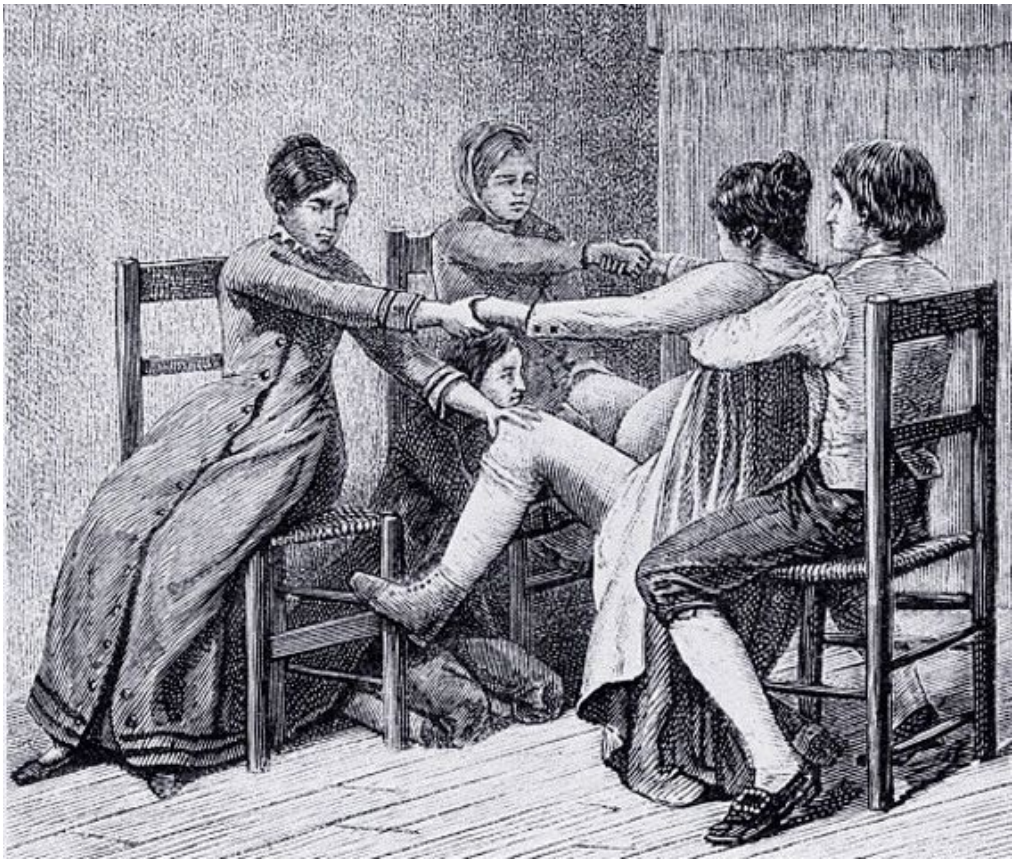
BENCHMARKING THE HYPERTENSIVE DISORDERS OF PREGNANCY – IMPROVING OUTCOMES FOR MOTHERS AND BABIES VIA CLINICAL PRACTICE CHANGE

©CHARLENE ELIZA THORNTON

A thesis submitted in fulfilment of the requirements for the award of the degree of Doctor of Philosophy, University of Western Sydney, 2010.



There is a clear need for further national and international studies, with agreed definitions, criteria and methods, which will be based on populations rather than hospitals…without such studies we are denied the considerable contribution that epidemiological methods can make in the elucidation of this complex group of diseases. (Davies 1971).

DEDICATION

To my supervisor, Professor Annemarie Hennessy, I would like to extend my heartfelt appreciation and admiration of her boundless enthusiasm, enormous intellect, endless patience, warm friendship and sincere support. This unique combination of attributes, enabled this project to come to fruition. For their intellectual support and contribution I would like to thank Associate Professor Peter von Dadelszen and Professor John Morley.

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STATEMENT OF AUTHENTICATION

The work presented in this thesis is, to the best of my knowledge and belief, original except as acknowledged in the text. I hereby declare that I have not submitted this material, either in full or in part, for a degree at this or any other institution.

(SIGNATURE)

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LIST OF ABBREVIATIONS

ACHCS	Australian Council on Health Care Standards
AIHW	Australian Institute of Health and Welfare
AMOSS	Australasian Maternity Outcomes Surveillance System
ANZICS	Australian and New Zealand Intensive Care Society
APACHE	Acute Physiology and Chronic Health Evaluation
APO	Acute pulmonary oedema
ARF	Acute renal failure
ASSHP	Australasian Society for the Study of Hypertension in Pregnancy
BCW	British Columbia Women's Hospital and Health Centre
BC	British Columbia
CDC	Centre for Disease Control and Prevention
CH	Chronic hypertension
CTH	Campbelltown Hospital
DRG	Diagnostic Related Groups
DNA	Deoxy Ribonucleic Acid
EDGE	Extracting Data and Generating Evidence
EU	European Union
GDP	Gross Domestic Product
GFR	Glomerular Filtration Rate
GH	Gestational Hypertension
HDP	Hypertensive Disorders of Pregnancy
HELLP	Haemolytic anaemia, elevated liver enzymes, low platelets syndrome
ICD-10	International Classification of Diseases 10th edition
ICD-10-AM	International Classification of Diseases 10th edition
ICU	Intensive Care Unit
IL	Interleukin
IUGR	Intrauterine Growth Restriction
LH	Liverpool Hospital
MDC	Midwives Data Collection
MMR	Maternal Mortality Ratio
NH	Nepean Hospital
NHMRC	National Health and Medical Research Council
NICU	Neonatal Intensive Care Unit
NPDC	National Perinatal Data Collection
NSW	New South Wales
PATH	Performance Assessment Tool for Quality Improvements in Hospitals
PE	Preeclampsia
PF	Plasma Fibronectin
PMR	Perinatal Mortality Rate
RCT	Randomised Controlled Trial
RNSH	Royal North Shore Hospital
RPAH	Royal Prince Alfred Hospital (Women and Babies)
SOMANZ	Society of Obstetric Medicine Australia and New Zealand
RR	Relative Risk
SP	Preeclampsia superimposed on chronic hypertension

THIS	Taiwan Healthcare Indicator Series
TNFα	Tumour necrosis factor alpha
UKOSS	United Kingdom Obstetric Surveillance System
WHO	World Health Organisation

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ABSTRACT

Background and aims

An average of one hundred and thirty four mothers and 410 babies die every day around the world as a result of the hypertensive disorders of pregnancy (HDP). This figure, whilst staggering, in no way reflects the degree of morbidity which results from these disorders. Randomised controlled data and isolated hospital specific databases can provide us with snapshots at single points in time within obstetric units and populations, but there is no routinely collected or mandatory dataset which accurately reflects the type and severity of complications which occur. Eclampsia, acute renal failure (ARF), acute pulmonary oedema (APO) and growth restriction are well documented complications of the HDPs, yet there considerable variation in the literature in describing their incidence. There is currently no consensus on treatment modalities which will optimise outcomes. Without accurate data, obstetric care providers and obstetric units are unable to be confident that the care they provide is optimal if they have no standard or benchmark against which to compare.

The aim of the study herein, was to establish a database across obstetric units and countries to determine if accurate rates of morbidity could be obtained, and if this data could be the basis for the establishment of 'best achievable outcomes'. Further, the utility of the audit and feedback process was tested as the means by which clinical practice change could be instituted.

Methods

A disorder-specific database was designed for data collection and collation. Following local area institutional human ethics committee approval at five obstetric units in the Sydney region of Australia and one obstetric unit in Vancouver, Canada, Individual Patient Data (IPD) methodology was utilised and detailed data on demographics, laboratory testing, pregnancy monitoring, treatments provided and delivery and postnatal progression data were collected over a 12 month period. The medical notes of women were identified utilising the International Classification for Diseases coding system. Eight clinical indicators were designed as the tools through which comparison could be made. Data were analysed with SPSS v.18® utilising standard statistical methodology as well as regression modelling where appropriate. Feedback sessions at each collaborating unit were held and potential factors influencing outcomes variations were discussed and analysed. The audit and feedback process was extended at one institution to test for cyclical improvement process over a three year period.

Results

Over 350 000 data points were collected on 1 728 pregnancies. Large treatment variations were seen between units in regard to the timing of the initiation of antihypertensive medication, the choice of medication used, the timing and type of delivery and the amount and type of intravenous fluid resuscitation. The incidence of outcomes such as eclampsia varied between units with rates ranging from 2-47/1 000 pregnancies complicated by a HDP. Variations were also seen in the rate of APO

(0-47/1 000 and ARF (2-46/1000). Acute pulmonary oedema was targeted at one unit where incidence was highest. Logistic modelling concluded intravenous fluid volume, resulting from unrestrictive fluid administration policies, was the determinant in the incidence of APO. This finding led to the implementation of more stringent fluid administration policies at the targeted unit followed by a fall in the incidence of APO over the time period of the study from 46/1 000 to 2/1 000 without an increase in ARF.

Conclusion

Individual patient data audit and benchmarking between obstetric units can be achieved. Detailed datasets can be used to assess the potential effect of treatment variations and clinical practice change can be successfully instituted based upon this methodology.

CHAPTER 1

BACKGROUND

1.1. HISTORICAL THEORIES OF DISEASE

The phenomenon of convulsions in the perinatal period was first noted by the ancient Greeks in the pre-Hippocratic Coan Prognosis. 'In pregnancy, drowsiness and headache accompanied by heaviness and convulsions, is generally bad' (cited in Chesley 1984, p.52). This was most likely an allusion to convulsions and the symptomatology of what we now call preeclampsia. The Greek Aetios in the 6th century AD noted 'a strong swollen pulse' (cited in Ong 2004, p.14) in women who subsequently convulsed, whilst the Roman Celsus in the 1st century AD and the Greek Galen in the 2nd century AD both make reference to convulsions which were life threatening (cited in Ong 2004). It was noted by the German physician Gaerbelkhouern in 1596 that there existed a type of epilepsy present only in the pregnant uterus, describing maternal pain to be like that of a rat gnawing at the heart (cited in Redman 1992).

The French physician Francois Mauriceau recorded a case of 'furious convulsions for a day and a half' in a 25 year old woman who died in her first pregnancy (cited in Chesley 1984, p.54). At this time it was believed that an excess of heated blood or malignant vapours were the cause of the convulsions. The tombstone of an English woman who died in 1638 in childbirth records her '…being shaken by the force of frequent convulsions…' (Lever 1843, p.12).

In the 18th century the growth of the pursuit of science and the role of obstetricians at the bedside of labouring women heralded the more frequent written recording of convulsions and their precursors. The convulsions of pregnancy were differentiated from those of epilepsy in 1739 by the Frenchman Bossier de Sauvages who is first credited with the use of the term eclampsia, most likely derived from the Greek for 'flash of lightning' or simply a translation for epilepsy (cited in Ong 2004), although the physician Varandesus is said to have used the term as early as 1619 in his Treatise on Gynaecology (cited in Chesley 1984). Mauriceau established that these convulsions occurred more frequently in first pregnancies, that antenatal convulsions were more life threatening than postnatal, that the fits were worse if the fetus was dead, and that if consciousness was not regained between fits, then the outcome was usually worse. The most prominent French midwife of the time, Madame de Bousier du Coudray, reported in 1773 that delivery was the only way to bring about the cessation of the convulsions (cited in Ong 2004).

In his report at Guy's Hospital in London in 1843, the British obstetrician Lever, demonstrated that protein in the urine, blurred vision, oedema and headaches were precursors of convulsions, yet concluded that this was caused by the kidney in a state of disease. This theory was supported by the German physician Frerichs (cited in Ong 2004) who in 1851 published a book on eclampsia as the symptom of end stage nephritis. Post mortem examination of the kidneys and the presence of liver damage distinguished eclampsia from nephritis at the close of the 19th century (Redman 1992).

The phenomenon that hypertension was raised in women before they fitted was noted with the development of the sphygmomanometer in the 19th century. This device enabled the measurement of blood pressure and heralded a new era in the understanding of this pregnancy disease (Chesley 1978).

William Tyler Smith noted that 'The state of the blood…may excite the convulsive disorder' (cited in Chesley 1978, p.54) and blood toxins such as water, fetal waste products, bacterial toxins, toxins released by the placenta, toxins released by the fetus, menstrual fluid and toxins released by damaged organs were recorded as the cause of convulsions. Other toxaemias of pregnancy were also noted such as hyperemesis, gingivitis, herpes and placental abruption, but by the 20th century the term "toxaemia" was synonymous with preeclampsia.

The term toxaemia was prominent for most of the 20th century and still lingers within obstetric terminology (Chesley 1978). The presumed release of toxins into the maternal circulation is a theory which is experiencing a regain in popularity in current research (Redman 1988).

1.2 RECENT THEORIES OF DISEASE

Placental aetiology theories dominated scientific research in the 20th century. In 1948 the American obstetrician Ernest Page argued that a shortage of maternal blood flow to the placenta was the cause of preeclampsia (cited in Redman 1992). Brosens (cited in Redman 1992) noted the role of the spiral arteries and their narrowing in the placental bed of women with preeclampsia. Redman (1992, p.28) refers to this theory

as the ‘sick placenta syndrome’, of an inadequate and/or malfunctioning placentation. The predominant hypothesis in this theory is that the normal pregnancy model of a high-flow/low pressure adaptation (to maximize the blood flow at the placental/maternal exchange) is deficient in women with preeclampsia, and that the trophoblast invasion is more superficial than in normal pregnancies. This theory is clearly evidenced by fetal growth restriction and in histological review of many preeclamptic placentas.

The greater majority of this human placental work has been accomplished *in vitro* via maternal blood, placental/decidual tissue or placental perfusion studies, due to the invasive nature of *in vivo* studies in the developing human. That animal work can link placental insufficiency with these concepts of circulating ‘toxins’ such as SFlt-1 (Makris et al. 2007; La Marca, Gilbert & Grainger 2008) has added support to the placental ‘toxin’ theory. Animal models for preeclampsia have been assumed to represent human placental disease (Phippard et al. 1986) and have been developed, (Orange et al. 2005) but they only partially succeed due to the almost exclusive natural development of preeclampsia to humans. These models may address specific potentially relevant mechanisms but are not truly representative of endogenous disease (La Marca, Gilbert & Grainger 2008).

The theory of preeclampsia as an excessive maternal inflammatory response causing placental changes has also gained steady momentum in the recent literature (Redman & Sargent 2009). The theory expounds that the syndromal effects are caused by a heightened immunological response which may be an extreme of the normal pregnancy adaptation: a systemic inflammatory response that causes endothelial cell

dysfunction and thereby all of the hallmark effects which are clinically apparent. Cytokines such as interleukin 6 and 10 (IL-6, IL-10), (Benian et al. 2002; Freeman et al. 2004; Makris et al. 2006) and factors such as tumour necrosis factor alpha (TNFα) (Beckmann et al. 2004), plasma fibronectin (PF), (Paarlburg et al. 1998; Madazli et al. 2005), free fetal DNA (Bauer et al. 2006) catecholamines (Pedersen et al. 1983), antithrombin III antibodies (Mello et al. 2005), s-flt-1 (Levine et al. 2004) or endoglin (Steinberg, Khankin & Karumanchi 2009) have been identified as potentially relevant as markers/predictors of disease and disease severity.

The transference of the results of this work from the laboratory into the clinical environment in the development of predictive markers of preeclampsia which can be used in early pregnancy has also been studied (Poon et al. 2009).

The concept of a single toxin or a combination of toxins (and hence the term toxaemia) may not be as outdated as one would expect. Alternatively, and more likely, a combination of factors may be involved in placental ischaemia/immunological responses and perhaps these were the toxins that William Tyler Smith may have been eluding to (cited in Chesley 1978, p.54).

The famous reference by German physician Zweifel (1916) to the ‘disease of theories’ is as relevant today as it was when first penned almost a century ago.

1.3 HISTORICAL TREATMENTS

Early treatments before the 18th century included bleeding or purging women. Treatment modalities during the 19th and early 20th centuries also included bleeding and purging as well as starvation, sedation, paralysis, diuresis, sweating, dietary restrictions of various components, spinal fluid drainage, oophrecomy, mastectomy, drowning, implantation of the uterus in the colon, emetics and blistering (Ong 2004)

During the time when convulsions were attributed to excess blood and fluid in the brain (pre 18th century) treatments were directed at ridding the body of excess fluid by purging, sweating, diuresis, starvation, the use of emetics and spinal fluid drainage (Chesley 1984). These methods also helped remove 'toxins' from the body when the toxaemia theory became popular and were also thought to reduce oedema wherever fluid accumulated. The use of sedatives and narcotics to sedate and paralyse became popular in the 19th century in an attempt to prevent convulsions. Before the advent of successful anaesthetising and labour induction techniques, women were sedated and left in a dark room to await the onset of spontaneous labour. Primitive induction techniques involving the insertion of objects into the cervix and vagina in order to induce labour and rupturing of the membranes were also used rather than awaiting natural labour in women who were convulsing. In the late 19th century, caesarean section was performed more regularly, yet the operation was performed too late on the majority of women, many of whom had already been convulsing for many hours and sometimes even days at home and was not a successful option in many cases for saving the life or either mother or baby. Other

surgical procedures included oophrectomy, mastectomy, renal decapsulisation, ventral suspension of the uterus and postpartum curettage (Ong 2004).

Low protein diets were recommenced to pregnant women when proteinuria was noted as a predictive sign in 1843, as a preventative measure against convulsions (Chesley 1984).

The introduction of magnesium sulphate ($MgSO_4$) by intravenous route by the American physician Lazard in 1925 heralded a new era in seizure management (Chesley 1984). Prior to its use in eclamptic women, $MgSO_4$ had been noted to control convulsions resulting from tetanus. Improved measures for inducing labour, surgical expertise and most importantly the introduction of anti-hypertensives in the 1950s have been the hallmarks of modern treatment. Outcome driven research does not appear in the literature to record the success of these treatments until the 1960s with the first international epidemiological study being conducted in 1971 (Davies).

1.4 CURRENT TREATMENTS

In the last two decades collaborative research has focused on refining drug therapies such as aspirin (Askie et al. 2007), $MgSO_4$ (Altman et al. 2002), antioxidants (Rumbold et al. 2006) and anti-hypertensives (Abalos et al. 2007) – although delivery of the fetus and placenta still remain, as they did in 1773, the only cure (Ong 2004).

1.5 TERMINOLOGY

A cyclical nature of the theories of aetiology has been reflected in the historical trend of ever changing terminology; preeclampsia, eclampsia, gestational hypertension, pregnancy induced hypertension, hypertension with or without proteinuria, proteinuria with or without oedema, toxaemia and preeclamptic toxaemia. The debate concerning terminology and disease categorisation dominated this niche literature during the 1980s and 1990s (Brown & Buddle 1997). The use of an internet search engine for consensus statements and guidelines for the diagnosis and treatment of hypertension in pregnancy produced 13,770 potential sites. Statements exist from: Australia and New Zealand (Lowe et al. 2008), Canada (Magee et al. 2008), the United States of America (USA) (Zamorski & Green 2002; Wagner 2004; Agency Healthcare Research and Quality 2000), the United Kingdom (UK) (Royal College of Obstetricians and Gynaecologists 2003, Milne et al. 2005; National Health Service 2001; Tuffnell et al. 2006), the International Society for the Study of Hypertension in Pregnancy (Brown et al. 2001), World Health Organisation (WHO 2008) and the Republic of South Africa (Woods 2006) and all use different terminology to refer to the Hypertensive Disorders of Pregnancy (HDPs). A scarcity of guidelines exist in those countries where the highest rates of maternal mortality occur, such as Afghanistan, Uganda, Ghana, Rwanda, Malawi, Nepal and Nigeria (Engender Health 2007).

> …the greatest contributors to bias and non-comparability of data are ignorance of pathogenesis, difficulties of diagnosis and the absence of a system of classification, universally accepted and applied. No published figures on the toxemias can be accepted at face value without enquiry into the clinical criteria and definitions used (Davies 1971, p. 33).

The Cochrane Collaboration Database of Systematic Reviews contains 29 reviews on the management of the HDPs (Duley, Williams & Henderson-Smart 1999; Duley & Henderson-Smart 1999; Duley & Gulmezoglu 2000; Bergel, Carroli, Althabe 2002; Duley, Gulmezoglu & Henderson-Smart 2003; Hofmeyr et al. 2003; Magee & Duley 2003; Matchaba & Moodley 2004; Duley, Henderson-Smart, Meher 2005; Meher, Abalos & Carroli 2005; Meher & Duley 2005; Hofmeyr, Atallah & Duley 2006; Li et al. 2006; Duley, Henderson-Smart & Meher 2006; Meher & Duley 2006a; Meher & Duley 2006b; Meher & Duley 2006c; Meher & Duley 2006d; Meher & Duley 2006e, Makrides, Duley & Olsen 2006; Churchill & Duley 2007; Steyn & Steyn 2007; Meher & Duley 2007; Duley, Henderson-Smart & King 2007; Duley et al. 2007; Abalos et al. 2007; Duley et al. 2007; Knight et al 2007; Rumbold et al. 2008). The main issue with consensus statements and guidelines is that whilst the intention is to guide clinical practice, the transition from paper to practise does not always occur. There have been studies (Davis, Homer & Brown 2002, Foy et al. 2004) conducted to determine if physicians and obstetricians follow guidelines and consensus statements even when one exists for their locality. The conclusion of these surveys was that clinicians practice a variety of diagnostic methodology and utilise a wide field of treatments independent of evidence existent.

> Such a loosely used and inclusive term as toxemias of pregnancy can no longer be regarded as precise or specific. It should be subjected to critical analysis, perhaps dissected out until there is nothing left (Herrick & Tillman 1935, p. 643)

For the purpose of the work herein the following terminology will be used: preeclampsia (PE), gestational hypertension (GH), chronic hypertension (CH) and preeclampsia superimposed on chronic hypertension (SP). The following table outlines these diagnostic groupings and the criteria used for diagnosis.

ASSHP	Criteria
Gestational Hypertension	≥140 and/or ≥90 mmHg on 2 separate occasions 4 hours apart, after 20 weeks gestation
Preeclampsia	≥140 and or ≥90 mmHg on 2 separate occasions 4 hours apart accompanied by **one or more** of the following: 1. Proteinuria (≥300 mg/24hr) 2. Renal insufficiency (serum/plasma Creatinine 0.09mmol/l) 3. Liver disease (abnormal LFTs or pain) 4. Neurological problems (eclampsia, Hyperreflexia with clonus, severe headache with hyperreflexia, persistent visual disturbance) 5. Haematological disturbance (thrombocytopaenia, DIC, haemolysis) 6. Fetal growth restriction
Chronic Hypertension	≥140 and/or ≥90mmHg pre-conception or before 20 weeks. May be essential or secondary
Preeclampsia Superimposed on Chronic Hypertension	≥140 and/or ≥90mmHg pre-conception or before 20 weeks. May be essential or secondary - With features of preeclampsia as previously detailed.

Table 1.1. Diagnostic features. Diagnostic features of the Australasian Society for the Study of Hypertension in Pregnancy (ASSHP) consensus statement

This terminology is that of the Australasian Society for the Study of Hypertension during Pregnancy (ASSHP) (Brown et al. 2000) which was amalgamated with the Obstetric Medicine Group of Australasia in 2004 and later amalgamated into the Society of Obstetric Medicine Australia and New Zealand (SOMANZ). At the time of data collection (2006/7), classification of these disorders was under the auspices of the Guidelines provided by ASSHP, although a 2008 version is now available under the SOMANZ name (Lowe et al. 2008).

1.6 CURRENT STATE OF PLAY

Features of the disease process can include hypertension, proteinuria, oedema, coagulopathy, liver dysfunction, kidney dysfunction, haemolysis, neurological symptoms (including scotomata and scintillations), headache, blindness, placental abruption, fetal growth restriction, eclamptic seizures, pulmonary oedema and cardiomyopathy. Some women will experience many of the signs and symptoms whilst others only one, yet we still define hypertension in pregnancy by the primary characteristic sign of raised blood pressure.

Nulliparity, maternal age greater than forty years, family history, new paternity, underlying renal disease, diabetes mellitus, multiple gestation, obesity, existing autoimmune disease, thrombophilic states and severe alloimmunisation all increase the risk of developing a HDP (Myers & Brockelsby 2004) Other possible risk factors include limited exposure to sperm (Einarsson, Sangi-Haghpeykar & Gardner 2003) , long inter-pregnancy interval (Basso et al. 2003), maternal work patterns (Higgins et al. 2002), the use of assisted reproductive techniques (van Disseldorp et al. 2010) and periodontal disease (Boggess et al. 2003).

Regardless of our knowledge of signs, symptoms, risk factors, treatments and theories of causality, well defined, systematically recorded outcomes for women and babies are not established across any national on international arena and it is this knowledge deficit which the work herein aims to address.

1.7 SCOPE OF DISEASE

1.7.1 MATERNAL AND PERINATAL MORTALITY

The HDPs affect between 7-10% of child bearing women worldwide (WHO 2008). In terms of maternal mortality, the HDPs result in approximately 134 maternal deaths every day, or 12% of the 424 000 maternal deaths which occur world-wide each year. Ninety-nine percent of these deaths occur in developing countries (Dolea & AbouZahr 2003; WHO 2008). We know that this discrepancy between maternal mortality ratios (MMR) in the developed, compared to the developing world, is the largest discrepancy of any health indicator (Kiklpatrick et al. 2002). In developing countries, causative factors for this rate are lack of effective antenatal care, scarcity of trained birth attendants, scarcity of medications to treat blood pressure and eclampsia and insufficient equipment to measure blood pressure or to administer medications when medications are available.

Whilst the scale of mortality is much smaller in developed countries (1% of all maternal deaths), the rate of maternal death attributable to the HDPs is similar (Sullivan, Hall & King 2007; Callaghan, MacKay & Berg 2008). Of the 104 direct deaths which occurred in the UK between 2000-2002, 17 cases or 16% were attributable to preeclampsia (National Institute of Clinical Excellence 2005). A study of contributing factors to maternal mortality in women with a HDP in the Netherlands (Schutte et al. 2008), found that in 96% of cases substandard care practices were present.

Data on maternal mortality in Australia are collected via the Maternal Deaths Data Collection. This is a non-mandatory reporting of maternal deaths which occur in Australia to the National Perinatal Statistics Unit (Sullivan, Hall & King 2007). The Australian Institute of Health and Welfare (AIHW) compiles these reports into a Maternal Death Report. Deaths are classified as:

a. Direct – deaths resulting from obstetric complications of the pregnant state eg. postpartum haemorrhage

b. Indirect – deaths resulting from pre-existing disease or disease that developed during the pregnancy but was not the direct result of the pregnancy eg. Type I or II diabetes

c. Incidental deaths – resulting from conditions which occurred during pregnancy but were not the result of the pregnancy eg. road accidents

In New South Wales (NSW) Australia in 2004 there were seven maternal deaths (8.2 per 100 000 pregnancies), none of which were directly attributable to a HDP (Centre for Epidemiology & Research NSW, 2007). It is estimated that maternal death in Australia is underreported in 30% of cases (King 2009) and that for every maternal death, there are 80 cases of severe morbidity.

1.7.2 MATERNAL AND PERINATAL MORBIDITY

Whilst we are aware of the mortality burden of the HDPs, our knowledge of the scale of morbidity associated with these disorders remains unclear. We do know that 17% of neonates admitted to Neonatal Intensive Care Units (NICU) in NSW in 2004 were admitted as the result of a maternal diagnosis of HDP (Centre for Epidemiology & Research NSW, 2007), yet the numbers of women who suffer acute renal failure (ARF), acute pulmonary oedema (APO), have an intra-cerebral event or leave hospital still medicated on anti-hypertensives remains unclear. We gain our knowledge of the incidence of these morbidities via what is reported in clinical trial data, for example APO rates were reported in the MAGPIE trial (Altman et al. 2002) or from published clinical audits such as the ARF rate in South Africa (Moodley 2004), yet these are only snapshots of a limited group of women recruited into clinical trials rather than representative of the wider population. Indicators of outcome for these large scale studies should be; 1. Broad ranging, 2. Chosen by multidisciplinary teams, 3. Address a specific research question and 4. Easy to obtain and accurately recorded. Achieving a smaller set of highly relevant clinical indicators for use in everyday clinical practise remains the challenge.

1.8 BENCHMARKING

1.8.1 DEFINITION

Historically is it thought that the term benchmarking was originally used in the cobbling trade, in which a customer would place their foot upon a bench and a mark would be made by which the shoe could be designed (Boxwell 1994). The term is also used by surveyors and archaeologists who place a mark – the benchmark - the place from which all measurements are taken. The benchmarking process as a theoretical science is attributed to the Xerox Corporation, and the Xerox manager, Robert C Camp in the late 1970s. In his book entitled Benchmarking: The search for Industry Best Practices That Lead to Superior Performance, Camp (1989) describes the process by which the Xerox Corporation sought to learn and copy from it's rivals, mainly Japanese Corporations, in order to improve manufacturing processes and thereby, corporate profit. The six sigma management strategy, a theory by which the focus is to achieve no more than a level of six errors by 100 000 units of production (Mikel 1988) is another commonly used quality improvement process which aims to improve quality and outcomes by removing errors from manufacturing and business process.

Camp (1989) identifies 11 steps in benchmarking:

1. Select subject ahead of monitoring period

2. Define the process

3. Identify potential partners

4. Identify datum sources

5. Collect data and select partners

6. Determine the gap

7. Establish process differences

8. Communicate

9. Adjust goal

10. Implement

11. Review/recalibrate

Simplistically, the benchmarking process involves studying the performance of one company and comparing this performance with another in order to establish what processes achieve optimal outcomes. These best practices are then adopted by the worse performers in order to increase productivity. The key themes are measurement, outcomes, identification of best practices, implementation and improvement (Anand & Kodali 2008). There is an underlying assumption here that the worst performers

have an innate desire to improve performance and to direct the lessons learnt through the process in order to achieve improvement.

Benchmarking has been described as both a snap-shot and as a continual process. The 'snap-shot' approach records the outcomes of an entity and the processes by which those outcomes are achieved over a pre-determined time-point. A highly functioning benchmarking process, by contrast, is continual in nature; one in which best practices are always evolving and the re-measurement of outcomes needs to be ongoing to keep up with this evolution. This continual benchmarking is often referred to as 'Best Practice Benchmarking' or 'Process Benchmarking' (Andersen & Pettersen 1996).

Benchmarking requires a major investment of both time and resources and needs to be done meticulously (Vaziri 1992; Vaziri 1993). The improvement in outcomes brought about by benchmarking has to be deemed worthy of the investment made in its undertaking. The commitment of stakeholders in the process also needs to be firm and resolute to justify the resources committed.

A comprehensive survey was commissioned by the Global Benchmarking Network in 2000 (Jarrar & Zairi 2001). The president of this network is Robert Camp, and it represents 22 countries. The survey was answered by 450 corporations in 40 countries and the results indicated that:

1. Significant benefits can be obtained from well instituted Best Practice Benchmarking – with 20% of projects resulting in increased profits of US$250 000.

2. 77% of companies use Mission and Vision Statements and conduct customer surveys as benchmarking tools.

3. 'Performance Benchmarking', 'Informal Benchmarking' and 'Best Practice Benchmarking' were the tools recognised most likely to be instituted by companies over the next three years by those companies not currently using them.

4. Benchmarking was a process which was now being used in many fields including: manufacturing, insurance, financial services, construction, banking, teaching and learning as well as the health sector.

Numerous benchmarking classification schemes exist, with Ananda and Kodali (2008) recording 61 variations, with the greater majority of these being tailored versions of the original proposed by Camp (1989); enhanced and modified to fit a variety of industries and situations. The model proposed in the work herein can be categorised as an 'Academic/research based model' – those models which are devised through the experience and knowledge of the user and adapted to individual application (Ananda & Kodali 2008).

1.8.2 CLINICAL INDICATORS

Clinical Indicators are the tools utilised in health sector benchmarking. They are measurement devices which can be applied across a multitude of settings due to their uniformity. There is a standardisation process which involves the establishment of strict definitions to ensure consensus of data being collected. They should serve to bring attention to a certain facet of what is being measured. In the healthcare setting, they usually bring attention to a type of morbidity or alternatively, an intervention (AIHW 2003).

Clinical indicators form the basis of quality improvement. The selection of clinical indicators is a process which needs to be undertaken in collaboration with other participants in the improvement process (Australian Council of Healthcare Standards - ACHCS 2003). An example of such a collaboration would be a multi-disciplinary team which would ideally include a consumer. Indicators need not only focus on negative outcomes but can also encompass positive health outcomes (ACHCS 2004).

Indicators need to be comprised of a definition of content, incorporated in the numerator and the denominator and a setting in which they are to be applied. The definitions need to be as unambiguous as possible to ensure that ‘true’ comparison is being undertaken.

1.8.3 BENCHMARKING IN THE HEALTH SECTOR

The use of benchmarking techniques has become more common place in the health sector since the 1990s. The use of these techniques has largely been pioneered in this sector in the USA through the publication of yearly reports such as the 'America's Best Hospitals' annual survey (US News and World Report 2009). Performance measurement systems which measure financial performance in the non-health sectors have been modified to consider multiple factors, not just financial benefit, such as consumer satisfaction, length of stay and morbidities, or as stated by Juran and Berwick (1990, p.29), 'Steal shamelessly and implement profusely the things that work in other industries'. The theory behind the transference of these techniques from manufacturing to health is that health care is also driven by improved performance outcomes in which monetary gain is replaced with less morbidity and better health for the consumer (Dey, Harihan & Despic 2008). It would be naïve to assume though that financial performance was not also at play in the health sector as better health (less morbidity, shorter hospital stay) also equates to less cost for the health care provider. This is a consideration not to be dismissed considering that the healthcare 'industry' comprises one-seventh of the Gross Domestic Product (GDP) of the USA (Dey, Harihan & Despic 2008) and 8% in Australia (AIHW 2009). The economic analysis of health care provider performance is the role of the health economist, who must weigh the equation faced with increasing health care costs and limited resources.

There are many examples of how benchmarking has been used in health care, which highlight the techniques involved and their effect. On an international scale, the

World Health Organisation (WHO) in 2003 instituted a practical tool to monitor and improve the performance of hospitals (Groene et al. 2008). The Performance Assessment Tool for Quality Improvements in Hospitals (PATH) was designed to enable clinical effectiveness, efficiency, staff orientation, responsive governance, safety and patient centeredness to be measured. Fifty-one hospitals from six regions completed the initial data collection. Data were compared between countries, but of greater relevance between regions of similar economic development. On a national level, the Taiwan Healthcare Indicator Series (THIS) System was developed to measure the performance of 111 hospitals in Taiwan and to gauge the acceptability of the process from a transparency perspective (Tung & Yang 2008). Other benchmarking initiatives target specific service providers, such as Intensive Care Units (ICU), in which the Acute Physiology and Chronic Health Evaluation (APACHE) model was developed in 1993 (Zimmerman et al. 2006). Now in version IV, the APACHE is a statistical model which benchmarks expected or predicted outcomes and compares them to actual outcomes. This type of predictive modelling is what fuels the casemix modelling incorporated in the Australian Diagnostic Related Groups (DRG) framework (AIHW 2009). This type of datum collection allows national comparisons to be made on predicted factors based on patient and hospital profiles. Sophisticated modelling utilising predictive event techniques such as Expected minus Observed' reporting methodology have assisted in the development of reports such as the Variable Life Adjusted Displays (Victorian Government Health Information 2008) which enable governing bodies to monitor flagged events within institutions to compare actual event episodes.

In a smaller, although no less important way, benchmarking is used in disease-specific areas on a regular basis. These are initiatives which are aimed at measuring the performance of individuals (Scholle et al. 2009) or units/services (Stow et al. 2006). Many programs call themselves benchmarking without a true understanding of the process involved. If data are collected, collated, and fedback to clinicians and the project ends, only half of the benchmarking process has been undertaken. Data on stroke rehabilitation (Bagg, Pombo & Hopman 2006), pain management (Meissner, Ullrich & Zwacka 2006) and diabetes outcomes (Rossi et al. 2008) for example, can all be reliably collected and comparisons made, but the cyclical processes of change implementation and re-evaluation need also to be undertaken to complete the process. An assessment of the promoters and motivators of clinical change in the face of measured outcomes needs also to be undertaken and will be addressed.

1.8.4 BENCHMARKING AND SURVEILLANCE OF OBSTETRIC MORBIDITY WORLDWIDE

1. **UK**

    a. The UK Obstetric Surveillance System (UKOSS) (Knight 2008) was established by the National Perinatal Epidemiology Unit and the Royal College of Obstetricians and Gynaecologists in 2005 to monitor rare obstetric conditions (one case per 2000 births) including amniotic fluid embolism, extreme obesity, myocardial infarction, pulmonary vascular disease, non-renal solid organ transplant, renal transplant, stroke in pregnancy, failed intubation, malaria, uterine rupture and

therapies for postpartum haemorrhage. Reporting to this system is voluntary, and return rates vary from 87-97% per annum since initiation. UKOSS is a surveillance system and does not compare outcomes across units.

2. USA

a. The Centre for Disease Control and Prevention (CDC) reports rates of hypertension (pregnancy associated) and hypertension (chronic) via the National Vital Statistics Report (US Department of Health and Human Services 2009). This mandatory report on all births in the USA does not include other markers of maternal morbidity.

b. A new initiative in the USA is a benchmarking database which will enable automatic extraction of coding from health records (Association of Women's Health, Obstetric and Neonatal Nurses 2008). Extracting Data and Generating Evidence (EDGE) will allow comparisons to be made across units within units and across time periods. This is not a mandatory system and reporting will be on a voluntary basis for those institutions which enrol in the program.

3. European Union (EU)

There is a vast variety of ways in which perinatal information is collected in the EU. In Belgium, the Czech Republic, Denmark,

Estonia, Finland, Germany, Hungary, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, the Netherlands, Norway, the Slovak Republic, Slovenia and Sweden, data collection is compulsory and is via a specialised birth registry, whilst in Austria, Cyprus, Finland, France, Hungary, Poland, and Spain, births data are collected from hospital coding systems. France, Italy and Spain regularly conduct surveys to collect perinatal information from medical records and from interviews with mothers. The definitions of morbidity vary between each datum collection system used and these methods are best utilised to monitor mortality rather than morbidity (Euro-Peristat Project 2008).

1.8.5 BENCHMARKING OBSTETRIC MORBIDITY IN AUSTRALIA – NATIONAL SURVEILLANCE

Surveillance of obstetric morbidity in Australia in monitored via eight different processes.

1. The National Perinatal Data Collection (NPDC) (AIHW 2007). This dataset is constructed from the datum collected under the Midwives Data Collection (MDC) (see Appendix I). In NSW, Australia, data are compiled into the NSW Mothers and Babies Report (NSW Department of Health 2007). The NSW MDC has been in operation since 1986 (known from 1985-1990 as the Maternal and Perinatal Data Collection). This is mandatory datum recorded on every birth ≥20 weeks gestation or >400grams (NSW Health 2005). It was

originally in paper form and more recently in electronic format. Data are entered into this system by the clinical midwives either at delivery or during the postnatal period. This is a limited dataset on events which occur during the initial birth hospitalisation. Morbidity data are non-specific in that severity of complications arising from diagnoses are not recorded. Two validation studies have been undertaken of the MDC, the first in 1990 (Pym & Taylor 1993) and the second in 1998 (NSW Department of Health). Both studies concluded that whilst the MDC was accurate in the majority of datum fields, such as birth date, birth weight and gender, there was a need to improve the reporting of maternal medical conditions, both obstetrically occurring and those pre-dating pregnancy. An independent validation study comparing maternal diagnoses as hand collected and those reported in the MDC conducted in 2003 over a six month period (Thornton et al. 2004), concluded that the MDC miscoded the type of hypertension women experienced in 56% of cases reviewed and under reported cases of hypertension by 12%. This type of datum collection also only collects statistics of the number of women experiencing a complication such as HDP. The current version of the MDC has the potential to capture the hypertensive diagnoses of chronic hypertension and pregnancy induced hypertension – proteinuric or non-proteinuric – terminology which is not in line with the current consensus or the previous consensus statements (Lowe et al. 2008). Information is not available on hypertensive medication prescription, rates of intra-uterine growth restricted neonates born to hypertensive mothers, maternal complications such as ARF, APO, intra-cerebral events such as

stroke, eclampsia, admission to high dependency or ICUs and length of stay variations.

2. The National Hospital Morbidity Dataset (NHMD) is a collection of datum collected by hospitals and reported to the AIHW. This dataset is comprised of codes recorded in the International Classification of Diseases Australian Modified Version 10 (ICD-10-AM) (ICD 2007), which refer to diagnoses and procedures assigned to an individual during a health care encounter. Obstetric outcomes have been assessed previously utilising this coding system. Taylor et al. (2005) examined data for accuracy of recording of perinatal morbidity and found that in relation to the accuracy of the recording or the maternal diagnosis or preeclampsia, the NHMD had a sensitivity of 50% and a specificity of 99.8%.

3. The AIHW also reports on specific pregnancy related conditions. In 2008 a report on gestational diabetes mellitus in Australia 2005-2006 (AIHW 2008) provided a snapshot of this condition for Australian women compiled from data collected under the NHMD and by the National Diabetes Services Scheme database. This review was based upon health care encounter coding and was not validated against medical records as reviewed by an independent assessor. The accuracy of the data produced by such reports is not known and to date there has never been such a review of the HDPs.

4. Australian and New Zealand Intensive Care Society (ANZICS) adult patient database is a voluntary database which records approximately 65% of adult

ICU admissions which was expanded in 2007 to include a coding for pregnancy status (Stow et al. 2006). The limitation on this dataset is that not all cases of serious morbidity are admitted to an ICU as there is no national guidelines in regard to which cases require mandatory admission to ICU and the voluntary status of this dataset questions it's validity.

5. The Australian Maternity Outcomes Surveillance System (AMOSS) is a National Health and Medical Research Council (NHMRC) grant-funded initiative established by the Perinatal & Reproductive Epidemiology Research Unit School of Women's & Children's Health University of NSW in order to record cases of serious maternal morbidity in Australia (AIHW 2009). This a voluntary database instituted to record the number of cases of conditions reported by maternity units which are considered to have an incidence of 1:1 000 births, including antenatal pulmonary embolus, amniotic fluid embolism, morbid obesity, cardiac disease, placenta accreta and peri partum hysterectomy. AMOSS is a surveillance system and does not compare outcomes across obstetric units.

6. The Australian Council on Healthcare Standards Australasian Clinical Indicators for Obstetrics (ACHS 2010) contains ten clinical indicators upon which voluntary reporting may occur to participating institutions. These indicators include:

    a. Induction of labour for other than defined reasons

b. primary non-elective caesarean section for failure to progress

c. primary caesarean section for fetal distress

d. primiparous patient - intact lower genital tract

e. primiparous patient – surgical repair of lower genital tract 1st degree tear

f. primiparous patient – surgical repair of lower genital tract 2nd degree tear

g. primiparous patient – surgical repair of lower genital tract 3rd degree tear

h. primiparous patient – surgical repair of lower genital tract 4th degree tear

i. term babies Apgar score (an assessment of the physical condition of a newborn infant; involves heart rate and muscle tone and respiratory effort and colour and reflexes) of four or below at five minutes

j. term babies transferred to a NICUs other than for congenital abnormality

Organisations which participate in this initiative provide date to the statistics unit of ACHS where they are analysed twice yearly. Comparative reports from all units which provide datum are provided as feedback. These results are used in hospital assessments such as the Evaluation and Quality Improvement Program (EQuIP).

7. The Victorian Government reports on the Victorian Maternity Services Performance Indicators annually (Victorian Government Department of Human Services 2009). This is a mandatory reporting of the following indicators:

    a. Rate of inductions in standard primiparae

    b. Rate of caesarean section in standard primiparae

    c. Rate of 3rd and 4th degree perineal tears in primiparae

    d. Term infants without birth defects who require additional care

    e. Rate of administration of antenatal corticosteroids to women < 34 weeks gestation

    f. Vaginal birth after primary caesarean section

    g. Perinatal mortality

h. Rate of women transferred to postnatal domiciliary care

i. Rate of women offered appropriate intervention in relation to smoking

j. Provision of appropriate breastfeeding support or advice

k. Rate of women receiving timely hospital antenatal clinic service

l. The rate of women who receive appropriate interpreter services

These indicators are reported on a hospital basis to allow clinicians and consumer appraisal and are one of the few series of indicators which reflect on both positive and negative outcomes.

8. The Women's Hospital Australasia (WHA) was formed in 1994. Membership is voluntary. Benchmarking involving a set of obstetric clinical indicators is a component of the undertakings of this group. The indicators include:

a. Caesarean section rates

b. Vaginal birth after caesarean section

c. Spontaneous vaginal birth

d. Induction of labour

e. Instrumental birth

f. Epidural for pain relief

g. General anaesthesia for caesarean section

h. Episiotomy and third or fourth degree perineal tear

i. Postpartum haemorrhage and transfusion

j. Apgar score

k. Hypoxic Ischaemic encephalopathy

The views of the WHA in regard to benchmarking maternity care are as follows:

> "Clinical indicators for maternity care should be used to monitor significant inter-unit variation and to detect both positive and negative trends over time with the aim of ultimately improving clinical outcomes. A national system, that ensures at least annual reporting of a core set of maternity indicators, needs to be enacted in Australia to monitor the safety and quality of hospital-based maternity care provision. Results should be reported by peer groups of hospitals in a format that shows the mean, variance around the mean, and gains to be made by shifting the mean in a positive direction. Each maternity service also needs to have in place a robust clinical governance framework to ensure that clinical teams have the capacity to review the outcomes for each indicator with the aim of improving clinical practice." (WHA 2007, p.XIII)

Unfortunately at present, the benchmarking initiative of the WHA does not include indicators which measure medical disease during pregnancy and only

two of the five Australian units which were assessed in the work herein are members of the WHA.

The common theme throughout all of these databases is the recording of the incidence of an event. Databases such as these can inform as to the number of cases of amniotic embolism, for example, but are not necessarily designed for the purpose of benchmarking *per se*. Some collections, such as the NSW Mothers and Babies Report do compare outcomes between health sectors or health services within a geographically defined area, yet the majority report on the state/area in which they were recorded as an entity. The voluntary reporting nature of many of these datasets also limits the validity of the data captured. The reliance upon clinicians to report events to mandatory bodies is a further limiting factor in the reliance upon existing datasets to establish standards of clinical care which reflect optimal outcomes.

There are obstetric audit initiatives which have been undertaken within Australia (De Lange et al. 2008) and internationally (Danel et al. 2003; Ekele & Ahmed 2004; van Roosmalen & Zwart 2009) in the obstetric setting. These undertakings have been conducted on a once-off basis aimed at determining rates of morbidity of a given condition over a specified time period. The focus of these studies is severe rare morbidity (i.e. those having an incidence of 1-2/1 000 births) or the occurrence of what are known as near-miss events (Penney & Brace 2007; Pattinson 2009). These are events which barring timely medical and/or surgical care would have resulted in death. The great majority of instruments devised and techniques used to monitor maternal morbidity (such as UKOSS and AMOSS) focus on this type of morbidity. The problem with this type of monitoring is that it takes the emphasis off many other types of maternal morbidity which are also life threatening, albeit much less rare,

such as APO and ARF. Datasets such as these also do not collect datum on pre-disposing factors, treatment variations and sequelae relevant to such events.

1.8.6 BENCHMARKING THE HDPS

With a paucity of reliable international, national or even state information collected on the HDPs how are we to assess the efficacy of the service we provide?

An epidemiological study of preeclampsia was undertaken in 1970 (Davies 1971). It concluded this disease affected women in all nations, although maternal mortality differed greatly.

For such a commonly occurring pregnancy complication, clinical outcomes and incidence of the HDPs have not been studied extensively. The oldest printed reference to an outcome evaluation of women following a HDP was by Herrick and Tillman (1935) of 930 women over a 15 year period. This study concluded that toxaemia affected women's long term vascular health and shortened their life expectancy. A study conducted in the USA over a 12 year period examining 0.7% of births (Zhang, Meikle & Trumble 2003) concluded that women with preeclampsia had a 3-35 fold increased risk of severe complications such as placental abruption, thrombocytopaenia, disseminated intravascular coagulation, pulmonary oedema and aspiration pneumonia. Outcomes for women with proteinuric compared to non-proteinuric disease presentations have also been undertaken (Buchbinder et al. 2002; Homer et al. 2007) without inclusion of other variations in disease presentation and with varying results. A 1997 study (Brown & Buddle) which utilised hand recorded

pregnancy details in a prospective fashion for 1 183 woman diagnosed in accordance with the ASSHP diagnostic criteria, reported that women with superimposed preeclampsia suffered more complications than other diagnostic categories and a greater number of fetal complications. This is a finding which has been replicated in other studies (Rey & Couturier 1994; Ray et al. 2001).

The outcomes for 4 589 women and their babies enrolled in the Calcium for preeclampsia prevention study (Hauth et al. 2000) showed that women with hypertension had increased maternal morbidity such as operative delivery, placental abruption, renal dysfunction and increased neonatal morbidity.

A database study of all women who were delivered in NSW over a two year period was undertaken (Roberts et al. 2008). Relying upon the MDC, this study concluded that 9.8% of women were affected by a HDP and that these women suffered greater major morbidity and mortality than the normotensive cohort. A South Australian study was conducted in 1998-2001 which examined the outcomes for 5 356 hypertensive women. Women were examined over a four year period relying upon data collected via the South Australian Perinatal Data Collection Form (Heard et al. 2003). Heard et al (2003) compared birth type, delivery gestation and neonatal characteristics such as birthweight, Apgar scores (an assessment of the physical condition of a newborn infant; involves heart rate, muscle tone, respiratory effort, colour and reflexes conducted at 1, 5 and 10 minutes of life) and special care nursery admissions between women with pregnancy hypertension and those with pre-existing hypertension. Women with pregnancy hypertension had a higher rate of operative delivery and worse perinatal outcomes. This study was also analysed utilising

multiple regression statistical techniques (Vreeburg et al. 2004). What studies such as these do not tell us though, is the variation in clinical practices which may have resulted in variable outcomes or the depth and variety of morbidity which occurs in these cohorts and which are not reported in datasets such as those used in these studies.

The common theme amongst all of these studies is that interunit, interstate or international comparisons were nor undertaken and whilst each study adds significantly to our knowledge of the HDPs at a given time point, none address the issue of outcome improvement across time or units.

1.9 HOW TO FACILITATE CHANGE IN THE CLINICAL ENVIRONMENT

1.9.1 THE PROCESS OF CHANGE

A true revolutionary for his time, Ernest Codman was a Boston physician who tried to establish a registry of all patients at the Massachusetts General Hospital in 1914 to determine whether treatments had been successful. Unfortunately for Codman his revolutionary ways created a threat to the existing *status quo* within his institution and he was expelled from the medical staff, yet his work is an example of early surveillance of health care performance (Brand 2009).

Patient safety became a focus issue for health care providers with two landmark events in the late 1990s. The first was the Bristol Royal Infirmary Inquiry (The Bristol Royal Infirmary Inquiry 2001) and the second was the publication of the Kohn, Corrigan and Donaldson study, To Err is Human, in 2001. The former identified the key issues of accountability and reporting in monitoring patient safety and outcomes. The latter named human and process systems failures as the two major causes of accidents in hospitals. Following this seminal work there was a large shift towards looking at patient outcomes as dependent upon the systems in place within an organisation rather than placing blame upon individuals for poor outcomes. The existence of protocols, checklists, simulations/drills, clinician feedback fora and a focus on the team rather than the individual, have all been effective processes utilised within the obstetric environment to improve outcomes (Main, Bloomfield & Hunt 2004; Main & Bingham 2008; Nielsen & Mann 2008; Clark et al. 2007; Factor

et al. 2000; Clark et al. 2008). Many of these techniques have been modified for medicine from those instituted in the aviation industry in attempts to improve flight safety (McGreevy et al. 2006; Sexton, Thomas & Helmreich 2000). The overall impression from the discussion of these different strategies is that no one strategy in isolation works effectively, but that practice improvement needs to be tackled on multiple fronts to be instituted effectively (Lomas et al. 1989; Chaillet et al. 2006; Althabe et al. 2008)

1.9.2 BARRIERS TO CHANGE

A series of key barriers to affecting change in the clinical environment have been identified:

1. Unwarranted variations in clinical practice due to lack of protocols or clinician consensus (Wennberg 2002)

2. Systems in which, due to protocol absence or non-adherence, clinicians' practise is not influenced by team approaches or driven by protocols (Sexton, Thomas & Helmriech 2000)

3. Reliance upon empirical rather than scientific knowledge (Kohn, Corrigan & Donaldson 2001)

4. Clinicians basing their practise on personal experience or the experience of mentors (Richens, Malone & Morrell 2004)

5. Lack of effective leadership (Kohn, Corrigan & Donaldson 2001)

6. Lack of team environment (Kohn, Corrigan & Donaldson 2001)

7. Lack of effective monitoring systems (Main & Bingham 2008) including the lack of or existence of ineffective or out dated datum collection systems to enable comparisons to be made.

8. Traditional medical training (Davis et al. 1999). Didactic medical education at both the undergraduate and postgraduate levels has been shown to be ineffective in instituting clinical practice change. Case discussions, role play and practical sessions were shown to be overall more effective means of training.

1.9.3 STRATEGIES WHICH HAVE BEEN USED TO IMPLEMENT CHANGE IN OBSTETRIC PRACTISE

1. Audit and feedback (Chaillet et al. 2006). Chaillet conducted a systematic review of 3 910 studies identified as describing techniques to change obstetric practice. Of this number, 1 596 incorporated the use of audit and feedback. Eleven of these studies evaluated whether audit and feedback actually resulted in practice change and in 82% of studies, positive change was noted. Strategies to ensure effective audit and feedback include the identification of a single event which occurs commonly to large numbers of patients (Robson, Scudamore & Walsh 1996). For these reasons, utilising

audit and feedback for changing obstetric practice in relation to the HDPs fits all of these criteria, as the HDPs affect 10% of women and practitioners come into contact with hypertensive women every day within the clinical domain.

2. Academic detailing – or expert training, was identified as the second most commonly used technique (829 studies). This can be from a local individual, but more commonly from a visiting scholar.

3. Guidelines/protocols – are generally only found to be effective in changing practice if they are well implemented (Richens, Malone & Morrell 2004). Means to achieve this are: widely publicised written and verbal dissemination of content, inclusion of multi-disciplinary staff in both writing and dissemination, and training sessions which involve practical displays. Merely writing a protocol and putting it into a clinical area is not an effective method of instituting change (Davis & Taylor-Vaisey 1997), but must be followed by written reminders and multiple methods of staff education.

Previous work examining the use of guideline implementation for the HDPs (Foy et al. 2004) concluded that the wide variation in practices in relation to the treatment of the HDPs makes guideline implementation challenging.

Another study of the effectiveness of the dissemination of consensus statements concerning the HDPs as reflected by clinical practice change was equally as disheartening (Davis, Homer & Brown 2002).

There could be several reasons why this is the case:

1. Disorders without known aetiology – without known causative factor or factors it is difficult to gain consensus upon optimal treatment, or consensus on nomenclature.

2. Wide variety of clinical and laboratory presentation – the mere fact there are several types of hypertension noted in pregnant women with a variety of clinical presentations and laboratory findings make the idea of an effective guideline which will cover all presentations implausible to many clinicians.

3. Lack of clinical trial evidence – the lack of randomised controlled trials (RCTs) investigating the HDPs and the paucity of reliable evidence make the writing of effective guidelines and protocols difficult.

This lack of clinical trial evidence disempowers clinicians in regard to treatment choice not only the prevention of rarer complications such as APO, but also in the provision of accurate advice to patients as to long term health benefits of treatment for both themselves and their babies.

CHAPTER 2

AIMS AND HYPOTHESES

2.1 THE AIMS OF THIS STUDY WERE TO:

1. Collect uniform data based upon pre-established criteria from five tertiary referral maternity units and one non-tertiary referral unit in NSW and Canada over a twelve month period for every woman diagnosed with a HDP

2. To determine via data analysis if outcomes, as measured by pre-determined clinical indicators, differed between units over this set period

3. To determine, via survey techniques, if clinicians were favourable to the process of benchmarking, audit and feedback as a scientific methodology upon which to institute protocol change

4. To choose one clinical indicator where outcomes differed significantly between two units and to explore these differences in detail based upon treatment and diagnostic data collected over the pre-determined twelve month period

5. To confirm the consistency of this outcome variation by conducting a second data collection period at both institutions over a second twelve month period

6. To use the process of feedback and education to determine if practitioners were receptive to protocol variation based upon this one clinical outcome variation between the two units

7. To determine if protocol variations instituted at the unit with the highest rate of morbidity (based upon this one clinical variation chosen) were adhered to by conducting a third audit at the target institution over a twelve month period

2.2 THE HYPOTHESES TO BE TESTED WERE:

1. That the benchmarking of a refined set of clinically relevant outcomes for women with the HDPs could be achieved across units

2. That obstetric units could work in collaboration in order to achieve this aim

3. That the benchmarking circle could be completed for one or more indicators for which there was significant difference between units

4. That clinical practice change or sustained improvement could result from this initiative

CHAPTER 3

METHODS

The methods section is presented under the headings of Experiment 1: The Benchmarking Initiative and Experiment 2: Clinician Attitudes

3.1 THE PHASES OF EXPERIMENT 1 – BENCHMARKING THE HDPS

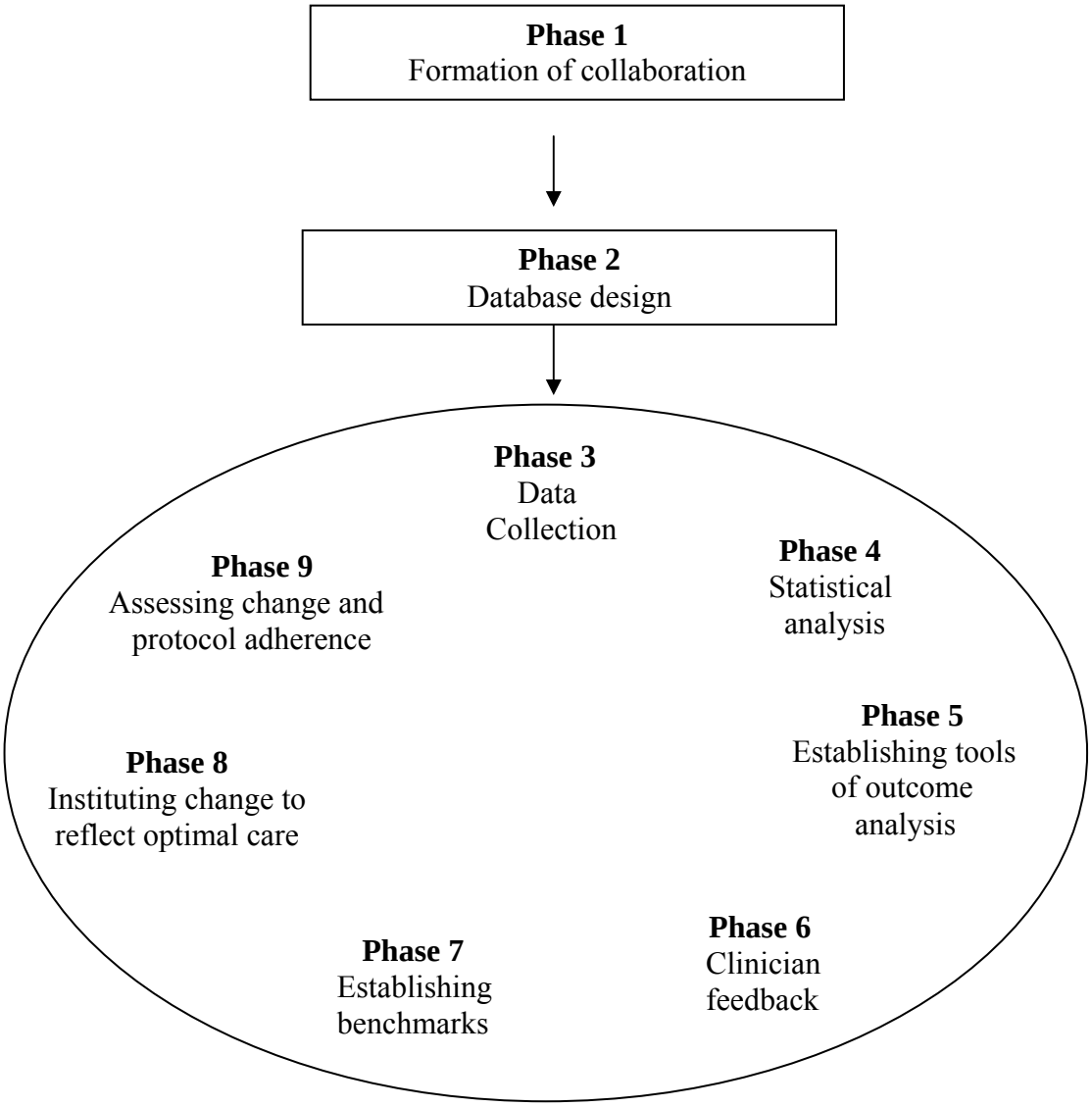


Figure 3.1. Phases of Experiment 1**.** The nine phases of Experiment 1 illustrating the progression of events

3.1.1 PHASE 1: THE FORMATION OF THE COLLABORATION

The five unit NSW collaboration commenced in 2002 through an interested partnership of obstetricians, midwives and obstetric physicians who met on a six monthly basis to share research results and proposals for future work. The proposal

for an outcomes driven project was presented to the group and representatives from Royal Prince Alfred Women and Babies (RPAH), Nepean Hospital (NH), Liverpool Hospital (LH), Royal North Shore Hospital (RNSH), and Campbelltown Hospital (CTH) agreed to collaborate on the project herein.

In 2004, the proposed project was presented at the International Society for the Study of Hypertension in Pregnancy bi-annual scientific meeting in Lisbon, Portugal, with an open invitation to participate in a comparison study. Representatives from the British Columbia Women's Hospital and Health Centre (BCW), Vancouver Canada expressed a strong interest in joining the collaboration. Clinicians from the Canadian unit voiced concerns regarding the outcomes for the women under their care. It was thought that the Canadian unit would have a similar ethnic and demographic profile to the units within NSW. This assumption proved to be correct.

	New South Wales	**British Columbia**
Population	6.3 million	4.3 million
Births state/province	90 600	41 000
Births hospital	RPAH 4 400	BCW 7 400
Neo Mortality	4.6/1 000	4.2/1 000
Smoking population	19%	19.3%
Life expectancy	Males75 years	Males 78 years
	Females 81 years	Females 83 years
Weekly earnings	$963 (AUD)	$952 (AUD)

Table 3.1. Demographics of units 1 and 6**.** Demographic comparison of New South Wales (Australian Bureau of Statistics 2009) and British Columbia (BC Stats 2009)

Face to face meetings and email communication cemented the collaborative group and institutional human ethics approval was sought and gained at the six target institutions.

3.1.2 PHASE 2 – DATABASE DESIGN

The data collection phase of the project was planned to be conducted over a one year period.

An excel database was designed for data collection.

The following considerations were taken into account when deciding upon datafields:

1. Data needed to be comprehensive without being overwhelming. Utilising the current incidence of hypertension during pregnancy and the total delivery rates at the collaborating institutions, it was estimated that >2000 medical records would be examined. To be able to achieve the completion of datum collection over a 12 month period, an estimated mean time of 36 minutes would need to be devoted to each medical record.

2. Datum collection would need to cover all parameters of clinical care which could potentially be affecting clinical outcomes. These parameters included medications prescribed, frequency and duration of prescription as well as blood pressure thresholds for medication initiation. Intravenous fluids,

including type, additives and volumes were also deemed to be potential factors in determining maternal morbidity.

3. Baseline measurement such as parity, height, weight, blood pressure at initial healthcare contact (booking visit) and pregnancy complications were all parameters which were deemed potential components of statistical modelling and potential defining parameters which would enable discrimination between units.

4. Delivery type and timing were important parameters as these treatment variations were judged to be defining variations of individual units.

5. Neonatal details including weight, length and head circumference were to be collected and centile calculations were made relying upon gestation at birth and gender. The centile calculations were made utilising the software designed by Beeby (Beeby, Bhutap & Taylor 1996) relying upon the NSW population centile calculations. The birth centiles of neonates born to Canadian women were also calculated utilising the NSW population centile calculations as well as those currently in use in BC (Arbuckle, Williams & Sherman 1993). A comparison of the two centile charts is outlined in the results section.

6. Symptomatology was to be recorded for diagnostics purposes in alignment with the consensus statement of the Australasian Society for the Study of Hypertension during Pregnancy (Brown et al. 2000) as outlined previously.

7. Relying upon the information contained in each medical record, all women were to be independently diagnosed upon established diagnostic criteria as noted above

8. Length of stay was to be recorded.

9. A free text notes section was also to be included to allow recording of information which may be deemed pertinent when the data checking process was undertaken.

The final version of the database consisted of 219 datafields, of which only two fields were the result of calculations from previous fields (body mass index (BMI kg/m^2) and centile calculations).

3.1.3 PHASE 3 – DATUM COLLECTION

The International Statistical Classification of Diseases and Related Health Problems, 10^{th} Revision, Australian Modification (ICD-10-AM) (National Centre for Classification in Health, 1998) was utilized to locate the cases of the women diagnosed with hypertension in pregnancy who delivered within the time period January 1^{st} 2005 to December 31^{st} 2005. In Canada, The International Statistical Classification of Diseases and Related Health Problems, 10^{th} Revision (ICD-10) was utilized for this purpose (WHO 2007). This coding system is the one utilized by clinical coders worldwide, when determining what diagnoses, procedures and treatments any patient undergoes in any transaction with a health care provider. For

the HDPs, this consists of a series of 19 different codes (see Appendix II for a full listing of codes). In assigning these codes to a medical encounter, the clinical coders of each institution are advised to code as per the documentation of the clinician (midwife or obstetrician) and not to assign codes based upon blood pressure readings only as documented in the medical notes.

Utilising ICD-10-AM and ICD-10 codes O10 through O16, lists of women were compiled by the health information departments at all six centres. These lists were used to obtain the medical records which were to be examined. Each physical medical record was examined by hand at each of the six collaborating institutions. All records in Canada were examined by the author on three separate visits. A 10% sample of records were re-examined in the Canadian unit by the primary supervisor of this project. Three reviewers collected data within the Australian units; the author and two research midwives. The data was collected in a uniform and pre-determined manner within the restrictions of the datafields. All queries were taken to the author for clarification. To ensure consistency of datum collection, inter and intra-assessor correlation analyses were performed on a 10% sample of results. Intra-assessor correlation was performed by a re-examination by the author of a 10% sample of all medical notes reviewed by the author. Inter-assessor correlation was performed by a re-examination of a 10% sample of all medical notes reviewed by the research midwives by the author. Cohen's Kappa correlation for agreement was calculated on these samples for all data points within this sample.

The datafields were populated as per all recorded information in the medical records. Biochemical and haematological results were as per printed laboratory results where

available and as per clinician recorded results in cases where printed results were not contained within the medical records. Antenatal length of time on medication was calculated from time of prescription until time of delivery or day of medication cessation, whichever occurred first. Medication dose, was the final dose prescribed of any given medication, even if numerous incremental changes had been made in medication dosages. If a woman had been admitted on numerous occasions in the antenatal period, the total length of antenatal stay was calculated from all antenatal admissions. Total postnatal length of stay was calculated from time of delivery until discharge, with additional days added for women who were readmitted postnatally. Diagnostic categorisation based upon the Canadian Guidelines (Magee et al. 2008) was also recorded for the Canadian dataset. In addition all Canadian women were re-categorised in accordance with Australasian criteria for consistency of diagnoses.

3.1.4 PHASE 4 - STATISTICAL ANALYSIS

All datafields were written in numerical code. Diagnostic groupings for example, were coded as 1. Preeclampsia, 2. Gestational hypertension, 3 Chronic hypertension and 4. Preeclampsia superimposed on chronic hypertension. The only text field was the free text notes field. All categorical fields were coded from 1 onwards, all dichotomous fields were coded as 1=yes and 2=no and all continuous variables were recorded as such.

Each individual unit's dataset was scrutinized for anomalies such as implausible birth weights for gestation and gender, blood pressure values outside the realm of possibility and datum entries which were not in alignment with prescribed coding

guidelines. This data cleaning process was assisted by the writing of data checking script in SPSS®. This process allowed for data checking where able, by returning to the medical record or data exclusion when data checking was not able. The six datum sets were then merged. The free text notes section of the database allowed for documentation to occur which assisted in this process. If an unusual or out of range result was recorded, a note was inserted into this section. For example, in the case of an extreme of birth weight, a note may have been recorded to indicate that this indeed was the correct birth weight.

Dichotomous variables were examined utilizing chi-squared analyses and relative risks (RR) were reported where appropriate. Continuous data was compared using means analyses for normally distributed data and non-parametric testing when parametric means were not available. Logistic modelling techniques were employed to examine dichotomous outcomes. All variables placed into models were compared using chi-squared analyses and only variables with statistically significant interactions were used. Statistical significance was assumed for p values<0.05. Correction of statistical significance for multiple comparisons was not undertaken as we adopted the view of Schulz & Grimes (2005), that in endpoints which are established *a priori* to data collection, such adjustments are not required. Adjustment for multiple comparisons is best suited to clinical trial data gathered following randomization. For endpoints which could be surrogate markers for other endpoints, such as length of stay for women with morbidity, statistically significant differences in surrogate markers are related to the morbidity rather than the HDP diagnosis *per se*.

3.1.5 PHASE 5 – ESTABLISHING TOOLS OF OUTCOME ANALYSIS

As benchmarks had never previously been established for this pregnancy complication, a series of clinical indicators needed to be established by which comparisons between units could be made. This work had previously been undertaken and published (Thornton et al. 2007). Therefore, the following, although the intellectual property of the author, is not new work undertaken within the body of the PhD study, except for the addition of Indicator Four (maternal eclampsia) which was deemed to be an important indicator of maternal morbidity and clinical care.

3.1.5.1 The 8 Clinical Indicators

Indicator One: Maternal Mortality Rate

Rationale: Used to determine direct and indirect death in women diagnosed with a hypertensive disorder of pregnancy: gestational hypertension, preeclampsia, chronic hypertension or preeclampsia superimposed on chronic hypertension.

Definition: Direct death: Deaths of women who were pregnant at the time of death or who died within 42 days of childbirth and who were diagnosed at the time of death, or retrospectively upon analysis following death, as having a hypertensive disorder of pregnancy and whose death was a direct result of the hypertensive disorder of pregnancy.

Indirect death: Deaths of women who were pregnant at the time of death or who died within 42 days of childbirth and who were diagnosed at the time of death, or retrospectively upon analysis following death, as having a hypertensive disorder of pregnancy and whose death was caused by other factor/s not directly attributable to the hypertensive disorder of pregnancy or it's complications. (Sullivan, Hall & King 2007)

Numerator: The number of maternal deaths directly or indirectly attributable to a hypertensive disorder of pregnancy.

Indicator Two: **Acute Pulmonary Oedema Rate**

Rationale: Is a serious and potentially life threatening complication of the HDPs.

Definition: New onset of pulmonary oedema in the presence of a hypertensive disorder of pregnancy occurring within the perinatal period. Acute pulmonary oedema was diagnosed by new moist crepitant rales (not explained by interstitial or other known lung disease and/or chest x-ray and/or decreased oxygen saturation by pulse oximetry. The diagnosis was dependent on the presence of two of the three diagnostic features as recorded by the attending physician.

Numerator: All women who are confirmed by physical and/or radiological findings as having new onset pulmonary oedema in the perinatal period who are diagnosed or subsequently diagnosed as having a hypertensive disorder of pregnancy.

Indicator Three: Acute Renal Failure Rate

Rationale: Reflects severity of end organ damage, intense vasospasm and endothelial glomerular dysfunction.

Definition: Glomerular filtration rate (GFR) decreased by 60% and/or new onset serum creatinine >0.09mmol/L (>75umol/L).

Numerator: All women with elevated creatinine or GFR (as defined) in the perinatal period and who are diagnosed or subsequently diagnosed as having a hypertensive disorder of pregnancy.

Indicator Four: Eclampsia Rate

Rationale: Reflects severity of end organ damage, intense vasospasm, severe hypertension and cerebral oedema.

Definition: Convulsions (seizures) occurring with pregnancy-associated high blood pressure and having no other cause.

Numerator: All women who are diagnosed as having experienced an eclamptic seizure in the perinatal period who are diagnosed or are subsequently diagnosed as having a hypertensive disorder of pregnancy.

Indicator Five: Perinatal Mortality Rate

Rationale: Used to determine perinatal deaths associated with maternal hypertension. Six percent of all perinatal deaths in NSW in 2005 occurred in women suffering from the hypertensive disorders of pregnancy: gestational hypertension, preeclampsia, chronic hypertension or preeclampsia superimposed on chronic hypertension (NSW Department of Health 2007).

Definition: PMR includes all perinatal deaths (stillbirths and neonatal deaths) which occur in the perinatal period: commencing at 20 completed weeks of gestation and ending 28 completed days after birth (AIHW 2007) who were born to women who were diagnosed at the time of death, or retrospectively upon analysis following death, as having a hypertensive disorder of pregnancy and whose baby died as a direct result of the hypertensive disorder of pregnancy as noted by medical record entries.

Numerator: All perinatal deaths (stillbirths and neonatal deaths) attributable to maternal hypertension.

Indicator Six: Admission of term neonates to Neonatal Intensive Care Units rate

Rationale: Admission of a full term baby to a NICU was deemed more relevant to severity of the hypertensive disorder than all admissions, as the majority of pre-term newborn babies are admitted to units regardless of health status.

Definition: All liveborn babies born at 37 or more completed weeks of gestation who have breathed or shown other signs of life, such as cord pulsation, beating of the heart, or voluntary muscle movement, and who have required admission to a NICU for reasons other than congenital abnormality born to mothers diagnosed at delivery or subsequently with a hypertensive disorder of pregnancy.

Numerator: All term babies admitted to NICUs born to mothers with a hypertensive disorder of pregnancy.

Indicator Seven: Birth weight below the 10th and 3rd percentiles rates

Rationale: Perinatal mortality and morbidity is increased in neonates and adults born below the 10th. Intrauterine growth restriction (IUGR) is also an important diagnostic tool for distinguishing preeclampsia from other hypertensive disorders of pregnancy.

Definition: The first weight of the fetus or newborn obtained from charts standardised for sex and gestation falling below the 10th centile (Beeby, Bhupal & Taylor 1998).

Numerator: All birthweights below the 10th centile for all women diagnosed or subsequently diagnosed with a hypertensive disorder of pregnancy.

Indicator Eight: Breast feeding at discharge rate

Rationale: Use of medications, ill-health and separation from the mother due to prematurity have been shown to be barriers to successful initiation and continuation of breastfeeding in mothers with medical conditions such as the hypertensive disorders of pregnancy. Breastfeeding initiation rates have previously been shown to be related to maternal diagnosis of medical disorders.

Definition: "Exclusive" breastfeeding was defined by the state in which an infant receives only breastmilk, whether direct or indirect by expressed breastmilk with no other liquids. "Predominant" breastfeeding was the term applied when an infant receives almost all of their nutrients from breastmilk but may have taken some other liquids. Discharge refers to the separation of the mother from the health institution including discharge from home visit programmes.

Numerator: This included all women who are exclusively or predominantly breastfed their infant/infants at time of discharge and who had been diagnosed or were subsequently diagnosed with a hypertensive disorder of pregnancy.

FOR ALL INDICATORS

Denominator for all maternal indicators: All women who have been diagnosed or are subsequently diagnosed with a hypertensive disorder of pregnancy. Each woman is counted as a single unit regardless of the number of fetuses conceived or delivered.

All figures can be calculated for an overall HDP rate or can be further defined for each diagnostic category of the ASSHP consensus statement.

Denominator for all neonatal indicators: All babies of 20 or more completed weeks gestation or of 400g or more birthweight whether liveborn or stillborn. For multiple births, each baby was counted as a separate unit when calculating rates of mortality and morbidity.

Context: Application can occur within single institutions, across multiple institutions as part of local governance, across government regions such as states or provinces or across nations.

3.1.5.2 Why choose these clinical indicators?

The aim of these indicators was to include a cross section of maternal and fetal/neonatal measures. The process for indicator establishment was based upon the SMART mnemonic, originally attributed to the management consultant Peter Drucker. The original mnemonic of 1. Specific, 2. Measurable, 3. Achievable, 4. Relevant and 5. Time Bound (Drucker 1954). was adapted for this clinical setting. The process utilized in the design of this set of indicators was SMAART

3.1.5.3 The SMAART concept

Standardised – The indicators would need to be devised with clear definition of terms and use of language applicable to any setting. The ASSHP consensus statement provided the diagnostic framework for HDP categorisation, whilst the wording of the indicators is also specific as to what is being measured and how the measurement was to occur. To assist in this standardisation process all terms used, such as 'term', 'perinatal', and 'direct' *etc*, were defined in accordance with the WHO . Terms which are standard definitions used by AIHW and NSW Health have been used where appropriate to make the indicators relevant to any context.

Measurable – The data upon which the indicators were to be based would need to be information collected on each woman regardless of database system employed or information available without reference to the medical record. Clear definition of individual indicators would need to include ways of measuring that particular outcome. The information collected by using these indicators would also need to be standardised within any given institution and between institutions and practitioners.

The presentation format and forum for the indicators should foster the sharing of information.

Analysable - the data collected would have to able to be analysed using standard and readily available tools in a variety of settings and by a number of practitioners utilising the tools. This prohibited the necessity of extensive statistical analysis of data. Basic incidence and descriptive statistics are all that is required.

Actionable - The concept of "actionability" is the most important in the development and relevance of any clinical indicator. The question needs to be asked continually – are these outcomes for which changes in clinical practice will have a discernible effect? It is futile to make measurement of events/outcomes in clinical practice for which change cannot be affected, if he aim of the measurement is to change practice.

The choice of indicators such as ARF, APO, eclampsia, perinatal mortality, birth weights and breastfeeding was based on the belief that treatment variations, (such as timeliness of instigating treatment, anti-hypertensive use, policies of restrictive fluid administration), as well as demographic variations in populations could be attributed to incidence variation between units/area/nations of measurement.

Reportable - The clinical outcomes measured must be able to be reported in a format and in a forum accessible and relevant to the audience addressed. The method of reporting must also be clearly established within the body of the indicator.

Trackable – Calculation of an outcome must be able to be replicated on a yearly/bi-yearly basis to allow comparison to be made over time periods. This will enable trends in disorder incidence and sequelae of the disorder to be examined over a given time period.

3.1.6 PHASE 6 - CLINICIAN FEEDBACK

Following data collection and analysis, a presentation time was booked with each obstetric unit. The results of each unit as per established clinical indicators were presented, as well as basic demographic information such as age, BMI (kg/m^2), smoking status, delivery type and medication prescription. These presentations were 'open forum' by design and multidisciplinary, including obstetricians, neonatologists, epidemiologists, scientific researchers, midwives, neonatal nurses and students. Thirty minute presentations were scheduled with an additional 15 minutes for questions and feedback. Phase 6 was the final phase for all NSW units involved in the project. The feedback process was cumulative, in that at the first presentation only the one unit's results were reported, whilst at the second presentation that unit's results as well as the previously reported units' results were reported. This process continued until at the sixth presentation, all units' results, were reported. The results of all six units were presented in both oral and poster format at national and international obstetric/neonatology conferences.

3.1.7 PHASE 7 – ESTABLISHING BENCHMARKS

Once clinician feedback had taken place, establishing 'best achievable' outcomes could now be established based upon the best outcomes for each indicator achieved by the six units.

The following phases of experiment 1 were only conducted between units 1 and 6.

3.1.8 PHASE 8 INSTITUTING CHANGE TO REFLECT OPTIMAL CARE.

Units 1 and 6 collaborated on achieving the 'best achievable' outcome for Indicator 2 – APO. The decision to target this indicator had already been established through previous collaborative work between the two units (Thornton et al. 2007). The difference in this outcome between the units, via statistical analysis of the causative factors, led to the targeting of this one particular feature of clinical care which differed between the two units. Statistical analysis of potential causative factors and treatment variations for this one indicator allowed for the collaborative development of a 'best practice' protocol.

3.1.9 PHASE 9 ASSESSING CHANGE AND PROTOCOL ADHERENCE.

Once policy change had been instituted in a unit, the process of individual patient data review had to be undertaken again. This was achieved by reviewing and collecting data on all hypertensive pregnant women at unit 6 for the calendar years 2006 and 2007. The policy change was instituted in 2007, so the review was

conducted for 2006 to confirm the level of morbidity occurring. The 2007 cohort was reviewed to assess policy change efficacy and adherence by clinicians. The steps outlined in Phases 3, 4 and 6 were applied again to this process.

3.2 EXPERIMENT 2 – ASSESSING CLINICIAN OPENNESS TO THE ULTILISATION OF BENCHMARKING TO IMPROVE CLINICAL OUTCOMES.

3.2.1 PHASE 1 DATUM COLLECTION

To determine the factors involved in the acceptance by clinicians of the principles of evidence based medicine, a survey was conducted. A one page questionnaire of nine questions was designed and administered to attendees of all feedback sessions at each unit as well as attendees at the Perinatal Society of Australia and New Zealand Annual Conference in Melbourne, Australia in 2007. The survey was designed upon the principles of simplicity and generalisability (Fink 2003), it was simple and involved ticking responses only. A one page questionnaire design format has been shown to be more readily accepted and completed by participants than longer surveys (Fink 2003).

3.2.2 PHASE 2 DATA ANALYSIS

The survey results were analysed utilizing chi-squared statistics to determine the clinician's familiarity with the benchmarking process and whether they would consider the results of an audit and feedback process, such as this, to be a stimulus for clinical change.

CHAPTER 4

RESULTS

4.1 EXPERIMENT ONE

4.1.1 PHASE 1: FORMATION OF COLLABORATION

Ethics approval was granted at each collaborating institution. The Chief Investigator at each institution was the obstetrician or renal physician, whilst the author was listed as a student co-investigator. List of all women coded under the ICD-10-AM (Australian Units) and ICD-10 (Canadian Unit), as having hypertension during pregnancy, were obtained from the Medical Information Department at each institution. The notes of each woman were recalled from the filing system and reviewed. Data were collected in line with those listed in Phase 2.

4.1.2 PHASE 2: DATABASE DESIGN

The following is a table of all of the fields of data collected by the database. The description is the detail of the datum item whilst the field type describes how the data were collected.

FIELD DESCRIPTIONS			FIELD TYPE
Antenatal Details			
	Demographics and readings	Age	Number
		Estimated date of confinement	Number
		Parity	Number
		Booking gestation (weeks)	Number
		Booking systolic blood pressure	Number
		Booking diastolic blood pressure	Number
		Booking urinalysis proteinuria	Number
		Booking weight	Number
		Booking height	Number
		Smoking status	Yes/No
		Pre-existing morbidity	Yes/No
		Morbidity details	Text
		History of hypertension in previous pregnancy	Yes/No
		Highest antenatal systolic blood pressure	Number
		Highest antenatal diastolic blood pressure	Number
		Gestation at which this occurred	Number
		Pregnancy complications No.1	Nominal
		Pregnancy complications No.2	Nominal
		Pregnancy complications No.3	Nominal
		Adverse pregnancy event	Yes/No
		Details	Nominal
	Blood values	Creatinine	Number
	(worst recorded)	Uric acid	Number
		Platelets	Number
		Alkaline Phosphatase	Number
		Aspartate Transaminase	Number
		Alanine Transaminase	Number
		Gamma-glutamyl Transferase	Number
	Medications	clonidine	Yes/No
		Maximum dose	Number
		Frequency prescribed	Number
		Days medicated	Number
		hydralazine	Yes/No
		maximum dose	Number

FIELD DESCRIPTIONS			FIELD TYPE
		Frequency prescribed	Number
		Days medicated	Number
		oxprenolol	Yes/No
		Maximum dose	Number
		Frequency prescribed	Number
		Days medicated	Number
		labetalol	Yes/No
		Maximum dose	Number
		Frequency prescribed	Number
		Days medicated	Number
		nifedipine	Yes/No
		Maximum dose	Number
		Frequency prescribed	Number
		Days medicated	Number
		methyldopa	Yes/No
		Maximum dose	Number
		Frequency prescribed	Number
		Days medicated	Number
		prazosin	Yes/No
		Maximum dose	Number
		Frequency prescribed	Number
		Days medicated	Number
		Oral diuretics	Yes/No
		Other	Text
		Intravenous diuretics	Yes/No
		Intravenous magnesium sulphate	Yes/No
		Hours prescribed	Number
		Intravenous diazoxide	Yes/No
		Dose	Number
		Intravenous hydralazine bolus	Yes/No
		Dose	Number
		Intravenous hydralazine infusion	Yes/No
		Time	Number
		Intravenous glycerine trinitrate	Yes/No
		Time	Number
	Intravenous fluids	Given	Yes/No
		Type	Nominal
		Additives inserted	Yes/No

FIELD DESCRIPTIONS			FIELD TYPE
		Volume in ml	Number
	Diagnostic parameters	Blood pressure	Yes/No
		Dipstick proteinuria	Yes/No
		Quantified	Yes/No
		Serum pl/cr ≥30 mg/mmol	Yes/No
		Oliguria	Yes/No
		Eclampsia	Yes/No
		Hyperreflexia with clonus	Yes/No
		Severe headaches with hyperreflexia	Yes/No
		Persistent visual disturbance	Yes/No
		Raised serum transaminases	Yes/No
		Severe epigastric/right upper quadrant pain	Yes/No
		Thrombocytopenia	Yes/No
		Disseminated intravascular coagulation	Yes/No
		Haemolysis	Yes/No
		Intra-uterine growth restriction <3rd centile	Yes/No
Delivery details		Gestation at delivery	Number
		Was delivery induced	Yes/No
		Type of delivery	Nominal
		Adverse event at delivery	Yes/No
		Details	Nominal
		Oral diuretics	Yes/No
		clonidine	Yes/No
		Dose	Number
		hydralazine	Yes/No
		Dose	Number
		Other	Yes/No
		Intravenous diuretics	Yes/No
		Intravenous magnesium sulphate	Yes/No
		Hours	Number
		Intravenous diazoxide	Yes/No
		Dose	Number
		Intravenous hydralazine bolus	Yes/No
		Dose	Number
		Intravenous hydralazine infusion	Yes/No
		Time	Number
		Intravenous glycerine trinitrate	Yes/No

FIELD DESCRIPTIONS			FIELD TYPE
		Time	Number
	Intravenous fluids	Given	Yes/No
		Type	Nominal
		Additives inserted	Yes/No
		Volume in ml	Number
		Steroids	Yes/No
Infant details		Status at birth	Nominal
	Baby 1	Status at discharge	Nominal
		Gender	Nominal
		Weight	Number
		Weight centile	Number
		Apgar1	Number
		Apgar2	Number
		Length	Number
		Length centile	Number
		Head circumference	Number
		Head circumference centile	Number
		Admitted to special care nursery	Yes/No
		Length of stay	Number
		Was the infant ventilated	Yes/No
		Type of feeding	Nominal
	Baby 2	Status at birth	Nominal
		Status at discharge	Nominal
		Gender	Nominal
		Weight	Number
		Weight centile	Number
		Apgar1	Number
		Apgar2	Number
		Length	Number
		Length centile	Number
		Head circumference	Number
		Head circumference centile	Number
		Admitted to special care nursery	Yes/No
		Length of stay	Number
		Was the infant ventilated	Yes/No
		Type of feeding	Nominal
	Baby 3	Status at birth	Nominal
		Status at discharge	Nominal

FIELD DESCRIPTIONS		FIELD TYPE
	Gender	Nominal
	Weight	Number
	Weight centile	Number
	Apgar1	Number
	Apgar2	Number
	Length	Number
	Length centile	Number
	Head circumference	Number
	Head circumference centile	Number
	Admitted to special care nursery	Yes/No
	Length of stay	Number
	Was the infant ventilated	Yes/No
	Type of feeding	Nominal
Postnatal details	Highest systolic blood pressure	Number
	Highest diastolic blood pressure	Number
	Day postnatal at which this occurred	Number
	clonidine	Yes/No
	Dose	Number
	Frequency prescribed	Number
	hydralazine	Yes/No
	Dose	Number
	Frequency prescribed	Number
	oxprenolol	Yes/No
	Dose	Number
	Frequency prescribed	Number
	labetalol	Yes/No
	Dose	Number
	Frequency prescribed	Number
	nifedipine	Yes/No
	Dose	Number
	Frequency prescribed	Number
	methyldopa	Yes/No
	Dose	Number
	Frequency prescribed	Number
	enalapril	Yes/No
	Dose	Number
	Frequency prescribed	Number
	minipress	Yes/No

FIELD DESCRIPTIONS			FIELD TYPE
		Diuretics	Yes/No
		Intravenous diuretics	Yes/No
		Intravenous magnesium sulphate	Yes/No
		Hours	Number
		intravenous diazoxide	Yes/No
		Dose	Number
		Intravenous hydralazine bolus	Yes/No
		Dose	Number
		Intravenous hydralazine infusion	Yes/No
		Time	Number
		Intravenous glycerine trinitrate	Yes/No
		Time	Number
	Intravenous fluids	Given	Yes/No
		Type	Nominal
		Additives inserted	Yes/No
		Volume in mls	Number
	Medications at discharge	Clonidine	Yes/No
		Dose	Number
		Frequency prescribed	Number
		Hydralazine	Yes/No
		Dose	Number
		Frequency prescribed	Number
		Labetalol	Yes/No
		Dose	Number
		Frequency prescribed	Number
		Nifedipine	Yes/No
		Dose	Number
		Frequency prescribed	Number
		Methyldopa	Yes/No
		Dose	Number
		Frequency prescribed	Number
		Oxprenolol	Yes/No
		Dose	Number
		Frequency prescribed	Number
		Enalapril	Yes/No
		Dose	Number
		Frequency prescribed	Number
		Other	Yes/No

FIELD DESCRIPTIONS			FIELD TYPE
		Details	Text
		Diuretics	Yes/No
		Adverse event postnatally	Nominal
	Blood values	Creatinine	Number
	(worst recorded)	Uric acid	Number
		Platelets	Number
		Alkaline Phosphatase	Number
		Aspartate Transaminase	Number
		Alanine Transaminase	Number
		Gamma-glutamyl Transferase	Number
		Notes	Text
		Length of stay	Number
Final coding		ICD-10-AM	Number
		ASSHP diagnoses	Number

Table 4.1. Datafields and descriptions. Datafields and descriptions of components of the HDP Database

4.1.3 PHASE 3 DATUM COLLECTION

4.1.3.1 Demographic differences between units.

The average family income for NSW is $1 176/week. The average age of birth for women is 32 years, 15% of women in NSW smoke during their pregnancy and the average rate of GDM affected pregnancies is 5%. (ABS Census 2006 and NSW Department of Health 2005). A comparison of the five Sydney units showed the following comparisons:

Unit 1 is situated in an inner city locality. The average family income for this area is $2 181/week. Women with HDP who deliver at this unit are of a mean age of 32 years, 10% smoked during their pregnancy, they had their first antenatal visit at 15 weeks, with a mean booking visit BMI (kg/m^2) of 26. These women had a mean booking blood pressure of 118/72 mm Hg and a GDM rate of 10%. Eighty-two percent of this population was Caucasian.

Unit 2 is located in a suburban area of Sydney with an average family income of $1 057/week. Women with HDP who deliver at this unit are of a mean age of 29 years, 12% smoked during their pregnancy, had their first antenatal visit at 15 weeks, with a mean booking BMI (kg/m^2) of 31. These women had a mean booking blood pressure of 122/72 mm Hg and a GDM rate of 19%. Seventy-six percent of this population were Caucasian.

Unit 3 is located in an outer suburban area of Sydney with an average family income of $1 285/week. Women with HDP who deliver at this unit are of a mean age of 29

years, 19% smoked during their pregnancy, had their first antenatal visit at 19 weeks, with a mean booking visit BMI (kg/m^2) of 30. These women had a mean booking blood pressure of 120/73 m Hg and a GDM rate of 11%. Eighty-nine percent of this population were Caucasian.

Unit 4 is located in an inner city locality. The average family income for this area is $2 219/week. Women with HDP who deliver at this unit are of a mean age of 31 years, 7% smoked during their pregnancy, they had their first antenatal visit at 15 weeks, with a mean booking visit BMI (kg/m^2) of 25. These women had a mean booking blood pressure of 118/71 mm Hg and a GDM rate of 8%. Eighty-nine percent of this population were Caucasian.

Unit 5 is located in an outer suburban area of Sydney. The average family income for this area is $1 156/week. Women with HDP who deliver at this unit are of a mean age of 28 years, 20% smoked during their pregnancy, they had their first antenatal visit at 17 weeks, with a mean booking visit BMI (kg/m^2) of 31. These women had a mean booking blood pressure of 116/71 mm Hg and a GDM rate of 14%. Eighty-five percent of this population were Caucasian.

The Canadian unit is located in Vancouver, eastern seaboard. The average family income for this area is $1 025/week (AUD). Women with HDP who deliver at this unit are of a mean age of 33 years, 15% smoked during their pregnancy, they had their first antenatal visit at 14 weeks, with a mean booking visit BMI (kg/m^2) of 26. These women had a mean booking blood pressure of 118/74 mm Hg and a GDM rate of 10%. Eighty-five percent of this population were Caucasian.

In this cohort, maternal age is positively correlated to booking systolic and diastolic blood pressure values (Pearson correlation p=0.01). The relative risk (RR) of developing GDM for women with a BMI (kg/m^2) of >30 when compared to women with a normal range BMI is 2.3 (95% CI 1.65-3.14).

In this cohort, smoking rates were highest in those units with a lower than state average or average family income (units 2 (LH), 3 (NH) and 5 (CTH)) .

4.1.3.2 Reliability of data collected

The ICD-10-AM lists of all women coded as having had a HDP during 2005 were examined at all institutions. Two individuals examined the medical records at each Australian institution and the inter-rater reliability of record examination was examined on a randomly chosen 10% of all cases (Kappa 0.96). At the Canadian institution a random sample of 10% of all cases was chosen during visits 1 and 2 and the intra-rater reliability was examined (Kappa 0.94). At the Canadian Institution during visit 3 a second individual was available for calculating an inter-rater reliability level on a randomly selected 10% of all cases examined (Kappa 0.96).

The intra-rater reliability was tested within a randomly selected sample of 10% of notes from the main note reviewer (author) at each site visited (Kappa 0.96) The Kappa statistic (Cohen 1960) was chosen as the test of reliability and reproducibility of data collected where each variable was categorised as a categorical variable and the correct response was decided upon by mutual agreement following re-evaluation of the medical record.

4.1.3.3 Data collected

Not all women met ASSHP criteria for inclusion following note review as indicated in Table 4.2. Exclusion reasons were based upon the absence of diagnostic criteria as recorded by attending medical personnel and through examination of laboratory results.

Unit number	**No. of notes reviewed**	**No. of women entered into database**	**No. of neonates entered into database***
1	536	472	498
2	204	174	179
3	398	375	388
4	204	170	181
5	162	128	132
6	449	409	441
Total	**1953**	**1728**	**1819**

*Including singletons, twins and triplets

Table 4.2. Notes reviewed and participants included**.** A representation of the total notes reviewed and the women and babies included in the dataset at each unit.

4.1.4 PHASE 4 STATISTICAL ANALYSIS

4.1.4.1 Unit descriptions

The basic unit descriptions are contained in Table 4.3

Unit name	**Unit number**	**Distance from Sydney GPO**	**Total deliveries 2005**	**Type of unit**
Royal Prince Alfred Women and Babies, Camperdown Australia (RPAH)	1	3 km	4 725	Tertiary referral
Liverpool Hospital, Liverpool Australia (LH)	2	41 km	2 902	Tertiary referral
Nepean Hospital, Penrith Australia (NH)	3	55 km	3 409	Tertiary referral
Royal North Shore Hospital, St. Leonards Australia (RNSH)	4	10 km	2 498	Tertiary referral
Campbelltown Hospital, Campbelltown Australia (CTH)	5	61 km	1 969	Non-tertiary
British Columbia Women's Hospital and Health Centre, Vancouver Canada (BCW)	6	21, 734 km	7 355	Tertiary referral

Table 4.3. Descriptions of individual units**.** Descriptive and geographic descriptions of all included units.

4.1.4.2.Rates of hypertensive women

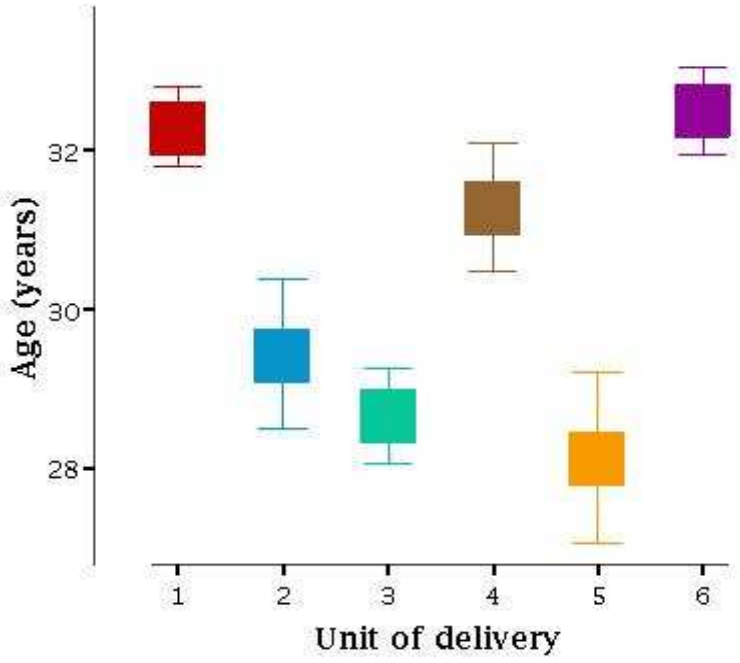
Unit number	Total HDP*	Preeclampsia †	Gestational hypertension†	Chronic hypertension †	Preeclampsia superimposed on chronic hypertension†
1	472 (10.0%)	190 (40.3%)	208 (44.1%)	45 (9.5%)	29 (6.1%)
2	174 (6.0%)	60 (34.5%)	77 (44.3%)	26 (14.9%)	11 (6.3%)
3	375 (11.0%)	135 (35.8%)	188 (50.3%)	32 (8.6%)	20 (5.3%)
4	170 (6.8%)	69 (40.8%)	72 (42.0%)	23 (13.6%)	6 (3.6%)
5	128 (6.5%)	50 (39.1%)	57 (44.5%)	12 (9.4%)	9 (7.0%)
6	409 (5.6%)	179 (43.8%)	153 (37.4%)	49 (12.0%)	28 (6.8%)
All units	*1728 (7.6%)	†683 (39.5%) *683 (3.0%)	†755 (43.7%) *755 (3.3%)	†187 (10.8%) *187 (0.8%)	†103 (6.0%) *103 (0.5%)

*Expressed as a percentage of all deliveries
†Expressed as a percentage of all HDP women

Table 4.4. Total women per unit and hypertension type. The total number of hypertensive women and the type of hypertension at each unit.

4.1.4.3 The demographic profile and baseline characteristics of the participants

Age, BMI (kg/m^2), smoking status, parity, race, gestation at booking visit and baseline blood pressure readings are displayed in Figures 4.1, 4.2, 4.3, 4.4, 4.5, 4.6, 4.7 and 4.8.

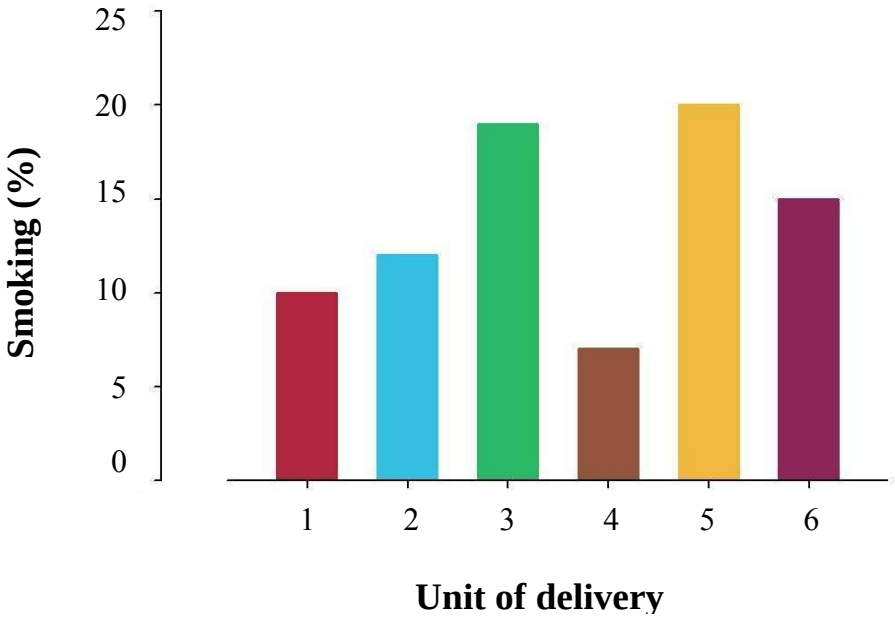


1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA between Units 1-6 p<0.001

Figure 4.1. Age of women at booking in years. The age of women at booking in total years at each unit.

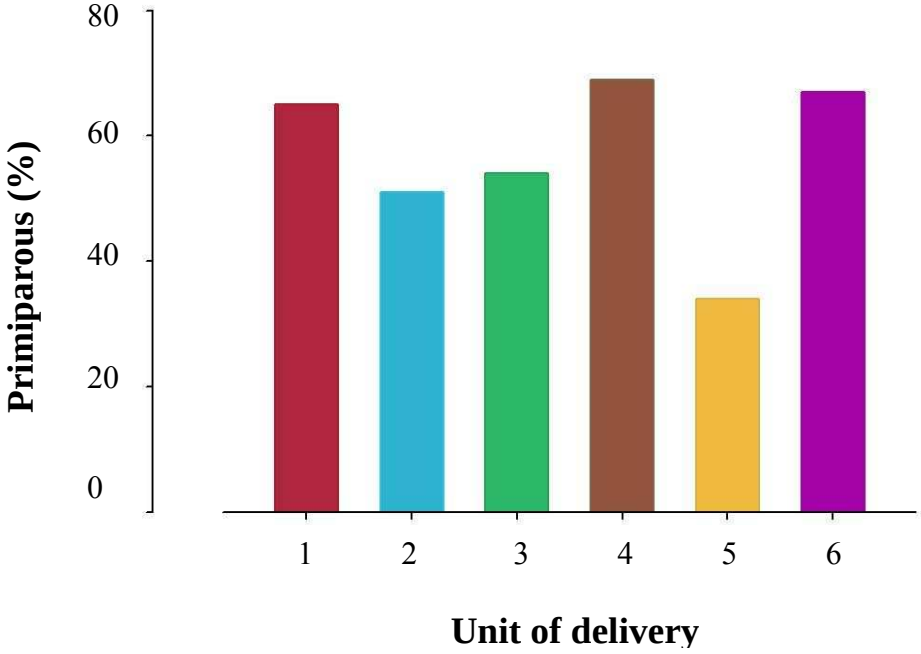
1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA between Units 1-6 $p<0.001$

Figure 4.2. Body Mass Index (BMI kg/m^2). The BMI of women at initial care provider visit at each unit.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis between Units 1-6 $p<0.0001$

Figure 4.3. Smoking status. The current smoking status of women as recorded at initial care provider visit at each unit.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BC
Chi-Squared analysis between Units 1-6 $p<0.0001$

Figure 4.4. Percentage of primiparous women. The percentage of women who were primiparous at each unit.

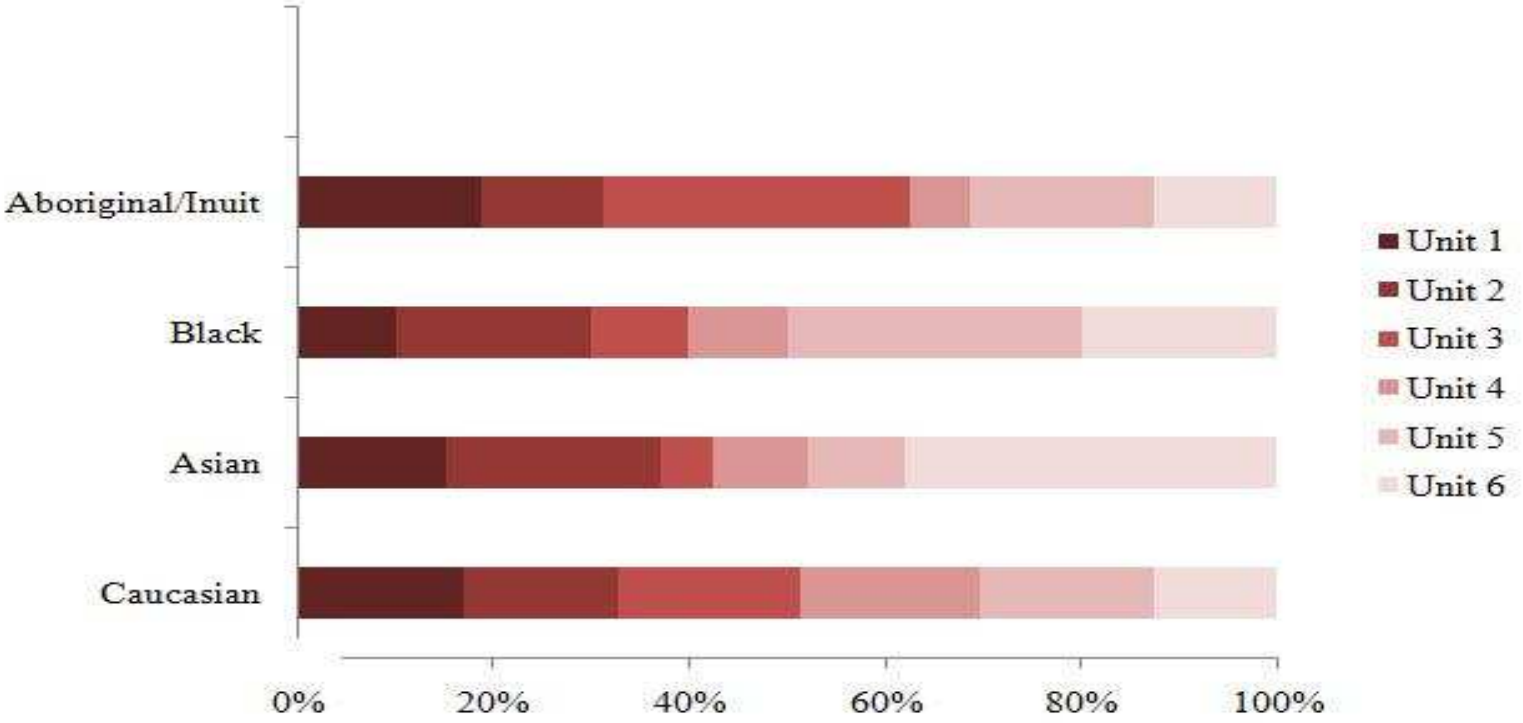
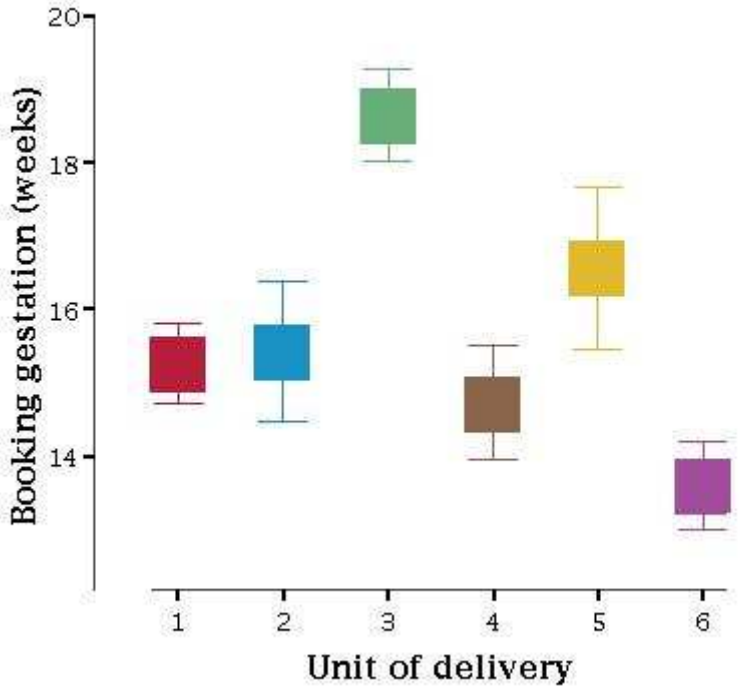
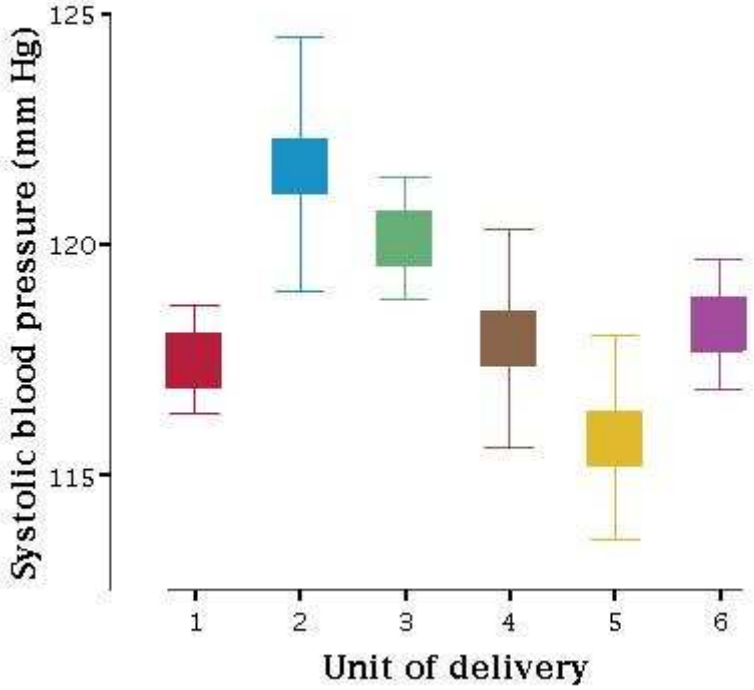


Figure 4.5. Race of women. The racial profile of women at each unit.



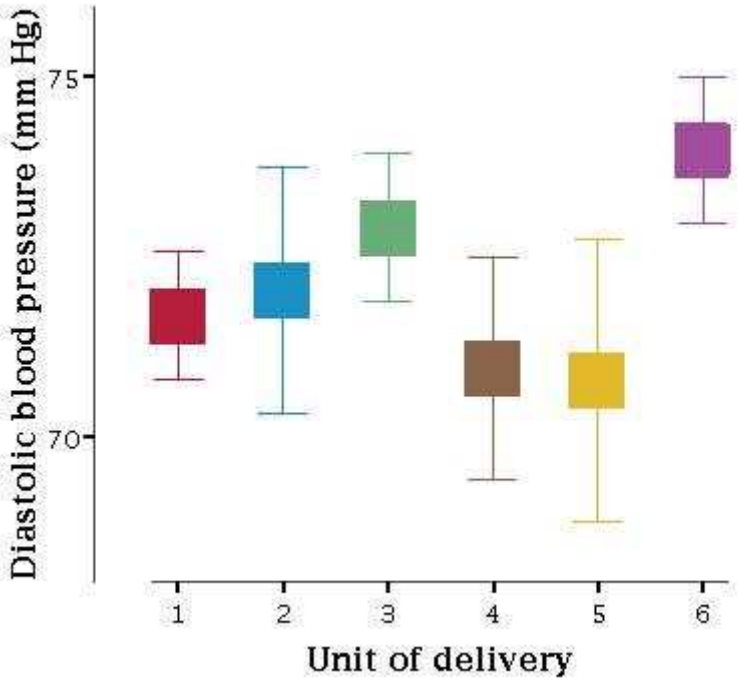
1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95%CI displayed, ANOVA between Units 1-6 p<0.0001

Figure 4.6. Gestation at initial care provider visit. The gestation of women at initial care provider visit at each unit.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA between Units 1-6 $p<0.001$

Figure 4.7. First systolic blood pressure. The systolic blood pressure readings of women at initial care provider visit at each unit.

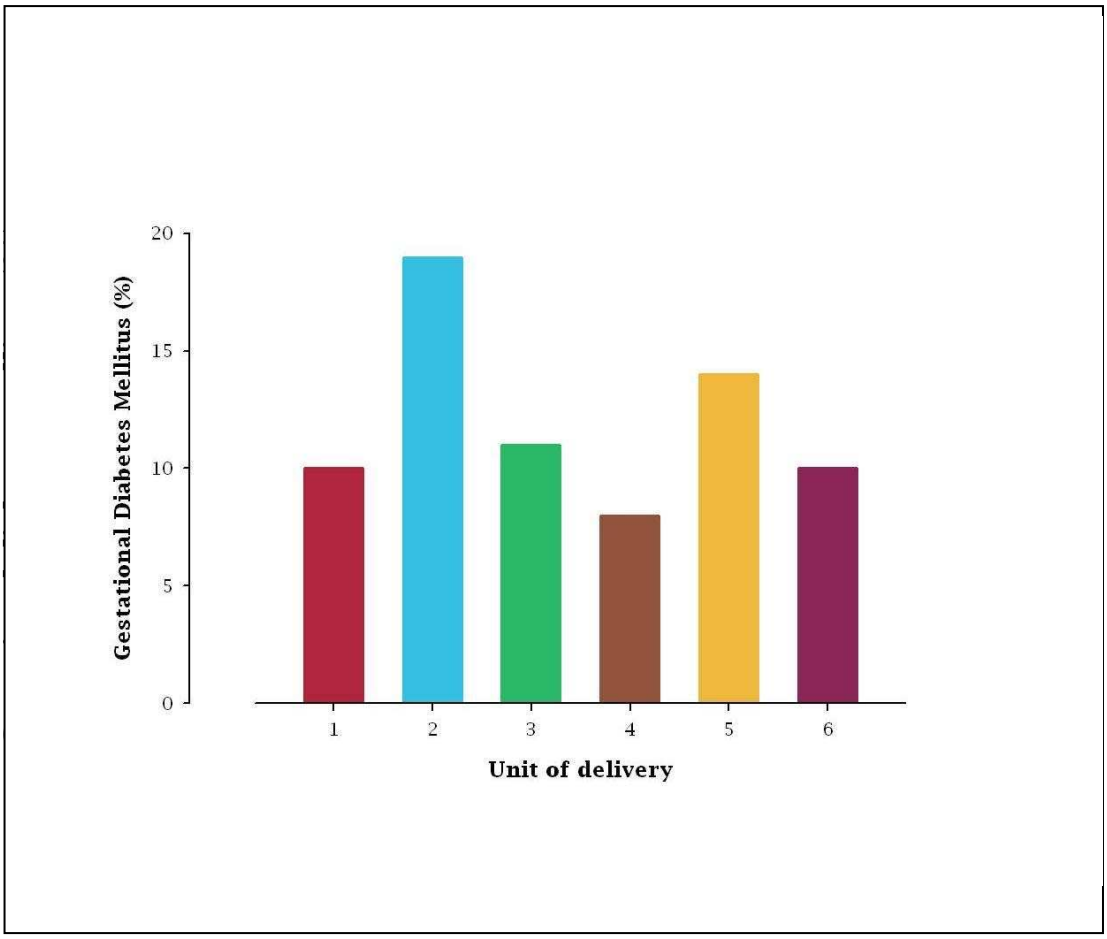


1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA between Units 1-6 p<0.002

Figure 4.8. First diastolic blood pressure**.** The diastolic blood pressure readings of women at initial care provider visit at each unit.

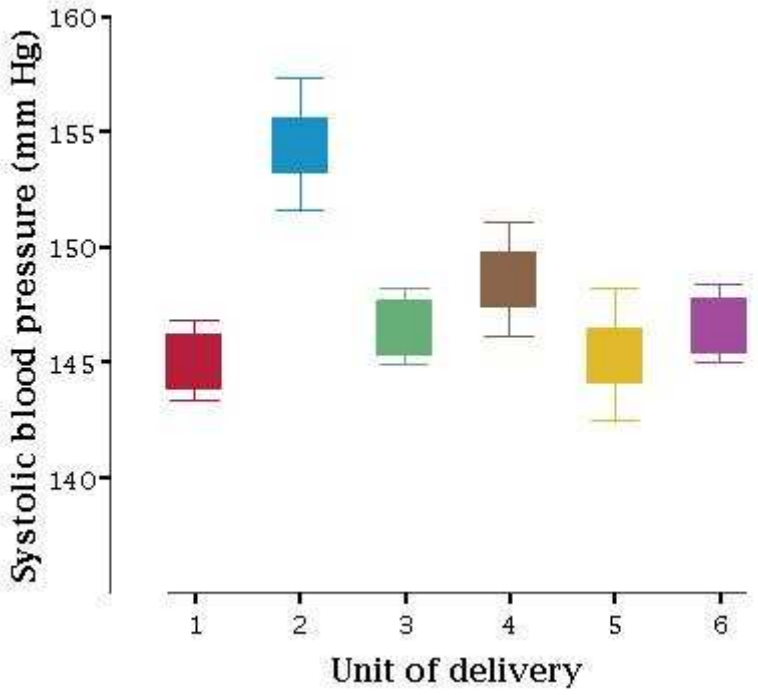
4.1.4.4 Pregnancy progression events

The rates of gestational diabetes mellitus and the highest recorded antenatal blood pressure are displayed in Figures 4.9,4.10 and 4.11.



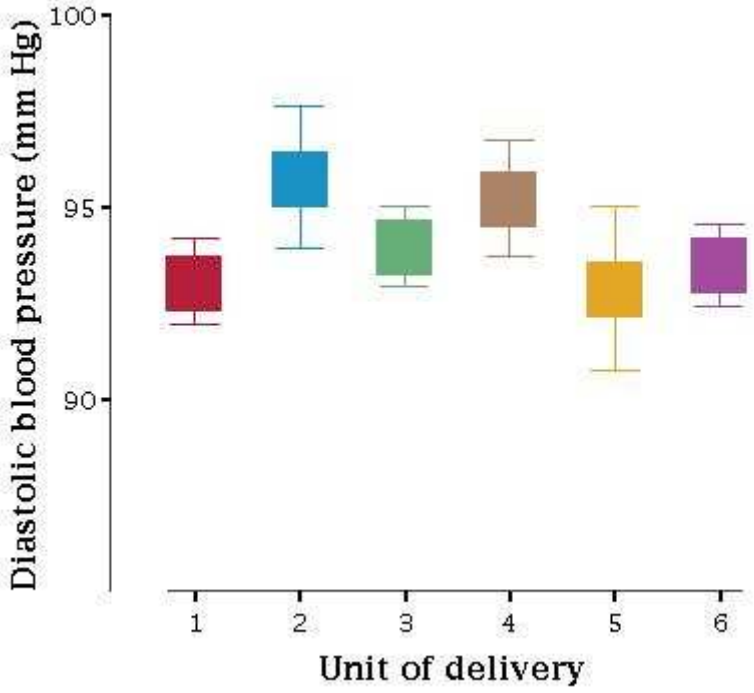
1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis p=0.01

Figure 4.9. Rates of diagnosis of gestational diabetes mellitus**.** The rate of diagnosis of gestational diabetes mellitus of women at each unit.



1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA p<0.001

Figure 4.10. Highest antenatal systolic blood pressure**.** The highest systolic blood pressure reading recorded in the antenatal period of women at each unit.



1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA p=0.046

Figure 4.11. Highest antenatal diastolic blood pressure**.** The highest diastolic blood pressure reading recorded in the antenatal period of women at all units.

4.1.4.5 Correlations

Correlations between demographic factors and blood pressures values.

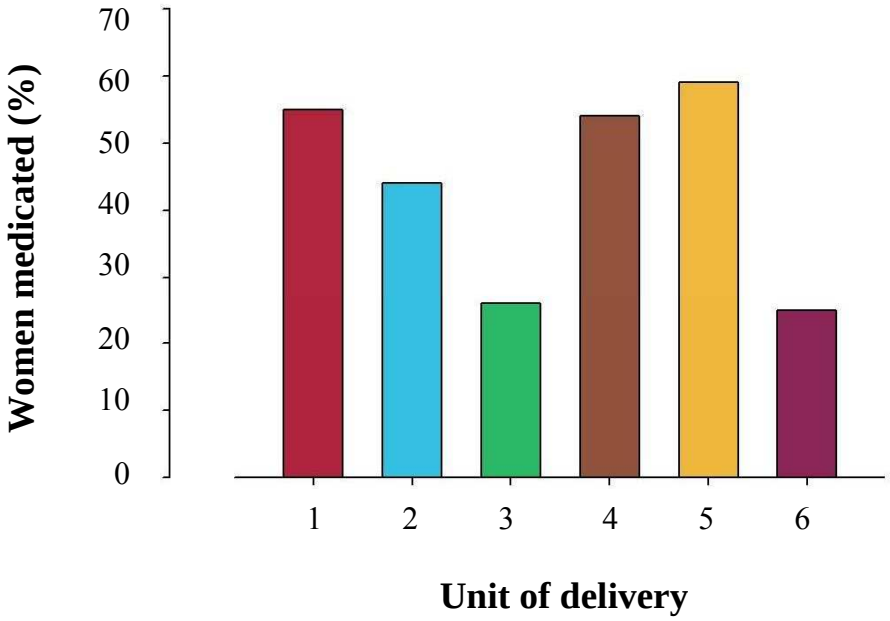
Age in years at booking visit is positively correlated (Pearson) at the 0.01 significance level with both booking systolic and diastolic blood pressure values.

Association between BMI (kg/m^2) and development of GDM.

For a BMI (kg/m^2)<25 (normal weight range), the RR of developing GDM in this cohort is 0.2 (0.16-0.28) in comparison to the rest of the cohort. For a BMI (kg/m^2) 25-29.9 (overweight range), the RR of developing GDM is 0.7 (0.53-1.04), not significant, whilst for a BMI (kg/m^2) >30 (obese), the RR of developing GDM is 1.8 (1.35-2.38).

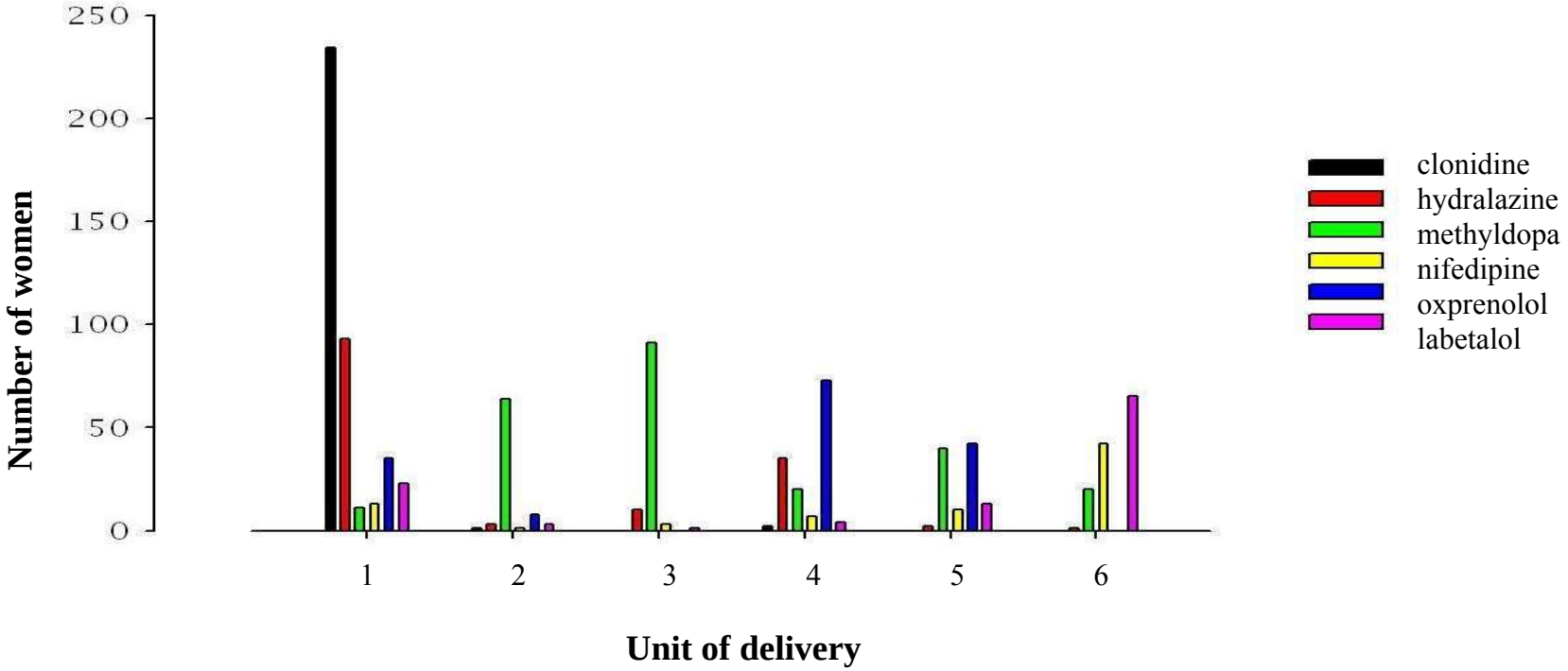
4.1.4.6 Anti-hypertensive medications in the antenatal period

The rates of anti-hypertensive medications prescribed, type of anti-hypertensive medication prescribed (by tablet), time on anti-hypertensive medication antenatally, number of different anti-hypertensive medications used and gestation at which prescription occurred are contained in Figures 4.12, 4.13, 4.14, 4.15 and 4.16 respectively.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Figure 4.12. Anti-hypertensive medication use in the antenatal period**.** The percentage of women who were prescribed anti-hypertensive medication in the antenatal period at each unit.

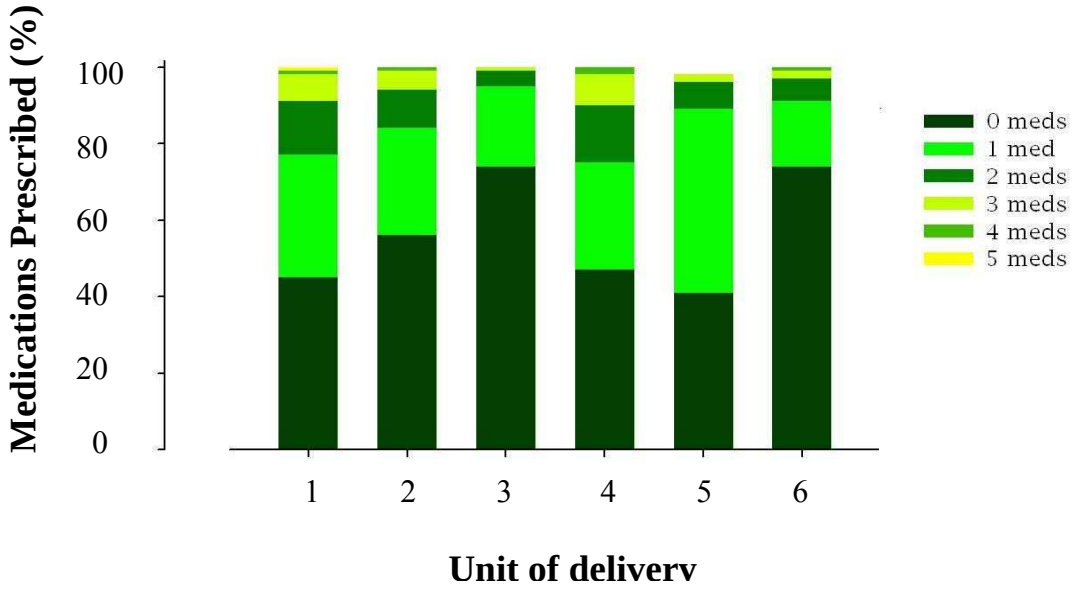


1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW

Figure 4.13. Type of anti-hypertensive medication prescribed. The type and number of anti-hypertensive medication prescribed to women in the antenatal period at each unit.

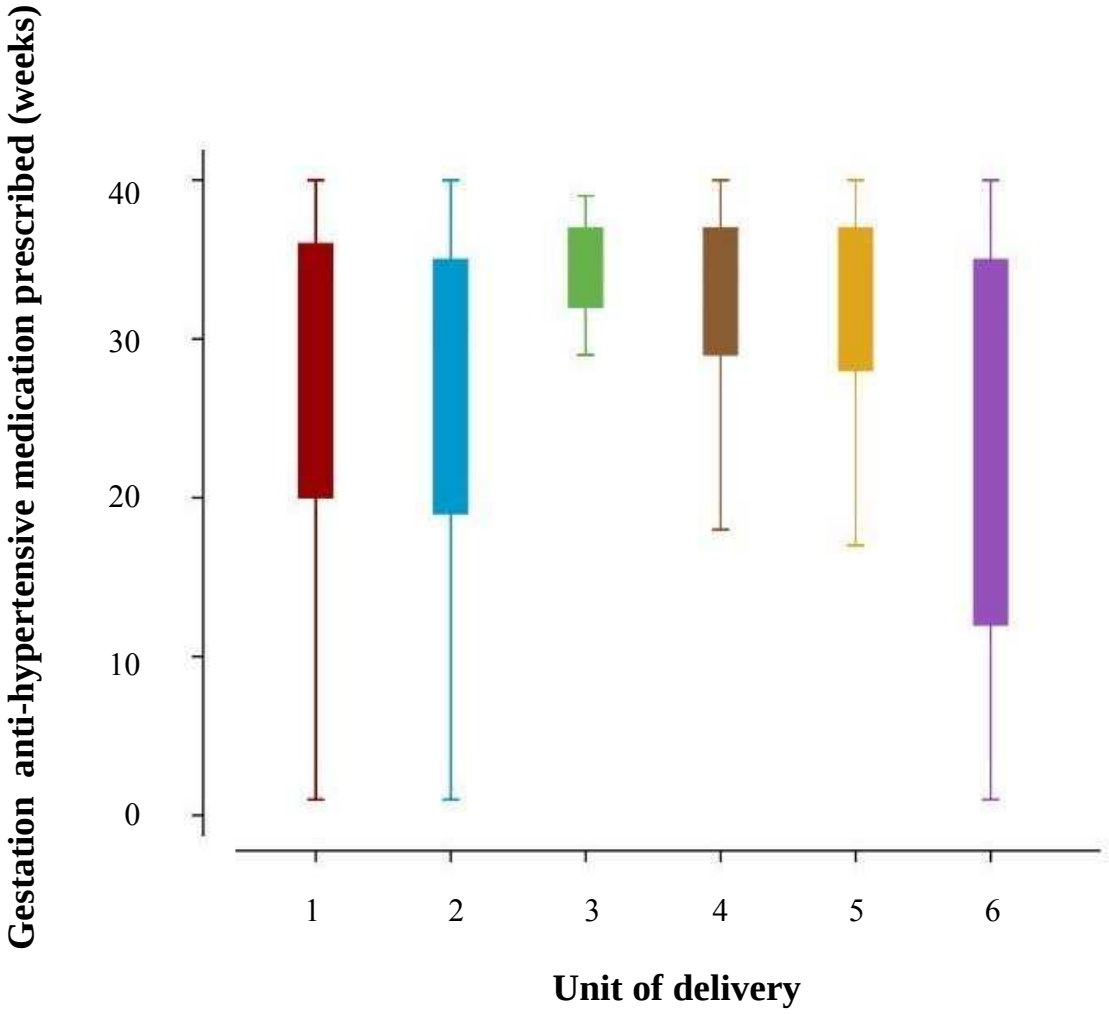
1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Kruskal-Wallis p<0.001 calculated on median length on medications in days

Figure 4.14. Time on anti-hypertensive medication antenatally. The length of time prescribed anti-hypertensive medication in the antenatal period for women at each unit.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Kruskal-Wallis p<0.001 calculated on median number of medications

Figure 4.15. Number of anti-hypertensive medications prescribed. The number of different anti-hypertensive medications prescribed for women in the antenatal period at each unit.

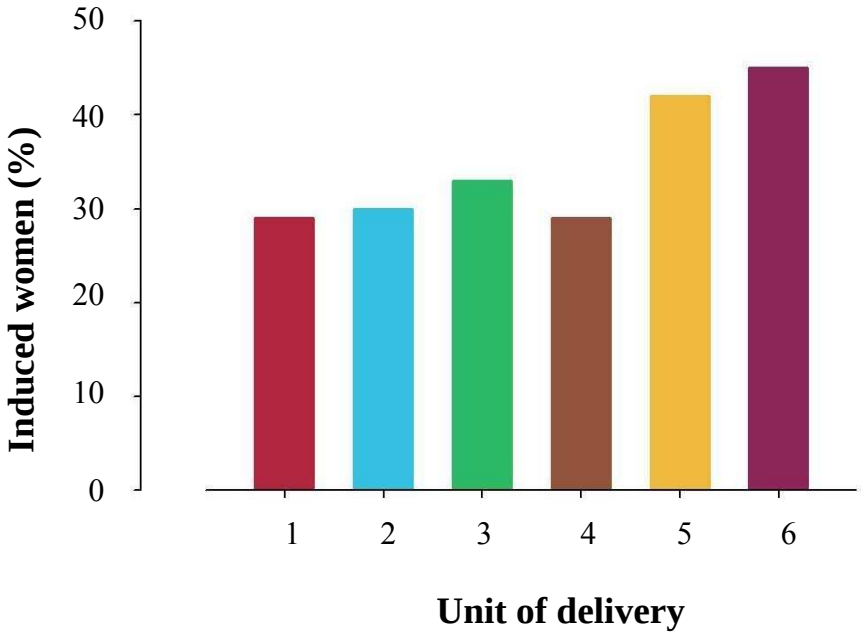


1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA p<0.001

Figure 4.16. Gestation anti-hypertensive medications prescribed. The gestation at which anti-hypertensive medication was first prescribed for women at each unit.

4.1.4.7 Delivery types and onset of delivery

An analysis of delivery types was undertaken, including the use of topical prostaglandins to induce labour and gestation at delivery. Results are displayed in Figures 4.17 and 4.18, and Tables 4.5, 4.6 and 4.7.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Figure 4.17. Induction of labour. The % of women who underwent the induction of labour process at each unit.

PE	GH	CH	SP	SS
71%	67%	78%	38%	<0.001

PE-preeclampsia, GH-gestational hypertension, CH-chronic hypertension, SP-preeclampsia superimposed on chronic hypertension, SS-statistical significance according to Chi-Squared test

Table 4.5. Induction of labour success and hypertensive diagnosis. The success of the induction of labour process categorised by type of hypertension diagnosed for women at each unit.

	ALL HDP	PE	GH	CH	SP
Total induced	595 (34%)*	257 (38%)*	250 (27%)*	51 (30%)*	37 (37%)*
Vaginal delivery	323 (54%)†	135 (52%)†	140 (56%)†	24 (47%)†	24 (65%)†
Instrumental vaginal delivery	78 (13%)†	31 (12%)†	33 (13%)†	10 (19%)†	4 (11%)†
Caesarean section	194 (33%)†	91(36%)†	77 (31%)†	17 (34%)†	9 (24%)†

PE-preeclampsia, GH-gestational hypertension, CH-chronic hypertension, SP-preeclampsia superimposed on chronic hypertension
*expressed as a % of all women in the cohort with that diagnosis
†expressed as a % of all women with that diagnosis who were induced

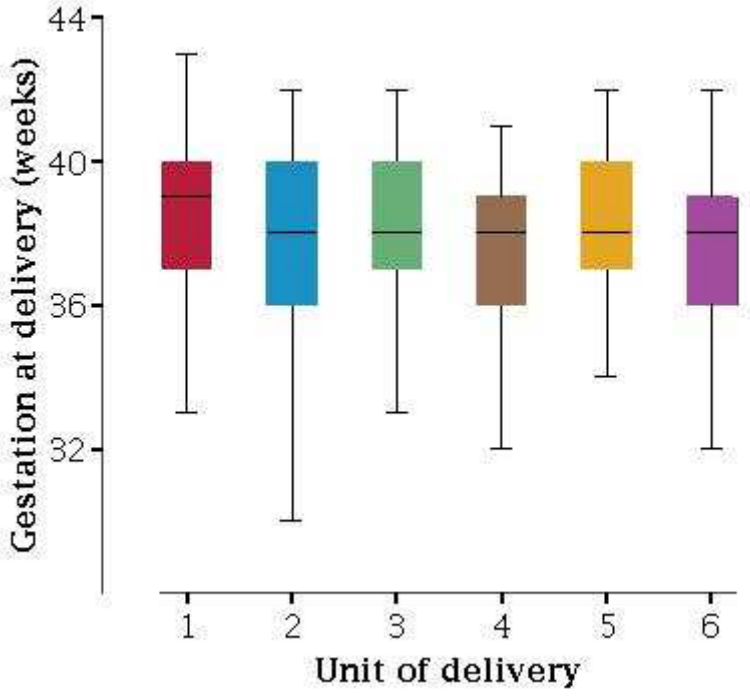
Table 4.6. Delivery types and hypertensive diagnosis. The delivery types of women induced categorised by the type of hypertension diagnosed at each unit.

The relative risk of an induction of labour resulting in a caesarean section for hypertensive women during the study period was 1.35 (95% CI. 1.04-1.76) in comparison to normotensive women.

	Unit 1	Unit 2	Unit 3	Unit 4	Unit 5	Unit 6
Vaginal delivery	42%	52%	46%	40%	64%	43%
Assisted vaginal delivery – ventouse & forceps	12%	7%	8%	10%	5%	12%
Caesarean section	46%	41%	46%	50%	31%	45%

1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared between units and delivery type $p<0.001$

Table 4.7. Delivery types. The delivery types of women at each unit.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Median and inter-quartile range displayed, Kruskal-Wallis p<0.001

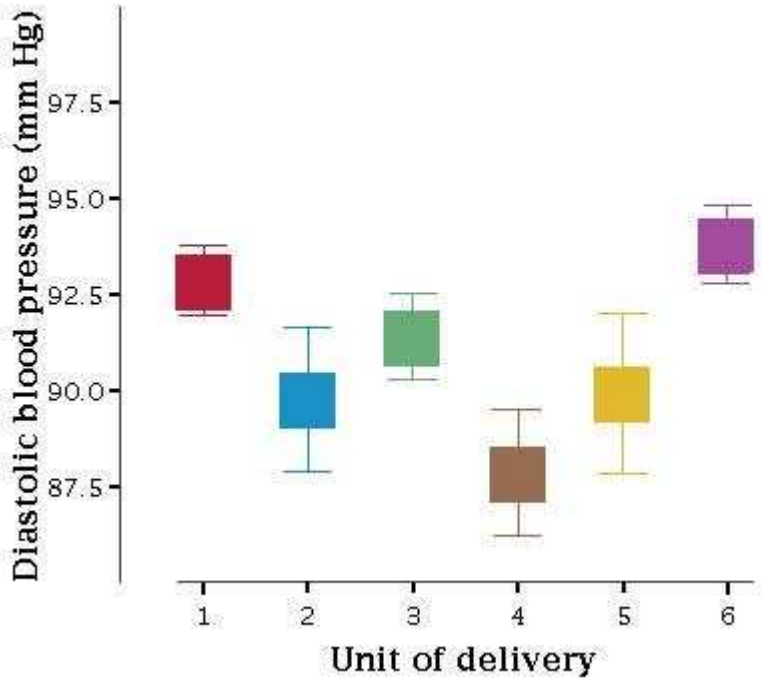
Figure 4.18. Gestation at delivery. The gestation at delivery for women at each unit.

4.1.4.8 Postnatal blood pressures

The postnatal systolic and diastolic blood pressures are displayed in Figures 4.19 and 4.20.

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA p<0.001

Figure 4.19. Highest postnatal systolic blood pressure. The highest recorded systolic blood pressure recorded in the postnatal period for women at each unit.

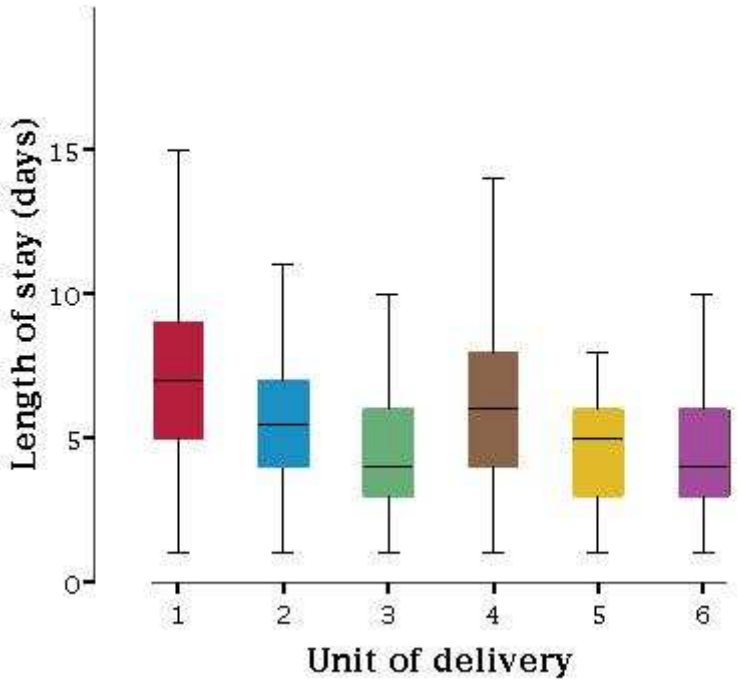


1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Mean and 95% CI displayed, ANOVA p<0.001

Figure 4.20. Highest postnatal diastolic blood pressure. The highest recorded systolic blood pressure recorded in the postnatal period for women at each unit.

4.1.4.9 Length of stay

An analysis of the total length of stay for women with a HDP was undertaken and is displayed in Figure 4.21.



1. RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Median and inter-quartile range displayed, Kruskal-Wallis p<0.001

Figure 4.21. Length of stay. The length of stay overall for all women.

4.1.4.10 Diagnostic features for women with preeclampsia at all units combined

Utilising the ASSHP diagnostic criteria, the diagnostic features of women are presented in Table 4.8.

Diagnostic feature	PE % of women	SP % of women
Hypertension Systolic blood pressure ≥140 and/or diastolic blood pressure ≥90 mmHg on 2 separate occasions 4 hours apart, after 20 weeks gestation	100%	100%
Proteinuria (≥300 mg/24hr)	77%	82%
Renal insufficiency (serum/plasma creatinine 0.09mmol/l)	10%	19%
Liver disease (abnormal LFTs or pain)	39%	18%
Neurological problems (eclampsia, hyperreflexia with clonus, severe headache with hyperreflexia, persistent visual disturbance)	33%	25%
Haematological disturbance (thrombocytopaenia, DIC, haemolysis)	28%	10%
Fetal growth restriction	10%	8%

PE-preeclampsia, SP-preeclampsia superimposed on chronic hypertension

Table 4.8. Diagnostic features for preeclampsia and preeclampsia superimposed on chronic hypertension. The diagnostic features of women diagnosed with either preeclampsia or preeclampsia superimposed on chronic hypertension at all units combined.

4.1.4.1 Adverse maternal events

Adverse events, other than those included in the indicator analyses included placental abruption, cerebrovascular event and postpartum haemorrhage (>500 ml). This data were subsequently included because of the evolving diagnostic criteria (SOMANZ 2008) which has now included placental abruption.

Unit number	**Placental abruption**	**Cerebrovascular event**	**Postpartum haemorrhage**
1	4 (1%)	1 (<1%)	33 (7%)
2	3 (2%)	0	10 (6%)
3	5 (1%)	0	31 (8%)
4	3 (2%)	0	15 (9%)
5	2 (2%)	0	6 (5%)
6	13 (3%)	1 (<1%)	69 (17%)
SS	0.904	0.547	**<0.001**

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
SS Statistical significance

Table 4.9. Adverse maternal events. The rate of adverse events which occurred over all natal periods for women at each unit.

4.1.5 PHASE 5 ESTABLISHING TOOLS OF OUTCOME ANALYSIS

4.1.5.1. Maternal mortality

There was no maternal mortality in any of the units during the year of surveillance

4.1.5.2. Acute pulmonary oedema

Unit Number	Rate of indicator
1	0/1 000
2	6/1 000
3	5/1 000
4	0/1 000
5	0/1 000
6	46/1 000

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Table 4.10. Acute pulmonary oedema. The rate of acute pulmonary oedema for women at each unit.

4.1.5.3. Acute renal failure

Unit Number	Rate of indicator
1	13/1 000
2	46/1 000
3	5/1 000
4	18/1 000
5	8/1 000
6	2/1 000

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Table 4.11. Acute renal failure. The rate of acute renal failure for women at each unit.

4.1.5.4. Eclampsia

Unit Number	Rate of indicator
1	2/1 000
2	6/1 000
3	5/1 000
4	12/1 000
5	47/1 000
6	2/1 000

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Table 4.12. Eclampsia. The rate of eclampsia for women at each unit.

At all units there were 12 cases of eclampsia. Of these 58% (7) occurred antenatally, 9% (1) intrapartum and 33% (4) postnatally. An examination of all potentially predictive factors was undertaken. Mean booking systolic BP was found to be lower

in those women who had an eclamptic seizure when compared to all other women (110 mmHg, SD 8.3 and 119 mmHg, SD 13.8 respectively). This was also consistent with mean booking diastolic BP (66 mmHg, SD 7.8 and 72 mmHg, SD 10.2). The highest recorded antenatal systolic BP was higher in those women who had an eclamptic seizure when compared to all other women (163 mmHg, SD 34.9 and 147 mmHg (SD 17.5 respectively).

Magnesium sulphate usage varied significantly between the units unit 1 RPAH 18.0%, unit 2 LH 8.6%, unit 3 NH 2.4%, unit 4 RNSH 3.5%, unit 5 CTH 9.4% 000 and unit 6 BCW 18.6% of all HDP women

4.1.5.5 Perinatal mortality rate

Unit Number	Rate of indicator
1	16/1 000
2	17/1 000
3	5/1 000
4	11/1 000
5	15/1 000
6	18/1 000

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Table 4.13. Perinatal mortality. The perinatal mortality rate for neonates at each unit.

4.1.5.6. Admission of term neonates to Neonatal Intensive Care Units

Unit Number	Rate of indicator
1	9%
2	12%
3	13%
4	3%
5	19%
6	7%

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Table 4.14. Neonatal Intensive Care Unit admission. The rate of admission of term neonates to a Neonatal Intensive Care Unit at each unit.

4.1.5.7a. Birth weight below the tenth centile

Unit Number	Rate of indicator
1	9%
2	20%
3	11%
4	18%
5	11%
6	17%

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Table 4.15. Birth weight below the tenth centile. The percentage of neonates with a birth weight below the tenth centile at each unit.

4.1.5.7b. Birth weight below the 3rd centile

Unit Number	Rate of indicator
1	3%
2	5%
3	4%
4	6%
5	3%
6	7%

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis $p<0.001$

Table 4.16. Birth weight below the third centile**.** The percentage of neonates with a birth weight below the third centile at each unit.

4.1.5.7c Examining antihypertensive medication choice and birth weight

An examination of the six predominant types of anti-hypertensive medication used and neonatal birth weight adjusted for gestation centile was undertaken.

Anti-hypertensive medication	*Median birthweight centile of neonates born to women medicated with this drug	*Median birthweight centile of neonates born to women not medicated with this drug	ss
Clonidine	46 (52.0)	45 (57.0)	0.682
Labetalol	26 (41.5)	46 (56.0)	**p<0.0001**
Methyldopa	39 (31.4)	47 (56.0)	**p=0.018**
Nifedipine	29 (58.0)	46 (57.0)	**p=0.007**
Hydralazine	38 (53.5)	46 (57.0)	p=.077
Oxprenolol	42 (54.5)	46 (57.0)	p=.092

*median centile and inter-quartile range, ss – statistical significance
Mann-Whitney U analysis

Table 4.17. Anti-hypertensive medication type and birthweight centile. A comparison of neonatal birthweight centiles between women medicated with anti-hypertensive medication during the antenatal period and those not medicated at all units.

An analysis of the relationship between birthweight centile and anti-hypertensive choice and length of antenatal prescription was undertaken. A (Pearson Chi-Squared) analysis compared the length of time medicated antenatally with the rate of delivery of an infant of birth weight $<10^{th}$ centile and showed a significant relationship of p=0.01 (OR 1.4, 95% CI 1.06-1.89). An examination of the six most commonly prescribed anti-hypertensives: labetolol, nifedipine, methyldopa, hydralazine, oxprenolol and clonidine, showed that this relationship only persisted for women prescribed methyldopa in the antenatal period (p=0.01, OR 1.5 95% CI 1.06-2.19). A

correlation between the number of days antenatally on methyldopa and birth weight centile did not show a significant relationship (Spearman's rho 0.021, p=.749).

Other factors shown to have an association with birth weight <10th centile were maternal BMI (kg/m^2) (Pearson correlation 0.214, p<0.0001), highest antenatal recorded systolic BP (Pearson correlation -0.78, p=0.002), highest antenatal diastolic blood pressure (Pearson correlation -0.11, p<0.0001) and maternal race (Asian or black) (Pearson Chi-squared p<0.0001). Logistic modelling was undertaken utilising step-wise approach for all variables which were found to have a significant relationship with the outcome variable. No variable was found to remain significant through this process.

4.1.5.8a. Breastfeeding at discharge

Unit Number	Rate of indicator
1	91%
2	68%
3	69%
4	92%
5	62%
6	93%

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
Chi-Squared analysis p<0.001

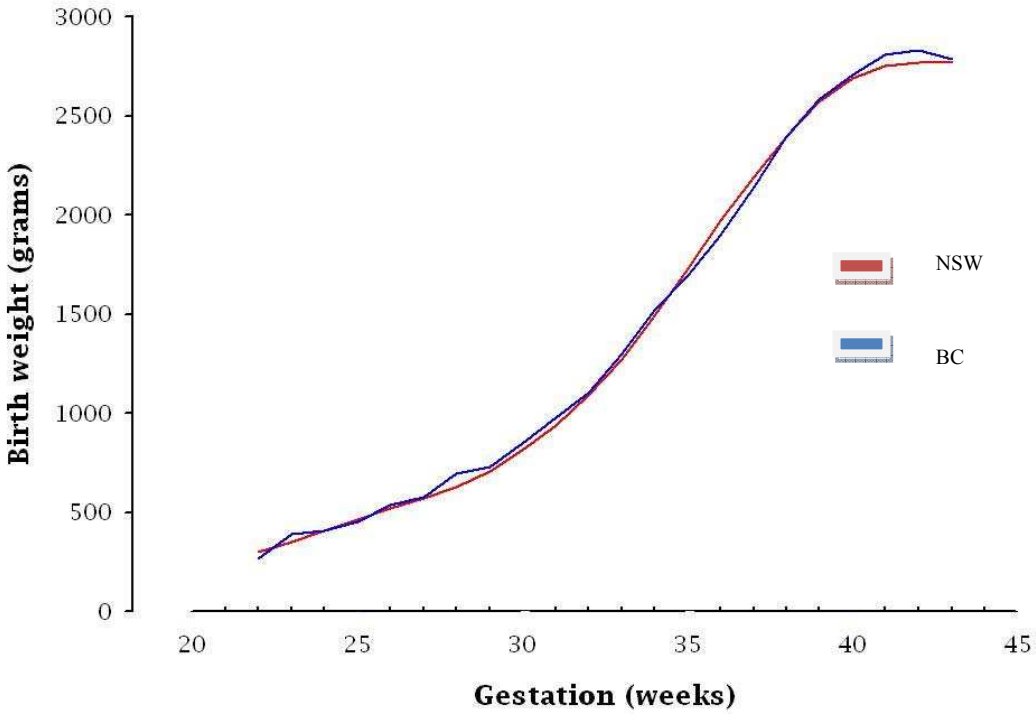
Table 4.18. Breastfeeding at discharge. The percentage of women breastfeeding at discharge from hospital at each unit.

4.1.5.8b. Breastfeeding initiation analysis

An analysis was undertaken to examine potential causative factors in the initiation of breastfeeding in this population. Maternal smoking had an significant negative association with breastfeeding initiation ($p<0.0001$), as did the delivery of a neonate pre-term ($p<0.0001$), maternal age >30 ($p<0.0001$), a maternal BMI(kg/m^2) of >30 ($p<0.0001$) and a maternal diagnosis of GDM ($p=0.03$). These factors were examined further in a binary logistic model. Having a BMI (kg/m^2) of >30 (obese) decreased the OR of successfully initiating breastfeeding by 2.4 ($p<0.0001$, 95% CI 1.82-3.25). Maternal age >30 years decreased the OR of successfully initiating breastfeeding by 0.57 ($p<0.0001$, 95% CI 0.426-0.760).

4.1.5.9 Birthweight centile chart comparison

An analysis of the birth weight percentile charts used in NSW and BC was undertaken to assure that there was no discrepancy between the NSW charts and those used within BC. All neonatal centiles herein were calculated upon the charts used within NSW.



Pearson correlation 0.979

Figure 4.22. Correlation of gestation/weight centile charts. The correlation of the centile for gestation and weight for females used in New South Wales and British Columbia.

This close alignment of the centiles was replicated for males also.

4.1.6 PHASE 6 CLINICIAN FEEDBACK

At Units 1, 2, 3, 4 and 5 clinician feedback occurred by way of 30 minute visual presentation sessions. Fifteen minutes was scheduled at the end of each presentation to allow for clinician feedback and questions. Data were presented graphically as per displayed results. Clinicians were invited to ask questions and provide feedback during and after the presentations.

	Unit 1	**Unit 2**	**Unit 3**	**Unit 4**	**Unit 5**	***Unit 6**
No. attended	78	15	53	21	42	34

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW
*face to face feedback session November 2008

Table 4.19. Clinician feedback attendance. The attendance rates for all clinicians who attended the feedback presentations sessions.

At unit 6 clinician feedback occurred in a different fashion due to geographical distance and extent of feedback.

Following data collection and analysis, a written report was provided to the lead clinician at unit 6. This clinician then used the information provided to construct and deliver an oral presentation to all obstetric/nursing/midwifery and neonatal clinicians across the BC province. This occurred by way of a visual and audio link-up between all obstetric units across BC.

4.1.7 PHASE 7 ESTABLISHING BENCHMARKS

The lowest rate of indicators 1 through 7 which occurred at any unit and the highest level of indicator 8 were established as the 'best achievable' towards which the other units should aim.

Indicator	'Best achievable'	Achieved by
Maternal mortality	0/1 000	All units
Acute pulmonary oedema	0/1 000	Units 1, 4 & 5
Acute renal failure	5/1 000	Unit 3
Eclampsia	2/1 000	Unit 6
Perinatal mortality	5/1 000	Unit 3
Admission of term neonates to Neonatal Intensive Care Units	3%	Unit 4
Birth weights <10th centile	9%	Unit 1
Birth weights <3rd centile	3%	Units 1 & 5
Breastfeeding at discharge	93%	Unit 6

1.RPAH, 2. LH, 3. NH, 4. RNSH, 5. CTH, 6. BCW

Table 4.20. 'Best achievable' indicators. The clinical indicators and the 'Best Achievable' rates achieved by all units.

The establishment of benchmarks was investigated further between units 1 and 6. Teleconference facility with the lead clinicians at both units was used to discuss results obtained. The identification of the issue of APO as an area of difference between units 1 and 6 was confirmed from the benchmarking process. The presentation of the results herein in regard to the incidence of APO clarified the extent of the issue within a unit where many practitioners expressed scepticism that the incidence of APO was an area of concern within their unit.

This issue was flagged for further analysis and Phase 4 was reinstituted at this point utilising logistic regression techniques. This step-wise process involves identifying potential factors which may potentially be included in the model. These factors were determined via discussion between the lead clinicians at both units 1 and 6.

	Women with APO	**Women without APO**	**SS**
Age (mean ±SD)	34±3.6	32±5.8	0.209
Final diagnosis of preeclampsia	**89%**	**42%**	**<0.0001**
Booking systolic BP (mean mmHg ±SD)	115± 15.2	118±16.1	0.512
Booking diastolic BP (mean mmHg ±SD)	74±10.2	74±10.4	0.939
Highest antenatal systolic BP (mean mmHg ±SD)	144±23.1	147±17.0	0.457
Highest antenatal diastolic BP (mean mmHg ±SD)	**88±11.9**	**93±11.6**	**0.048**
Body Mass Index (weight/height^2) (mean units ±SD)	26±5.2	26±5.8	0.529
Parity - %primiparous*	68%	67%	1.000
Delivery gestation (mean weeks ±SD)	**36.7±4.48**	**37.3±2.82**	**0.014**
Delivered via caesarean section*	**68%**	**44%**	**0.034**
Induction of labour attempted*	47%	45%	1.000
Failed induction of labour*	**70%**	**31%**	**0.031**
Highest postnatal systolic BP (mean mmHg ±SD)	**165±12.6**	**151±15.9**	**<0.001**
Highest postnatal diastolic BP (mean mmHg ±SD)	96±11.1	94±10.3	0.387
Intravenous $MgSO_4$ administration*	42%	39%	0.872
Total intravenous fluids administered (median & Inter Quartile range ml)	**9210 (6720)**	**5200 (5688)**	**<0.001**
Acute renal failure	2/1000	2/1000	1.000
Length of hospital stay (mean days ±SD)	**7.3±2.7**	**5.9±3.8**	**0.048**

APO acute pulmonary oedema, $MgSO_4$ magnesium sulphate, SD standard deviation

Table 4.21. Details for all women with/without acute pulmonary oedema at unit 6. Demographics, parameters, treatments and outcomes for women with APO at each unit.

The factors highlighted in the Table 4.21 were those which were used in the regression model. Length of stay was not included as this was determined to be affected by the development of APO rather than independent of it. Comparing Units 1 and 6, there were no other statistically significant differences other than in the volume of intravenous fluids administered suggesting the possibility of overuse of intravenous fluids at Unit 6.

	Unit 1	**Unit 6 women with APO**	**Unit 6 women without APO**	SS
Antenatal fluids administered ml*	1 000 (1 000)	6 204†	2 400 (2 653)	†
Intrapartum fluids administered ml*	1 000 (2 875)	4 276 (3 000)	2 775 (2 290)	**<0.001**
Postpartum fluids administered ml*	1 000 (2 000)	4 625 (4 000)	2 400 (2 803)	**0.003**
Total fluids administered ml*	2 100 (3 000)	9 210 (6 720)	5 200 (5 688)	**<0.001**

*median and interquartile range reported
†n=2, unable to calculate interquartile range or SS
Unit 1 RPAH, Unit 6 BCW
APO acute pulmonary oedema
SS statistical significance calculated between women at BCW only

Table 4.22. Intravenous fluid volume administration. The intravenous fluid administration volumes for all women at units 1 and 6.

4.1.8 PHASE 8 INSTITUTING CHANGE TO REFLECT OPTIMAL CARE

The results obtained from the analysis of the relationship between the development of APO and fluid administration were presented to unit 6. These results were further presented to the clinicians of BC via the province wide presentations described previously. Following this presentation, a sub-committee was formed to develop a new protocol for the administration of intravenous fluids in women with hypertension. This new protocol was introduced across the province of BC in December 2006. The new protocol reflected the limited fluid administration practices of unit 1; under which intravenous fluids are monitored strictly in hypertensive women and an 80/ml per hour policy was introduced at unit 6.

4.1.9 PHASE 9 ASSESSING CHANGE AND PROTOCOL ADHERENCE

Following the introduction of the new fluid administration policy protocol, a review of adherence to the protocol was undertaken. Unit 6 was attended in April and September 2008 and Phases 3,4 and 6 were undertaken for the 2006 and 2007 cohort of hypertensive women. The 2006 cohort was examined to determine if APO rates were similar to 2005 and 2007 cohort was examined to determine the extent of protocol adherence. The demographic and baseline characteristics of the women at unit 6 are compared in Table 4.23 to determine homogeneity of population between sampling years

	Unit 6 2005 cohort	**Unit 6 2006 cohort**	**Unit 6 2007 cohort**
Age	33 (5.7)	33 (6.0)	34 (5.6)
Body Mass Index	26.4 (5.81)	26.7 (6.35)	26.8 (5.65)
Primiparous	67%	67%	66%
Smoking*	27%	10%	8.5%
Gestation at initial care provider visit	13.6 (6.08)	13.7 (6.13)	13.6 (7.2)
Systolic blood pressure at initial care provider visit	118 (14.2)	118 (15.9)	118 (16.3)
Diastolic blood pressure at initial care provider visit	74 (10.3)	72 (10.4)	75 (9.9)
Highest antenatal systolic blood pressure	147 (17.3)	144 (18.3)	145 (17.8)
Highest antenatal diastolic blood pressure	93 (11.0)	92 (12.1)	95 (11.4)
Induction of labour rate	45%	44%	46%
Caesarean section rate	45%	46%	46%

*statistical significance reached for this parameter only $p<0.001$

Table 4.23. Demographic details and baseline characteristics unit 6. The demographic details and the baseline characteristics of all women at unit 6 for 2005, 2006 and 2007.

As the incidence of APO was the indicator being targeted at unit 6, an assessment of cases was made for the three years of the study and is displayed in Table 4.24.

	Unit 6 2005	**Unit 6 2006**	**Unit 6 2007**
Acute pulmonary oedema	46/1 000 (19 cases)	30/1 000 (11 cases)	2/1 000 (1 case)

Table 4.24. Acute pulmonary oedema rates at unit 6. Acute pulmonary oedema rates at Unit 6 before and after the introduction of the new fluid administration protocol.

To determine if the protocol change was being adhered to, an analysis of the amount of intravenous fluid was undertaken and is displayed in Table 4.25.

	Unit 6 2005		**Unit 6 2006**		**Unit 6 2007**	
	Women with APO	Women without APO	Women with APO	Women without APO	Woman with APO	Women without APO
Antenatal fluid administered (ml)	6204*	2400 (2653)	8144 (4964)	3310 (3913)	0*	500 (2043)
Intrapartum fluids administered (ml)	4276 (3000)	2775 (2290)	3300 (3878)	3000 (2000)	9313*	2200 (1857)
Postnatal fluids administered (ml)	4625 (4000)	2400 (2803)	5815 (6841)	2075 (1950)	15688*	1950 (1956)
Total fluids administered (ml)	9210 (6720)	5200 (5688)	11665 (15407)	5235 (4495)	25001*	3900 (3973)

Median and interquartile range reported
*interquartile range not reported due to small number of cases

Table 4.25. Intravenous fluids administered and acute pulmonary oedema. A comparison of the volume of intravenous fluids administered at Unit 6 for women with and without APO.

4.2 EXPERIMENT 2

As detailed previously, a survey of all clinicians who attended a feedback session at their unit and attendees at the Perinatal Society of Australia and New Zealand Annual Conference 2007 (Melbourne, Australia) was undertaken. The actual survey can be found in Appendix III.

	No. of respondents
Midwife	92
Obstetric resident	13
Obstetric registrar	19
Obstetric staff specialist	29
Maternal-Fetal medicine specialist	12
Neonatologist	1
Paediatrician	2
Medical researcher	3
Student	2
Total	173

Table 4.26. Survey respondents. The professional groupings of the survey respondents for all units.

	No. of respondents	**<12 months**	**12 months – 5 years**	**>5 years-10years**	**>10 years –2o years**	**>20 years**
Length of time in this profession	173	8%	12%	29%	33%	20%

Table 4.27. Length of time in current profession. The length of time in current profession for survey respondents for all units.

	No. of respondents	Daily	Weekly	Monthly	<Monthly
How often do you have clinical care contact with women with a HDP	173	59%	30%	2%	9%

Table 4.28. Frequency of clinical care. The frequency of clinical care for all survey respondents at all units.

	No of respondents	Yes	No	Unsure
Existence of protocol	169	85%	1%	14%

Table 4.29. Existence of protocol. The answer al all survey respondent at all units when asked: 'Does your unit have a protocol for managing the HDPs?'

	No. of respondents	All of the time	Most of the time	Some of the time	Never followed
Compliance with protocol	157	30%	66%	3%	1%

Table 4.30. Protocol compliance. The answer of all survey respondents when asked: 'If there is a protocol present, is it followed?'

	No. of respondents	Within the last 12 months	Within the last 5 years	Unsure
Frequency of protocol review	161	48%	17%	35%

Table 4.31. Frequency of protocol review. The answer of all survey respondents when asked: 'If there is a protocol present, when was it last reviewed?'.

	No. of respondents	Hospital protocols	Published results of randomised control trials	Published results from systematic reviews and meta-analyses	Personal experience	Experience of colleagues
Evidence	163*	86%	42%	36%	37%	16%

*Total respondents=163, although respondents could respond to as many categories as required to answer question

Table 4.32. Type of evidence care provided based upon. The answer of survey respondents when asked: 'If you are a clinician providing care to women with a HDP, do you base your clinical care upon?'

	No. of respondents	Yes very familiar	Yes somewhat familiar	Heard the term but not familiar	Never heard the term
Benchmarking familiarity	173	14%	39%	34%	13%

Table 4.33. Benchmarking familiarity. The answer of survey respondents when asked: 'Are you familiar with the benchmarking process?'

	No. of respondents	**Yes**	**No**	**Maybe**	**Unsure**
Practise change	165	61%	0%	35%	4%

Table 4.34. Practice change. The answer of survey respondents when asked: 'If it could be shown to you that outcomes following a HDP were different between units through a process of individual patient note review and feedback, would you consider changing your practice according to the practices of units with better outcomes?'

Cross tabulations were undertaken to see if respondent's answers varied dependant upon their profession.

	Midwife	Obstetric Resident	Obstetric Registrar	Obstetric Staff Specialist	Maternal-Fetal Medicine Specialist
Hospital protocols	95%	100%	79%	97%	100%

Table 4.35. Profession and basis for clinical practice. A comparison of profession and what evidence clinicians base their clinical practice upon.

	Midwife	Obstetric Resident	Obstetric Registrar	Obstetric Staff Specialist	Maternal-Fetal Medicine Specialist
Yes	64%	8%	69%	62%	100%
Maybe	30%	92%	31%	38%	0%
Unsure	6%	0%	0%	0%	0%

Table 4.36. Profession and practice change. A comparison of profession and willingness to changes clinical practice based upon results obtained from benchmarking initiatives.

CHAPTER 5

DISCUSSION

5.1 DEMOGRAPHICS

This study aimed to examine a cohort of hypertensive pregnant women at six obstetric units and to utilise the clinical data to establish benchmarks for clinical care. Further, this study sought to determine if an audit of best achievable outcomes at each unit was a useful tool which could be utilised to effect positive change in the health outcomes for these women and babies. Both of these aims were achieved.

One thousand, seven hundred and twenty eight pregnancies at six units over a 12 month period resulted in more than of 350 000 data points being collected. This enabled a detailed and exact analysis of clinical and laboratory parameters to occur, including treatment options and durations. Most importantly this study concentrated on options of care that are amenable to change and for which there is evidence of disagreement between clinicians and protocols at various obstetric care units.

Although the units are identified in this thesis, the presentations at each unit occurred in a de-identified manner deliberatively designed to diminish identification of the units and to encourage co-operation.

5.1.1 AGE, INCOME AND BMI (KG/M^2)

The demographic differences between units demonstrated in this study were statistically significant, despite the close geographical proximity of the five NSW units. Major correlations were found between maternal age and booking systolic and diastolic blood pressure readings. Although the income of each woman was not collected in this dataset, the socio-economic profiles of the geographic location of each hospital enabled conclusions to be made. The women in higher income areas had the lowest rates of GDM, and the lowest mean BMI (kg/m^2). The correlations between higher income and lower rates of overweight and obesity have been documented previously and are attributed to greater access to health care services (Van Doorslaer et al. 2008, Villar & Quintana-Domeque 2009), although this relationship may be questionable. Women with higher incomes are also more likely to seek medical care earlier in their pregnancies with many receiving pre-pregnancy counselling and assisted reproductive technology (ART) treatment (Omurtag et al. 2009, Hammoud et al. 2009). The issue of the potential effects of ART would be further amenable to examination, if this was a parameter on the NSW MDC, although this was not a subject of this study.

This study illustrates that BMI (kg/m^2) is also related to breastfeeding, with obese (BMI>30) women having lower success rates of breastfeeding initiation. This finding is supported in the literature (Bartick & Reinhold 2010, Kitsantas & Pawloski 2010).

5.1.2 SMOKING

According to the ABS Census 2006, 20% of all Australians over the age of 15 smoke cigarettes daily and 18% of women smoke. This statistic was reflected in these data, with smoking rates varying between 7-20% in this cohort. These variations in smoking rates are also associated with the socio-economic status of the geographic area of the units, and these findings are supported in the literature (Mohsin & Bauman 2005, Delpisheh, Kelly, Rizwan and Brabin 2006). Cigarette smoking has been noted previously as being protective against preeclampsia (Engel et al. 2009), yet in this dataset, the units with the higher smoking status had the highest rates of preeclampsia.

5.1.3 INCIDENCE OF HDP

The incidence of HDP and the type of HDP varied between units. HDP rates varied between 6-11% at individual units with an overall HDP rate of 7.5%. Preeclampsia rates varied between 2-4% at individual units with an overall preeclampsia rate of 3%. Preeclampsia rates are quoted in the literature as being between 5-7% of all pregnancies, regardless of geographical variations (Sibai et al. 1997, Roberts & Cooper 2001, Redman & Sargent 2005) and the rate for NSW in 2005 was 5.5% (NSW Department of Health 2005) and BC 5.4% (British Columbia Statistical Agency 2001). The variation in rates seen between the units is most likely due to coding ascertainment rather than ‘true’ variations in rates of HDP.

The medical records in NSW and BC are assigned ICD codes in accordance with what is written in the notes by medical and allied health personnel, not purely from the discharge summary, if one is present. The clinical coders examine all aspects of the notes looking for written entries which relate to the various ICD codes. If a medical or allied health individual notes 'preeclampsia', for example, in a woman's notes, this woman is then coded for preeclampsia. If no such entry is noted, even if the woman has elevated blood pressures and other diagnostic criteria, none of the ICD codes for elevated blood pressure will be appropriately assigned. This phenomenon has been examined previously (Thornton et al. 2004) and it was shown that the use of the ICD coding resulted in a significant miscoding for the occurrence of a HDP. The accuracy of hospital database ICD recording of maternal conditions was also examined by Taylor et al. (2005). The kappa for agreement between Inpatient Statistics Collection ICD coding and medical note review was 0.64 for preeclampsia, with a reported sensitivity of 50% in the Taylor et al. (2005) study. The two units with the higher rates of HDP (RPAH and NH), are those units which provide in-service to the clinical coders on a regular basis concerning the signs and symptoms of the various presentations of HDP. Other units do not follow this in-service practice, and this could be an explanation for the lower rates of HDP noted from the utilisation of the ICD coding. The only way to overcome this ascertainment issue would be to audit the medical records of all deliveries which occurred within an institution over a defined time period (minimum one year), to determine truly accurate rates of HDP within that institution.

As outlined previously, the NSW MDC only records preeclampsia and chronic hypertension and does not take into account gestational hypertension or

superimposed preeclampsia, and is, therefore, not an accurate reflection of disease incidence, as it is reflected in the current SOMANZ Guidelines (Lowe et al 2008). The rates quoted for hypertension in pregnancy for NSW (NSW Department of Health 2006) are rates obtained from the NSW MDC, not from the ICD coding.

Risk factors for the development of a HDP are many and varied. Some potential contributing factors, such as maternal age, parity and weight are routinely collected data. Other potential factors, such as the use of ART and barrier contraception, are not. Due to the many varied risk factors and the infinite number of other unknown factors at play in the development of a HDP, the composition of datasets would need to be very detailed in order to capture both known and, as yet, unknown potential risk factors. Statistical modelling from the dataset examined herein, was unable to define a 'risk profile'. Modelling which may be able to elucidate a 'risk profile' would only be able to be successfully undertaken on a much larger and more detailed dataset. There is evidence from large datasets in the literature (Tyldum, Romindstad & Slordahl 2010, Nilsen et al. 2009), in which data linkage on datasets of 300 000 women link birth data and pregnancy outcomes. The risk with all large routinely collected datasets is that more complex medical complications of pregnancy are more likely to be incorrectly coded, as supported by validation studies undertaken (Roberts et al. 2008, Pym & Taylor 1993). Pregnancy hypertension linkage projects can only be improved if an accurate dataset of women is obtained through medical record review, or from prospective database collection from a large population.

5.2 INDICATOR OUTCOMES

5.2.1 MATERNAL MORTALITY RATE

There was no incidence of maternal mortality at any of the units during the period of audit. This outcome reflects the overall relatively low rates for maternal death noted in the literature for developed countries (NSW Department of Health 2005, British Columbia Statistical Agency 2001, NICE 2005). The MMR in Australia is 4/100 000 live births and in Canada 8/100 000 live births (WHO 2010), whilst in developing countries such as Afghanistan, the MMR is 1 600/100 000 live births, the highest rate in the world. The median world rate is 44/100 000 live births (WHO 2010). The generalisability of the indicators to be used worldwide is reflected in the inclusion of this indicator, so whilst it might be not seen as a useful measure in the developed world, it may be viewed differently in the developing world.

5.2.2 ACUTE PULMONARY OEDEMA

Acute pulmonary oedema rates in NSW varied little between units during the datum collection period; unit 1 RPAH 0/1 000, unit 2 LH 6/1 000, unit 3 NH 5/1 000, unit 4 RNSH 0/1 000 and unit 5 CTH 0/1 000, although the APO rates in unit 6 BCW varied between 46/1 000-3/1 000 (2005-2007). The overall rates for APO for NSW or BC province are not available as they have not been published on a population dataset in the literature, and are not collected in any routine dataset.

Acute pulmonary oedema has been linked to increased maternal age, delivery via caesarean section, BMI (kg/m^2), parity, undiagnosed cardiomyopathy (Sciscione et al. 2003), multiple gestation, corticosteroid use, colloid therapy and $MgSO_4$ use (Yeast et al. 1993). Endothelial damage, and resulting fluid leakage into the alveolar space, contributes to the development of APO in hypertensive women (Cotton et al. 1984, Benedetti et al. 1985), largely due to preeclampsia being an endothelial disease (Roberts et al. 1989). Iatrogenic causes, linked to liberal, non-restrictive intravenous crystalloid fluid administration policies, have also been noted (Tuffnell et al. 2006). The negative inotropic effect attributed to commonly used anti-hypertensive medications in the obstetric setting, such as nifedipine (Abbas et al. 2006) and labetalol (Le Bret et al. 1992), has also been questioned as a contributing factor in the development of APO. Non-steroidal anti-inflammatory drugs (NSAIDS) have been implicated in the development of post partum preeclampsia (Makris et al. 2004) and their potential anti-diuretic effect may be a further factor in the development of APO.

A full examination of causative factors utilising logistic regression techniques will be discussed in section 5.3, as this indicator was the focus of further examination re the effectiveness of the audit and feedback technique at unit 6 BCW. The culmination of the use of these techniques was a significant improvement in the rate of APO at unit 6 BCW, highlighting the effectiveness of these techniques in informing clinicians and ultimately improving the health outcomes for women.

5.2.3 ACUTE RENAL FAILURE

Acute renal failure rates varied in this cohort between unit 1 RPAH 13/1 000, unit 2 LH 46/1 000, unit 3 NH 5/1 000, unit 4 RNSH 18/1 000, unit 5 CTH 8/1 000 and unit 6 BCW 2/1 000. The rates for ARF for NSW or BC are not available as they have not been published on a population dataset in the literature and are not collected in any routine dataset.

Renal perfusion (renal plasma flow) increases in normal pregnancy with a subsequent rise in GFR (Davison 1987). Glomerular enlargement and endothelial swelling occur in women with preeclampsia and GFR is reduced (Sheehan 1980). Basement membrane damage (loss of fenestration and charge selectivity), has been postulated as the reason for the presence of proteinuria (Karumanchi et al. 2005). These nephrotic changes can proceed to ARF in preeclamptic women, although the rate at which this actually occurs in unknown. Case series have been published (Naqvi et al. 1996) on women with preeclampsia and the associated diagnosis of ARF, yet the incidence of ARF in any given obstetric population has not been reported. The range for serum creatinine in normal pregnant women is 44-73 umol/L (De Swiet et al. 2002), reflecting the increase in GFR. Therefore, a value of >90 umol/l is considered indicative of a significant degree of renal impairment (Kincaid-Smith 1991). This is considered ARF without the need for biopsy for confirmation for the purposes of this study and is in accordance with clinical practice guidelines (Lowe et al. 2008).

The significantly higher rate of ARF at unit 2 LH was a point of discussion when the results were presented at this unit. From this discussion it was evident that clinicians

at this unit placed lesser value upon the serum creatinine results obtained from hypertensive women and relied more upon symptomatology, the presence of proteinuria and uric acid concentrations to assign diagnoses and commence treatment. This higher rate of ARF was not associated with higher rates of any other adverse outcome in this unit, nor progression to acute dialysis.

The association between a pregnancy hypertension diagnosis and long term renal disease has been the focus of several studies with varying findings. Van Pampus and Aarnoudse (2005) and Jacquemyn et al. (2004) found no association between preeclampsia/haemolytic anaemia, elevated liver enzymes and low-platelets syndrome (HELLP) and long term renal disease, whilst Shmaas and Maayah (2000) found high rates of microalbuminuria (23%) in previously preeclamptic women compared to 3% in those women who had normal pregnancies. Suzuki et al. (2008) in a study without controls, found high rates of focal segmental glomerulosclerosis, IgA nephropathy and nephrosclerosis on renal biopsy in postmenopausal women who had previously had preeclampsia.

Two recent reviews (Vikse et al. 2006, Vikse et al. 2008) showed that women with a prior history of preeclampsia had a greater risk of both end stage renal disease and of requiring a renal biopsy. Future modelling, and/or clinical audit within and between obstetric units could identify factors that contribute to the development of ARF in preeclampsia. Such research should influence practice favorably and would help lower long term morbidity and mortality. Such modelling may also enable the identification of women who have undiagnosed underlying renal disease which becomes unmasked during pregnancy as preeclampsia.

5.2.4 ECLAMPSIA

Eclampsia rates varied in this cohort between unit 1 RPAH 2/1 000, unit 2 LH 6/1 000, unit 3 NH 5/1 000, unit 4 RNSH 12/1 000, unit 5 CTH 47/1 000 and unit 6 BCW 2/1000. Eclampsia rates in the UK have been reported as 4.9/10 000 births (Knight, 2007), in comparison to a mean rate of 5.2/10 000 births within this dataset. Eclampsia rates are also available from RCTs. The overall eclampsia rate in both arms of the MAGPIE trial was 14/10 000, 63% of cases occurring in countries with a high PMR (40/1 000). In a national surveillance study in the UK (Douglas & Redman 1994), the case fatality rate from eclampsia was 1.8% and the PMR was 54/1 000 births. In the population studied herein there was one case of perinatal mortality associated with maternal eclampsia (PMR 80/1 000 births to women with eclampsia, or 0.6/1 000 births to women with a HDP).

There was large variation in $MgSO_4$ usage; unit 1 RPAH 18.0%, unit 2 LH 8.6%, unit 3 NH 2.4%, unit 4 RNSH 3.5%, unit 5 CTH 9.4% and unit 6 BCW 18.6% of all HDP women. The low incidence of eclampsia made logistic regression, to evaluate influencing factors, unable to be conducted.

Unit 5 (CTH), which had the highest rate of eclampsia, six cases over the 12 month period examined (47/1 000), had a $MgSO_4$ usage rate of 9.4% of all HDP women . The presentation of these results to this unit culminated in an analysis of all eclamptic cases. An examination of the choice of anti-convulsant used at this unit (not always $MgSO_4$) and an evaluation of other management practices was undertaken, which will hopefully resolve this high relative rate.

Magnesium sulphate usage for eclampsia prophylaxis has attracted considerable criticism in the literature (Yeast et al. 1993, Benedetti, Kates & Williams 1985 & Rice 2006), due to unacceptable toxicity risk in the face of very low eclampsia rates. In Australia there has not been any adverse drug reactions from $MgSO_4$ reported to the Australian Government Therapeutic Goods Administration (Australian Government Department of Health and Aging Therapeutic Goods Administration 2009). In contrast, the Institute of Safe Medication Practices (Rice 2006) in the USA maintains a database of incidents relating to $MgSO_4$ of which there are over 50 cases, some of which resulted in maternal death or persistent vegetative state involving the use of $MgSO_4$ diluted in intravenous fluids.

There are both short and potential long term sequelae of having experienced an eclamptic seizure. A recent study followed women 6-24 months after they experienced an eclamptic seizure (Andersgaard et al. 2009). Ten percent of women reported persistent amnesia, 22% reported loss of memory, 11% experienced visual disturbances and 10% had ongoing headaches. Long term consequences may include structural changes to the white matter of the brain, with lesions seen in 40% of women and loss of cerebral tissue in 25% of women six weeks post-partum (Loureiro et al. 2003, Demirtas, Gelal & Vidinli 2005, Zeeman 2009).

5.2.5 PERINATAL MORTALITY RATE

Overall PMR in NSW was 9/1 000 (NSW Department of Health 2005) and 4/1 000 in BC (British Columbia Statistical Agency 2001) in the year examined. This difference can be explained by the fact that the definition of PMR differs in the

Canadian setting in that 28 weeks gestation is considered viable. In the Australian setting 20 weeks is the age of viability for statistical purposes. The Australian definition was applied for the purposes of this study to Canadian data. PMR in HDP affected pregnancies in this cohort were unit 1 RPAH 16/1 000, unit 2 LH 17/1 000, unit 3 NH 5/1 000, unit 4 RNSH 11/1 000, unit 5 CTH 15/1 000 and unit 6 BCW 18/1 000. Ninety three precent of cases of perinatal mortality occurred in infants born pre-term, OR 169 (95% CI 9.52-30.07), whilst 70% of cases of perinatal mortality in this cohort occurred in neonates born with a birth for gestation /gender adjusted centile of $<10^{th}$ with an OR of 3.2 (95% CI 0.69-15.20). These very large confidence intervals are the result of the relatively small size of the PMR in this study. In a study of 247 cases of perinatal death in an Australian setting (De Lange et al. 2008), the OR for growth restriction was 3.9 (3.12-4.99) and pre-term birth of 22.1(18.16-26.76) respectively. Due to the low PMR in this study, regression techniques were not applied in an examination of causality.

5.2.6 ADMISSION OF TERM NEONATES TO NEONATAL INTENSIVE CARE UNITS

The NSW rate of term admission of neonates to NICU was 8% in 2004 (NSW Department of Health 2005). The rate in this study unit 1 RPAH 9%, unit 2 LH 12%, unit 3 NH 13%, unit 4 RNSH 3%, unit 5 CTH 19% and unit 6 BCW 7%. The highest rate of 19% occurred in the non-tertiary unit, so therefore, these babies were only admitted to a special care/level 2 nursery. Originally when this indicator was decided upon, it was thought that it would be a useful marker of neonatal wellness at varying institutions. The admission of a term neonate is an indicator of significant

neonatal morbidity. Comparisons made between pre-term and term neonates admitted to NICUs, have shown that term neonates who require NICU admission perform worse in measures of gross motor control than their pre-term counterparts. Term neonates admitted to NICUs also have lower Apgar scores at one and five minutes than pre-term neonates (Persson & Stromberg, 1995).

Potentially there are serious consequences of a term NICU admission on the family and specifically on the initiation and future success of breastfeeding (Cregan et al. 2002). It has been well documented that the admission of a neonate to an NICU is an extremely stressful experience for the family (Gale et al. 2004, Raines 1996, Cescutti-Butler & Galvin 2003). The alterations to the parenting role and the change in expectations of the pathway of events following delivery, are also drastically altered. So significant is this stress, that scales of measurement have been created such as the 'Parental Stressor Scale' (Miles & Brunssen 2003) and support network building initiatives, such as 'buddy' systems, have been implemented in some centres to assist and educate parents in coping and management skills (Preyde & Ardal, 2003).

At unit 5 CTH, the unit with the highest level of admissions, it was discussed at the feedback session that all neonates born via caesarean section at that unit were admitted to the special care nursery whilst awaiting the return of their mothers from the recovery unit.

This practice explained why that unit had the highest rate of admission, but also brought emphasis to the affect that individual hospital protocols and practices have

upon indicator results, something which is not always evident when examining the statistics, rather than the unit, in depth. The medical records provide a profile of the woman and baby, but do not make reference to the protocols of the Unit in which treatment is being delivered. A detailed understanding of the clinical environment in which care is delivered can only be gained through the collaborative processes of feedback and discussion. An indicator outcome, such as NICU admission for term neonates, should be interpreted in the context of the protocols operative at that site

5.2.7 BIRTH WEIGHT BELOW THE TENTH AND THIRD CENTILES

The rates of birth weight below the 10th centile were unit 1 RPAH 9%, unit 2 LH 20%, unit 3 NH 11%, unit 4 RNSH 18%, unit 5 CTH 11% and unit 6 BCW 17% of all neonates born to hypertensive women. The rates of birth weight below the 3rd centile unit were 1 RPAH 3%, unit 2 LH 5%, unit 3 NH 4%, unit 4 RNSH 6% unit 5 CTH 3% and unit 6 BCW 7%. Discussion at feedback sessions centred around the potential causative factors for these variations. It was suggested that there may be large variations in the centile charts used in NSW when compared to those used in BC, yet these charts correlated well. Ethnicity, anti-hypertensive medication usage and smoking status were also suggested as potential contributing factors and were used in logistic modelling. Birth weights below 10th and 3rd centiles did vary significantly between units, yet statistical modelling utilising the data obtained in this study was not found to add any knowledge to the causative factors for the low birth weight rates in this study.

Birth weight below the 10th and 3rd centiles of weight for gestational age and gender is associated with both short and long term health consequences for that individual.

Whilst survival rates for low birth weight babies have improved in developed countries since the introduction of NICUs, morbidity in the short and long term remains higher than babies or normal birth weight (Payne et al. 2010). Poor postnatal growth, necrotising enterocolitis, intracranial haemorrhage, chronic lung disease and retinopathy still remain significant risks for low birth weight babies. This dataset under examination did not collect detailed data on the short or long term health of the babies born to women with a HDP.

Being born at a very low birth weight (that is small for gestational age (SGA)) in comparison to appropriate for gestational age (AGA) is associated with an increase in mortality of 4.52 fold (Regev et al. 2003). In a study of 2 764 babies born at very low birth weights (Regev et al. 2003), 53.4% of the SGA babies were born to women with an HDP. Babies born below the 10th and 3rd centiles are more likely to require NICU admission with attendant separation from the mother.

The concept of significant long term adult morbidity issues for low birth weight individuals is encapsulated in what is known as the Fetal Origins of Adult Disease theory (Barker et al. 1989, Barker 2002, Barker 2003) or more currently the more encompassing Developmental Origins of Health and Disease theory. Whether this is a consequence of events in-utero, or the result of neonatal/paediatric 'catch-up' weight gain (Huxley, Schiell & Law 2000), there exists a relationship between low birth weight and adult cardiovascular and metabolic disease. A relationship between

low birth weight and emotional and behavioural problems at age 12 has also been highlighted by Sabet et al. (2009) in a cohort of 1 029 adolescents in South Africa. Poorer educational outcomes and lower mean Intelligence Quotient (IQ) scores were also seen in SGA infants when compared to their normal birth weight controls in a prospective cohort study in the USA (van Burk et al. 2009). The Australian Raine Study Cohort has identified the existence of multiple relationships between the uterine environment, neonatal development and child and adolescent health outcomes (Blake et al. 2000, Hickey et al. 2009)

The issue of the potential effect of different pharmacological treatments upon fetal weight is an area of concentrated research and discussion. Systematic reviews, including a Cochrane review, have examined the benefit of aiming pharmacological treatment to maintain varying levels of blood pressure and these treatment effects on fetal size (von Dadelszen et al. 2000, Magee, Orntein & von Dadelszen 1989, Magee & Duley 2000, von Dadelzsen & Magee 2002, Magee 2007, Abalos et al. 2007). The consensus of these reviews is that more RCTs need to be conducted in order to answer the questions regarding the effect of the degree of blood pressure control in both chronically hypertensive women, and in women who develop hypertension during their pregnancy. A current multi-centered, international RCT "Control of Hypertension in Pregnancy Study (CHIPS)", is aimed at whether "tight" or "less tight" control of diastolic blood pressure is associated with better or worse outcomes in women with non-proteinuric hypertension and chronic hypertension and their babies (UBC 2009). The CHIPS study will examine short term outcomes.

The literature informs us that the beta-blocker, atenolol (Butters, Kennedy & Rubin 1990, Lip et al. 1997, Butters 1990) and the ACE inhibitors (Barr & Cohen 1991) have been associated with reduced weight at birth and fetal renal perfusion, and for this reason, the use of these drugs should be avoided during pregnancy. In this current study 14 different anti-hypertensives were prescribed to hypertensive women in the antenatal period, including atenolol and the ACE inhibitors. Anti-hypertensive choice is clearly based upon unit preference, protocol or historical biases in lack of trial evidence. The Cochrane Review of drugs for the treatment of very high blood pressure in pregnancy concluded that; 'Until better evidence is available, the choice of anti-hypertensive should depend on the clinician's experience and familiarity with a particular drug, and on what is known about adverse effects' (Duley et al. 2006, p.1).

An examination of the birth weight centiles of the babies born to women medicated with anti-hypertensives in the antenatal period compared to those not medicated, showed that for the babies of women medicated with labetalol, methyldopa and nifedipine, birthweight centiles were lower. However the effect of drug choice on birth weight was lost when combined within a model including race, smoking and blood pressure readings. Drug choice should be informed by the results of RCTs or, by collaborative multi-centred outcome driven data collections such as that herein. In order to determine the 'perfect' drug for the treatment of the HDPs, or at least to establish the non-inferiority of drugs used, large scale datasets are required. At the very least, datasets such as these, and the use of the audit and feedback processes, should inform and warn clinicians about the use of drugs which have been shown to be harmful such as atenolol and ACE inhibitors.

Regardless of treatment variations, the rates of low birth weights illustrated in this dataset, which reflect the population average or better is commendable in compromised pregnancies where prematurity and growth restriction are likely consequences.

5.2.8 BREASTFEEDING AT DISCHARGE

The breastfeeding rates in this study were; 1 RPAH 91%, unit 2 LH 68%, unit 3 NH 69%, unit 4 RNSH 92%, unit 5 CTH 62% and unit 6 BCW 93% of all neonates born to hypertensive women.

The World Health Organisation (WHO 2010) recommends exclusive breastfeeding for the first six months of life, with continued breastfeeding for up to two years of age. The benefits of breastfeeding include both short term and long term advantages over infant formula. Passive immunity, protection against gastrointestinal and allergic diseases and thereby decreased morbidity are seen amongst breastfed infants (WHO 2010). Long term benefits include lower rates of adult hypertension, Type II diabetes and obesity, as well as higher cognitive ability (Neutzling et al. 2009).

Breastmilk has been shown to be of great immunological and nutritional value to pre-term infants, yet the initiation of breastfeeding in mothers of pre-term infants has been shown to be impaired due to insufficient lactogenesis markers when compared to women who deliver at term (Persson & Stromberg 1995). Significant numbers of babies delivered to women with a HDP are pre-term. In this study, 23% of babies were born pre-term. Research has shown that breastfeeding initiation rates in ‘high

risk' pregnancies, including preeclampsia, is lower than in the general population (Britton et al. 2007). Women with a HDP are therefore, at a two-fold risk in that they are unwell and often have pre-term deliveries. Therefore, there is the problem of compromised infants whose requirement for breast milk is high, coupled with compromised mothers who have physiological barriers to breastmilk production. This combination makes the initiation of breastfeeding in hypertensive women of great importance and a priority amongst health professionals.

Breastfeeding rates vary greatly between countries with initiation rates as low as 4% in Thailand and as high as 99% in Russia (Dyson, McCormick & Renfrew 2005). The majority of research conducted into breastfeeding initiation has examined term babies, yet reports on pre-term breastfeeding initiation have shown that rates in this population are significantly lower than in term neonates (Fruman et al. 2002, Espy & Senn 2002), even though breastmilk has been shown to reduce the rates of sepsis in babies by 27%. There are many different reasons for the success/failure to initiate breastfeeding including cultural, personal, social, attitudinal and socio-economic. The countries with the highest breastfeeding initiation rates are those with structured public policy supporting initiation and continuation/support programs such as exists in Sweden (Galtry 2003).

A systematic review of interventions to promote breastfeeding has shown that only increased antenatal education increased the rates of breastfeeding initiation by 1.57 (95% CI 1.15-2.15) when compared to standard care (Pate 2009).

In this study, breastfeeding rates varied between 68-93%. With the exception of the Canadian unit, the other five units are in close proximity to each other and the disparity of breastfeeding rates is a serious issue, as this showed the greatest discrepancy of any parameter measured between units. The disparity of income and the cascade of resulting health, access and education implications resulting from such is highly evidenced by this result. When these results were discussed with clinicians at feedback sessions, the opinion was strongly expressed that lack of resources for postnatal lactation support at some units was the potential reason for low breastfeeding initiation rates. This argument was refuted by the high rates of breast feeding in the Canadian unit (93%) in which there is a low presence of midwives and lactation consultants in the hospital setting. Under this system, such health personnel are only available in the community setting. This finding is not supported in the literature where it has been found that increased support both in and out of the hospital setting increased initiation rates and length of time breastfeeding (Britton et al. 2007)

From the logistic modelling undertaken, to successfully initiate breastfeeding a woman in this study was over 30, did not smoke and was in a healthy weight range. This correlates with the demographics of the units which had the higher breastfeeding initiation rates; units 1, 4 and 6. These findings are well supported in the literature. Oddy et al. (2006) found that overweight and obese women in Australia were more likely to have discontinued breastfeeding by six months of age, a finding supported by Li et al. (2003), Donath & Amir (2008), Hilson, Rasmussen & Kjolhede (2004). Women who smoke initiate breastfeeding at lower rates and for those who do initiate breastfeeding, the length of breastfeeding is lower in smokers

than in non-smokers – including those women who quit smoking during their pregnancy (Bosnjak et al. 2009, Giglia, Binns & Alfonso, 2006, Baghurst et al. 2007). Baghurst et al. 2007 concluded that higher socio-economic status (including education, type of housing and family income) was the largest predictor of breastfeeding success.

5.2.9 SUMMARY

The most and least favourable outcomes did not cluster within any one unit, but were dispersed amongst all units. This suggests the possibility that any one unit may achieve a zero MMR, low PMR, zero APO, zero ARF, low eclampsia rate, average birth weights, avoidance of NICU admission and high rates of breastfeeding. Such outcomes have already been achieved and are attainable through vigilance of care, education of clinicians and the accumulation of large datasets upon which multi-factorial analyses can be undertaken, although factors such as socio-economic status and lifestyle choices of patients are confounders which must always be taken into account.

5.3 THE AUDIT AND FEEDBACK PROCESS

In unit 6 BCW, the relatively high rate of APO was targeted as the indicator for which it was thought that clinical practice change could be reflected in improved outcomes. Through the utilisation of audit, feedback, policy change, re-audit and feedback it can been seen that this process was successful in a) affecting practice change and b) improving outcomes for HDP women over the three year time frame of the study.

The development of APO was directly linked at unit 6 BCW to the liberal fluid replacement evident at that unit. Median volumes of in excess of 9 litres were administered to women prior to the onset of APO. Logistic regression techniques identified that these volumes were the only significant variable contributing to the development of APO and, this was supported by the reduction in the incidence of APO once the restrictive fluid practice changes were implemented. Feedback from clinicians at unit 6, prior to the practice change being implemented, indicated reservations that limiting fluid replacement may affect renal function by altering renal perfusion. ARF rates at unit 6 BCW did not vary from 2005 to 2007; 2/1 000 cases in each year.

The use of audit and feedback in the obstetric setting has been successfully utilised as a quality improvement tool previously. In two South African studies (Pattison et al. 2006, Say et al 2009) a daily audit of severe obstetric morbidity events was instituted, including the completion of a form for ‘near miss’ events over a two year period. When the two year period was complete, protocols were changed to reflect

methods to improve care. Following the introduction of these new protocols, the audit and feedback process was continued for a further two years. Over the four year period, the MMR fell, as did the medical personnel error rate.

A systematic review of the effectiveness of audit and feedback (Jamtvedt et al. 2006a, Jamtvedt et al. 2006b) included 118 RCTs of this process and concluded that audit and feedback is effective in achieving positive practice change when protocol adherence was low to begin with, and is optimal when feedback is intensive. This review also concluded that feedback is most effective when given nearest the time of decision making and when the clinician had previously agreed to have their practice audited.

A RCT in 19 South American maternity hospitals, conducted to assess the optimal method of improving evidence based guideline adherence (Althabe et al. 2008), concluded that a combination of interventions including audit, feedback, workshops, use of local opinion leaders and reminders was effective in improving outcomes. The use of a strong local opinion leader can also be highlighted in the improvement of APO rates in the Canadian unit, as this facet of the study would not have been feasible without the presence and support of such an individual in this distant unit. This combination of audit and feedback accompanied by a local opinion leader was also highlighted by Chaillet et al. (2006) (in a systematic review of how best to implement guidelines in obstetric practice), as the most effective methodology.

The success achieved by audit and feedback in this study was also significantly accredited to the timeliness of the study. The previous protocol for intravenous fluid

administration at unit 6 BCW had not been reviewed or updated for several years. As is the predicament of many obstetric personnel, clinicians were not aware of their current outcomes due to a deficit of regular audit. Even if this audit process been in place, they would not have had any established benchmarks against which to compare their results. The benchmarking process undertaken allowed clinicians to see what outcomes were being achieved in other units and gave the impetus for the local opinion leader to revise the existing protocol and through the use of evidence based practice, to facilitate the implementation of this protocol on a province wide basis. Conducting an audit following the implementation of the new policy and feeding these results back to clinicians, allowed clinicians to view the changes which could be achieved through this process.

5.4 CLINICIAN ATTITUDES TOWARDS BENCHMARKING AS A TOOL FOR OUTCOME IMPROVEMENT

The survey conducted at all institutions and at an international conference provided an insight into clinician's attitude towards the use of benchmarking techniques. The number of attendees (169) and, the multi-disciplinary representation of those who attended, was viewed as encouraging of clinician's willingness to be involved in the process. The range of attendees at individual sessions was 20-100 people. The formation of the collaboration itself at six institutions also highlighted the success of the necessity for a local opinion leader to facilitate the process. This issue is further highlighted by the fact that the feedback sessions in the Canadian unit were attended by 20 people, yet a local opinion leader who is dedicated to the issue at hand, as occurred in unit 6 (BCW), carried great impetus for change. The level of seniority of those attending (82% having >5 years clinical experience) as well as the fact that 59% of attendees provided daily clinical care to pregnant women, were both viewed as positive signs that clinicians were willing to have their practice audited and were willing to be involved in feedback sessions. Each unit had a HDP protocol in existence and 85% of respondents knew this, although only 30% thought that the protocol was followed all of the time. This level of response is documented in the literature also. A study of obstetricians knowledge of protocols in relation to caesarean section guidelines (Lomas et al. 1989), revealed that 87% of respondents knew of the existence of a protocol but only 67% correctly knew of the contents and that the existence of the protocol alone did not result in clinical practice change. This finding was a reflection of those of Davis, Homer & Brown 2002 and Foy et al. 2004.

When asked what evidence clinicians base their clinical decisions on, 86% responded hospital protocols. The importance of the hospital protocol was highlighted via this survey. All professional groupings indicated that it was the hospital protocol upon which they based their clinical practice. Protocols and guidelines should be based upon the best level of evidence available as per the National Institute of Clinical Excellence Grading of the Evidence guidelines (NICE, 2009). The best recommended level of evidence is high quality meta-analyses of RCTs when available. The least recommended level of evidence is expert opinion. In this survey, 53% of respondents indicated that they based their clinical decision making on either their own opinion or the opinion of others. The response to question 5, that 87% had at least heard of the benchmarking process and that 96% would either change their practice if shown that outcomes could be improved by adopting the practices of other units or at least consider change, was considered a highly positive response to the process of change. The profession of the individual did affect this response, with 100% of Maternal-Fetal Medicine Specialists indicating their willingness to use this methodology as a basis for clinical practice change and only 63% of midwives indicating the same response. Whilst only one unit was actually involved in the phases of the study which instituted change and audited its effect, these results highlight how these techniques could be used across all institutions.

5.5 STUDY LIMITATIONS

The limitations of this study largely revolve around the size of the dataset and the relatively rare incidence of many of the outcomes chosen to be examined. A larger dataset would allow more meaningful comparisons of rare events and would give greater statistical power to regression analyses examining issues such as anti-hypertensive drug choices. Such analyses would empower clinicians to make clinical change based upon the most reliable evidence to hand within this current context. The size of the dataset and the limitation of only being conducted over a one year period also limited the tools which could be applied for the establishment of accurate benchmarks.

5.6 OTHER CLINICAL OUTCOMES

In undertaking this study it became apparent that there were many other clinical outcomes which could have been measured which were amenable to change. Anti-hypertensive drug choice, and the factors influencing such, was an issue that was noted on multiple occasions when analysing medical entries in records. The basis of drug choice and the perceived effect of medications on the recordings of fetal heart rates and patterns via cardio tomography (CTG), became apparent. A further study was initiated to examine if the anti-hypertensive drug clonidine had any effect on CTG readings. The results of this study are contained in Appendix IV.

5.7 FUTURE DIRECTIONS

- Many clinicians expressed an interest in further examination of the factors influencing the success of the induction of labour process in HDP women and the factors influencing the higher than average caesarean section rates these women experience.

- An analysis concerning the validity of the current disorder categorisation system in use in Australasia in classifying women according to outcomes or disease severity will also be undertaken.
- Ongoing comparison of outcomes amenable to clinical practice change should be seen as an issue of high importance amongst collaborators and a continuation of the collection of accurate data followed by audit and feedback is vital.

- An examination of the ability of units to maintain clinical change also is required at unit 6 BCW to determine if the initial reduction in APO was able to be maintained in the long term.

- A long term project examining the health outcomes for women and their offspring remote from pregnancy is also planned for the future. Such a project would encompass both the use of the individual patient data methodology combined with the use of health data linkage to determine the long term health status of women.

CHAPTER 6

CONCLUSIONS

‘A little learning is a dangerous thing;
Drink deep, or taste not the Pierian spring;
Their shallow draughts intoxicate the brain,
And drinking largely sobers us again.’

Alexander Pope 1709

Facilitating change in the clinical environment is one of the greatest challenges to the proponents of quality improvement. The completion of quality improvement means going beyond data collection, analysis and reporting to take clinical improvement and change to the next level by ensuring that reliable data are being used to inform clinical practice decisions.

A twelve month sample of data on 1 762 pregnancies from six units has been able to supply ample data to enable the benchmarking of outcomes and to inform clinicians of their units current status in regard to certain aspects of the care of women with hypertension during pregnancy. Additional data points collected on each pregnancy also allowed statistical modelling to be conducted to determine if causative factors for outcomes could be identified. The multitude of results obtained from this detailed, hand-collected dataset from multiple units, has not been produced previously. This dataset has enabled perinatal morbidity for women with hypertension to be examined in detail on an individual patient level.

This study has also shown that accurate data (as assessed by statistical agreement) can be gained from individual patient records. The accuracy of the data obtained by

this methodology cannot be replicated by any current source of computer-based obstetric information. These data can be utilised to make meaningful comparisons between units, utilising established clinical indicators, based upon the principles of being standardised, measurable, analysable, actionable, reportable and trackable. This set of well defined clinical indicators can be utilised both nationally and internationally, and as the Canadian example has illustrated, are replicable on a year to year basis.

The targeting of APO in the Canadian unit enabled the benchmarking process to be undertaken and this highlighted the effectiveness of the audit and feedback process in affecting clinical practice change by, namely dramatically improving outcomes and by reducing morbidity related to the incidence of APO in hypertensive women. The targeting of causative factors, such as fluid replacement strategies, identified by this dataset, is the methodology by which this improvement was able to occur.

It has been demonstrated that a multi-disciplinary partnership of researchers within obstetric units in both the Sydney region and internationally can collaborate on a research study. To enable this to occur, individuals must be willing to have the outcomes of their individual units exposed to peer scrutiny. Such a partnership can at times be discordant, as the acceptance of the results herein did at times vary dependent upon the type of result obtained and the inherent culture in existence within the unit. Favourable results were accepted without query, whilst less favourable outcomes were at times met with disbelief, yet the power of numbers and the statistical analysis of such, is evidence which is hard to refute. The force of a local opinion leader in shaping the culture of the practices within obstetric units is a

powerful one and practice change relies not only upon the strength of the data, but also upon the willingness of clinicians to be accepting of change and improvement.

The use of an anonymous survey has allowed clinicians to provide useful feedback concerning their views towards how they learn and the foundations upon which they base their clinical decisions.

In summary, benchmarking of outcomes following a diagnosis of HDP can be achieved within obstetric units and the audit and feedback process can be used with success to change clinical practice and achieve better health for women and their babies.

CHAPTER 7

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APPENDICES

APPENDIX I NSW MIDWIVES DATA COLLECTION

Policy Directive

NSW HEALTH

Department of Health, NSW
73 Miller Street North Sydney NSW 2060
Locked Mail Bag 961 North Sydney NSW 2059
Telephone (02) 9391 9000 Fax (02) 9391 9101
http://www.health.nsw.gov.au/policies/

Midwives Data Collection (MDC) Reporting and Submission Requirements

Document Number	PD2005_636
Publication date	05-Dec-2005
Functional Sub group	Clinical/ Patient Services - Maternity Clinical/ Patient Services - Records Clinical/ Patient Services - Information and data
Summary	Midwives Data Collection (MDC) is a statewide surveillance system which monitors patterns of pregnancy care, services and pregnancy outcomes.
Replaces Doc. No.	Midwives Data Collection (MDC) [PD2005_192] Midwives Data Collection - 1 January 1998 [PD2005_117]
Author Branch	Demand and Performance Evaluation Branch
Branch contact	Margie Luke
Applies to	Area Health Services/Chief Executive Governed Statutory Health Corporation, Affiliated Health Organisations - Non Declared, Community Health Centres, NSW Dept of Health, Private Hospitals and Day Procedure Centres, Public Hospitals
Audience	Administration, nursing, midwives, medical records
Distributed to	Public Health System, Community Health Centres, NSW Ambulance Service, NSW Department of Health, Public Hospitals, Private Hospitals and Day Procedure Centres
Review date	05-Dec-2010
Policy Manual	Not applicable
File No.	05/1804
Previous reference	N/A
Status	Active

Director-General

This Policy Directive may be varied, withdrawn or replaced at any time. Compliance with this directive is **mandatory** for NSW Health and is a condition of subsidy for public health organisations.

Policy Directive NSW HEALTH

MIDWIVES DATA COLLECTION (MDC) REPORTING AND SUBMISSION REQUIREMENTS

1. Introduction

1.1 From 1 January 2006, this Policy Directive rescinds and replaces Policy Directives PD2005_117 and PD2005_192 concerning the NSW Midwives Data Collection.

1.2 This policy directive applies to reporting of births to the NSW Midwives Data Collection (MDC) from 1 January 2006.

1.3 This policy directive should be distributed to all staff involved in collecting and supplying data for the MDC. This includes staff of obstetric and neonatal units and medical record staff.

2. Scope of the MDC

The MDC is a state wide surveillance system, which monitors patterns of pregnancy care, services and pregnancy outcomes. The MDC is an on-going collection, which is managed on a calendar year basis.

MDC forms, or an electronic equivalent, must be completed for all births in NSW, including live born babies regardless of gestational age and stillbirths of at least 20 weeks gestation OR 400 grams birth weight. In the case of multiple births, a separate form must be completed in full for each baby.

3. Reporting Methods

3.1 Paper submission of data is via the NSW Midwives Data Collection Form (MR44/PR16). The form comes in a four part set with the original copy sent to the NSW Department of Health and duplicate copies for hospital medical records, parents and Early Childhood Centres.

The form is available from Salmat (incorporating Government Printing Service) and may be obtained from their Fast Forms Division, fax (02) 9743 8603 or phone (02) 9743 8777.

3.2 Electronic submission of data must comply with the electronic file layout specifications for the MDC. Before the Department can accept regular submission of electronic data a three month sample of test data must be submitted. Approval for receipt of electronic data will be given when the test data has demonstrated to be in the correct format and of sufficient quality.

4. Reporting requirements

4.1 Reporting of all births in NSW is a requirement of the NSW Public Health Act, 1991.

4.2 Data submitted on hard copy (paper) should be submitted within 30 days of the baby being discharged.

4.3 Electronic data should be submitted no later than two months after the close of each quarter. For example January to March data should be submitted by 1 June. The following table summarises the due dates for electronically submitted MDC Data.

Time period	Date of data submission
January to March	1 June
April to June	1 September
July to September	1 December
October to December	1 March (the following year)

4.4 Data should be submitted to:

Midwives Data Collection
Data Collections and Quality Section
Demand and Performance Evaluation Branch
NSW Health Department
Locked Bag 961
North Sydney 2059

5. Data items to be reported

5.1 Demographic details of mother

- Unit record number of Mother
- Hospital name
- Hospital code
- First name
- Family name
- Address
- Mother's date of birth
- Indigenous status

Previous pregnancies

- Previous pregnancy greater than 20 weeks
- Number of previous pregnancies greater than 20 weeks
- Was the last birth by caesarean section
- Total number of previous caesarean sections

This pregnancy

- Estimated date of confinement (EDC)
- Was antenatal care received?
- Duration of antenatal care
- Diabetes mellitus
- Gestational diabetes
- Chronic hypertension
- Pregnancy-induced hypertension (proteinuric)
- Pregnancy-induced hypertension (non-proteinuric)
- Smoking
- Number of cigarettes smoked

Labour and delivery

- Onset of labour
- Type of augmentation or induction of labour
- Indication for induction of labour
- Presentation at birth
- Analgesia for labour
- Type of birth
- Main indication for caesarean section
- Anaesthesia for delivery
- Perineal status
- Episiotomy
- Surgical repair of the vagina or perineum
- Management of the 3rd stage

Baby

- Unit record number of Baby
- Baby birth date
- Sex
- Plurality
- Plurality number
- Birth weight
- Estimated gestational age
- Apgar
- Resuscitation of baby

Maternity care

- Model of care
- Mother referred from another hospital
- Referral hospital
- Referral before or after the onset of labour
- Place of birth

Postnatal care of mother and baby

- Postpartum haemorrhage requiring blood transfusion
- Birth defect
- Admitted to SCU/NICU
- If admitted to NICU: was a birth defect the main reason for admission
- Vitamin K
- Hepatitis B birth dose

Discharge status of mother and baby

- Discharge status of mother
- Discharge status of baby
- Mother's date of discharge or transfer
- Hospital mother transferred to
- Infant feeding on hospital discharge
- Baby's date of discharge or transfer
- Hospital baby was transferred to
- Baby transferred by NETS

5.2 The reporting of these data items must comply with instructions provided in the MDC Instruction Manual.

6. Data quality

6.1 Data quality checks are made to ensure that all fields are complete and there are no inconsistencies in the data within a particular record.

6.2 Incomplete records or records with errors, will be returned to the hospital of origin and must be completed and/or corrected and returned to the Data Collections and Quality Section of the Department of Health within the time stipulated.

6.3 In order to validate the enumeration of births, each year a list of reported births are sent to each hospital and validated against the hospital birth register.

7. Security of data

7.1 The Privacy Manual (Version 2) – NSW Health (Policy Directive 2005_593 – June, 2005) and the Privacy Management Plan (Policy Directive 2005_554 - March, 2005) must be observed for all data relating to the MDC.

7.2 Data sent in a hard copy (paper) format must be kept secure at all times. This means records must be sent by secure post (or courier) using a service that records the name of persons handling the data.

7.3 Data sent in an electronic format should not be sent by Internet e-mail unless authorised in advanced. Data submitted be e-mail should contain encryption and password protection. The password must be provided separately.

8. MDC information – access and dissemination

8.1 Summary information for NSW is published annually in the NSW Mothers and Babies Report.

8.2 De-identfied unit record data are provided to the AIHW National Perinatal Statistics Unit for inclusion in the national perinatal data collection.

8.3 De-identified unit record data may be obtained via the Health Outcomes Information Statistical Toolkit (HOIST), which is accessible by staff of the Department and Area Health Services subject to signing of a confidentiality agreement.

8.4 Access to de-identified MDC unit record data may also be sought by written request to the Director, Centre for Epidemiology and Research.

8.5 Information about the MDC, and other data collections, may be found on-line on HealthNet or HealthWeb (the NSW Health Intranet and Internet sites):

- HealthNet: http://internal.health.nsw.gov.au/im/ims/mdc/index.html
- HealthWeb: http://www.health.nsw.gov.au/im/ims/mdc

9. Further information

For further information about this policy directive or the MDC, contact:

Margie Luke
Manager, Data Collections and Quality Section
Demand and Performance Evaluation Branch

Phone: (02) 9391 9765
e-mail : mluke@doh.health.nsw.gov.au

or requests may be faxed to the Data Collections and Quality section on (02) 93919070.

Robyn Kruk
Director-General

APPENDIX II - ICD-10 HYPERTENSION CODES

Chapter XV

Pregnancy, childbirth and the puerperium (O00-O99)

Oedema, proteinuria and hypertensive disorders in pregnancy, childbirth and the puerperium (O10-O16)

O10 **Pre-existing hypertension complicating pregnancy, childbirth and the puerperium**

Includes: the listed conditions with pre-existing proteinuria

Excludes: that with increased or superimposed proteinuria (O11)

O10.0 **Pre-existing essential hypertension complicating pregnancy, childbirth and the puerperium**

Any condition in I10 specified as a reason for obstetric care during pregnancy, childbirth or the puerperium

O10.1 **Pre-existing hypertensive heart disease complicating pregnancy, childbirth and the puerperium**

Any condition in I11.- specified as a reason for obstetric care during pregnancy, childbirth or the puerperium

O10.2 **Pre-existing hypertensive renal disease complicating pregnancy, childbirth and the puerperium**

Any condition in I12.- specified as a reason for obstetric care during pregnancy, childbirth or the puerperium

O10.3 **Pre-existing hypertensive heart and renal disease complicating pregnancy, childbirth and the puerperium**

Any condition in I13.- specified as a reason for obstetric care during pregnancy, childbirth or the puerperium

O10.4 **Pre-existing secondary hypertension complicating**

pregnancy, childbirth and the puerperium

Any condition in I15.- specified as a reason for obstetric care during pregnancy, childbirth or the puerperium

O10.9 Unspecified pre-existing hypertension complicating pregnancy, childbirth and the puerperium

O11 Pre-existing hypertensive disorder with superimposed proteinuria

Conditions in O10.- complicated by increased proteinuria
Superimposed pre-eclampsia

O12 Gestational [pregnancy-induced] oedema and proteinuria without hypertension

O12.0 Gestational oedema

O12.1 Gestational proteinuria

O12.2 Gestational oedema with proteinuria

O13 Gestational [pregnancy-induced] hypertension without significant proteinuria

Gestational hypertension NOS
Mild pre-eclampsia

O14 Gestational [pregnancy-induced] hypertension with significant proteinuria

Excludes: superimposed pre-eclampsia (O11)

O14.0 Moderate pre-eclampsia

O14.1 Severe pre-eclampsia

O14.9 Pre-eclampsia, unspecified

O15 Eclampsia

Includes: convulsions following conditions in O10-O14 and O16
eclampsia with pregnancy-induced or pre-existing hypertension

O15.0 Eclampsia in pregnancy

O15.1 Eclampsia in labour

O15.2 Eclampsia in the puerperium

O15.9 Eclampsia, unspecified as to time period

Eclampsia NOS

O16 Unspecified maternal hypertension

APPENDIX III – SURVEY

Benchmarking the Hypertensive Disorders of Pregnancy (HDPS)

1. Does your unit have a protocol for managing the HDPs?

□ Yes □ No □ Unsure

2. If there is a protocol present, is it followed?

□ All of the time □ Most of the time □ Some of the time □ Never followed

3. If there is a protocol present, when was it last reviewed?

□ Within the last 12 months □ Within the last 5 years □ Unsure

4. If you are a clinician providing care to women with a HDP, do you base your clinical care upon - (tick as many options as appropriate for you)

□ Hospital protocols
□ Published results of RCTs
□ Published results from systematic reviews and meta-analyses
□ Personal experience
□ Experience of colleagues

5. Are you familiar with the benchmarking process?

□ Yes very familiar □ Yes somewhat familiar □ Heard the term but not familiar □ Never heard the term

6. If it could be shown to you that outcomes following HDP were different between units through a process of individual patient note review and feedback, would you consider changing your practice according to the practices of units with better outcomes? (ie less morbidity)

□ Yes □ No □ Maybe □ Unsure

7. What is your profession?

□ Midwife □ Obstetric resident □ Obstetric registrar □ Obstetrician/staff specialist
□ MFM specialist □ Renal physician □ Other (please state)
_____

8. How long have you been working in the above area?

□ less than 12 months □ 12 months-5 years □ 5-10 years □10-20 years □>20 years

9. How often do you have clinical care contact with women with a HDP?

□ Daily □ Weekly □ Monthly □ Less than monthly

Thank you for taking the time to complete this survey. Your feedback is invaluable to the provision of improved care to women and their babies following hypertension during pregnancy.

APPENDIX IV – JOURNAL ARTICLE:

Thornton, C, Makris, A, Tooher, J, Ogle, R, Hennessy, A 2010, 'Does the anti-hypertensive drug clonidine affect the short term variation of CTG recordings?', *Australian and New Zealand Journal of Obstetrics and Gynaecology*, accepted for publication June 2010.

ANZJOG Proof

The Australian and New Zealand Journal of Obstetrics and Gynaecology

ANZJOG

Does the anti-hypertensive drug clonidine affect the short term variation of CTG recordings?

Journal:	*The Australian and New Zealand Journal of Obstetrics and Gynaecology*
Manuscript ID:	ANZJOG-2010-0061.R1
Manuscript Type:	Original Manuscript
Keywords:	cardiotocograph, hypertension, clonidine

Does the anti-hypertensive drug clonidine affect the short term variation of CTG recordings?

Short Title: Drug effect of clonidine on short term variation of CTGs

Word count: Abstract: 256, main 1391

Tables: two

Key words: Pre-Eclampsia; Fetal Monitoring; Cardiotocography; Hypertension; Anti-hypertensive Agents

Abstract

Background: Cardiotocographic (CTG) recordings of the fetal heart remain standard obstetric practice among hypertensive women. Changes in the short term variation (STV) of the fetal heart are often attributed to the effect of anti-hypertensive medications, regardless of the fact that this principle has never been validated.

Aim: To assess the STV of CTG recordings pre and post the anti-hypertensive medication clonidine.

Methods: Forty hypertensive pregnant women, already receiving the anti-hypertensive clonidine were recruited. CTGs were conducted pre and post dose administration. The CTGs were assessed by the Sonicaid Team® automated CTG analysis (Oxford Instruments, UK) to avoid CTG assessor bias. Baseline FHR (delta change from pre and post dose) and STV were compared using SPSS v.14® utilising student t-tests.

Results: No statistical difference was found in the pre and post baseline FHRs (p=0.48). The mean delta baseline heart rates before and after drug administration was -0.54 bpm. The STV of the CTGs recorded pre and post clonidine dose was also not affected by administration of the drug (p=0.34). The mean delta STV before and after drug administration was 0.39 ms. Two women received betamethasone 12 mg intramuscularly within the 12 hour period prior to CTG recordings to enhance fetal lung maturity. The mean STV for the fetuses of these women pre-drug was 4.8 ms and 13.2 ms post administration. This was the largest delta seen in all STVs recorded in this dataset.

Conclusion: The anti-hypertensive drug clonidine does not alter baseline FHRs or effect the STV of the fetal heart rate in hypertensive pregnant women.

Introduction

Cardiotocogram (CTG) recordings of the fetal heart remain standard obstetric practice for assessment of fetal well being, regardless of the doubtful utility of such practice in reducing neonatal mortality[1, 2]. CTG recordings are commonly made on the fetuses of women experiencing pregnancy complications and management decisions are often made upon the subjective analysis of fetal heart patterns. The short term variation (STV) of the fetal heart rate (FHR) pattern reflects the neurological status of the fetus and represents both the status of the sympathetic and parasympathetic nervous systems[3]. STV is affected by both extrinsic factors including maternal medical conditions (such as gestational diabetes and preeclampsia) and maternal drug use[4] and by intrinsic factors such as intra-uterine growth restriction[5]. The accuracy of the estimate of STV via visual assessment by clinicians is questionable[6-9] and the determination of STV can be optimised through the use of automated software assessment[5, 10].

Hypertension during pregnancy currently complicates around 10% of all pregnancies and between 25-50% of all hypertensive women will resultantly be medicated with anti-hypertensives[11]. The anti-hypertensive clonidine, is a first-line drug used in this setting. Clonidine is a centrally acting alpha-1-receptor agonist. It acts primarily by stimulating the alpha-2-adrenoreceptors at the depressor site of the vasomotor centres[12]. This results in a decrease of sympathetic outflow from the brain and an increase in baroreceptor activity. Additionally, clonidine stimulates peripheral alpha-1 adrenergic receptors, which

is reflected by a small transient pressor effect when given pareneterally. Clonidine acts relatively rapidly: the patient's blood pressure declines within 30 to 60 minutes after an oral dose, the maximum decrease occurring within 2 to 4 hours. The plasma level of clonidine hydrochloride peaks in approximately 1 to 2 hours and the plasma half-life ranges from 12 to 16 hours[13]. Studies conducted at Royal Prince Alfred Hospital, Sydney, and other institutions have shown that clonidine is safe to use in pregnancy[14,15] and in comparison with alphamethyldopa or placebo, has no detrimental effect on neonatal survival, Apgar scores, neonatal blood pressure or rebound hypertension[16],[17,18].

Previous work has addressed the effect of labetalol, hydralazine, isradipine, methyldopa, pindolol, propanalol, diazoxide and clonidine on CTG recordings[19,20], although no previous published work has been designed to specifically assess the effect of anti-hypertensives on the FHR and STV utilising automated analysis technology. The effect of maternal administration of steroids for fetal indications has been previously addressed with varying impacts of STV[4,21]. The use of automated analysis of CTG tracings, a method shown to accurately predict neonatal acidosis and remove assessor bias, has only been utilised in one previous trial[22]. .

Therefore, the aim of this before and after study was to determine if the oral anti-hypertensive agent clonidine had a measurable effect on FHR patterns and STV in the fetuses of women with gestational hypertension and preeclampsia.

Methods

Following local area institutional ethics approval and with full written consent, hypertensive women currently medicated on clonidine were recruited from the antenatal ward and from the day stay assessment unit. Hypertension was diagnosed in accordance with the Society of Obstetric Medicine of Australia and New Zealand (SOMANZ) (formerly Australasian Society for the Study of Hypertension in Pregnancy) Consensus Guidelines[23]. In summary – this study was limited to women of greater than 20 weeks gestation who had new onset hypertension with at least two readings of 140 mmHg systolic and/or 90 mmHg diastolic of blood pressure 2 hours apart. Women with chronic hypertension were excluded from the study. CTG recordings were conducted for a 60 minute period prior to drug administration. Following oral clonidine administration, a second CTG was recorded for a period of 90 minutes. The automated anaylsis facility of the Sonicaid Team® automated CTG analysis (Oxford Instruments, UK) was used to determine FHR and STV pre and post drug administration. The Sonicaid Team® automated CTG automatically notifies users after a 10 minute period of heart rate recording whether criteria has been met for the recording. For the purpose of this study – the results of this automated analysis were limited to the results available following the pre-stated periods (at the 60 minute point pre-dose and at the 90 minute point post-dose). Data was analysed with SPSS v.14® utilising student t-tests with statistical significance assumed for p values<0.05.

Results

Forty women were recruited, consented and took part in the study. Nineteen received a final diagnosis of preeclampsia and 21 had gestational hypertension with no evidence of end-organ damage. The age and gestation of the women are outlined in Table 1. There was no statistically significant difference in the mean baseline FHR pre clonidine administration was 129 bpm ± 9 (Range 115-152 bpm) and 128 ± 10 bpm (range 109 – 148 bpm) post administration. STV was also not statistically different pre and post clonidine (9.76 ± 3.0 ms (range 4.5-16.3 ms) and 10.25 ± 3.1 ms (range 4.6 – 17.6 ms) pre and post clonidine dose respectively) (Table 2). Only two women received intra-muscular steroids for the enhancement of fetal lung maturity in the 12 hour period prior to CTG recordings. The baseline FHR of these fetuses was not altered pre and post clonidine dose but the STV increased by 7.6 ms and 8.8 ms following clonidine administration. This finding was not able to be analysed for statistical purposes due to the small sample size but these were the largest differences seen between STVs recorded in this dataset.

Discussion

This study demonstrates that clonidine administered orally to hypertensive regnant women does not effect the fetal heart rate nor the short term variability of antenatal CTG assessments.

The options available to clinicians to assess fetal well being in the hypertensive disorders of pregnancy include umbilical and cerebral artery Doppler assessment, biophysical profiling and CTG tracings[24]. The most commonly

used for time efficiency and cost is the CTG, although CTGs are unreliable predictors of poor neonatal outcome with reported positive predictive values of 8.7% for babies requiring ventilation[1].

The altered CTG pattern found in recordings made on hypertensive women may be attributable to a number of factors; the inherent placental insufficiency related to new onset hypertension or the effect of anti-hypertensives which alter maternal haemodynamics and may be reflected in the fetal heart pattern as well as the common comorbidity of intrauterine growth restriction[25].

Inter-rater agreement on CTG recordings is poor. In normal recordings agreement kappa is 0.48, whilst in pathologic tracings kappa falls to 0.42[6] and agreement between assessors was found to be lowest when assessing the variability characteristic[9]. Automated analysis of this component of CTG recordings is an effective tool in removing assessor bias[5,10].

A previous systematic review conducted on this topic also concluded that anti-hypertensive medication does not alter fetal heart rate or patterns. Maternal antenatal corticosteroid administration has been associated with changes in fetal heart rate variation. Dawes *et al*[4] found that dexamethasone administration caused a rise in fetal heart rate variation. This finding was confirmed by Magee etal[19] who uncovered a similar effect from betamethasone. Whilst Mulder *et al*[26] saw a decrease in variability from betamethasone which was not affected by fetal age. Our study found increased STV in both fetuses of mothers exposed to betamethasone. These increases

were the largest of the cohort, and whilst they may be the result of a Type 1 error, they do reflect what has been found previously and it is an issue that requires further study.

An audit of anti-hypertensive medications used in four tertiary referral obstetric units in the Sydney region[11] showed that 45% of hypertensive women were medicated in the antenatal period. Anti-hypertensive choice remains largely an institution idiosyncratic choice. Clonidine is not as commonly used in obstetrics as methyldopa or oxprenolol, but on the basis of prior randomised clinical trials[16,17,18] it is the drug of choice at Royal Prince Alfred Women and Babies (Australia) with a current birth rate of >5000 deliveries, a hypertension rate of 10% and where 55% of hypertensive women are medicated[11].

The anti-hypertensive medication clonidine does not alter the STV of the fetal heart and changes in STV and other fetal heart rate patterns may be indicative of altered maternal or fetal haemodynamic stability and require further clinical investigation. Attributing altered fetal heart rate patterns to anti-hypertensives medication alone may be dangerous clinical practice and consideration should be given to other potential pathological changes which may be occurring to the fetus.

Acknowledgements

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Table 1 Details of women and treatment.

	Age (years)	Gestation (weeks)	Diagnosis	Dose of clonidine (μg)
All women	33 ± 5.5	35 ± 3	GH – 52% PE – 48%	114±41.6
			Booking BP	**Max BP**
GH	32.2 ± 1.4	36 ± 1.0	110 ± 2.3 / 68 ± 2.0	154 ± 3.5 / 99 ± 2.3
PE	33.8 ± 1.0	34.1 ± 0.8	115 ± 2.0 / 72 ± 2.0	155 ± 2.0 / 96 ± 2.0

GH - gestational hypertension
PE – preeclampsia

Table 2 Fetal heart rates (FHR) and short term variation (STV)

	Pre clonidine	Post clonidine	SS
FHR	129bpm	128bpm	0.48
STV	9.76ms	10.25ms	0.34

bpm – beats per minute, ss – statistical significance

Does the antihypertensive drug clonidine affect the short term variation of CTG recordings?

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