that apply and were they followed?]

1. Relevant standards are outlined in the text above.

[In your opinion, did [Dr B] appropriately manage [Mrs A's] antenatal care?]

2. [Mrs A's] antenatal care was not appropriately managed.

[Was it reasonable in the circumstances that [Dr B] did not diagnose foetal growth retardation in the antenatal period?]

3. Fetal growth retardation is commonly missed with routine antenatal care. [Dr B's] error lay in using a diagnostic modality, ultrasound, in which he was inadequately trained and then relying upon this to alleviate any concerns he or [Mrs A] had about the baby's growth.

[Was it reasonable in the circumstances that [Mrs A] was not referred for specialist assessment in the late antenatal stage?]

4. Recognition of the diagnosis of Intrauterine Growth Retardation would have led to specialist referral. The error was in not making the diagnosis.

[In your opinion, did [Dr B] appropriately manage [Mrs A's] labour?]

5. I believe the management of [Mrs A's] labour was appropriate.

[What did the CTG tracings at the time of [Mrs A's] admission show and were they normal or abnormal?]

6. The CTG abnormalities witnessed in the hour prior to birth were not evidence of severe, acute hypoxic stress, but were a reflection of the already brain damaged foetal state.

[Was it reasonable in the circumstances that [Dr B] did not call [Dr C], the on-call specialist, until approximately 9.50pm?]

7. For the reasons outlined above the CTG did not suggest an immediate life threatening situation. Appropriate management would have been observation for a short period to note any progression of the CTG changes, followed by vaginal examination, rupture of the membranes and then specialist consultation.

[Mrs A] states in her evidence ‘I was worried [IUGR] may have been happening in this pregnancy because I was not as big as last time’. There is an old saying in medicine ‘Always listen carefully to the patient, he or she is telling you the diagnosis.’ In maternity care this saying is particularly true, and any persisting concern that a patient has must always be very carefully considered. In my experience any woman who thinks she is either significantly bigger, or significantly smaller than she was in her last pregnancy is usually correct. This simple observation is very important as the vast majority of bad obstetric outcomes occur in babies that are either too big or too small.

In this case the past history of a baby under 3000gm coupled with the mother’s concerns and the clinical findings should have raised the alarm. I believe if [Dr B] had relied on his clinical skills and not been confused by his use of ultrasound, he may well have made the correct diagnosis. The time for intervention in this case was a month before delivery.”

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## Appendix 1 – Royal College of Obstetricians and Gynaecologists Guidelines

**Table 4. Recommended classification of fetal heart rate features and cardiotocograph appearance**

Fetal heart rate features	Baseline (bpm)	Variability (bpm)	Accelerations	Decelerations
Normal	110-160	≥ 5	present	none
Non-reassuring	100-109 161-180	<5 for ≥ 40 but <90 minutes		early variable single prolonged ≤ 3 minutes
Abnormal	<100 >180	<5 for ≥ 90 minutes		atypical variable late single prolonged > 3 minutes

**CTG classification.**

Normal CTG	all four features are reassuring
Suspicious CTG	one CTG feature is non-reassuring, the remainder are normal
Abnormal CTG	$\geq 2$ non-reassuring features or $\geq 1$ abnormal feature

**Source:** Royal College of Obstetricians and Gynaecologists. The use of electronic fetal monitoring. The use and interpretation of cardiotocography in intrapartum fetal surveillance. Evidence-based Clinical Guideline Number 8. London: RCOG Press, 2001.

**Appendix 2 – Public Hospital Training Course 1994****Protocol for Cardiotocogram (CTG) interpretation and recommendation action.**

Classification	Baseline	Baseline Variability “Band Width”	Decelerations	Action
Normal	110-155 bpm	10-20 bpm 5-20	none	Continue recording
Intermediate	100-110 155-180	>20 bpm <5 bpm	early mild variable <sup>1</sup>	<u>Any two</u> >30 mins; FBS. Repeat 1 hour <sup>2</sup> .
Abnormal	<100 bpm with accelerations >180 bpm	<5 bpm, no accelerations in the absence of sedation.	severe variable <sup>3</sup> late	<u>Any two</u> >30 mins; FBS. <u>Any two</u> >15 mins; FBS. Repeat 1 hour <sup>2</sup> .
Preterminal	Persistent bradycardia, no accelerations exceeding 100 bpm and believe this will be persistent. ↓ variability			Deliver.

1. <60 dropped beats, <60 seconds duration.
  2. FBS earlier if CTG deteriorates.
  3. >60 dropped beats, >60 seconds.
- 

### **Additional independent advice to Commissioner**

In response to my provisional opinion, Dr B disputed a number of Dr Ferguson's conclusions. In particular, Dr B asked me to review the medical literature he had provided to ACC's Medical Misadventure Advisory Committee concerning the use of clinical assessment, fundal heights and BPD to screen for babies small for their gestational age ("SGA"). I therefore sought further independent advice from Dr Ferguson, who provided the following additional independent advice:

"Thank you for the opportunity to read the literature review assembled by [Dr B] with regard to the role of clinical measurement of Symphysis to Fundal Height (SFH), and the role of ultrasound measurement of Biparietal diameter (BPD) as screening tools to detect Small For Gestational Age (SGA) fetuses, and Intrauterine Growth Retardation (IUGR).

[Dr B] requests that I review my opinion in the light of the evidence he presented, with regard to two issues:

- 1) Did the clinical observations of fundal height require that a competent practitioner would refer [Mrs A] for a specialist growth assessment .
- 2) Can BPD assessment add any assurance in the detection of a small baby.

#### 1) Clinical Observations of Symphysis Fundal Height

In addition to reading the material I plotted [Dr B's] clinical observations on two SFH growth chart nomograms. One is an early chart from [the women's hospital – see Appendix 3], that I believe has not been updated since 1976, and the other was from my own literature resources, sourced from a Scandinavian population ... Hakansson 1995 [see Appendix 4]. I also took the opportunity to obtain a 'blind' opinion on the two graphs and the original clinical recordings from [Dr ...], an experienced GPO colleague recently retired from obstetrics, whom I was aware had made a discipline of constructing an SFH foetal growth chart for every pregnancy he managed.

Both graphs show all [Dr B's] recordings to be within the normal range. Most research validating the use of SFH as a screening tool has used a cut-off point of below the 10<sup>th</sup> centile in screening for SGA babies. Some have used the 25<sup>th</sup> centile. The Swedish graph showed none of [Dr B's] measurements were below the 25<sup>th</sup> centile and the [women's hospital] graph showed all the recordings were above the 50<sup>th</sup> centile.

One study that demonstrated a greater screening sensitivity (picking up 73% of infants whose birth weight was subsequently shown to be below the 10<sup>th</sup> centile) used a methodology of responding to any single recording that was 3 or more centimetres below the mean for gestation. None of [Dr B's] recordings were 3cm below the mean. Thus none of the numerous quoted studies, which would have included tens of thousands of women patients, would have identified [the baby] as a case of severe intrauterine growth retardation, if we apply their stated methodologies. The 'blind' opinion from [Dr ...] (he was unaware of all other details of the case), was that he considered the recordings would have served in his experience as an indicator of possible risk, to which he would have sought an additional risk factor – such as poor obstetric history, hypertension or decreased fetal movements – before he would have ordered an ultrasound scan to assess growth.

Widely quoted studies, of which [Dr B's] literature review is a fair representation, suggest only 25% of cases of intrauterine growth retardation will be detected with routine antenatal care in general population studies. With careful attention to detail and measurement technique and the use of standard graphs customised to the patient's height, weight, parity and ethnicity, detection rates improve to detect 50% to 70% of cases (ref. Gardosi 1999, Thompson 1997).

To my knowledge no such customized nomagrams are used or are available in New Zealand and I do not believe it is standard practice in New Zealand to graph the SFH measurements. The literature review therefore confirms that one of the most important goals of antenatal care – identification of the growth retarded baby – cannot be expected to be achieved in 50% of cases, despite the best standard of normal care in a primary health setting, in a population considered to be 'low risk'.

In my previous opinion I had stated 'the baby had severe IUGR and the clinical observations recorded by [Dr B] reflected this reality'. I believe that in the same way that most opinions of the abnormal CTG trace retrospectively identify it (wrongly in my view) as signalling a scenario of severe acute brain damage because of the known outcome, so I have also found it equally easy to interpret the fundal height recordings as clearly demonstrating IUGR. However the objective application of research methodologies as outlined above suggest that it was never so clear cut. It is certainly true that every practitioner will bring a different threshold of concern to these recordings, but there is no clear guide from the obstetric medical literature as to when action should be taken. The New Zealand referral guidelines, that cover every known obstetric condition, do not offer any guidance as to firm indications for ultrasound referral. The matter is left entirely to the judgement and experience of the practitioner.

Thus it seems easy to identify that at 37 weeks, with SFH measurement at 33 cm, and an increase of only 3 cm over 7 weeks that this baby might be growth retarded or small. [Dr B] does however point out that this coincided with descent of the fetal head, which without doubt does influence the measured fundal height. Prior to this, at 36 weeks, the SFH of 34 cm is not abnormal, and subsequent to this the weekly measurements from 37 to 40 weeks appeared to show steady growth of 1 cm per week, which also is normal.

(In the Swedish growth chart this follows the normal growth curve at or above the 25<sup>th</sup> centile.) My conclusion from all of this is that at best we could expect about 50% of competent practitioners to have made an early referral for ultrasound growth assessment in the absence of any other compelling risk factor.

One of the papers quoted by [Dr B] included in its methodology the ability to detect a baby that for example had been following the 90<sup>th</sup> centile and then dropped to the 50<sup>th</sup> (Thompson 1997), and another was sensitive to situations in which the SFH recording remained in the normal range, but the slope of the curve was flatter than on the 10<sup>th</sup> centile line (Gardosi 1999). This changes focus from absolute notions of a normal range at any given gestation, to the increment of serially plotted fundal heights.

One of [Dr B's] papers (Fescina 1987) demonstrated clearly just how subtle the fall off in fundal height can actually be in known cases of IUGR, especially when considering asymmetrical IUGR (as in the baby's case). With this in mind it is possible to look at the charts of [the baby's] growth and conclude that the slope of the growth curve set by the first two measurements, between 30 weeks and 36 weeks, changes significantly from 36 to 40 weeks, and I believe this provides some evidence that the underlying problem in this case was manifest prior to 36 weeks. With the benefit of hindsight it is possible to offer the interpretation that the early clue to this tragedy lay in the single recording at 30 weeks, that was clearly above average. The crucial intervening measurements were not made by the locum (12/1/98, 23/1/98) and indeed would only have been useful if the same practitioner had recorded them. Inter-observer error is a well known shortcoming of the technique of measuring SFH, and has probably been one factor that has led to a widespread under appreciation of the value of measuring fundal height. On a growth chart customised for this initial higher than average recording, the subsequent measurements may have been more strikingly abnormal. [the baby's] birth measurements confirm the abnormal pattern of growth that occurred. At 51 cm his height was in the 50<sup>th</sup> to 90<sup>th</sup> centile range, and suggest he was genetically intended to be bigger than average. At 30 weeks the fundal height may well have been reflecting this prior to the onset of the growth impairment. His head circumference, at 33cm, was less than the 10<sup>th</sup> centile and weight at 2290 gms was dramatically below the 3<sup>rd</sup> centile. I believe the mismatch between his length and head circumference adds further evidence to the suggestion that an early cerebral insult had occurred. There is a rather confusing letter in the file from ACC, in which a Paediatric Radiologist whose opinion was sought by their advisory committee advised upon the timing of the brain injury relative to the known abnormalities in the CT scan, most notably the brain atrophy. In the letter she states that 'the earliest time to show the definite presence of subsequent atrophy would be about six weeks'. Again this suggests the brain damage occurred sometime around, or prior, to 35 weeks gestation, and had time to impact upon the growth of [the baby's] head circumference.

## 2) Measurement of BPD in Screening for SGA Infants

In regard to the value of measuring BPD in detecting intra-uterine growth retardation I do not believe the literature review supports this in any way, in fact several of the papers



highlight its deficiencies (Fescina 1987). Nevertheless [Dr B] is correct in stating that it provides screening sensitivity that is not dissimilar to the value of SFH measurement. However I believe the point is that if one has access to the modality of ultra-sound then the technique which should be used is the one that is shown to be the most valuable, i.e. measurement of abdominal circumference. This clearly is the gold standard for detecting intra-uterine growth retardation and is most useful when in fact the 3 key parameters of BPD (Biparietal Diameter), AC (abdominal circumference) and FL (femur length) are all measured and compared. A graph of [Dr B's] BPD measurements [see Appendix 5] show that the measurements were steadily moving up the 50<sup>th</sup> percentile and I believe this will have only offered false reassurance to [Dr B] that all was well, given that the question had arisen in his mind as to the adequacy of the baby's growth.

### SUMMARY

My opinion of [Dr B's] management has been changed by graphing his original clinical measurements, review of the literature and examination of my own decision making. I don't believe that he can be held to be incompetent for failing to diagnose a condition that is routinely missed in 50% of cases.

Without recognition of [the baby's] severe IUGR one cannot describe as incompetent the aspects of labour management that followed, such as allowing 10 days of post maturity, no early vaginal examination and rupture of membranes on admission, and delay in seeking a specialist opinion on the abnormal CTG trace. All of this in retrospect can be seen to be unwise, but do however represent normal management of a labour perceived as 'normal'. Questions could be asked about the absence of any further antenatal assessment when [the baby] was more than a week overdue. It is common practice to consider either an antenatal CTG or an ultrasound assessment (Biophysical Profile) when monitoring a pregnancy between 41 and 42 weeks gestation.

To my previous defence of his interpretation of the CTG trace, I wish to add a point of clarification. Whilst the CTG was abnormal it did not signify acute life threatening progression of fetal asphyxia. In the absence of information about the status of the amniotic fluid (ie presence of meconium) a CTG trace such as this has a very poor predictive value for acute fetal distress. In a multiparous woman in active labour it was appropriate to observe the progress of the trace for approximately 30 minutes as clearly either improvement or deterioration would dictate management. The fact he observed it for a further 30 minutes was of no consequence to the outcome of the case.

The evidence of the neonatal brain CT scan showing cerebral atrophy and the comments of the Paediatric Radiologist firmly place the onset of the mechanism of [the baby's] brain injury at least 6 weeks prior to birth.

I am however critical of [Dr B's] use of ultrasound. At best it provided a distraction both from a more considered interpretation of the clinical evidence he had gathered, and from [Mrs A's] own anxieties about the situation. At worst it misled him from his own clinical suspicions about [the baby's] growth, by providing BPD's that appeared to be growing along the 50<sup>th</sup> centile. As I stated previously this provided no reassurance

about the presence of (asymmetrical) growth retardation, and may have prevented a referral at that point to a radiologist who would have easily made the diagnosis. Rather than helping him in his endeavour to be amongst the 50% of practitioners that evidence tells us would make this diagnosis, his use of ultrasound ensured that he was amongst the 50% that would miss it.

The clinical diagnosis of the SGA infant, or intrauterine growth retardation, is a major focus of good antenatal care. Low birth weight (<2500gm) is associated with 75% of all poor perinatal outcomes, and the condition accounts for approximately 10% of perinatal mortality. Importantly it is a cause of death and morbidity in which outcomes can be improved by timely intervention in some, but not all cases. Unfortunately there will be many practitioners who are less conscientious than [Dr B] in carefully recording fundal heights and we are told in the evidence that in the experience of one Consultant Obstetrician only about half of the 'LMCs' bother to record it.

In my experience the value of not only SFH measurements but also maternal girth measurements in giving clues to the presence of an intrauterine growth disorder (either too small or too big) is greatly underrated, especially when the same practitioner makes all of the measurements. Perhaps increasing reliance on ultrasound in recent years has made practitioners less focused on the basic clinical skills of antenatal care.

Even amongst practitioners who carefully measure uterine growth it is relatively uncommon to plot growth curves in pregnancies considered 'normal' or 'low risk'. As I have explained I think the only chance [the baby] had of being diagnosed correctly when he needed it – probably around 36-37 weeks, would have been with the use of a growth chart of SFH measurements. This would have shown a fall off in the slope of the growth curve beyond 36 weeks, compared to that prior. As the research correctly indicates, there are no useful absolute levels of SFH that can be used as indications for referral because of the very wide range of normal.

This case highlights a very important issue in primary obstetric care and I believe all practitioners would benefit from:

- i) a reminder of the importance of careful SFH measurement.
- ii) early recourse to graphing the results if there is the slightest concern – particularly looking for a fall off in growth rate, even when absolute measurements are within the 'normal' range.
- iii) early referral for ultrasound assessment if there are either clinical concerns or additional risk factors.

#### **APPENDIX:     References**

- 1) Hakansson A, Aberg A, Nyberg P, Schersten B – A new Symphysis-fundus height growth chart based on a well defined female population with ultrasound dated singleton pregnancies. *Acta Obstet Gynecol Scand.* (1995) 74, 682-686.

- 2) Thompson M.L, Theron G.B, Fatti L.P. Predictive value of conditional centile charts for weight and fundal height in pregnancy in detecting light for gestational age births. *European Journal of Obs. Gynae and Reproductive Biology* (1997) 72, 3-8.
- 3) Gardosi J, Francis A. Controlled trial of fundal height measurement plotted on customized antenatal growth charts. *Br J Obstet Gynaecol* (1999) vol 106, 309-317.
- 4) Fescina R.H, Martell M, Martinez G, Lastra L, Schwarcz R. Small for dates: Evaluation of different diagnostic methods. *Acta Obstet Gynecol Scand* (1987) 66: 221-226.

...”

Appendix 3

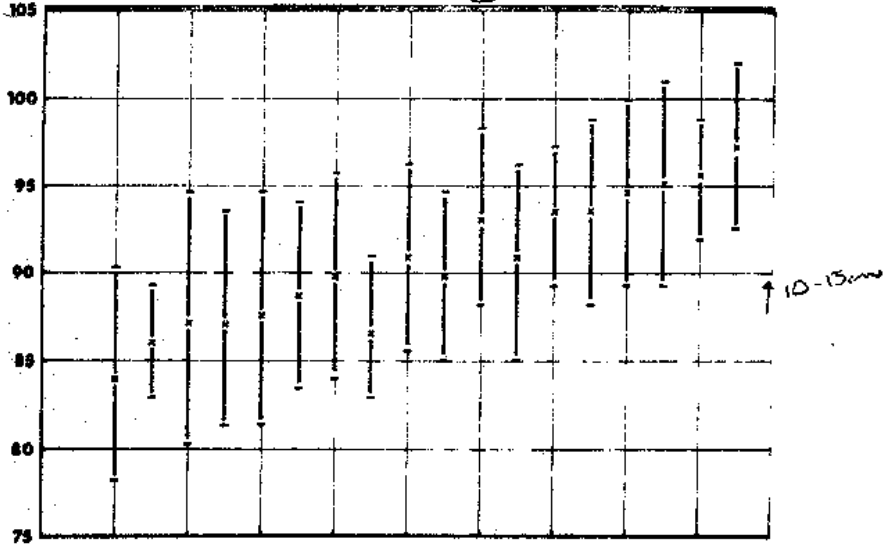
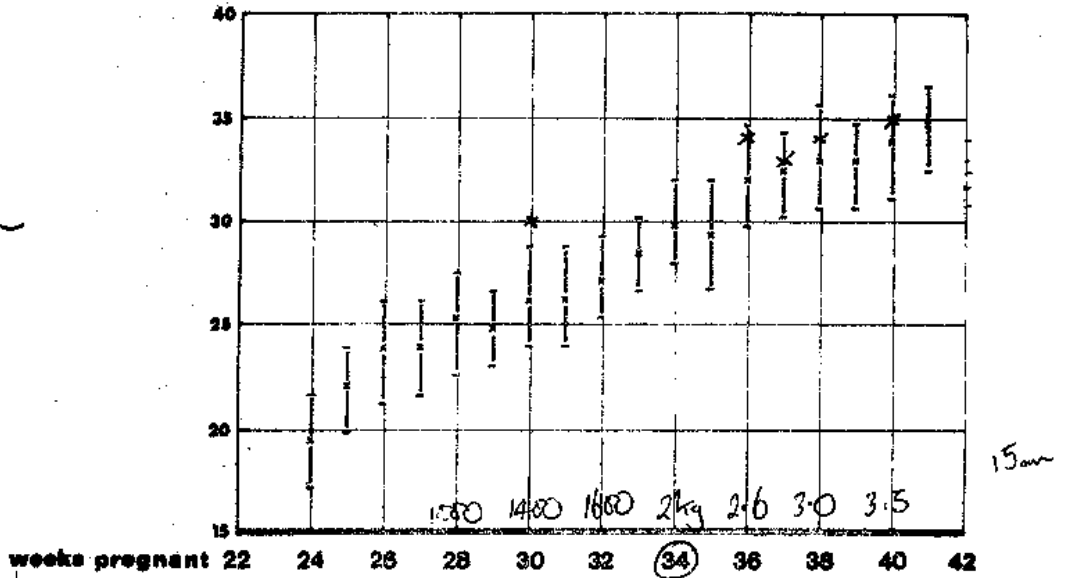
Graph ①



POSTGRADUATE SCHOOL OF OBSTETRICS & GYNAECOLOGY

FUNDAL HEIGHT & GIRTH STANDARDS 1976 ( $\pm$  1.S.D.)

AVERAGE WEEKLY FUNDAL HEIGHT



AVERAGE WEEKLY GIRTH

## Appendix 4

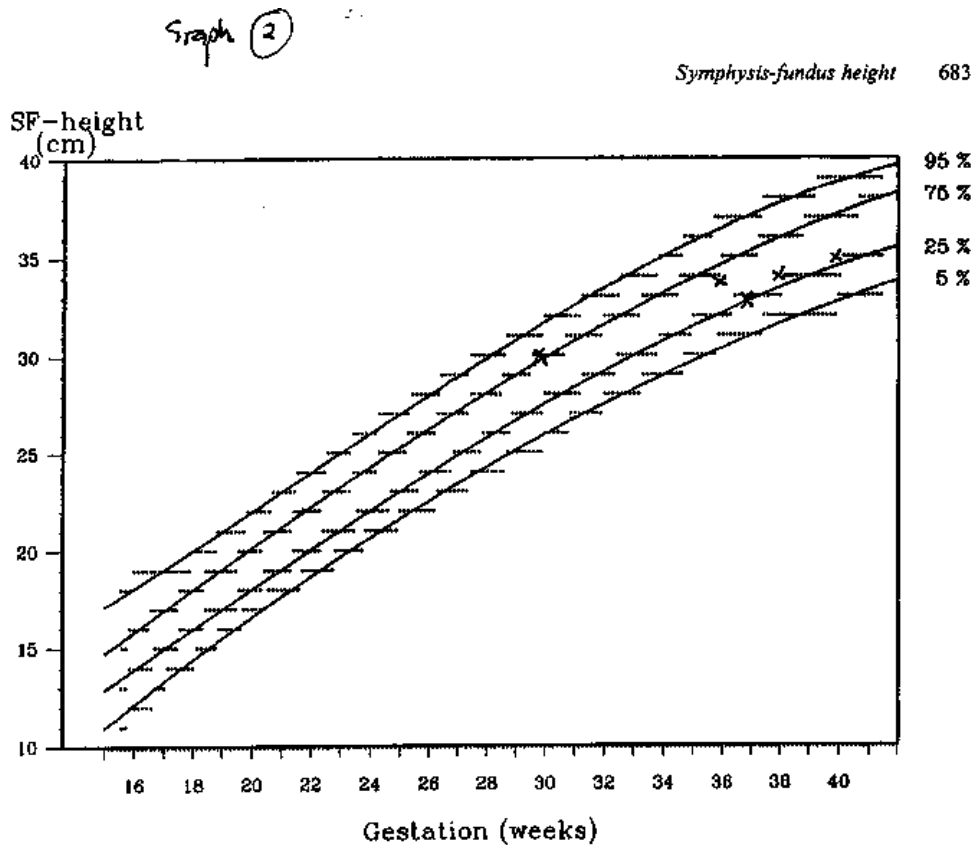


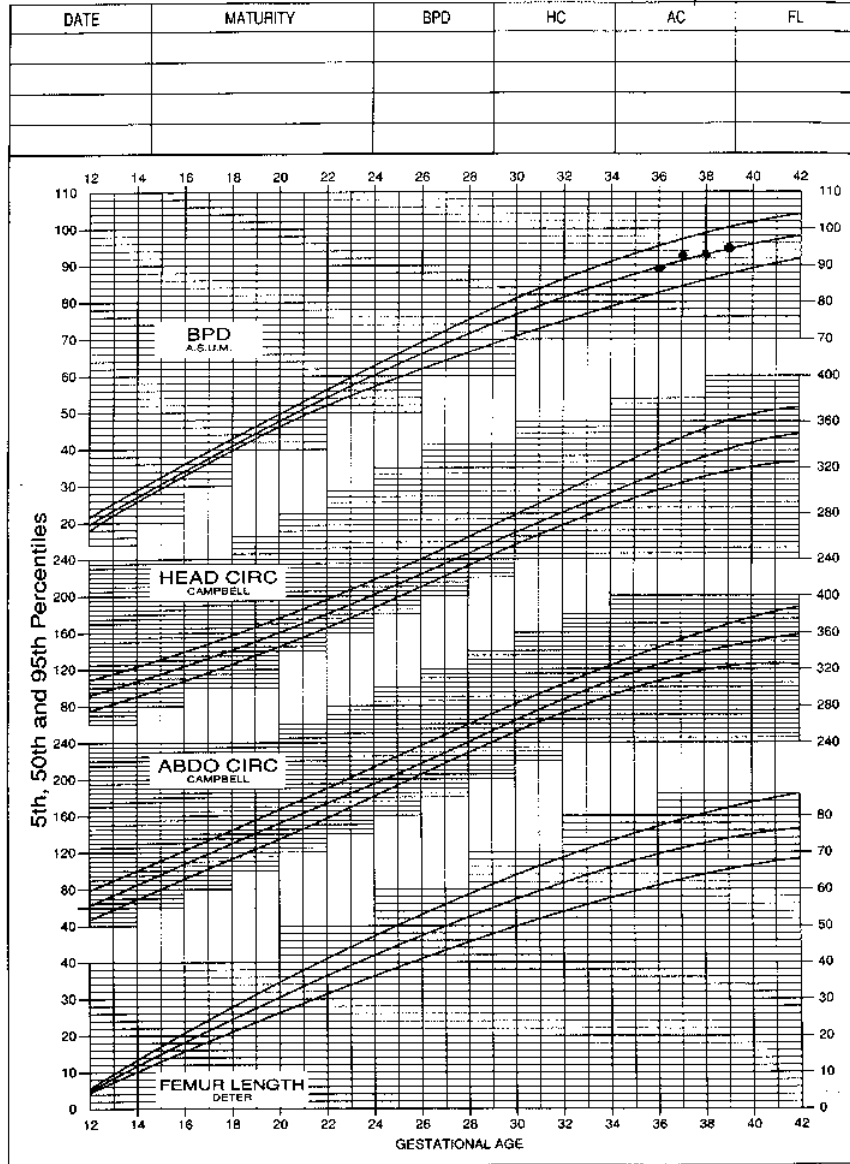
Fig. 1. Symphysis-fundus (SF) height growth chart based on 403 ultrasound-dated singleton pregnancies. The 5th, 25th, 75th, and 95th percentiles from 109 to 290 days of pregnancy are shown. The dotted, broken lines indicate the estimated values for each day of pregnancy, and the smooth, continuous lines are fitted by a cubic regression model.

Appendix 5

Graph 3

Attach patient identification label here

**SERIAL GROWTH CHART**



SERIAL GROWTH CHART

5.07.05

## Code of Health and Disability Services Consumers' Rights

The following Right in the Code of Health and Disability Services Consumers' Rights is applicable to this complaint:

### *RIGHT 4*

#### *Right to Services of an Appropriate Standard*

- 1) Every consumer has the right to have services provided with reasonable care and skill.*
- 

## **Opinion: No breach – Dr B**

### *Management of Mrs A's labour*

In my opinion, Dr B exercised reasonable care and skill in his management of Mrs A's labour.

Dr B arrived in the delivery suite at approximately 8.50pm. The CTG showed a borderline tachycardia (rapid heart rate) and deep type 1 dips which mirrored the contraction intensity. Dr B did not consider the CTG indicated a need for urgent action, and continued to monitor it. At approximately 9.30pm, Dr B examined Mrs A and found that she was 2cm dilated, and that the baby's head was facing down and had begun to engage. He continued to monitor the contractions in the hope that the CTG trace might improve. When the trace did not improve, he decided to refer Mrs A to a specialist because, although he did not believe that the baby was in imminent danger, "it seemed increasingly unlikely that the situation would spontaneously improve". Dr B contacted Dr C at approximately 9.50pm.

My general practitioner advisor considered that Dr B's management of Mrs A's labour was reasonable. He advised me that Dr B acted appropriately in contacting Dr C, the on-call specialist, when he did. My advisor gave that advice on the basis that, although the CTG was abnormal, it did not suggest an immediate life-threatening situation. According to my advisor, the CTG did not reach the criteria outlined in the Royal College of Obstetricians and Gynaecologists guidelines, or in the women's hospital guidelines, that would trigger immediate delivery. My advisor considered that the baby had sustained the damage that led to his cerebral palsy with spastic quadriplegia prior to the onset of labour. He advised that any alterations in the management of the labour would not have significantly changed the outcome for the baby.

I note my advisor's comment:

"In retrospect of the delivery of a baby with obvious brain damage it would be very easy to emphasise the abnormal features of this CTG trace, and assume that more rapid intervention would have improved the outcome.

...

The CTG abnormalities witnessed in the hour prior to birth were not evidence of severe, acute hypoxic stress, but were a reflection of the already brain damaged foetal state.”

My advisor also observed that Dr C delivered the baby by Caesarean just under an hour after he arrived at the hospital. In my advisor’s view, this suggested that even Dr C did not think in the first instance that this was an immediately life-threatening abnormality. He noted:

“CTG appearances commonly associated with acute severe hypoxic brain damage would have prompted a ‘crash’ LSCS [Caesarean section] that would have seen the baby delivered in less than half this time.”

I am guided by my advisor’s comments. In my opinion, Dr B managed Mrs A’s labour appropriately and contacted Dr C in a timely fashion, and therefore did not breach the Code.

Having concluded that Dr B acted appropriately in contacting Dr C when he did, I do not accept the allegation that delayed referral to a specialist caused the baby to sustain cerebral palsy and spastic quadriplegia. I note that my advisor considers that the baby was already brain damaged prior to the onset of labour, and I accept that advice.

---

## **Opinion: Breach – Dr B**

### *Antenatal care*

At 36 weeks’ gestation, Dr B starting using an ultrasound machine in his rooms to measure the diameter of the baby’s head. Dr B advised that his main reason for using ultrasound at 36 weeks is to diagnose breech presentations, and that he did not use the baby’s BPD measurements specifically to exclude IUGR. I note, however, that Dr B was concerned about the baby’s growth because he continued to measure the baby’s BPD in conjunction with Mrs A’s fundal height, beyond 36 weeks. The BPD measurements, combined with an increase in fundal height that Dr B considered to be average for a baby near term, reassured him that the baby was growing appropriately. However, the baby suffered severe intrauterine growth retardation, and when he was delivered at 41 weeks he weighed only 2290gms.

In his initial advice, my general practitioner advisor stated:

“[Mrs A’s] baby had severe intrauterine growth retardation and the clinical observations recorded by [Dr B] reflected this reality.

...

Unfortunately [Dr B] had insufficient skills with ultrasound to convert his concerns about the baby’s growth into a reliable interpretation of the ultrasound findings. It



shows a very deficient understanding of abnormalities of foetal growth to assume that normal growth of head circumference parameters provides any assurance at all of satisfactory foetal growth and uteroplacental perfusion [the blood flow between mother and baby].

The appropriate response to any clinical concerns about foetal growth would be referral to a radiologist qualified to assess foetal growth. Detection of IUGR would then have prompted specialist referral, and most probably induction of labour prior to term. Early antenatal intervention may have provided the possibility of a better outcome.”

When he responded to my provisional opinion, Dr B disputed my advisor’s conclusion that his clinical observations reflected the reality that the baby was suffering severe intrauterine growth retardation. He argued that the medical literature he had submitted to the Committee supported his view that his recordings of Mrs A’s fundal height did not suggest the need for referral for growth assessment.

Dr B also disputed my advisor’s conclusion that normal BPD growth did not provide any assurance of satisfactory foetal growth and uteroplacental perfusion. He submitted that the medical literature suggested that BPD measurements do provide some reassurance of adequate fetal growth, and that BPD measurements alone are “at least as sensitive as fundal height measurements and clinical assessment” in detecting babies small for their gestational age.

Dr B also disputed Dr Ferguson’s conclusion that the appropriate response to any clinical concern was referral to a radiologist qualified to assess fetal growth. Dr B stated that not every patient with a less than average growth in fundal height is referred for specialist ultrasound assessment, and that his clinical findings did not reach his “clinical threshold” for referral. Dr B submitted that the “published evidence” suggested his actions were reasonable.

In light of Dr B’s submissions, I asked my independent advisor to review the literature Dr B had provided to the Committee, and to reconsider his original advice.

Dr Ferguson considered that Dr B’s literature review was a “fair representation” of widely quoted studies. My advisor stated that those studies suggest that routine antenatal care will detect only around 25% of cases of IUGR, and that careful attention to detail, measurement technique, and the use of standard graphs customised to the patient’s height, weight, parity and ethnicity, could improve detection rates to 50% to 70% of cases.

Dr Ferguson plotted Dr B’s measurements of Mrs A’s fundal height on two different growth chart nomograms. Dr B’s recordings were within the normal range on both charts. On one, none of Dr B’s measurements were below the 25<sup>th</sup> percentile, and on the other, all Dr B’s recordings were above the 50<sup>th</sup> percentile. Dr Ferguson further noted that none of Dr B’s recordings were 3cm or more below the mean for gestation, which one study found was a measurement providing greater screening sensitivity. Dr Ferguson stated:

“Thus none of the numerous quoted studies, which would have included tens of thousands of women patients, would have identified [the baby] as a case of severe intrauterine growth retardation, if we apply their stated methodologies.”

My advisor therefore revisited his original conclusion that Dr B’s measurements of Mrs A’s fundal height “reflected [the] reality” that the baby was suffering severe IUGR. Dr Ferguson acknowledged that because he knew the baby had been born with severe IUGR, it had been easy to interpret the fundal height recordings as clearly demonstrating IUGR. My advisor agreed with Dr B that an objective application of research methodologies suggested his conclusion was “never so clear cut”.

In relation to his original advice that the appropriate response to “any clinical concerns about foetal growth” would have been referral to a radiologist, my advisor acknowledged that the obstetric medical literature provided no clear guide as to when action should be taken. Nor did New Zealand referral guidelines offer clear guidance for ultrasound referral, referral being left entirely to the judgement and experience of the practitioner.

However, Dr Ferguson remained critical of Dr B’s use of ultrasound. My advisor did not agree that the medical literature supported Dr B’s view that measuring BPD assisted in the detection of IUGR. My advisor pointed to papers Dr B had submitted to the Committee which highlighted the deficiencies of the method. My advisor stated:

“... [T]he point is that if one has access to the modality of ultra-sound then the technique which should be used is the one that is shown to be the most valuable, i.e. measurement of abdominal circumference. ... [The BPD measurements] were steadily moving up the 50<sup>th</sup> percentile and I believe this will have only offered false reassurance to [Dr B] that all was well, given that the question had arisen in his mind as to the adequacy of the baby’s growth.”

In fact, the baby suffered asymmetrical growth retardation. Dr B did not measure the baby’s abdominal circumference or femur length because he acknowledged not having the necessary training or skill to make reliable measurements. Dr B acknowledged his limitations in using ultrasound, and stated that he understands that when a baby is suffering asymmetrical IUGR, the baby’s head growth is less affected than abdominal circumference. With that in mind, it is difficult to understand how, on the basis of his BPD measurements, Dr B concluded the baby was growing adequately. His limited use of ultrasound was insufficient to provide him with reassurance.

My advisor considered that Dr B was using “a diagnostic modality, ultrasound, in which he was inadequately trained”. Dr Ferguson stated:

“At best it provided a distraction both from a more considered interpretation of the clinical evidence he had gathered, and from [Mrs A’s] own anxieties about the situation. At worst it misled him from his own clinical suspicions about [the baby’s] growth, by providing BPD’s that appeared to be growing along the 50<sup>th</sup> percentile. As I stated previously this provided no reassurance about the presence of (asymmetrical) growth retardation, and may have prevented a referral at that point to a radiologist who would

have easily made the diagnosis. Rather than helping him in his endeavour to be amongst the 50% of practitioners that evidence tells us would make this diagnosis, his use of ultrasound ensured that he was amongst the 50% that would miss it.”

I accept Dr B’s argument that the diagnosis of growth retardation is often not made and cannot reasonably be expected to always be made. I also accept that a “false reassurance” is not necessarily the result of failing to exercise reasonable skill and care. However, there is a distinction between false reassurance which occurs despite the exercise of reasonable skill and care, and false reassurance which results from a failure to exercise reasonable skill and care. In my view, Dr B was falsely reassured because he inappropriately relied on ultrasound, a diagnostic modality in which he was insufficiently trained, and in doing so he failed to exercise reasonable care and skill.

According to my advisor, Dr B should have referred Mrs A to a radiologist qualified to assess foetal growth. Detection of IUGR would then have prompted specialist referral, and most probably induction of labour prior to term. In my advisor’s view, the baby may have had a better outcome with early antenatal intervention.

I accept my expert advice. In my opinion, Dr B was concerned enough to use a diagnostic method he was not fully qualified to use, and at that point needed to refer Mrs A to a radiologist qualified to accurately measure foetal growth.

I also note my advisor’s comments:

“There is an old saying in medicine ‘Always listen carefully to the patient, he or she is telling you the diagnosis’. In maternity care this saying is particularly true, and any persisting concern that a patient has must always be very carefully considered. In my experience any woman who thinks she is either significantly bigger, or significantly smaller than she was in her last pregnancy is usually correct. This simple observation is very important as the vast majority of bad obstetric outcomes occur in babies that are either too big or too small.”

In my opinion, Dr B did not provide Mrs A with an appropriate standard of care. First, he used ultrasound beyond his capabilities and was falsely reassured that the baby was growing adequately. He should have referred Mrs A to a radiologist qualified to assess the baby’s growth. Secondly, Dr B failed to heed Mrs A’s concerns. Mrs A knew “something wasn’t right”, yet Dr B continued to reassure her otherwise. I endorse my advisor’s view that doctors need to listen carefully to their patients, particularly in maternity care.

Accordingly, in my opinion Dr B breached Right 4(1) of the Code in relation to his management of Mrs A’s antenatal care.

## **Actions taken**

In my provisional opinion, I recommended that Dr B apologise in writing for his breach of the Code, the apology to be sent to my Office for forwarding to Mrs A. In response, Dr B advised me that the knowledge that earlier antenatal intervention may have led to a better outcome for the baby had been a huge burden to him over the last four years. He doubted he could adequately convey to Mrs A and her family his sorrow and distress over this tragic matter, especially given that he feels that Mrs A blames him entirely for the baby's cerebral palsy. I also note that in late 1998, Dr B wrote to Mrs A and her family, stating that he was "painfully aware that if [he] had referred [Mrs A] for a specialist opinion in pregnancy [the baby] may have been delivered early and may not now have cerebral palsy". I have considered the circumstances and reached the view that there is little to be gained by requesting Dr B to apologise again.

Dr B also advised me that after the baby's birth he asked a local specialist to review his obstetric practice on a case-by-case basis, and that the specialist had continued that oversight until Dr B ceased intrapartum obstetrics about six months after the baby's birth. Dr B indicated his willingness to have the Medical Council review his obstetric practice.

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## **Further actions**

- A copy of this opinion will be sent to the Medical Council of New Zealand, with a recommendation that the Council consider whether a competence review of Dr B's obstetric practice is warranted.
- A copy of this opinion, with all identifying features removed, will be sent to the Royal New Zealand College of General Practitioners, the Royal Australian and New Zealand College of Obstetricians and Gynaecologists, and the Maternity Services Consumer Council, and will be placed on the Health and Disability Commissioner website, [www.hdc.org.nz](http://www.hdc.org.nz), for educational purposes.