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Maternal deaths in Australia 2000–2002

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The Australian Institute of Health and Welfare is Australia's national health and welfare statistics and information agency. The Institute's mission is *better health and wellbeing for Australians through better health and welfare statistics and information*.

The AIHW National Perinatal Statistics Unit (NPSU) is a collaborating unit of the AIHW established in 1979. The NPSU aims to improve the health of Australian mothers and babies through the collection, analysis and reporting of information on reproductive, perinatal and maternal health. The NPSU maintains national data collections on perinatal health, maternal deaths, congenital anomalies and assisted reproduction technology. NPSU is located at Sydney's Children Hospital. NPSU is situated within the School of Women's and Children's Health, Faculty of Medicine, The University of New South Wales.

Please note that as with all statistical reports there is the potential for minor revisions of the data presented in the *Maternal deaths in Australia 2000–2002* report. Please refer to the online version at <www.npsu.unsw.edu.au>.

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Preface

This thirteenth triennial report on maternal deaths in Australia presents a summary of maternal deaths in Australia for the period 2000–2002. Improved general health status and reproductive patterns, together with access to appropriate general and specialised health care, have contributed to a progressive reduction in the incidence of maternal mortality in Australia. Despite improvements in the maternal mortality ratio, since reporting commenced in 1964, it is important to note that pregnancy is associated with death for approximately 30 women per year, often unpredictably. Regrettably, the disparity in overall health outcomes experienced by Aboriginal and Torres Strait Islander Australians compared to other Australians is also seen in high rates of maternal mortality.

Maternal mortality is often used as a measure of a country's overall health and development status. Reducing maternal mortality is a key goal of the United Nations Millennium Project to which Australia is a signatory. To be able to measure Australian progress towards this goal we must be able to measure, monitor and report accurately on maternal mortality. The Australian Council for Safety and Quality in Health Care (ACSQ) which has been succeeded by the Australian Commission on Safety and Quality in Health Care (ACSQHC), includes maternal deaths in hospital as one of its key sentinel events. This is the second report the ACSQ has funded, demonstrating a commitment to monitoring the safety and quality of maternity care in Australia. In addition, the ACSQ and now the ACSQHC is supporting Australia's first national workshop on maternal deaths, which will occur in 2006. This commitment will ensure ongoing improvements in measuring, monitoring and reporting on maternity care in Australia.

This report follows a framework agreed to by the AIHW National Advisory Committee on Maternal Mortality (NACMM), and differs from previous publications by presenting deaths by cause of death, rather than classification of death. This is part of a program of quality improvement for monitoring of maternal deaths in Australia.

Seeking to avoid loss of women's lives in childbearing, and minimising damage to their health, remain issues of importance for maternity service providers in Australia. It is hoped that this report will support improved practice in maternity care by providing information to practitioners to improve the quality and safety of health care during pregnancy and the puerperium.

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The current chair of the National Advisory Committee on Maternal Mortality (NACMM) is Associate Professor James King. We would like to gratefully acknowledge his valuable contribution to the report as editor, clinician and epidemiologist.

This report is the result of hard work, generosity of time and commitment by the members of the national committee. The publication has been peer reviewed by the expert members of the national committee and invited experts. Their critical and constructive comments added to the quality and authority to this publication and their valuable contribution is gratefully acknowledged.

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We would also like to thank the following three academics who were invited to provide input on relevant chapters in which they have expertise:

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This report was a team effort with a number of people from the National Perinatal Statistics Unit (NPSU) involved with development of the database, data entry and preparation of the report. We would like acknowledge the major contribution by Sue Kildea who coordinated the preparation of the report during 2005, Emma Slaytor and Eydis Konráðsdóttir for preparation of the data and background information, Jishan Dean for developing the maternal deaths database and data extraction and programs, Paula Laws for provision of perinatal data, Chun Chen for formatting the report and Narelle Grayson for reviewing the report.

The AIHW National Perinatal Statistics Unit (NPSU) is a formally affiliated institution of the University of New South Wales (UNSW) and is linked to the School of Women's and Children's Health. We would like to acknowledge the support of the NPSU by the School of Women's and Children's Health, UNSW and the Sydney Children's Hospital.

We would also like to acknowledge financial support from the former Australian Council for Safety and Quality in Health Care (ACSQ). The former Council has been succeeded by the Australian Commission on Safety and Quality in Health Care (ACSQHC). Information about the ACSQHC can be found at their website: www.safetyandquality.gov.au .

Abbreviations

ABS	Australian Bureau of Statistics
ACAM	Australian Centre for Asthma Monitoring
ACSQ	Australian Council for Safety and Quality in Health Care
ACSQHC	Australian Commission on Safety and Quality in Health Care
ACT	Australian Capital Territory
AFE	Amniotic fluid embolism
AIHW	Australian Institute of Health and Welfare
ANZCA	Australian and New Zealand College of Anaesthetists
APH	Antepartum haemorrhage
ARIA	Accessibility/Remoteness Index of Australia
ASA	American Society of Anesthesiologists
ASTB	Australian Safety Transport Bureau
BMI	Body mass index
BP	Blood pressure
CEMACH	Confidential Enquiries into Maternal and Child Health
C/S	Caesarean section
CT Scan	Computed tomography scan
DIC	Disseminated intravascular coagulopathy
DVT	Deep vein thrombosis
EDS	Edinburgh depression scale
EPDS	Edinburgh postnatal depression scale
g	Gram
GM	Grand multipara
GP	General practitioner
HELLP	Haemolysis, elevated liver enzymes and low platelet count
kg	Kilogram
ICD–9	International Classification of Diseases, ninth revision
ICD–10	International Statistical Classification of Diseases and Related Health Problems, tenth revision
L	Litre
M	Multipara
MDG	Millenium Development Goal
mmHg	Millimetre of mercury
mg	Milligram
MMR	Maternal mortality ratio

MMRWG	Maternal Mortality Review Working Group
MSU	Mid-stream urine
N	Nullipara
NACMM	AIHW National Advisory Committee on Maternal Mortality
NCIS	National Coroners Information System
NHMD	AIHW National Hospital Morbidity Database
NPSU	AIHW National Perinatal Statistics Unit
NSW	New South Wales
NT	Northern Territory
P	Primipara
PTE	Pulmonary thromboembolism
PPH	Postpartum haemorrhage
Qld	Queensland
RANZCOG	Royal Australian and New Zealand College of Obstetricians and Gynaecologists
RANZCP	Royal Australian and New Zealand College of Psychiatrists
RCOG	Royal College of Obstetricians and Gynaecologists
RR	Respiratory rate
RWH	Royal Women's Hospital
SA	South Australia
STMMC	State and Territory Maternal Mortality Committees
Tas	Tasmania
TTP	Thrombotic thrombocytopenic purpura
UK	United Kingdom
UNSW	University of New South Wales
USA	United States of America
Vic	Victoria
WA	Western Australia
WHO	World Health Organization

Summary: Maternal deaths in Australia 2000–2002

This is the thirteenth triennial report on maternal deaths in Australia, presenting a summary of maternal deaths in Australia for the period 2000–2002. Death from pregnancy is rare in Australia compared to many countries in the region and the world. There were 95 maternal deaths reported to the AIHW National Perinatal Statistics Unit, of these 84 deaths were directly or indirectly related to or aggravated by the pregnancy or its management and occurred while the woman was pregnant or within 42 days of the termination of the pregnancy. Another three maternal deaths, known as incidental deaths were from accidental or incidental causes not related to the pregnancy or its management; and eight were late maternal deaths which occurred between 43 and 365 days post termination of pregnancy from causes related to or aggravated by the pregnancy or its management (Figure 1).

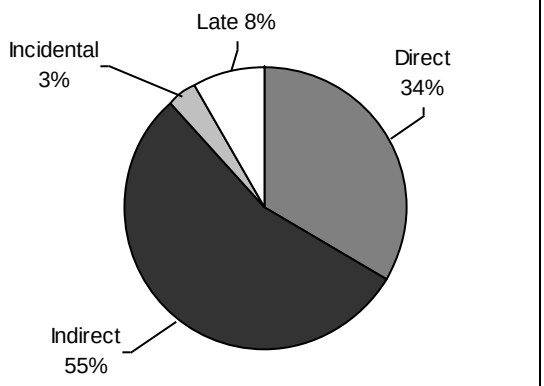

Figure 1: Relative proportion of maternal deaths by type of death, Australia, 2000–2002

The figures presented in this report are not entirely comparable to previous reports because of continued improvements in ascertainment of maternal deaths and changes in the classification of deaths for some causes. This is most evident in the change in the distribution of indirect and incidental maternal deaths over the last two triennia (Table 1).

Table 1: Maternal deaths by type of death, Australia, 1997–2002

Type of death	1997–1999	2000–2002
Direct cause	34	32
Indirect cause	30	52
Incidental	28	3
Late	5	8
Total	**97**	**95**

The maternal mortality ratio (MMR) is the number of (direct and indirect) maternal deaths over the number of women who gave birth during the same time period. For the period 2000–2002 the MMR was 11.1 deaths per 100,000 women who gave birth. This is the highest recorded since the 1979–1981 triennium. However, there has been a change in classification of deaths from incidental to indirect for some causes in recent triennia. Also, there is fluctuation in the number of deaths from some causes over time due to small number variations and the sporadic nature of deaths. There has also been enhanced reporting of maternal deaths due to the inclusion of some conditions because of advances in the knowledge of their aetiology in relation to pregnancy. The age-specific MMR peaked at 32.8 per 100,000 women who gave birth for women aged 40–50 years and lowest for women aged 20–24 years (4.3 per 100,000 women who gave birth).

The major causes of direct and indirect maternal deaths were amniotic fluid embolism (AFE), haemorrhage from obstetric and other causes and cardiac disease and infection (Figure 2). The number of direct maternal deaths due to AFE increased from seven deaths in the 1997–1999 triennium to 10 in the 2000–2002 triennium. The number of indirect maternal deaths due to cardiac disease increased from seven deaths in the 1997–1999 triennium to 11 in the 2000–2002 triennium while those due to infection doubled from five deaths in the 1997–1999 triennium to 10 deaths in the 2000–2002 triennium.

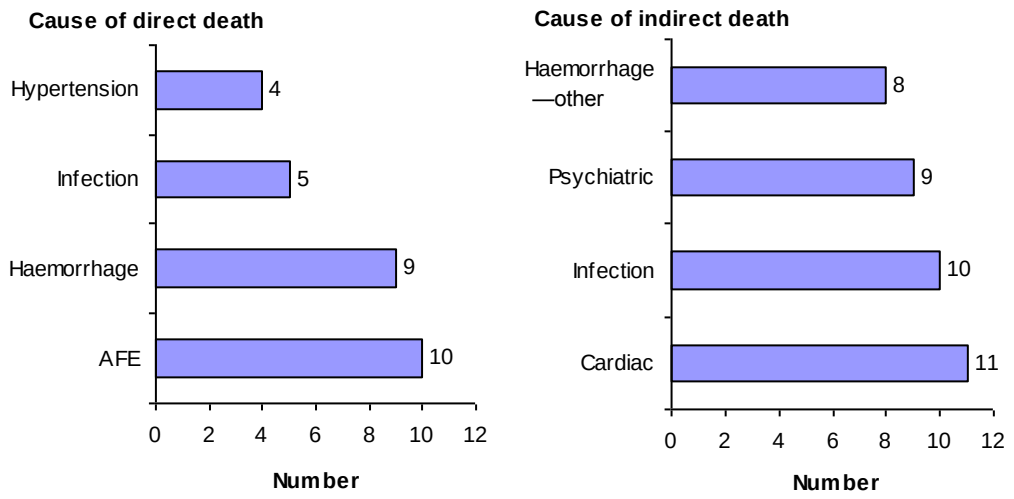

Figure 2: Leading causes of direct and indirect maternal deaths, Australia, 2000–2002

Mortality rates of Aboriginal or Torres Strait Islander women remained unacceptably high and were 4.5 times as high as for other women (MMR 34.8 deaths per 100,000 Indigenous women who gave birth versus 7.7 deaths per 100,000 other women who gave birth) when reported over the six year period 1997–2002. The very high mortality rates have been identified in previous reports. The 13 deaths identified in the 2000–2002 triennium were made up of one incidental and 12 direct and indirect deaths. This equalled one direct or indirect maternal death per 2,177 Aboriginal or Torres Strait Islander women who gave birth.

The 2000–2002 triennium has seen a decline in maternal mortality due to hypertensive disorders, thrombosis and thromboembolism and motor vehicle accidents compared to the 1997–1999 triennium. Deaths from motor vehicle accidents decreased from six in the previous triennium to one during the 2000–2002 triennium.

Section A: Introduction

Background to the report

This is the thirteenth triennial report on maternal deaths in Australia, presenting a summary of maternal deaths in Australia for the period 2000–2002. Maternal deaths continue to be rare in Australia with 95 included in this report. A steep decline in maternal deaths from 275 to 106 was reported for Australia between the first triennial *Report on maternal deaths in Australia 1964–66* and the fourth twelve years later for the 1976–1978 triennium. Since then the total number of deaths have remained stable ranging from 84 to 100 for each three year period. Information on Indigenous status was first collected for the 1970–1972 report; however, ascertainment remains incomplete.

This is the second report to be produced under the auspices of the Australian Institute of Health and Welfare and the AIHW National Advisory Committee on Maternal Mortality (NACMM). Reports prior to the 1997–1999 triennium were undertaken by the National Health and Medical Research Council (NHMRC). This is the second report in the AIHW maternal death series. The AIHW NACMM includes members nominated by the Royal Australian and New Zealand Colleges of Obstetricians and Gynaecologists (RANZCOG) and Psychiatrists, Australian College of Midwives, Australian and New Zealand College of Anaesthetists (ANZCA), Royal College of Pathologists of Australasia, all State and Territory Mortality Committees (STMMC), former Australian Council for Safety and Quality in Health Care and AIHW National Perinatal Statistics Units as well as other key technical and consumer representation as detailed in Appendix 1.

The report was funded by the former Australian Council for Safety and Quality in Health Care (ACSQ) who funded with the NHMRC the 1997–1999 report. The former Council has been succeeded by the Australian Commission on Safety and Quality in Health Care (ACSQHC). This funding enables national reporting of maternal deaths as part of the framework for monitoring the safety and quality of maternity care in Australia. The importance of this is reflected in the continuing disparity in maternal death rates between Indigenous and non-Indigenous Australians that has been evident since reporting commenced. It also reinforces the relevance of the Millennium Declaration which was adopted during the United Nations Millennium Summit in September 2000. The fifth Millennium Development Goal (MDG) is to improve maternal health by reducing maternal mortality by 75% by 2015. There is an ongoing need to identify factors in Australia that are preventable or ameliorable to change in an effort to reduce maternal mortality.

In Australia, maternal deaths are notified to multidisciplinary STMMC (Appendix 3) by medical practitioners and midwives, hospitals, health departments, coronial and post mortem investigations, perinatal and hospital morbidity collections and from the Registrar of Births, Deaths and Marriages. Individual committees are responsible for conducting confidential death enquiries that assign each death a principal cause and classification; and identify avoidable factors. The information reviewed includes hospital admissions, autopsy, toxicology, police and coroners' reports, and ancillary information.

The AIHW NACMM receives information on maternal deaths from the STMMC. These data are validated against mortality and morbidity data. A standard set of information is requested by AIHW NPSU from the STMMC that is based upon the National Maternal Death

Reporting Form. A subcommittee of the NACMM, the Maternal Mortality Review Working Group (MMRWG) (Appendix 2) review all maternal deaths for national consistency; allocating principal and contributory causes of death and the classification of death. The AIHW NPSU compiles this information into a national report with expert input from the AIHW NACMM.

Aims of this report

The aims of this report are to:

- collate maternal mortality data for the period 1 January 2000 to 31 December 2002 from the STMMC
- maximise ascertainment and validity of maternal death reporting in Australia
- provide an epidemiological overview of maternal deaths in Australia
- provide statistical information to assist practitioners to
  - reduce maternal mortality
  - counsel women who are contemplating pregnancy
- reference relevant evidence-based maternity care guidelines.

Methodology

The methodology of this report has followed a similar pattern to previous reports. It contains epidemiological data on maternal deaths, the use of illustrative vignettes, clinical commentary and referral to published guidelines for further education on specific clinical management where available and relevant. The use and selection of illustrative cases or vignettes has changed over the last two reports in terms of number, specificity and repetition. The number of vignettes in most cases has been reduced to several per major cause of death, vignettes often composite information from several deaths and are reported once in the report. This allows vignettes to be selected to both illustrate the circumstances of the women's death and to be used as a tool for quality improvement of medical and midwifery practice whilst maximising confidentiality. Further details on the method used in this report are included in Section E.

The United Kingdom model of Confidential Enquiries into Maternal and Child Health (CEMACH) is internationally considered the gold standard in professional self-audit and maternal death reporting. The Australian Maternal Deaths Series is largely based upon the UK *Why mothers die* series of reports. However, there are some critical differences in the methods used by CEMACH and the NACMM which limit the depth of the enquiry of the Australian report and thus comparability to the UK report. The UK model is driven by both a history of 'professional self evaluation' by the Royal Colleges of Obstetricians and Gynaecologists (RCOG) and Midwives; and a government requirement that all maternal deaths should be subject to Confidential Enquiry (CEMACH for the 2000–2002 triennium) in which all health professionals are required to participate. This process is underpinned by the use of a comprehensive standard enquiry form which is completed by all professionals involved in the care of the deceased women and is then assessed by sets of honorary regional assessors. Each set of regional assessors includes obstetric, anaesthetic, pathology and midwifery assessors who review additional case materials and are charged with commenting

on the case and evaluating the clinical management and resources of the organisation responsible for the care of the deceased (Lewis G (ed.) 2004). This is then reviewed at a central level and written up into the triennial report. The method of enquiry is described in detail in the *Why mothers die. The sixth report on Confidential Enquiries into Maternal Deaths in the United Kingdom 2000–2002. The Confidential Enquiries into Maternal and Child Health* (CEMACH) (Lewis G (ed.) 2004).

The Australian report like the UK report is an observational study of maternal health. The value of this type of study has been questioned in the past, however the capacity 'to identify patterns of practice, service provision and public health issues that maybe causally related to maternal deaths' through a form of sentinel event reporting is critical to our understanding of these rare and catastrophic events (Lewis G (ed.) 2004). Often safety issues concerning rare events are best addressed through observational study. The analogy of the investigation of a maternal death to that of an aeroplane crash made in the *Why mothers die. The sixth report on Confidential Enquiries into Maternal Deaths in the United Kingdom 2000–2002* remains apt with the investigation of a maternal death revealing 'crucial information on the scientific, medical social and personal factors that could lead to better health' and responses to prevent further untimely deaths (Lewis G (ed.) 2004). This type of reporting does not preclude the use of randomised control trials or systematic reviews produced by the Cochrane Pregnancy and Childbirth Group, but acknowledges that not all the problems identified in maternal death reports 'including prevention of thromboembolic disease and treatment of amniotic fluid embolism' can be addressed by randomised trials. Many of the causes of maternal deaths are very rare and may never be prevalent enough to be subject to formal scientific study. However, other rare causes of severe morbidity and death with prevalence of less than 1 in 2000 births maybe be better addressed by the establishment of a rare obstetric event surveillance system. Such a system was established in the UK in 2005 and is known as United Kingdom Obstetric Surveillance System (UKOSS). CEMACH goes on further to state in *Why mothers die. T he sixth report on Confidential Enquiries into Maternal Deaths in the United Kingdom 2000–2002* that 'inevitably, recommendations for care to avoid such deaths in the future rely on lesser levels of evidence, and frequently 'expert opinion' (Lewis G (ed.) 2004). This does not mean that the report is not evidence-based, merely that, necessarily, the evidence cannot be in the form of a randomised control trial or case-control study, owing to the relative rarity of the condition' (Lewis G (ed.) 2004).

Structure of this report

The NACMM has made two significant changes to the presentation of the report. Maternal deaths are presented in one of the four summary chapters on direct, indirect, incidental and late maternal deaths (Section B); and are reported by cause of death rather than classification of death (Section C). Section D has also been modified in response to NACMM recommendation for the 2000–2002 report to not include chapters on the *Epidemiology of maternal deaths in Australia*, *trends in reproductive health and maternity care in Australia* and *International comparisons* as the information would not have significantly changed since publication of the 1997–1999 report.

Australian definitions of maternal mortality

Maternal death is defined in the tenth revision of the International Statistical Classification of Diseases and Related Health Problems (ICD–10) as:

> *The death of a woman while pregnant or within 42 days of the termination of pregnancy, irrespective of the duration and the site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes (WHO 1992).*

This definition includes deaths of women from pregnancy, terminations of pregnancy, miscarriage and ectopic pregnancy, but excludes deaths associated with assisted reproduction technologies where pregnancy has not occurred.

In line with international conventions, the maternal mortality ratio is calculated using direct and indirect deaths combined, but excluding incidental and late maternal deaths (See: Box 1). In the 1997–1999 report, the Australian maternal mortality ratio was calculated for the first time in accordance with the World Health Organization (WHO) recommendations (i.e. excluding incidental deaths). In all Australian reports on maternal deaths prior to 1997–1999, incidental deaths were included in the definition and calculation of MMR. Caution must therefore be taken when comparing MMR from triennia prior to 1997–1999 with MMR from the 1997–1999 triennia onwards. In the 2000–2002 report the MMRs from previous triennia have been recalculated to reflect this change in the standard of reporting.

In line with current international trends and in recognition of the fact that modern life-sustaining procedures and technologies can delay death, ICD–10 introduced the late maternal death category. A late maternal death is defined as:

> *The death of a woman from direct or indirect obstetric causes more than 42 days but less than one year after termination of pregnancy (WHO 1992).*

The definition of a late maternal death does not include deaths from incidental causes.

Late maternal deaths are particularly relevant to developed countries such as Australia where advances in clinical care and medical technology mean that women who have suffered a severe morbid event during pregnancy or childbirth may not die within the 42-day period. The inclusion of the late maternal death category allows these deaths to be reported. Death related to psychiatric illness are often reported and included in this category.

Reporting of late maternal deaths in Australia has been variably adopted by states and territories since 2000, with phased-in national reporting occurring over this period. The purpose of including late maternal deaths in the report is to enhance the monitoring and awareness of these deaths. It is probable that late maternal deaths are under-reported in Australia. The NACMM requests information on all late maternal deaths, including those that are classified as incidental deaths. The request for information on late deaths currently classified as incidental is to allow for the potential to change the classification in the future.

Classification of maternal deaths in Australia

Four maternal death categories are used in Australia and are based on ICD–10 definitions (WHO 1992). They are presented in Box 1.

Box 1: Classification of maternal deaths

Direct deaths

Result from obstetric complications of the pregnant state (pregnancy, labour and puerperium), from interventions, omissions, incorrect treatment or from a chain of events resulting from any of the above.

e.g. eclampsia, amniotic fluid embolism, rupture of the uterus, postpartum haemorrhage

Indirect deaths

Result from pre-existing disease or disease that developed during pregnancy and which was not due to direct obstetric causes, but which was aggravated by the physiological effects of pregnancy.

e.g. heart disease, diabetes, renal disease

Incidental deaths

Result from conditions occurring during pregnancy, where the pregnancy is unlikely to have contributed significantly to the death, although it is sometimes possible to postulate a distant association.

e.g. road accidents, some malignancies

Late maternal death

Death of a woman from direct or indirect obstetric causes more than 42 days but less than one year after termination of pregnancy.

In practice it is often difficult for expert committees to assign a maternal death category. The NACMM maternal mortality working group reviews every maternal death, classifies and assigns principal and or contributing causes of death using the information and advice provided by the STMMC. The NACMM has followed the CEMACH practice of being inclusive in the classification of indirect deaths. For example, death from an asthma attack in some instances may have been due to some effect of the pregnant state of the woman, but in others, it may be unrelated and thus may be classified as an indirect death in the former example, and an incidental death in the latter. Other difficult areas include psychiatric-related deaths, where a self-administered overdose could be unintentional or intentional suicide, and may or may not have been due to some effect of the pregnant state of the woman. Deaths from cancers and tumours can also be difficult to classify, as some can be affected by pregnancy and others are thought to be unaffected by pregnancy. This inclusiveness should be taken into account when making international comparisons aside from the United Kingdom.

Measuring maternal mortality

Maternal mortality is an internationally accepted measure of maternal health. There are three measures of maternal mortality in widespread use: the maternal mortality ratio, the maternal mortality rate, and the lifetime risk of maternal death. All measures need to be defined to enable meaningful comparison between countries. The following measures have been described in a WHO report:

> *The most commonly used measure is the maternal mortality ratio, that is the number of [direct and indirect] maternal deaths during a given period per 100,000 live births during the same period. This is a measure of the risk of death once a woman has become pregnant.*
>
> *The maternal mortality rate, that is, the number of [direct and indirect] maternal deaths in a given period per 100,000 women of reproductive age during the same period, reflects the frequency with which women are exposed to risk through fertility.*
>
> *The lifetime risk of maternal death takes into account both the probability of becoming pregnant and the probability of dying as a result of that pregnancy cumulated across a woman's reproductive years. In theory, the lifetime risk is a cohort measure but it is usually calculated with period measures for practical reasons. It can be approximated by multiplying the maternal mortality rate by the length of the reproductive period (around 35 years) (WHO et al. 2000 p. 5).*

In the 2000–2002 triennium report the maternal mortality ratio has been used to measure and describe maternal deaths.

Numerator

The numerator used in this report is the total number of direct and indirect maternal deaths that occurred in the triennium, noting that the Australian definition of indirect maternal deaths is more inclusive than the definition used in some countries. Deaths due to psychiatric causes have been classified as indirect rather than incidental from 1997 onwards. Deaths due to some cancers and tumours have been classified as indirect rather than incidental, from 2000 onwards. Both these changes are in line with changes occurring in the United Kingdom. The Royal College of Obstetricians and Gynaecologists have a study group on cancer in pregnancy which concluded that certain cancers are aggravated by pregnancy (Lewis G (ed.) 2004). NACMM has followed the RCOG recommendations in this area.

Denominator

The denominator used for calculating maternal mortality measures differs across the world depending on the data available. The most appropriate denominator for estimating maternal mortality rates is the number of women at risk, that is, the number of pregnant or recently pregnant women. However, this figure is not accurately known, the unknown component being the number of pregnancies ending before 20 weeks gestation.

The WHO definition specifies that the number of live births or the number of total births (live births plus fetal deaths) can be used as the denominator (WHO 1992). Where both denominators are available the WHO report suggests that both calculations be made.

In Australia, accurate population data are available on the total number of women who gave birth resulting in at least one birth (including live births and stillbirths) of either 20 or more completed weeks of gestation or 400 grams or more birthweight. The formula for calculating the MMR in Australia is:

Numerator: Number of direct and indirect maternal deaths x 100,000

Denominator: Total number of women who gave birth

The MMR in Australia is usually calculated for a three year period or a triennium, which is the term used in this report. Table 2 shows the denominator data used in this report. This includes the total number of women who gave birth to either a live birth or stillbirth of either 20 or more completed weeks of gestation or 400 grams or more birthweight during the 2000–2002 triennium.

Table 2: Number of women who gave birth by maternal age, Australia, 2000–2002

Age group (years)	2000	2001	2002	Total	Per cent
<20	12,647	12,441	12,227	37,315	4.9
20–24	39,301	38,720	38,055	116,076	15.4
25–29	79,295	74,881	71,791	225,967	30.0
30–34	78,585	80,263	83,225	242,073	32.1
35–39	36,572	36,517	37,831	110,920	14.7
≥ 40	6,614	7,153	7,551	21,318	2.8
Not stated	58	96	78	232	0.0
Total	**253,072**	**250,071**	**250,758**	**753,901**	**100.0**

Source: AIHW National Perinatal Statistics Unit, National Perinatal Data Collection.

This is similar to the measure used by the CEMACH in the UK where the denominator is described as 'maternities: the number of pregnancies that result in a live birth at any gestation or stillbirths occurring at or after 24 weeks of completed gestation' (Lewis G (ed.) 2004).

Maternal mortality rates

In Australia, mortality rates are calculated by the Australian Bureau of Statistics using death certificate data from the Registrars of Births, Deaths and Marriages. For this report maternal mortality is calculated through deaths notified to the NACMM. The overall maternal death rate is based upon reported direct and indirect maternal deaths.

Erratum-*Maternal deaths in Australia 2000-2002*

Section B: Epidemiology of maternal deaths

Maternal deaths by state and territory

The information provided on Page 13 about why data for Western Australia were not published in Tables 10, 11 and 12 is incorrect. It states that Western Australia only provided permission for data to be published over two triennia, 1997-2002 (i.e. in Table 12). Therefore, the data for Western Australia were suppressed in Tables 10 and 11. The data were also suppressed in Table 12 to maintain confidentiality of the Australian Capital Territory data, since the Australian Capital Territory did not provide permission to publish data in Tables 10, 11 and 12.

However, Western Australia did provide permission to publish data in Tables 10, 11 and 12. Therefore, the statement on page 13 should read: 'The Australian Capital Territory (ACT) did not give permission to publish data in Tables 10, 11 and 12 because of the small number of deaths and concerns about reporting based on place of death rather than place of usual residence. Western Australia (WA) gave permission to publish data in these tables. However, to maintain the confidentiality of the ACT data, the data for WA have been suppressed'.

Section B: Epidemiology of maternal deaths

There were 95 maternal deaths in the triennium 2000–2002 which met the inclusion criteria defined in Box 1. Eighty-four of these deaths were either direct or indirect (Table 3).

Table 3: Maternal deaths by type of death, Australia, 2000–2002

Type of death	Number
Direct	32
Indirect	52
Total	**84**

In addition, there were three incidental deaths and eight late maternal deaths (two direct and six indirect). An additional four late incidental deaths were reported to the committee which did not satisfy the inclusion criteria of the ICD–10 definition (WHO 1992) and are excluded from this report.

Maternal mortality ratio

The MMR was 11.1 deaths per 100,000 women who gave birth (Table 4). This was one maternal death per 8,975 women who gave birth.

Table 4: Maternal mortality ratio, Australia, 2000–2002

Type of death	Number of deaths	Maternal mortality ratio[(a)]
Direct and Indirect	84	11.1

(a) Per 100,000 women who gave birth.

The MMR has risen from 8.4 per 100,000 women who gave birth in the 1997–1999 triennium to 11.1 per 100,000 women who gave birth in the 2000–2002 triennium. The increase in MMR is a result of an increase in the number of maternal deaths classified as indirect deaths during the triennium (Table 5). This increase in indirect deaths can be partly explained by changes in the classification of some conditions and improved reporting; however an actual rise in indirect deaths cannot be ruled out. Since 1973–1975 direct maternal deaths have almost halved from 60 to 32.

Table 5: Maternal mortality ratios, Australia, 1973–2002

Triennium	Direct deaths	Indirect deaths	Number of women who gave birth	Maternal mortality ratio[(a)]
1973–1975	60	32	726,690	12.7
1976–1978	52	35	678,098	12.8
1979–1981	54	34	682,880	12.9
1982–1984	42	25	713,985	9.4
1985–1987	32	30	726,642	8.5
1988–1990	37	33	754,468	9.3
1991–1993	27	22	769,253	6.2
1994–1996	46	20	767,448	9.1
1997–1999	34	[(b)]30	758,030	8.4
2000–2002	32	52	753,901	11.1

(a) Per 100,000 women who gave birth.

(b) Data updated to include two indirect deaths in the 1997–1999 triennium that were notified or amended after the 1997–1999 report was published.

Maternal mortality ratio for use in international comparisons

The World Health Organization (WHO) defines the MMR as the number of direct and indirect maternal deaths during a given time period per 100,000 live births. The WHO definition uses live births as the denominator and differs from the denominator used in Australia which is the number of women who gave birth during a given time period. For the purposes of international comparison the MMR is calculated in this section using the WHO criteria for the denominator. The number of live births in Australia during the period 2000–2002 was 761,390. The MMR for Australia for the period 2000–2002 is 11.0 deaths per 100,000 live births. This compares regionally to an estimated MMR for 2000 of 240 deaths per 100,000 live births for Oceania. The maternal mortality ratio for the world in 2000 was estimated at 400 deaths per 100,000 live births (WHO 2004b). The MMR of the developed regions (including Australia, New Zealand, USA, Europe, Japan, and Canada) in 2000 was an estimated 20 deaths per 100,000 live births compared to an estimated 440 deaths per 100,000 live births in the developing regions (WHO 2004b).

Maternal mortality ratios 1973–1975 to 2000–2002

Table 6 presents trends in direct and indirect maternal mortality ratios between 1973–1975 and 2000–2002. The ratio of death per 100,000 women who gave birth has also been reported for incidental and late maternal deaths. Though several fluctuations have occurred, the direct maternal death ratio has almost halved over the reporting period. The indirect maternal death ratio has increased over the last two triennia as the incidental death ratio has decreased.

The maternal mortality ratios for direct and indirect deaths in 2000–2002 were 4.2 and 6.9 per 100,000 women who gave birth, respectively (Table 6).

Table 6: Maternal mortality ratio per 100,000 women for direct and indirect deaths, and ratio per 100,000 women who gave birth for incidental and late maternal deaths, Australia, 1973–2002

Triennium	Direct maternal mortality ratio[(a)]	Indirect maternal mortality ratio[(a)]	Incidental death ratio[(a)]	Late death ratio[(a)]
1973–1975	8.3	4.4	6.2	—
1976–1978	7.7	5.2	2.8	—
1979–1981	7.9	5.0	1.5	—
1982–1984	5.9	3.5	3.8	—
1985–1987	4.4	4.1	3.3	—
1988–1990	4.9	4.4	3.4	—
1991–1993	3.5	2.9	4.7	—
1994–1996	6.0	2.6	4.4	0.3
1997–1999	4.5	[(b)]4.0	3.7	0.7
2000–2002	4.2	6.9	0.4	1.1

(a) Per 100,000 women who gave birth.

(b) Data updated to include two indirect deaths in the 1997–1999 triennium that were notified or amended after the 1997–1999 report was published.

Age-specific and age-standardised mortality rates

Age-specific mortality rates are calculated to present a more accurate summary measure of maternal mortality. Maternal mortality varies by age and it is important to relate the deaths at each age group to the number of women in the population at that age. This has particular relevance in Australia where maternal age has increased over time and there has been a decline in maternal mortality. The term 'specific' is used to indicate that these rates are specified according to age. The methods for the calculation are detailed in Appendix 6. Fluctuation in the age-specific rates is in part due to the effect of the small numbers. Age-specific mortality rates have been calculated separately for direct deaths, indirect deaths and combined direct and indirect deaths (Tables 7–9).

Age-standardisation is a measure used to control for variation in the age distribution of different populations (Appendix 6). When looking at maternal mortality and how the rate has changed since 1973–1975 it is important to control for the changes in the distribution of the ages of women who are giving birth. For example, in 1973–1975 women aged 20–24 years made up 19.7% of the population compared with 15.2% in 2000–2002. Controlling for this difference in age distribution allows appropriate comparisons to be made between triennia. The age-standardised rate for direct maternal deaths has decreased from 0.74 to 0.25 deaths per 100,000 female population over the 30-year period from 1973–1975 to 2000–2002. The decrease in the age-standardised rate reflects improvements in maternity care and maternal outcome of the period rather than the changing ages of the population of women who are giving birth.

Table 7: Age-specific and age-standardised maternal mortality rates: direct maternal deaths, Australia, 1973–2002

Triennium	Direct deaths	15–19 years	20–24 years	25–29 years	30–34 years	35–39 years	40–44 years	Age-standardised rate[(a)]
1973–1975	66	0.25	1.10	1.14	0.42	0.57	1.00	0.74
1976–1978	52	0.22	0.58	0.70	0.80	0.41	0.73	0.58
1979–1981	54	0.17	0.80	0.60	0.74	0.39	0.54	0.54
1982–1984	42	0.11	0.75	0.62	0.23	0.45	0.08	0.37
1985–1987	32	0.20	0.25	0.40	0.52	0.27	0.00	0.28
1988–1990	37	0.05	0.35	0.52	0.59	0.26	0.06	0.31
1991–1993	27	0.00	0.09	0.44	0.28	0.34	0.16	0.22
1994–1996	46	0.11	0.33	0.48	0.69	0.47	0.10	0.37
1997–1999	34	0.00	0.20	0.27	0.37	0.58	0.14	0.27
2000–2002	32	0.10	0.05	0.38	0.45	0.36	[(b)]0.13	0.25

(a) Directly age-standardised to the Australian female population aged 15 to 44 years at 30 June 2001.

(b) Includes 1 woman aged over 44 years.

Note: Rates expressed per 100,000 female population.

For indirect deaths, the age-standardised rate decreased from 0.35 to 0.16 deaths per 100,000 female population aged 15-44 years over the 23-year period 1973–1975 to 1994–1996 (Table 8). The age-standardised rate has risen since 1994–1996 to 0.41 deaths per 100,000 female population in the 2000–2002 triennium. This is in part due to changes in classification of some conditions from incidental deaths to indirect deaths.

Table 8: Age-specific and age-standardised maternal mortality rates: indirect maternal deaths, Australia, 1973–2002

Triennium	Indirect deaths	15–19 years	20–24 years	25–29 years	30–34 years	35–39 years	40–44 years	Age-standardised rate[(a)]
1973–1975	30	0.06	0.42	0.57	0.31	0.53	0.19	0.35
1976–1978	35	0.11	0.64	0.53	0.27	0.25	0.46	0.37
1979–1981	34	0.17	0.39	0.65	0.36	0.30	0.09	0.33
1982–1984	25	0.11	0.27	0.34	0.29	0.32	0.00	0.22
1985–1987	30	0.30	0.20	0.75	0.16	0.11	0.00	0.25
1988–1990	33	0.05	0.20	0.52	0.54	0.26	0.06	0.27
1991–1993	21	0.15	0.09	0.24	0.32	0.15	0.05	0.17
1994–1996	20	0.00	0.10	0.19	0.55	0.09	0.00	0.16
1997–1999	[(b)]28	0.16	0.05	0.41	0.37	0.27	0.05	0.22
2000–2002	52	0.15	0.26	0.75	0.90	0.22	0.13	0.41

(a) Directly age-standardised to the Australian female population aged 15 to 44 years at 30 June 2001.

(b) Data do not include two indirect deaths in the 1997–1999 triennium that were notified or amended after the 1997–1999 report was published.

Note: Rates expressed per 100,000 female population.

The age-standardised rate for the combined direct and indirect maternal deaths has decreased from 1.09 to 0.66 deaths per 100,000 female population aged 15–44 years over the 30-year period 1973–1975 to 2000–2002 (Table 9).

Table 9: Age-specific and age-standardised maternal mortality rates: direct and indirect maternal deaths, Australia, 1973–2002

Triennium	Direct & indirect deaths	15–19 years	20–24 years	25–29 years	30–34 years	35–39 years	40–44 years	Age-standardised rate[a]
1973–1975	96	0.30	1.52	1.71	0.74	1.11	1.17	1.09
1976–1978	87	0.33	1.22	1.23	1.07	0.65	1.20	0.95
1979–1981	88	0.33	1.19	1.25	1.10	0.69	0.63	0.86
1982–1984	67	0.23	1.03	0.97	0.53	0.77	0.08	0.59
1985–1987	62	0.51	0.46	1.15	0.68	0.38	0.00	0.52
1988–1990	70	0.10	0.56	1.04	1.13	0.52	0.11	0.58
1991–1993	48	0.15	0.19	0.68	0.60	0.49	0.21	0.39
1994–1996	66	0.11	0.43	0.67	1.23	0.56	0.10	0.53
1997–1999	[b]62	0.16	0.25	0.68	0.75	0.84	0.19	0.49
2000–2002	84	0.20	0.26	1.13	1.35	0.62	[c]0.31	0.66

(a) Directly age-standardised to the Australian female population aged 15 to 44 years at 30 June 2001.

(b) Data do not include two indirect deaths in the 1997–1999 triennium that were notified or amended after the 1997–1999 report was published.

(c) Includes 1 woman aged over 44 years.

Note: Rates expressed per 100,000 female population.

Maternal deaths by state and territory

The majority of states and territories publish maternal deaths in their maternal and perinatal reports, with some reports including case reviews and MMRs. This is the first time that state or territory of death has been included in the *Maternal deaths in Australia* report (Table 10). These findings need to be interpreted with some caution as the numbers are small and there is potential for statistical fluctuation over time. The Australian Capital Territory (ACT) did not give permission to publish data because of the small number of deaths and concerns about reporting based on place of death rather than place of usual residence. Western Australia (WA) gave permission to publish data combined over two triennia for the period 1997–2002. Data have therefore been omitted for both states in the following three tables to satisfy these reporting requirements. The classification of deaths may differ from those published in state and territory reports due to the NACCM classifying some deaths differently to the state and territories.

Table 10 shows the number of direct and indirect maternal deaths and MMR by state and territory for the 2000–2002 triennium. Data on late maternal deaths are also presented.

Table 10: Maternal deaths by state and territory, 2000–2002

State and territory	Number of women who gave birth	Direct and indirect deaths[(a)]	MMR[(b)]	Direct, indirect & late deaths[(a)]	Ratio[(b)] inc. late deaths
NSW	255,427	23	9.0	28	11.0
Vic	184,703	17	9.2	18	9.7
Qld[(c)]	145,754	23	15.8	24	16.5
WA	73,708	n.p.	n.p.	n.p.	n.p.
SA[(d)]	52,426	8	15.3	8	15.3
Tas	17,045	3	17.6	3	17.6
ACT	13,806	n.p.	n.p.	n.p.	n.p.
NT	11,032	1	9.1	1	9.1
Total	**753,901**	**84**	**11.1**	**92**	**12.2**

(a) Numbers may differ from those published in state and territory reports due to possible differences in the classification of maternal deaths by the NACMM compared to the STMMC.

(b) Per 100,000 women who gave birth.

(c) Includes one death where the birth occurred in NSW and the woman was transferred and died in Qld.

(d) The South Australian Maternal, Perinatal and Infant Mortality Committee, Maternal Subcommittee enquiries into maternal deaths during the period 1997–2002 has not found recurring causes or system deficiencies in its review of deaths.

n.p. Not published.

Table 11 shows the distribution of maternal deaths and MMR by state and territory, as classified by the NACMM, for the 1997–1999 triennium. These data were not published in the *Maternal deaths in Australia 1997–99* report.

Table 11: Maternal deaths by state and territory, 1997–1999

State and territory	Number of women who gave birth	Direct and indirect deaths[a]	MMR[b]	Direct, indirect & late deaths[a]	Ratio[b] inc. late deaths
NSW	257,961	29	11.2	29	11.2
Vic	183,969	12	6.5	13	7.1
Qld	142,770	15	10.5	16	11.2
WA	75,525	n.p.	n.p.	n.p.	n.p.
SA[c]	55,047	4	7.3	5	9.1
Tas	18,246	1	5.5	1	5.5
ACT	13,952	n.p.	n.p.	n.p.	n.p.
NT	10,560	1	9.5	1	9.5
Total	**758,030**	**66**	**8.7**	**70**	**9.2**

(a) Numbers may differ from those published in state and territory reports due to possible differences in the classification of maternal deaths by the NACMM compared to the STMMC.

(b) Per 100,000 women who gave birth.

(c) The South Australian Maternal, Perinatal and Infant Mortality Committee, Maternal Subcommittee enquiries into maternal deaths during the period 1997–2002 has not found recurring causes or system deficiencies in its review of deaths.

n.p. Not published.

Table 12 shows the distribution of maternal deaths and MMR by state and territory, as classified by the NACMM, over the two triennia, 1997–2002.

Table 12: Maternal deaths by state and territory, 1997–2002

State and territory	Number of women who gave birth	Direct and indirect deaths[a]	MMR[b]	Direct, indirect & late deaths[a]	Ratio[b] inc. late deaths
NSW	513,388	52	10.1	57	11.1
Vic	368,672	29	7.9	31	8.4
Qld[c]	288,524	38	13.2	40	13.9
WA	149,233	n.p.	n.p.	n.p.	n.p.
SA[d]	107,473	12	11.2	13	12.1
Tas	35,291	4	11.3	4	11.3
ACT	27,758	n.p.	n.p.	n.p.	n.p.
NT	21,592	2	9.3	2	9.3
Total	**1,511,931**	**150**	**9.9**	**162**	**10.7**

(a) Numbers may differ from those published in state and territory reports due to possible differences in the classification of maternal deaths by the NACMM compared to the STMMC.

(b) Per 100,000 women who gave birth.

(c) Includes one death where the birth occurred in NSW and the woman was transferred and died in Qld.

(d) The South Australian Maternal, Perinatal and Infant Mortality Committee, Maternal Subcommittee enquiries into maternal deaths during the period 1997–2002 has not found recurring causes or system deficiencies in its review of deaths.

n.p. Not published.

Direct maternal deaths

There was a decline in the maternal mortality ratio of direct deaths from the 1964–66 triennium to the 1985–1987 triennium. Since 1985–1987, the ratio has remained below 5.0 per 100,000 women who gave birth, with the exception of 1994–1996 when the ratio was 6.0 per 100,000 women who gave birth. The maternal mortality ratio for direct deaths was 4.2 deaths per 100,000 women who gave birth in the 2000–2002 triennium (Table 13).

Table 13: Direct maternal deaths, Australia, 1964–2002

Triennium	Direct maternal deaths	Maternal mortality ratio[(a)]
1964–1966	202	30.3
1967–1969	166	23.3
1970–1972	150	19.0
1973–1975	60	8.3
1976–1978	52	7.7
1979–1981	54	7.9
1982–1984	42	5.9
1985–1987	32	4.4
1988–1990	37	4.9
1991–1993	27	3.5
1994–1996	46	6.0
1997–1999	34	4.5
2000–2002	32	4.2

(a) Per 100,000 women who gave birth.

Cause of direct maternal deaths

There were 32 direct maternal deaths in the 2000–2002 triennium (Table 14). This was two less deaths than reported in the 1997–1999 triennium.

The causes of direct death were amniotic fluid embolism (n=10), obstetric haemorrhage (n=9), infection (n=5), hypertensive disorders of pregnancy (n=4), pulmonary thromboembolism (n=2), with one death due to anaesthesia and one death due to ectopic pregnancy (Figure 3).

Table 14: Cause of direct maternal deaths, Australia, 2000–2002

Cause of death	Number
Amniotic fluid embolism	
Amniotic fluid embolism	10
Total	*10*
Obstetric haemorrhage	
Postpartum haemorrhage	2
Postpartum haemorrhage with arterial bleeding at caesarean section (C/S)	2
Postpartum haemorrhage with uterine rupture	1
Placenta percreta	3
Placental abruption	1
Total	*9*
Infection	
Septicaemia: Streptococcus pyogenes	2
Septicaemia: *Escherichia coli*	1
Septicaemia: Group A Streptococcus	1
Septic shock secondary to faecal peritonitis	1
Total	*5*
Hypertensive disorders of pregnancy	
Pre-eclampsia / eclampsia	3
Hypertension with cerebral and brain stem haemorrhage	1
Total	*4*
Pulmonary thromboembolism	
Pulmonary thromboembolism	2
Total	*2*
Deaths associated with anaesthesia	
Epidural abscess	1
Total	*1*
Early pregnancy deaths	
Ruptured ectopic pregnancy	1
Total	*1*
Total	**32**

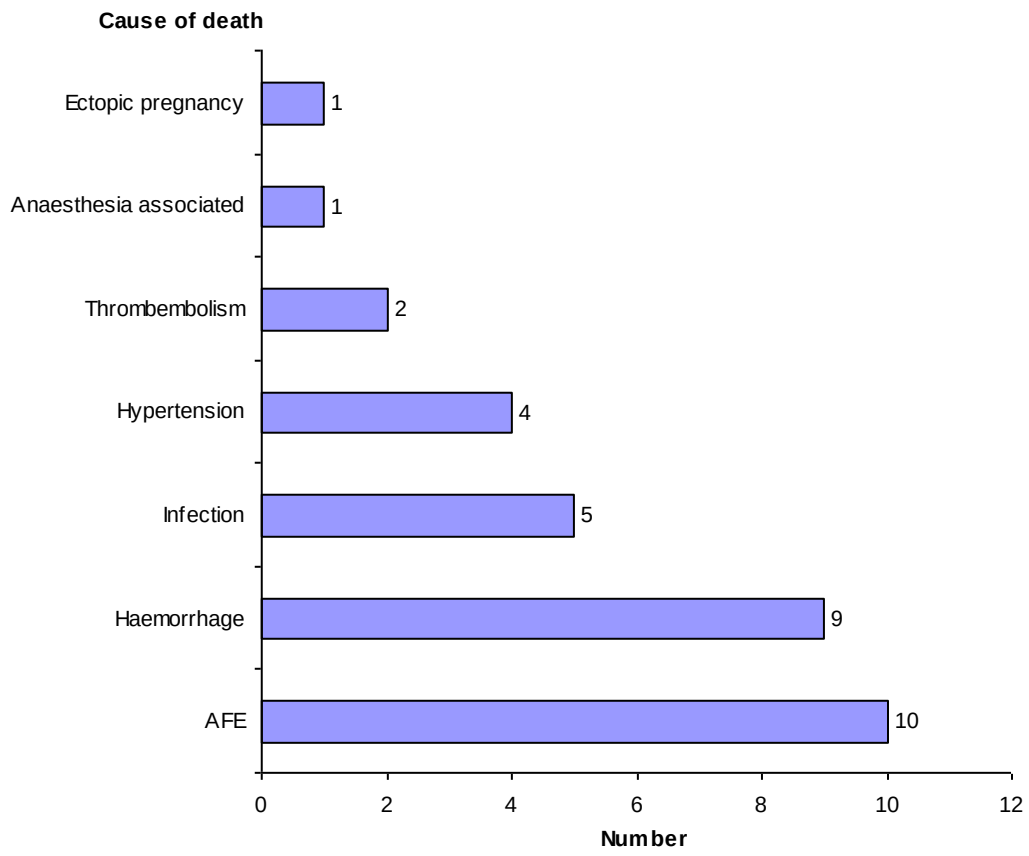

Figure 3: Cause of direct maternal deaths, Australia, 2000–2002

Parity by age group

During the 2000–2002 triennium, 72% (n=23) of the women classified as having a direct death had between one and three previous pregnancies resulting in a birth and 25% (n=8) had four or more previous pregnancies resulting in births (Table 15). One of the two teenage deaths was of a nulliparous woman.

Table 15: Direct maternal deaths by parity and age group, Australia, 2000–2002

	Parity (number)			
Age group (years)	**0**	**1–3**	**≥ 4**	**Total**
15–19	1	1	—	2
20–24	—	1	—	1
25–29	—	6	2	8
30–34	—	7	3	10
35–39	—	6	2	8
40–44	—	1	1	2
≥ 45	—	1	—	1
Total	**1**	**23**	**8**	**32**

Indirect maternal deaths

The maternal mortality ratio for indirect deaths in Australia has fluctuated between 2.7 to 6.9 per 100,000 women who gave birth during the last thirty years (Table 16). Between the 1964–1966 and 1973–1975 triennia, indirect and incidental deaths were not separately classified, therefore, a maternal mortality ratio for indirect deaths cannot be reported for those triennia.

The increase in the ratio since 1994–1996 is in part due to changes in classification of some causes of death from incidental to indirect. There has been increasing interest in examining maternal deaths which do not initially appear to be related to the pregnancy, but on closer examination may well be directly or indirectly associated with the pregnancy or its complications. Examples of this would be deaths due unintentional and intentional harm such as a drug overdose in former and domestic violence in the latter, where the precipitation or contributing factor was the pregnancy. In the 2000–2002 triennium, deaths such as this have been classified as indirect rather then incidental. This change in classification involved deaths due to psychiatric causes from 1997 onwards and deaths due to some cancers and tumours from 2000 onwards. In the 2000–2002 triennium, three deaths were caused by cancers or tumours that would have been classified as incidental in previous reports. The NACMM will continue to maximise efforts to obtain comprehensive ascertainment of these deaths and to ensure national consistency in reporting.

Table 16: Indirect maternal deaths, Australia, 1973–2002

Triennium	Indirect maternal deaths	Maternal mortality ratio[(a)]
1973–1975	32	4.4
1976–1978	35	5.2
1979–1981	34	5.0
1982–1984	25	3.5
1985–1987	30	4.1
1988–1990	33	4.4
1991–1993	22	2.9
1994–1996	20	2.6
1997–1999	[(b)]30	4.0
2000–2002	52	6.9

(a) Per 100,000 women who gave birth.

(b) Data updated to include two indirect deaths in the1997–1999 triennium that were notified or amended after the 1997–1999 report was published.

Cause of indirect maternal deaths

There were 52 indirect maternal deaths in the 2000–2002 triennium (Table 17). This represents an increase from the previous triennium of 22 deaths. The rise is in part due to changes in classification of some deaths, though an actual increase cannot be ruled out. The main causes of indirect deaths were deaths due to cardiac disease (n=11), infection (n=10), psychiatric causes (n=9), haemorrhage–other (n=8), cancers or tumours (n=3) and asthma (3) (Figure 4).

Table 17: Cause of indirect maternal deaths, Australia, 2000–2002

Cause of death	Number
Cardiac disease	
Cardiac failure	3
Aortic arch dissection	2
Myocardial infarction	2
Myocarditis	2
Ischaemic heart disease	1
Rheumatic heart disease	1
Total	*11*
Infection	
Septicaemia	4
Meningitis	2
Herpes simplex viral infection	2
Adult respiratory distress syndrome/ pneumonia	2
Total	*10*
Deaths from psychiatric causes	
Hanging	6
Drug overdose / poisoning	2
Pedestrian in motor vehicle accident	1
Total	*9*
Other haemorrhage	
Intra-cerebral haemorrhage	2
Subarachnoid haemorrhage	2
Cystic medial necrosis of abdominal aorta	1
Splenic artery aneurysm rupture	1
Left subclavian artery dissection	1
Intra-abdominal haemorrhage	1
Total	*8*
Other indirect causes	
Asthma	3
Cancers or tumours	3
Homicide / assault (head injury)	2
Thrombotic thrombocytopenic purpura	1
Systemic lupus erythematosus	1
Epilepsy	1
Hypertension (intra-cerebral haemorrhage)	1
Hyperammonaemic encephalopathy	1
Pulmonary embolism	1
Total	*14*
Total	**52**

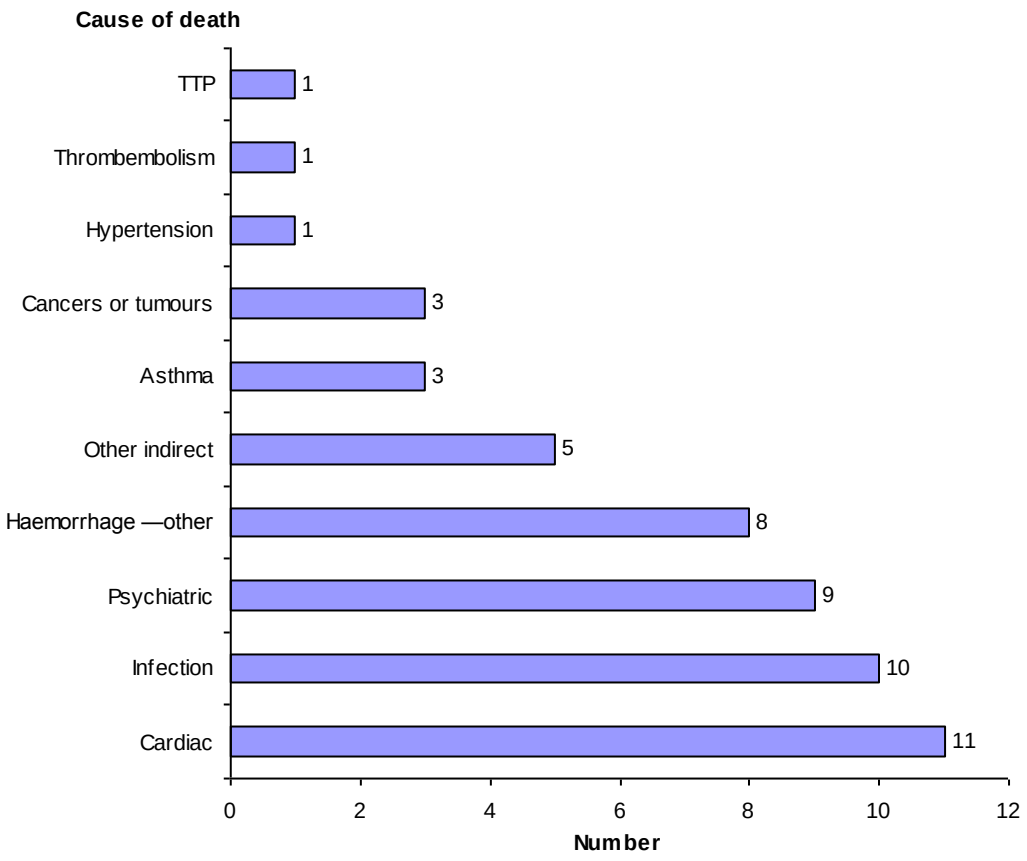

Figure 4: Cause of indirect maternal deaths, Australia, 2000–2002

Parity by age group

The majority of women classified as having had an indirect maternal death in the 2000–2002 triennium were parous with 62% (n=32) having had between one and three previous pregnancies resulting in a birth, and 12% (n=6) having had four or more previous pregnancies resulting in births (Table 18). Only 12% (n=6) were nulliparous.

Table 18: Indirect maternal deaths by parity and age group, Australia, 2000–2002

	Parity (number)				
Age group (years)	**0**	**1–3**	**≥ 4**	**Not stated**	**Total**
15–19	1	—	—	1	2
20–24	2	2	—	—	4
25–29	3	10	—	3	16
30–34	—	14	4	2	20
35–39	—	3	2	1	6
40–44	—	3	—	1	4
Total	**6**	**32**	**6**	**8**	**52**

Incidental maternal deaths

There were three incidental maternal deaths in the 2000–2002 triennium. The causes of the incidental maternal deaths were synovial sarcoma, multiple injuries sustained in a motor vehicle accident, and one death where the cause was undetermined, but was thought to be due to an arrhythmia (Table 19).

Table 19: Incidental maternal deaths, Australia, 2000–2002

Age group (years)	Principal cause of death	Gestation (weeks)	Type of delivery, baby outcome and comments
20–34	Synovial sarcoma	16	Induced abortion
<20	Multiple injuries from motor vehicle accident	26	Emergency caesarean section, stillbirth. Woman found on the roadway beside the vehicle – no information about seatbelt usage.
20–34	Undetermined, possible arrhythmia	6	Not delivered

There has been a marked decline in incidental maternal deaths between the 1997–1999 and 2000–2002 trienniums from 28 to 3 (Table 20). This is due to both changes in classification practices and possibly a real decline in incidental deaths. There is potential for some deaths to be able to be classified into either indirect or incidental categories such as when there is not clinical consensus about the aetiology of the case or where more recent data suggest a condition should now be considered aggravated by the physiological aspects of pregnancy, as in some of the cancers. Thus some deaths classified as incidental, e.g. cancers, in previous triennia have been classified as indirect during the 2000–2002 triennium.

Table 20: Incidental maternal deaths, Australia, 1973–2002

Triennium	Number of women who gave birth	Incidental maternal deaths
1973–75	726,690	45
1976–78	678,098	19
1979–81	682,880	10
1982–84	713,985	27
1985–87	726,642	24
1988–90	754,468	26
1991–93	769,253	36
1994–96	767,448	34
1997–1999	758,030	28
2000–2002	753,901	3

Clinical comment and best practice

It is reassuring to see a decrease in maternal deaths resulting from motor vehicle accidents. In the 1997–1999 triennium there were five deaths involving motor vehicle accidents with all women thought not to be wearing seatbelts. It is legislated throughout Australia that seatbelts be worn by all car passengers, including pregnant women. Individual state and territory transport authorities publish brochures demonstrating the correct way to wear a seatbelt in pregnancy. Brochures are also available in languages other than English and for

Aboriginal and Torres Strait Islander women. The correct position to wear a seatbelt is for the lap strap to be sitting over the thighs, across the pelvis and below the unborn child. The sash strap should be placed above the stomach and between the breasts (ASTB 2004). Antenatal education should incorporate this important information as early in pregnancy as possible, preferably at first contact with the care provider.

Late maternal deaths

There were eight late maternal deaths in the 2000–2002 triennium (Table 21). Ascertainment of these deaths is thought to be incomplete. The ratio of those reported to NACMM was 1.1 per 100,000 women who gave birth compared to 4.7 per 100,000 maternities for the United Kingdom during the 2000–2002 triennium (Lewis G (ed.) 2004).

Two late maternal deaths were classified as direct maternal deaths and six were classified as indirect maternal deaths.

Table 21: Late maternal deaths, Australia, 1994–2002

Triennium	Number of women who gave birth	Number of maternal deaths	
		Direct	Indirect
1994–96	767,448	—	2
1997–1999	758,030	1	4
2000–2002	753,901	2	6

Cause of late maternal deaths

Late maternal deaths in the 2000–2002 triennium were caused by cardiac disease (n=4), thrombotic thrombocytopenic purpura (TTP) (n=2) and one each from infection and suicide (Table 22).

Table 22: Cause of late maternal deaths, Australia, 2000–2002

Cause of death	Number
Cardiac	
Peripartum cardiomyopathy	2
Severe pulmonary hypertension	1
Cardiac tamponade	1
Total	*4*
TTP	
TTP	2
Total	*2*
Infection	
Adult respiratory distress syndrome	1
Total	*1*
Suicide	
Overdose	1
Total	*1*
Total	**8**

Table 23 shows more detail on the late maternal deaths in the 2000–2002 triennium.

Table 23: Late maternal deaths, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Method of birth and baby outcome	Days postpartum at death
Direct				
20–34	TTP	Abruption pre-eclampsia, multi-system failure	Emergency C/S, live birth	49
Indirect				
20–34	Adult respiratory distress syndrome	Asthma, staphylococcal pneumonia	Spontaneous vaginal birth, live birth	48
20–34	Suicide	Overdose	Emergency C/S, live birth	49
20–34	TTP	Haemorrhage from VasCath, ventricular dysfunction, pericardial effusion	Spontaneous vaginal birth, live birth	52
20–34	Peripartum cardiomyopathy		Vacuum extraction, live birth	78
20–34	Peripartum cardiomyopathy	Mitral valve replacement	Elective C/S, live birth	102
≥ 35	Severe pulmonary hypertension	Mixed connective tissue disease, cardio-respiratory failure	Emergency C/S, live birth	135
≥ 35	Cardiac tamponade	Aortic dissection	Elective C/S, live birth	143

Demographic profile

This section presents data on all 95 maternal deaths during the 2000–2002 triennuim, unless stated otherwise. In some tables the MMR has been reported. In these tables the incidental maternal deaths and late maternal deaths have been excluded.

Maternal age

The ages of the 95 women who died in the 2000–2002 triennium ranged from 17 to 50 years with a median age of 30.0 years and a mean age of 30.6 years.

Table 24 shows deaths by age group the deaths were classified as direct and indirect. Eleven per cent of all the maternal deaths were of women aged less than 25 years, 64% were aged 25–34 years and 25% were aged 35 years and older. The MMR was highest for women aged 40–50 years (32.8 per 100,000 women who gave birth) and lowest for women aged 20–24 years (4.3 per 100,000 women who gave birth).

Table 24: Maternal deaths[(a)] by age group, Australia, 2000–2002

Age group (years)	Number of deaths[(a)]	Per cent	Number of women who gave birth	Per cent of total number of women who gave birth	MMR[(b)]
15–19	4	4.8	37,315	4.9	10.7
20–24	5	6.0	116,076	15.4	4.3
25–29	24	28.6	225,967	30.0	10.6
30–34	30	35.7	242,073	32.1	12.4
35–39	14	16.7	110,920	14.7	12.6
40–50	7	8.3	21,318	2.8	32.8
Not stated	0	. .	232	. .	. .
Total	**84**	**100.0**	**753,901**	**100.0**	**11.1**

(a) Direct and indirect maternal deaths.

(b) Per 100,000 women who gave birth.

. . Not applicable.

Maternal deaths among Aboriginal and Torres Strait Islander women

There were 13 deaths reported of Aboriginal or Torres Strait Islander women in the 2000–2002 triennium. Of the 13 deaths, four were categorised as direct maternal deaths, eight as indirect maternal deaths and one as an incidental death. This is one maternal death (direct and indirect) for every 2,177 Aboriginal or Torres Strait Islander women who gave birth during the triennium. The MMR for Aboriginal and Torres Strait Islander women was 45.9 per 100,000 Aboriginal and Torres Strait Islander women who gave birth. These deaths are discussed in the chapter entitled *Maternal mortality in Aboriginal and Torres Strait Islander women*.

Maternal deaths among overseas-born women

Fifty-seven (60%) of the women who died were born in Australia, 21 (22%) were born outside Australia and in 17 (18%) cases country of birth was not known. During the same period 2000–2002, 77.2% of all births in Australia were to women who were born in Australia compared to 73.1% (57/78) of maternal deaths (where country of birth was known). The number of deaths varied by country of birth. There were three maternal deaths from women born in Vietnam and also the Philippines; two each from India and New Zealand; and one maternal death from Bahrain, China, Ethiopia, Hong Kong, Iraq, Lebanon, Scotland, South Africa, Sri Lanka, Tonga and the UK respectively. These findings need to be interpreted with some caution as the numbers are small and the duration of residency in Australia is not known. No subgroup analyses were undertaken due to small numbers and missing data.

Remoteness Area of usual residence

Remoteness area categories are based on road distance from a locality to the closest service centre in five classes of population size (AIHW 2004b). The classifications are: Major Cities of Australia, Inner Regional Australia, Outer Regional Australia, Remote Australia and Very Remote Australia.

Table 25 presents maternal deaths by remoteness area of usual residence. Sixty-one per cent of the deaths occurred in Major cities in Australia, where 66% of the population live compared to seven per cent in Remote and Very Remote areas where three per cent of the population usually reside. These data do not establish any relationship between risk of death and usual residence and need to be interpreted with some caution as the numbers are small.

Table 25: Maternal deaths by Remoteness Area of usual residence and type of death, Australia, 2000–2002

	Number of deaths				
Remoteness Area	**Direct**	**Indirect**	**Incidental**	**Total**	**Per cent**
Major Cities	23 (1)	34 (5)	1	58	61.1
Inner Regional	4 (1)	8	2	14	14.7
Outer Regional	3	12 (1)	—	15	15.8
Remote / Very Remote	3	4	—	7	7.4
Not stated	1	—	—	1	1.0
Total	**34**	**58**	**3**	**95**	**100.0**

( ) No. of late deaths.

Gestational age at death

The gestational age by type of death is shown in Table 26. For 29 (30.5%) of the deaths gestational age was not known. The distribution of deaths varied by type of death, with the majority of indirect deaths occurring at less than 20 weeks gestation compared to 28-36 weeks gestation for direct deaths.

Twenty-eight women died undelivered (Table 27). Of these deaths 54% (n=15) were in pregnancies where the gestational age was <20 weeks and 46% (n=13) were in pregnancies where the gestational age was ≥20 weeks (Table 27). Sixty-seven women (71%) died following birth or termination of pregnancy, with 25% (n=17) of this group dying within 24 hours of birth or termination of pregnancy and 12% (n=8) classified as late maternal deaths.

Table 26: Maternal deaths by gestational age by type of death, Australia, 2000–2002

Gestational age (weeks)	Direct	Indirect	Incidental	Total
<20	1	17	2	20
20–27	2	7	1	10
28–36	12	13	—	25
>36	6	5	—	11
Not stated	13	16	—	29
Total	**34**	**58**	**3**	**95**

Table 27: Maternal deaths by timing of death and gestational age, Australia, 2000–2002

	Gestational age (weeks)					
Timing of death	<20	20–27	28–36	≥37	Not stated	Total
Died undelivered	15	6	6	1	—	28
Within 24 hours of birth or termination of pregnancy	1	—	7	5	4	17
2–6 days postpartum	1	1	5	1	7	15
7–42 days postpartum	2	1	4	—	12	19
>42 days postpartum	—	—	1	2	2	8
Not stated	1	2	2	2	4	8
Total	**20**	**10**	**25**	**11**	**29**	**95**

Pregnancy outcome

The pregnancy outcomes of the 61 women who delivered prior to their death included 53 women who had live born infants and eight women whose infants were stillborn (Table 28).

Table 28: Maternal deaths by pregnancy outcome, Australia, 2000–2002

Outcome	Number	Per cent
Live birth	53	55.8
Stillbirth	8	8.4
Spontaneous abortion	2	2.1
Induced abortion	2	2.1
Ectopic	1	1.1
Undelivered	28	29.5
Not stated	1	1.1
Total	**95**	**100.0**

Cause of maternal deaths

Figure 5 shows maternal deaths in Australia by cause and type of death for 2000–2002.

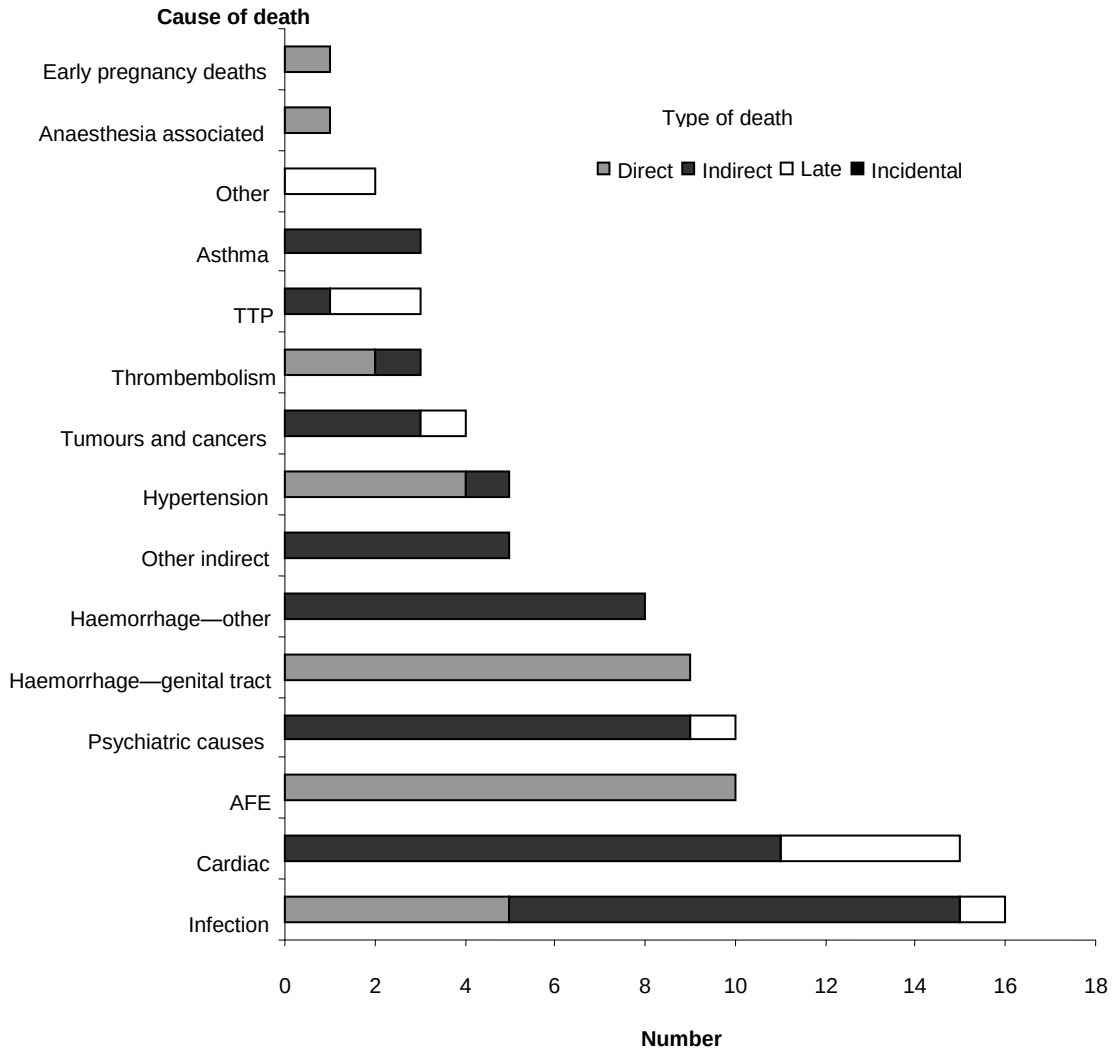

Figure 5: Maternal deaths by cause and type of death, 2000–2002

Contributing factors

There is no agreed national definition for the term 'contributing factors' to a maternal death. The definition currently used by the NACMM is:

> *Some departure from the accepted standard of satisfactory care by the woman, practitioner or institution, which may have contributed to the death (Slaytor et al. 2004).*

This report presents results of the STMMC considerations concerning the presence of contributing factors for individual cases. The NACMM is aware of inconsistencies between (and within) states and territories in their approaches, and is therefore unable to provide a comprehensive report on this subject. The findings need to be interpreted with some caution.

A contributing factor was considered to be 'possibly present' or 'certainly present' in 46 (48%) cases: 19 direct maternal deaths, 22 indirect maternal deaths and one incidental maternal death. More than one contributing factor was reported in 15 cases. The categories of contributing factors are listed in Table 29.

Table 29: Contributing factors as reported by the State and Territory Maternal Mortality Committees, 2000–2002

System		Number
Facilities	Lack of human resources (e.g. lack of available staff)	2
	Lack/delay in access to health services due to rural location	2
	Lack of facilities, equipment or consumables	1
Logistical systems	Communication breakdown between health services	2
	Lack of transport between health care facilities	1
	Lack/delay in access to health services due to rural location	1
Health personnel	Inappropriate clinical management	11
	Lack of expertise, training or education	5
	Insufficient antenatal care	1
	Communication breakdown between health services	1
	Other	1
Personal/Family	Delay in woman seeking help	10
	Refusal of treatment or admission	7
	Insufficient antenatal care	3
	Risk behaviour (alcohol, drugs)	3
	Other	3
Total[(a)]		**54**

(a) More than one contributing factor was reported in 15 cases.

Other considerations

Postmortem examinations

Postmortem examinations were undertaken for 69% (n=66) of the 95 maternal deaths. Twentyfive (74%) of the direct maternal deaths and 39 (67%) of the indirect maternal deaths underwent postmortem examination. Two of the three incidental maternal deaths had postmortem examinations.

Coronial inquests

Coronial inquests are mandated for: all violent deaths, all unnatural deaths, all deaths within 24 hours of having an anaesthetic and all deaths where the medical practitioners cannot give a cause of death. For the 2000–2002 triennia 53 of the 95 deaths (56%) were investigated by a state or territory coroner. Of the 34 direct maternal deaths, 21 (62%) were subject to a coronial inquest while 32 (55%) of the 58 indirect maternal deaths had coronial inquests. There were no coronial inquests into the incidental maternal deaths. NACMM holds the view that the coroner should be notified in all unexpected maternal deaths.

Section C: Cause of maternal deaths

Haemorrhage

Introduction

Worldwide, it is estimated that 515,000 women die in pregnancy and childbirth per year with 97% of these deaths occurring in developing countries (JHPIEGO Corporation 1999–2003; MacDonald & Starrs 2002). It is estimated that obstetric haemorrhage accounts for 25–50% of these maternal deaths worldwide with the rate of mortality varying by country and development indices. The major cause of death is postpartum haemorrhage (PPH) (JHPIEGO Corporation 1999–2003; Schuurmans et al. 2002). Obstetric haemorrhage is also a problem for developed countries. In Australia it is a principal cause of maternal mortality responsible for 20–28% of direct deaths since 1973–1975 (Sullivan et al. 2004). Obstetric haemorrhage, including antepartum haemorrhage (APH) and postpartum haemorrhage (PPH) can be sudden, unpredictable and catastrophic.

Antepartum haemorrhage usually results from either placental abruption or placenta praevia. The incidence of placental abruption is thought to be increasing and has been reported as 0.5–1% of all singleton pregnancies and 1.22% of twin pregnancies (Hall & Wagaarachchi 2002). This condition is seen more commonly in women having their first baby, older women, multiple pregnancy, women who smoke, women who use cocaine, those with a history of first-trimester bleeding, hypertension or pre-eclampsia, trauma and previous placental abruption (Hall & Wagaarachchi 2002; Neilson 2003).

Placenta praevia has an incidence of approximately 0.48% of all pregnancies (Hall & Wagaarachchi 2002). Placenta percreta/accreta are the most severe forms of this condition. Placenta praevia and the morbidly adherent conditions are more commonly seen in older women, women with multiple pregnancy, women with previous placenta praevia, and women who have had one or more prior caesarean section deliveries (Bennett & Sen 2003; Lewis et al. 2004). The risk of placenta praevia rises with subsequent caesarean sections, with a prospective study showing a fivefold increase (0.44% to 2.54%) in placenta praevia following one caesarean section (Chattopadhyay 1993). In this study Placenta praevia was complicated by accreta in 10% of cases following one caesarean section but this increased to 59% of cases when there had been more than one previous caesarean sections performed (Chattopadhyay 1993).

This chapter also reports on haemorrhage from sites other than the genital tract. Previously, these may have been classified as *other* indirect deaths and reported in other chapters as circulatory deaths and in the case of abdominal aortic aneurysm possibly as a cardiac death (Lewis et al. 2004; Slaytor et al. 2004). However, for this triennium, the Committee decided to include all haemorrhagic deaths in this chapter.

In the first part of this chapter, deaths due to obstetric haemorrhage originating from the genital tract are reported, and in the second part, deaths due to haemorrhage from other sites is reported.

Summary of the findings for 2000–2002

In the 2000–2002 triennium, there were 18 deaths in which obstetric haemorrhage was attributed as a principal or contributing cause of death. There were nine direct maternal deaths principally attributed to obstetric haemorrhage and nine direct maternal deaths where haemorrhage was recorded as a contributing factor. Amniotic fluid embolism (AFE) was the principal cause of death in eight of these cases. The other case was a late maternal death (49 days following birth without discharge from hospital), where TTP was the principal cause.

Table 30 presents an overview of maternal deaths where obstetric haemorrhage was either a principal or contributing cause of death. Many of these cases were complex cases with mixed presentations including APH, placenta praevia, AFE and subsequent PPH.

Table 30: Maternal deaths due to obstetric haemorrhage, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Comments	Parity[(a)]	Gestation (weeks)	Birth outcome
Direct						
≥ 35	Haemorrhage		Abruption, Twin pregnancy, APH	M	34	Stillbirth × 2
20–34	Haemorrhage		Placenta percreta, disseminated intravascular coagulopathy (DIC)	M	n.s.	Live birth
20–34	Haemorrhage		Placenta percreta — invasion to bladder and cervix, APH, massive transfusion	M	34	Live birth
20–34	Haemorrhage		Placenta percreta — invasion to bladder, APH 24/40, previous PPH	M	24	Live birth
20–34	Haemorrhage		PPH, refusal to accept blood products, thalassaemia, APH — abruption following fall	P	37	Stillbirth
≥ 35	Haemorrhage	Caesarean section	PPH, pre-eclampsia, C/S, fibroids - bladder adherent to uterus, arterial bleeding at C/S	M	28	Live birth
20–34	Haemorrhage		PPH, retained placenta, manual removal, hysterectomy, torn cervix at autopsy	GM	40	Live birth
≥ 35	Haemorrhage		PPH, uterine rupture, birth in remote area with poor weather delaying transfer	GM	Term	Live birth
≥ 35	Haemorrhage	AFE	PPH following intra-partum uterine rupture	M	40	Live birth
20–34	AFE	Haemorrhage	PPH mucosal tear, uterine rupture	GM	36	Live birth
20–34	AFE	Haemorrhage	Grade IV placenta praevia, APH, PPH, DIC	M	36	Live birth
≥ 35	AFE	Haemorrhage	Grade III placenta praevia, APH, PPH, DIC	P	34	Live birth
20–34	AFE	Haemorrhage	PPH, DIC, hysterectomy, possible air embolism	M	38	Live birth
20–34	AFE	Haemorrhage	PPH, hysterectomy, pelvic haematoma, DIC, multi-organ failure	P	n.s.	Live birth
20–34	AFE	Haemorrhage	Twin pregnancy, APH, placental abruption, PPH	P	32	Live birth x 2
20–34	AFE	Haemorrhage	PPH, DIC, hysterectomy	P	n.s.	Live birth
20–34	AFE	Haemorrhage	PPH secondary to uterine atony, DIC	P	Term	Live birth
Late direct						
20–34	TTP	Haemorrhage	Placental abruption (49 days pp) pre-eclampsia, multi-system failure	P	34	Live birth

(a) P = primipara, M = multipara, GM = grand multipara.

n.s. Not stated.

In the triennium 1997–1999, there were eight direct maternal deaths primarily attributed to obstetric haemorrhage, with PPH judged to be the principal cause of death in all cases. In one other death (due to AFE), PPH was recorded as a contributing factor. There were no deaths due to antepartum haemorrhage in the 1997–1999 triennium.

Antepartum haemorrhage

In the 2000–2002 triennium, there was one case in which placental abruption was the principal cause of haemorrhage. In three other cases abruption was a contributing cause of death with PPH, AFE and TTP (late death) as the principal cause of death.

Deaths from APH also included three women who had placenta praevia percreta as the principal cause of death. One case involved placental invasion of the parametrial tissues and two cases involved invasion of the bladder, which in one case extended to involve the cervix. All three women were documented as having had a previous caesarean section. All three women experienced significant haemorrhage requiring intensive resuscitation, hysterectomy and in one case ligation of the internal iliac arteries.

Two other women had placenta praevia (Grade III and IV) as a contributing factor in their deaths, with AFE being classified as the principal cause of death in both cases. Neither of these women had had previous caesarean sections though both had previous uterine surgery. Both of these cases involved antepartum haemorrhage, emergency caesarean section, coagulopathy and PPH.

Postpartum haemorrhage

Five women had PPH as the principal cause of death. Two of these deaths were associated with uterine rupture, one was associated with bleeding at caesarean section, one was complicated by a refusal to accept blood products for religious reasons and the other involved a retained placenta with a torn cervix being identified at autopsy. Three of the five women with PPH had labour induced (one for fetal death in utero). Postpartum haemorrhage was a contributing factor in the deaths of 10 women, three of whom had placenta percreta as the principal cause of death and eight women had a principle cause of death of AFE, one of whom had a uterine rupture identified at postmortem.

Illustrative cases

The following cases illustrate the complex nature of the deaths that were attributed to haemorrhage during the 2000–2002 triennium.

A woman who had a previous caesarean section, presented at 23 weeks gestation with APH. She was diagnosed with placenta praevia. She had two further minor APHs and at 34 weeks gestation was admitted, contracting with small bright vaginal blood loss. She proceeded to an emergency caesarean section where a live baby was born. She died despite massive transfusions and multidisciplinary involvement. Autopsy showed placenta praevia percreta, with erosion into the posterior wall of the urinary bladder.

Principal cause: Placenta percreta.

Contributing cause: PPH.

Maternal death classification: Direct.

A woman was admitted at 37 weeks gestation with abdominal pain. She had fallen the day before and an ultrasound scan confirmed a placental abruption with a fetal death in utero. Thalassaemia had been identified during the antenatal period. Labour was induced and the woman experienced a PPH. Medical management was complicated by the woman's religious status as a Jehovah's Witness and consequent refusal of any blood products. She was transferred for treatment in a hyperbaric unit. Despite intensive resuscitation efforts she continued to deteriorate with metabolic acidosis and cardiac arrest. An autopsy concluded the cause of death to be PPH complicated by a refusal to accept blood transfusions because of religious beliefs.

Principal cause: PPH.

Contributing causes: Abruption, religious beliefs restricting treatment options.

Maternal death classification: Direct.

A primigravida was admitted with placental abruption at 32 weeks gestation. An emergency caesarean section was performed and two live babies delivered. Following delivery there was moderate ongoing blood loss despite intravenous and intramyometrial oxytocics. Postpartum haemorrhage continued and the woman was returned to theatre for laparotomy and subtotal hysterectomy. She required major resuscitation including multiple blood products. A diagnosis of disseminated intravascular coagulopathy (DIC) was made and she was transferred to the intensive care unit. She developed acidosis, inotrope resistance and finally cardiac arrest. Autopsy found histological evidence of squames in the maternal circulation. The cause of death was attributed to multi-system failure occurring as a consequence of DIC complicating PPH. Amniotic fluid embolism was thought to be the precipitating factor.

Principal cause: AFE.

Contributing cause: Abruption.

Maternal death classification: Direct.

A woman in her third pregnancy was diagnosed with gestational diabetes and commenced on insulin at 32 weeks gestation. At 38 weeks an ultrasound suggested a large-for-dates baby with estimated fetal weight of 4.3 kg. Labour was induced at 40 weeks with Prostaglandin E 2 mg and the woman gave birth to a live infant weighting 4.6 kg. Moderate shoulder dystocia was noted and the woman collapsed with cardiac arrest 10 minutes later. Despite resuscitation, signs of DIC developed and she died one-and-a-half hours later. Autopsy showed extensive retroperitoneal haemorrhage with uterine, cervical and anterior vaginal tears involving the bladder. Numerous pulmonary vessels and three uterine vessels contained squames, indicative of amniotic fluid embolus.

Principal cause: Haemorrhage, uterine rupture.

Contributing cause: AFE.

Maternal death classification: Direct.

Summary of the findings for 2000–2002

A further 14 maternal deaths are included in this chapter. There were nine indirect deaths where haemorrhage from a site other than the genital tract was the principal cause of death. Three direct deaths and one indirect death resulted from hypertension as the principal cause of death with haemorrhage as a contributing factor. One additional late maternal death resulted from TTP as the principal cause of death with haemorrhage from insertion of a VasCath as a contributing cause of death. These deaths are summarised in Table 31.

Table 31: Maternal deaths due to other haemorrhage, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death and comments	Gestation (weeks)	Method of birth and birth outcome
Direct				
20–34	Hypertension: eclampsia	Haemorrhage: intracranial	27	Live birth
<20	Hypertension	Haemorrhage: bilateral cerebral and brain stem	32	Undelivered
≥ 35	Hypertension: pre-eclampsia	Haemorrhage: ruptured subcapsular hepatic haematoma	32	Undelivered
Indirect				
20–34	Haemorrhage: subarachnoid	Ruptured anterior communicating artery aneurysm, cerebral oedema	5–6	Undelivered
20–34	Haemorrhage: subarachnoid	Right occipital subarachnoid haemorrhage	2 weeks postpartum	n.s.
20–34	Haemorrhage: ruptured splenic artery aneurysm		28	Undelivered
20–34	Haemorrhage: intracerebral		36	Undelivered
20–34	Haemorrhage: intracerebral	Inoperable intracerebral arterio-venous malformation	24	Undelivered
20–34	Haemorrhage: subdural	Assault	16	Undelivered
20–34	Haemorrhage: ruptured abdominal aorta	Cystic medial necrosis of abdominal aorta	26	Undelivered
≥ 35	Haemorrhage: intrathoracic, dissection subclavian artery		38	Undelivered
≥ 35	Haemorrhage: intra abdominal, autopsy could not find site of haemorrhage	Cardiac arrest 30 mins after admission, in labour	39	Stillbirth following emergency C/S
≥ 35	Hypertension	Haemorrhage: intracerebral, streptococcal pneumonia	n.s.	Live birth
Late indirect				
20–34	TTP	Haemorrhage from VasCath, ventricular dysfunction, pericardial effusion	52 days postpartum	Spontaneous vaginal birth, live birth

n.s. Not stated.

Illustrative cases

A woman in her third pregnancy had an uncomplicated pregnancy until 28 weeks gestation when she experienced sudden onset of epigastric pain. She was admitted to hospital with a differential diagnosis including placental abruption, cholelithiasis or gastroenteritis. An ultrasound of the abdomen, three hours after admission, noted 'a moderate amount of free fluid in the abdomen, the cause of which is not certain'. Her haemoglobin was 47 g/L and 13 hours post admission she was found collapsed with no cardiac output. Despite resuscitation she died in the operating theatre one-and-a-half hours after her collapse. The autopsy found the splenic artery showed thinning of the wall with rupture of an aneurysm and haemorrhage into the wall of the vessel. There was no obvious pre-existing pathology of the wall including vasculitis or cystic medial degeneration; no haemosiderin identified in the areas of haemorrhage; excessive numbers of neutrophils suggested an onset of haemorrhage between 8–48 hours prior to death.

Principal cause: Ruptured splenic artery aneurysm.

Maternal death classification: Indirect.

Clinical comment

There is justification for continuing concern regarding the contribution of haemorrhage to maternal mortality in Australia. At the first sign of complications, early care by an experienced, multidisciplinary team is essential. The severity of blood loss is often underestimated and, as a consequence, resuscitation and invasive monitoring is suboptimal. In situations where it is known that blood product transfusion will be refused on religious grounds, the most senior medically qualified person should attend the birth and multidisciplinary management may be required. Antenatal assessment and counselling are also indicated.

Best practice

Placenta accreta

A RANZCOG statement on placenta accreta (RANZCOG 2003) recommends:

- Where there is an increased risk of placenta accreta (for example, an anterior placenta praevia after one or more previous caesarean sections), delivery should occur in a place with the necessary facilities.
- Such facilities would include:
  - an ability to cope with the requirement for high volume blood transfusion
  - capability for provision of other blood products (e.g. platelets, clotting factors)
  - availability of appropriate specialised obstetric expertise.
- A multidisciplinary approach might be needed, including urologists, gynaecological oncologists and interventional radiologists. As with all patients at risk of major antepartum haemorrhage, those with suspected placenta accreta should be encouraged to remain close to the hospital that has been recommended for giving birth in the third trimester of pregnancy.

In all women with a history of previous caesarean section, the site of the placenta should be accurately identified by ultrasound scan before another caesarean section is performed. In those with placenta praevia and one or more prior caesarean sections, an experienced surgeon should perform the operation, with the anaesthetist and theatre staff alerted and cross matched blood available at the time of surgery (Lewis G (ed.) 2004).

Active management of the third stage

A Cochrane review showed active management of the third stage, when compared with physiological management, decreases the risk of PPH and blood transfusion (Prendiville et al. 2003). Active management includes the administration of prophylactic oxytocics, early cord clamping and cutting, and controlled cord traction.

Refusal of blood products

The latest CEMACH report from the UK includes Guidelines for the management and treatment of obstetric haemorrhage in women who decline blood transfusion (Lewis G (ed.) 2004). Key points include:

- identification of these beliefs antenatally
- booking at a hospital that is able to provide interventional radiology, cell salvage and surgical expertise
- maximising iron stores and knowing the placental site antenatally
- consultant obstetrician and anaesthetist involvement at birth with preference being given to a vaginal birth
- active management of the third stage of labour
- prompt investigation and treatment of bleeding
- pharmacological interventions.

Additionally, these guidelines stress the importance of open communication, respecting the woman's wishes and supporting the relatives and the staff if the woman dies (Lewis G (ed.) 2004).

Key learning points

- Placenta praevia accreta can be expected to be encountered more commonly by obstetricians throughout the world with the trend of rising caesarean section rates globally (Weerasekera 2000).
- Prevention and treatment of PPH includes:
  - preventing antenatal anaemia
  - discussing the management of the third stage of labour antenatally so that the woman and her family can make informed decisions
  - avoiding episiotomies unless necessary
  - all units having protocols for massive haemorrhage with regular multidisciplinary emergency drills
  - uterine atony treated with bimanual compression of the uterus and oxytocic agents

- compression of the abdominal aorta to be tried while waiting for further treatment (American Academy of Family Physicians 2000; Hall 2004)
- there are increasing reports in the literature of the application of uterine compression sutures when treating PPH; several different techniques are described (Mousa & Alfirevic 2003)
- additional measures include radiological embolisation of the bleeding vessels, radical surgery or ligation of the uterine or internal iliac arteries (Allam & B-Lynch 2005).

- Thorough assessment of any pregnant woman presenting with abdominal or back pain of unknown origin, must include the possibility of a leaking or ruptured abdominal or thoracic aneurysm.

Resources:

- NSW Health recommends active management of third stage of labour for all women (NSW Health 2002). The framework for prevention, early recognition and management of PPH can be found on their website <http://www.health.nsw.gov.au/policies/PD/2005/PD2005_264.html>.
- A RANZCOG statement entitled Management of the third stage of labour (RANZCOG 2003) recommends active management according to standard regimens. It can be found on their website <http://www.ranzcog.edu.au/publications/collegestatements.shtml>.
- The UK Guidelines for the management and treatment of obstetric haemorrhage in women who decline blood transfusion (Lewis 2004). The guidelines can be found at <http://www.cemach.org.uk/publications.htm>.

Infection

Introduction

Maternal mortality due to infection has slowly declined in the developed world and deaths from puerperal sepsis, septic abortion and post-operative sepsis are now rare. Improved socioeconomic conditions, increased access to safe termination of pregnancy, improved aseptic techniques and the development of antibiotics have all contributed to this decline (De Costa 2002). Group A streptococcus or streptococcus pyogenes has been the organism responsible for many puerperal infections along with many other organisms including staphylococci, gonococci, anaerobes, other streptococci and gram negative organisms.

In the developing world, the rise in numbers of pregnant women with HIV/AIDS is influencing rates of infection in pregnancy. Internationally, more than half of the 30 million adults living with HIV/AIDS are women of child-bearing age. During pregnancy these women are more at risk of other infections, in particular urinary tract infections, bacterial pneumonia and any of the HIV-related opportunistic infections. There have been no reported maternal deaths from HIV/AIDS in Australia.

Summary of the findings for 2000–2002

In the 2000–2002 triennium there were 19 cases in which infection was judged to be the principal or contributory cause of death (Table 32). Five of these deaths were classified as direct maternal deaths and ten as indirect maternal deaths. An additional indirect death was a late death (48 days postpartum). Three of the deaths were associated with asthma, which is discussed in more detail in the chapter *Other indirect causes of maternal death*. Three additional deaths had infection as a contributing cause; one was a direct death due to an epidural abscess and is presented in the chapter on *Deaths associated with anaesthesia*. The other two were indirect deaths including one due to a pulmonary embolus and one which was a cardiac death.

The median age of the 16 women with infection as the principal cause of death was 30 years (range 19–40 years). Three of the women were primigravida, eight between one and four previous pregnancies resulting in a birth, three had five or more previous pregnancies resulting in births and this information was unavailable in two cases. Two women died antenatally at 25 and 32 weeks gestation, seven died four or less days postpartum and seven died 8–48 days postpartum.

Table 32: Maternal deaths due to infection, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Organism / comments	Method of birth and birth outcome
Direct				
<20	Infection, septicaemia	Liver failure, pre-eclampsia, possible acute fatty liver	*Escherichia coli* urinary tract infection	Emergency C/S, live birth
20–34	Uterine infection, pneumonia, septicaemia	Thrombosis and thromboembolism	Group A streptococcus Homebirth without medical or midwifery involvement for personal reasons	Spontaneous vaginal birth, live birth
20–34	Infection, septicaemia	Caesarean associated	Septic shock secondary to faecal peritonitis	Elective C/S, live birth
≥ 35	Infection, septicaemia		Streptococcus pyogenes	Spontaneous vaginal birth, live birth
20–34	Infection, septicaemia		Streptococcus pyogenes	Spontaneous vaginal birth, live birth
≥ 35	Anaesthesia associated	Infection, suppurative basal meningitis	Epidural abscess; methicillin resistant *staphylococcus aureus*.	Elective C/S, live birth
Indirect				
20–34	Infection, septicaemia and pneumonia	Asthma, acute fatty liver, perforated duodenal ulcer	*Staphylococcus aureus* and *Haemophilus influenzae* A	Undelivered
20–34	Infection, septicaemia	Previous splenectomy, pulmonary and pelvic thromboemboli	Group A streptococcus	Elective C/S, live birth
20–34	Infection	Multi-organ failure, coagulopathy	Hepatic necrosis secondary to fulminating herpes simplex II infection	C/S, live birth
20–34	Infection	Pulmonary embolism	Herpes simplex encephalitis	C/S, stillbirth
20–34	Infection, pneumonia	Asthma, adult respiratory distress syndrome	Influenza A	Elective C/S, live birth
20–34	Infection, pneumonia	Asthma, pulmonary oedema, multi-organ failure	*Staphylococcus aureus*	Elective C/S, live birth
20–34	Infection, septicaemia	Early pregnancy death, coagulopathy, adult respiratory distress syndrome	*Plasmodium falciparum* Malaria	Spontaneous abortion
≥ 35	Infection, meningitis	Thrombocytopenia, cerebral infarction	Cryptococcus neoformans	Emergency C/S, twins live birth
20–34	Infection, meningitis	Cerebral oedema, diabetes insipidus, chronic left otitis media, pneumonia, sepsis	Pneumococcal meningitis, *Streptococcus pneumoniae*	Undelivered
20–34	Infection, septicaemia	Severe focal coronary atherosclerosis, thrombocytopenia, multi-organ failure	*Pseudomonas aeruginosa* gram negative endotoxic shock	C/S stillbirth
20–34	Pulmonary embolism	Infection, bilateral bronchopneumonia		Undelivered
≥ 35	Cardiac	Infection, myocarditis or postpartum cardiomyopathy	Possibly viral	Induced, C/S, live birth
Late indirect				
20–34	Infection, pneumonia	Asthma, adult respiratory distress syndrome, multi-organ failure	*Staphylococcus aureus*, died 48 days postpartum	Stillbirth twins

Illustrative cases

Sepsis

A primigravida was admitted to her local hospital at 28 weeks gestation with excessive vomiting and dehydration. A provisional diagnosis of a urinary tract infection was made and a mid-stream urine (MSU) was sent to pathology. She was commenced on the intravenous antibiotic cephalothin; however, only one dose was given before she discharged herself from hospital on the day after admission. No further antibiotics were given to the woman. The MSU subsequently grew *Escherichia coli*. The woman presented three days later in preterm labour and was transferred to a tertiary hospital. An emergency caesarean section was performed for fetal distress. She was administered cephalothin intra-operatively.

Post-operatively she was investigated for pre-eclampsia and acute fatty liver of pregnancy. Her condition deteriorated with abnormal liver function tests, neurological impairment, respiratory failure and hypotension which failed to respond to treatment. Incomplete treatment of urinary tract infection at first admission, and failure to identify a urinary tract infection causing overwhelming sepsis on second admission, were contributing factors.

Principal cause: *Escherichia coli* septicaemia.

Contributing causes: Liver failure, pre-eclampsia, possible acute fatty liver.

Maternal death classification: Direct.

A woman with a history of splenectomy had a caesarean section at term for an unstable lie. She received a single dose of peri-operative prophylactic cephalosporin during the procedure. In the days following surgery she developed septicaemia due to group A streptococcus and despite transfer to a tertiary hospital on the fourth post-operative day she died of overwhelming sepsis.

Principal cause: Group A streptococcal septicaemia.

Contributing causes: Pulmonary, pelvic thromboembolus.

Maternal death classification: Indirect.

Puerperal sepsis

A woman presented hospital six days postpartum with fever (39.4°C), rigors and myalgia. She was sent home following a diagnosis of the 'flu'. She re-presented the following day with hypotension (70/50 mmHg) and swelling of her arms. Over the next 24 hours the swelling increased markedly with bloody blisters appearing. She was transferred to an intensive care unit with a provisional diagnosis of septic shock, possible toxic shock and rhabdomyolysis. She was treated with an adrenaline infusion and intravenous haemaccel, ampicillin, metronidazole and gentamycin. As her condition deteriorated she was transferred to a tertiary hospital. On arrival, her pulse was 150 and respiratory rate >40; swollen oedematous arms with right ulnar nerve palsy; right leg massively swollen, blistered and purpuric; unable to feel or move left leg and suprapubic tenderness. She required intubation and ventilation and developed ventricular arrhythmias. Her condition continued to deteriorate and resuscitation was unsuccessful. The postmortem report stated that blood cultures subsequently grew streptococcus pyogenes.

Principal cause: Streptococcus pyogenes septicaemia.

Maternal death classification: Direct.

A woman had spontaneous onset of preterm labour at 32 weeks gestation and gave birth to a live infant. On the following day she complained of dizziness and 'after-pains', which were relieved by oral analgesia. The pains increased late on day two and were associated with nausea and diarrhoea. Over the next two days her condition deteriorated. The woman was transferred to intensive care where she became progressively tachycardic, hypotensive and peripherally cyanosed with a purpuric rash. Despite intensive resuscitation attempts including multiple antibiotics (ceftriaxone, penicillin, metronidazole, gentamycin), volume replacement, intubation, ventilation, inotropes and haemodialysis she developed multi-organ failure. She died four days following birth and blood cultures grew streptococcus pyogenes.

Principal cause: Streptococcus pyogenes septicaemia.

Maternal death classification: Direct.

Pneumonia

A woman with a history of smoking 15–20 cigarettes per day and daily marijuana use had been prescribed a salbutamol inhaler for asthma in the previous year. She had minimal antenatal care and was suffering from respiratory symptoms with a cough and black sputum. She had been advised to rest and take cold and flu tablets, and ibuprofen. She died at home, undelivered at 35 weeks gestation. The autopsy found that death was the result of pneumonia and staphylococcus aureus septicaemia.

Principal cause: Infection: septicaemia and pneumonia.

Contributing cause: Acute fatty liver of pregnancy.

Terminal event: Perforated acute bleeding duodenal ulcer.

Maternal death classification: Indirect.

Meningitis

A woman aged over 34 years developed headaches, vomiting, slurred speech and an unsteady gait at 29 weeks gestation. At 32 weeks gestation she presented in preterm labour and was transferred from a regional hospital to a tertiary hospital for an emergency caesarean section of healthy twins. Postpartum the woman's neurological symptoms worsened and cryptococcus neoformans meningitis was diagnosed. Despite active management with amphoterocin, flucytosine and intensive therapy support, she deteriorated and died on day 18 postpartum.

Principal cause: Infection: meningitis.

Contributing causes: Thrombocytopenia, cerebral infarction.

Maternal death classification: Indirect.

Herpes simplex

The herpes simplex virus was the cause of two deaths during the 2000–2002 triennium. One was associated with herpes simplex encephalitis and the other with hepatic necrosis secondary to fulminating herpes simplex II infection.

A woman presented with fever and malaise at 31 weeks gestation. She progressively developed abnormal liver function tests and persistent pyrexia, unresponsive to antibiotics. Over the subsequent nine days her condition deteriorated and she was transferred to another hospital. The woman developed encephalopathy, coagulopathy and, following an abnormal

cardiotocograph, had an emergency caesarean section with a liveborn infant. Liver biopsy showed fulminant hepatic necrosis with herpes simplex II infection. Despite intensive multidisciplinary input she progressively deteriorated with fulminant herpes simplex II virus, hepatitis, hepatic failure, coagulopathy, enterococcal bacteraemia and multi-organ failure.

Principal cause: Hepatic necrosis secondary to fulminating herpes simplex II infection.

Maternal death classification: Indirect.

Malaria

A woman was admitted to hospital at six weeks gestation on return from South-East Asia. She had a four-day history of a febrile illness and had not taken antimalarial prophylaxis, due to pregnancy. *Plasmodium falciparum* was detected on thick film and she was treated with quinine. She was hospitalised with thrombocytopenia, coagulopathy and hepatitis and was given fresh frozen plasma and platelets. She had a spontaneous abortion on day two of the admission and experienced ongoing vaginal bleeding. She developed shortness of breath on day four and a chest X-ray showed bilateral pleural effusions and lower lobe infiltrates, and acute respiratory distress syndrome was diagnosed. Sudden deterioration with cardiac arrest led to intensive resuscitation but severe hypotension could not be reversed. She died on day four post spontaneous abortion.

Principal cause: Septicaemia.

Contributing cause: Malaria.

Maternal death classification: Indirect.

Clinical comment and key learning points

Infection is a major cause of maternal mortality in Australia. Early and systematic investigation of non-specific symptoms in pregnant women is necessary in order to exclude sepsis. It must be remembered that, even with excellent management, not all deaths from infection can be prevented.

Sepsis

Sepsis should be suspected in any postpartum woman who becomes febrile and feels unwell. Following appropriate cultures, broad-spectrum antibiotic treatment must be instituted if sepsis is suspected, with early involvement of a multidisciplinary team. Full courses of broad-spectrum antibiotics should be used in situations of suspected or established sepsis. Single-dose antibiotic prophylaxis should not be confused with therapeutic regimens.

Pneumonia

Difficulties in diagnosis and subsequent delay in appropriate treatment may reflect the difficulty distinguishing between symptoms related to physiological changes associated with pregnancy and symptoms due to pneumonia. Whilst the use of X-ray is generally not recommended during pregnancy the potential benefit of such measures should always be weighed up against the risks of not using them in serious clinical situations (Lowe 2004). Penicillins, macrolides and cephalosporins are the main antimicrobials recommended for treatment of antepartum community-acquired pneumonia (Yost et al. 2000).

Herpes simplex

Herpes simplex hepatitis is difficult to diagnose, can be fatal and is thought to be exacerbated by the impaired immunity of pregnancy (Kaufman et al. 1997). Diagnosis is made by liver biopsy with fever, nausea and vomiting, abdominal pain, leukopaenia, thrombocytopenia, coagulopathy and a marked rise in serum transaminase are usually present. Early diagnosis and treatment with specific antiviral medication may prevent progression and mortality.

Malaria

Pregnant women are at greater risk of malaria than non-pregnant women (Brabin & Verhoeff 2002). In areas that have high levels of malaria transmission, women will often have some immunity but are still vulnerable to the complications of this infection including anaemia, hypoglycaemia, central nervous system complications, pulmonary oedema, pregnancy loss, sequestration of parasites in the placenta, preterm birth, low birthweight and high infant mortality (WHO 2004). Women without pre-existing immunity to malaria have a higher risk of developing serious malarial infection which can result in maternal death (WHO 2004). The most lethal malaria parasite is *plasmodium falciparum*; infection with this organism in a non-immune pregnant woman can lead to rapid death (Brabin & Verhoeff 2002, WHO 2004). The increased risk of acquiring infection and developing severe disease persists for at least 70 days postpartum (Diagne et al. 2000).

Advice about antimalarials in pregnancy is very complex and depends on gestation, region of travel and planned activities at destination (related to exposure). Generally, pregnant women should be advised to avoid travel to areas where they may be exposed to malaria. If such exposure is unavoidable they should receive specialist advice regarding the risks of travel and methods for minimising the risk of malaria infection including mosquito avoidance measures and prophylactic antimalarials which are safe in pregnancy.

Prophylaxis is usually commenced two weeks prior to entering a malarious area and continued for four weeks after leaving (Yung et al. 2004). Where indicated, women should also be educated regarding symptoms and signs of malaria so they can seek early medical attention and treatment.

Splenectomy

Asplenic patients are at risk of overwhelming sepsis and can progress to irreversible shock within hours. Recognition of the susceptibility of an asplenic patient is important so early investigation and early administration of appropriate antimicrobial therapy can ensue. The increased risk of infection remains life long after a splenectomy.

Cardiac disease

Introduction

The prevalence of cardiac disease in pregnancy is reported to vary from 0.4% to 4.0% of all pregnancies and is a leading cause of maternal mortality in the United Kingdom (UK), United States of America (USA) and Australia (Ramsey et al. 2001). The maternal mortality rate from cardiac disease in the UK declined from the 1950s to the mid–1980s, mostly resulting from the decreasing incidence of rheumatic fever, which was the principal cause of 84% of cardiac deaths in 1952–1954 but which did not cause any deaths in 2000–2002 (de Swiet & Neilson-Piercy 2004).

In contrast to the fall in deaths from rheumatic heart disease, there has been a rise in deaths due to ischaemic, congenital and other cardiac disorders. Since the 1980s improved medical treatment and surgery for women with congenital cardiac disease has enabled such women to survive into reproductive age (Lupton et al. 2002). The increase in women with cardiac disease becoming pregnant has led to an increasing number of maternal deaths related to cardiac disease. This is likely to continue and worsen as maternal age increases and as women with serious congenital lesions, partly corrected in early life, become pregnant.

The cardiovascular changes that occur in normal pregnancy include a major increase in blood volume and cardiac output, 40% in singletons and more for women with multiple pregnancies. For a woman with pre-existing cardiac disease, these increases add further burden to an already compromised heart. In addition, anaemia, infections, hypertension and major surgery (for example caesarean section) are common in pregnancy and add further to cardiac demands (Ramsey et al. 2001).

Women with previous cardiac surgery and prosthetic valves in situ, particularly the older valves in the mitral position, are at increased risk antenatally of a poor outcome. Anticoagulation is necessary; however warfarin is not usually recommended at any stage of pregnancy, although its dangers for the fetus are less after the first trimester. It is usually replaced with heparin or low-dose heparin which, while having no fetal side effects, is less effective in providing adequate anticoagulation and reducing maternal complications (Lupton et al. 2002). There have been many cases of serious thrombotic events arising from prosthetic valves, despite the use of effective doses of unfractionated and low molecular weight heparin (Lupton et al. 2002).

Summary of the findings for 2000–2002

Cardiac disease has been the leading cause of indirect maternal mortality in Australia over the 30 years from 1973–2002. There were 15 deaths in the current triennium in which cardiac disease was judged to be the principal cause of death (Table 33). Fourteen of these 15 deaths were classified as indirect deaths and four of these deaths were late maternal deaths. One late maternal death, where cardiac failure resulted from peripartum cardiomyopathy, was classified as a direct death. Twenty-one per cent of all indirect deaths (excluding late deaths) were due to cardiac conditions (n=11). There were two deaths during the 2000–2002 triennium in women with rheumatic heart disease.

The median age of women who died from a cardiac condition in 2000–2002 was 32 years (range 25–43). Four women died antenatally and the remaining 11 deaths occurred in the postpartum period (one following a perimortem caesarean section).

Table 33: Maternal deaths due to cardiac disease, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Parity[(a)]	Gestation (weeks)	Method of birth and birth outcome	Days post-partum
Indirect						
20–34	Myocardial ischaemia possible arrythmia	Systemic lupus erythematosus, coronary artery disease, myocarditis	M	10	Twin pregnancy fetal death in utero, undelivered	. .
20–34	Cardiac failure, pulmonary oedema	Asthma, atrial septal defect repaired at 16 years	M	16	Not delivered	. .
20–34	Cardiomegaly, cardiogenic shock	Atrial septal defect, mitral valve repair, sick sinus syndrome, pacemaker	P	32	Emergency C/S, live birth	2
20–34	Cardiac failure	Congenital heart disease, transposition of the great vessels, ventricular septal defect	M	26	Not delivered	. .
20–34	Myocarditis, cardiac arrhythmia	Undiagnosed heart disease, cardiac hypertrophy, acute heart failure	n.s.	34	Not delivered	. .
20–34	Aortic dissection		P	40	Induced, emergency C/S fetal retrieval, live birth	< 1 hour
20–34	Rheumatic heart disease cardiogenic shock	Died during valve replacement, left ventricular failure	M	28	Emergency C/S at 28/40, live birth	1
20–34	Aortic arch dissection	Marfan's syndrome	P	n.s.	Elective C/S, live birth	8
≥ 35	Myocardial infarction		M	n.s.	Elective C/S, live birth	8
20–34	Ischaemic heart disease		n.s.	n.s.	Not stated, live birth	19
≥ 35	Myocarditis or postpartum cardiomyopathy	Possible viral infection	P	n.s.	Induced, emergency C/S, live birth	34
Late direct						
20–34	Cardiac failure	Peripartum cardiomyopathy	M	Term	Induced, vacuum extraction, live birth	78
Late indirect						
20–34	Peripartum cardiomyopathy with cardiac failure	Rheumatic heart disease, mitral valve replacement, chronic atrial fibrillation	P	38	Elective C/S, live birth	102
≥ 35	Severe pulmonary hypertension	Mixed connective tissue disease, Raynaud's disease	P	n.s.	Emergency C/S, live birth	135
≥ 35	Cardiac tamponade	Hypertension in pregnancy, haemo-pneumothorax 18 months prior to death	P	35	Emergency C/S, live birth	143

(a) P = primipara, M = multipara.

n.s. Not stated.

. . Not applicable.

In the previous triennium (1997–1999), 40% of all indirect deaths (n=7) were due to cardiac disease and one direct death was due to puerperal cardiomyopathy. In a further two indirect deaths, cardiac conditions were contributing factors.

Illustrative cases

Congenital heart disease

A woman had a history of congenital heart disease with previous repair of both an atrial septal defect and the mitral valve. She had been diagnosed with sick sinus syndrome and treated with a pacemaker as she experienced chronic atrial fibrillation. At 32 weeks of pregnancy she developed vomiting, anorexia, shortness of breath and palpitations. Her liver function tests had deteriorated and an ultrasound of the liver showed congestive changes consistent with right heart failure. She underwent an urgent caesarean section. During the operation she experienced episodes of bradycardia and decreased cardiac output requiring two episodes of cardiopulmonary resuscitation. Her condition deteriorated and intensive resuscitation was unsuccessful. Postmortem examination confirmed significant underlying heart disease with moderate cardiomegaly. Her underlying heart condition had resulted in chronic passive venous congestion of the liver and fibrosis.

Cause of death: Cardiac failure.

Contributing causes: Congenital heart disease, sick sinus syndrome.

Maternal death classification: Indirect.

A woman had surgery in early childhood for congenital transposition of the great vessels and a ventricular septal defect. She attended a routine antenatal visit at 26 weeks of pregnancy and no abnormal symptoms were documented. The following day she experienced a sudden episode of breathlessness, collapsed and died. The autopsy stated she had cardiac failure caused by congenital heart disease which was exacerbated by the cardiovascular stresses of pregnancy. The presence of cardiac enlargement and fibrosis suggested that there had been longstanding impairment of cardiac function.

Cause of death: Cardiac failure.

Contributing causes: Congenital heart disease, transposition of the great vessels, ventricular septal defect.

Maternal death classification: Indirect.

A woman in her first pregnancy had an uneventful pregnancy and elective caesarean section. She was known to have Marfan's syndrome and was discharged from hospital in apparently good condition. Five days postpartum she developed chest pain and sensory changes to her left leg. An aortogram showed a dissecting thoracic aortic aneurysm and an operation for an aortic stent was uneventful. Cardiac arrest occurred 24 hours post-operatively and resuscitation was unsuccessful. A postmortem was not performed.

Cause of death: Aortic arch dissection.

Contributing causes: Marfan's syndrome, aneurysm of aortic arch.

Terminal event: Cardiac arrest.

Maternal death classification: Indirect.

Myocardial infarction

A woman collapsed at home eight days following an uncomplicated caesarean section. She had complained of vague chest and abdominal pain the day before her collapse and death.

The clinical diagnosis in the emergency room was massive pulmonary embolism but emergency sternotomy and surgical exploration could not identify the cause of death. The autopsy examination of the heart muscle showed minor changes consistent with myocardial ischaemia.

Cause of death: Myocardial infarction.

Maternal death classification: Indirect.

Peripartum cardiomyopathy

A woman was admitted to hospital at 37 weeks gestation with increasing dyspnoea and complained of having a cough for four weeks. She was diagnosed with cardiac failure secondary to peripartum cardiomyopathy and was transferred to a tertiary centre, where she remained untill her death, 78 days later.

Cause of death: Peripartum cardiomyopathy.

Maternal death classification: Indirect.

Clinical comment and best practice

The contribution of cardiac disease to maternal mortality will require continuing consideration in subsequent reports. As more women with 'corrected' congenital heart disease and older women with ischaemic heart disease (related to diabetes, hypertension, obesity and smoking) present for antenatal care, complications are likely. There are increasing numbers of women delaying childbearing until aged 35 years or older. This has resulted in increasing numbers of women presenting in pregnancy with acquired heart disease including ischaemic heart disease. Early multidisciplinary specialist care including a cardiologist and/or obstetric physician and consultant obstetrician is recommended for pregnant women with cardiac disease. This did not appear to occur for several women presented in this chapter.

Peripartum cardiomyopathy is usually diagnosed when a woman develops congestive heart failure during the last month of pregnancy or first five months postpartum, in the absence of pre-existing heart disease and when no other cause of the cardiac failure can be found (Ramsey et al. 2001). Peripartum cardiomyopathy is more common in older, obese, hypertensive, multiparous women who present with signs of cardiac failure, often in the first three months postpartum (Ramsey et al. 2001; de Swiet & Neilson-Piercy 2004). Clinicians should be alert for signs of increasing fatigue, tachycardia, tachypnoea, dyspnoea or pulmonary oedema (Ramsey et al. 2001). Maternal mortality for women diagnosed with this condition ranges from 25–50%. For those women who recover, there is a high rate of recurrence in further pregnancy (Elkayam 2001). For these women, extensive evaluation and preconception counselling with a specialist is required, with advice usually against further pregnancy.

Key learning points

- Pre-pregnancy counselling should be offered to all women with cardiac disease who are considering pregnancy. Important factors include the specific cardiac lesion, a history of surgical correction and the pre-pregnancy functional cardiac status.

- Pregnant women with cardiac disease require close monitoring by a cardiologist and/or obstetric physician and an experienced obstetrician throughout the pregnancy, birth and postpartum period.
- Anaemia should be prevented in women who have cardiac disease in pregnancy.
- Isolated systolic hypertension should not be ignored in women with cardiac disease.
- Termination of pregnancy services need to be readily available for women whose cardiac condition places them at risk from pregnancy.
- It is important to consider dissecting aortic aneurysm in women presenting with atypical chest pain.
- Warfarin is classed as a Category D drug in pregnancy and is contraindicated. It has been associated with a specific embryopathy following exposure at 6–9 weeks post conception.
- Low molecular weight heparin must be administered in an adequate dose and frequency. This is particularly important for women with prosthetic aortic valves, which are known to predispose to thrombosis.
- Pulmonary hypertension is an ominous risk factor in pregnancy with high risk of mortality despite appropriate treatment.

Amniotic fluid and air embolism

Introduction

Amniotic fluid embolism (AFE) is a dramatic, rare and often fatal condition that is a major cause of maternal mortality in Australia and other developed countries. The diagnosis of AFE is based on clinical evidence as there is no specific laboratory test or feature that distinguishes it from other conditions (Tuffnell 2002). In many cases a characteristic series of events may be highly suggestive of the diagnosis, and swift recognition, treatment and transfer to an intensive care unit may improve the outcome.

In the past, the mortality rate was around 60–80%, however more recent publications report lower mortality rates of 16–30% (Tuffnell 2003; Vlies 2004; Marcus et al. 2005). A population-based study in California identified 53 cases of amniotic fluid embolism among 1,094,248 births (an incidence of just over 1 in 20,000). The maternal mortality rate was 26% with 87% of the survivors recovering fully by the time of discharge and 13% requiring long-term follow-up for some residual problem (Gilbert & Danielsen 1999; Tuffnell 2003). In women who survive a clinical episode of AFE it can be very difficult to establish the diagnosis with certainty.

Amniotic fluid embolism is considered to have the characteristics of an anaphylactic reaction, usually manifesting as a severe systemic response to the presence of material of fetal origin from the amniotic fluid, in the maternal circulation. A rise in tryptase levels may be seen, perhaps in response to fetal antigens in the maternal circulation (Marcus et al. 2005). Clinical presentation typically begins in labour with the sudden appearance of one or more of an acute cardio-respiratory collapse, cyanosis, hypotension, confusion, a seizure and/or dyspnoea. This will usually be followed within minutes by haemorrhage and severe coagulopathy leading to DIC. Sudden profound fetal distress is usual, compelling the need for urgent delivery at a time when the woman is already severely compromised (Davies 2001).

Summary of the findings for 2000–2002

Over the past 12 triennia in Australia, AFE has remained a leading cause of maternal mortality. In the two most recent triennia (1994–1996, 1997–1999) AFE has been the principle cause of death for 15 women.

In the 2000–2002 triennium there were 11 deaths associated with AFE. Ten were direct deaths in which AFE was judged to be the principal cause of death; one other direct death had AFE as a contributing cause of death with the principal cause being haemorrhage from a ruptured uterus (Table 34). In one of these cases AFE seemed the most likely cause of death but it was possible that air embolism also occurred and contributed significantly to the death. In every case AFE was diagnosed on autopsy. The women were aged between 20 and 39 years, with a median age of 30 years.

Labour had been induced in three women, two with prostaglandins, and documentation was unavailable in the third case. A fourth woman collapsed following artificial rupture of the membranes in labour. Information on induction or augmentation was not available in two cases and not applicable in four cases where the women had caesarean sections without labour. Complications included two women with placenta praevia, both of whom

experienced minor bleeding several times during the pregnancy, and another woman who presented at 32 weeks with an abruption.

Fetal death and severe morbidity are common in this condition. However, this was not evident in Australia during the 2000–2002 triennium, as all women had liveborn infants with one set of twins being born. One baby, born following caesarean section, did not survive and was classified as a neonatal death.

The UK, with 2.6 times the number of births of Australia in 2000–2002, has seen a significant decrease in AFE from 17 deaths in the 1994–1996 triennium to eight in 1997–1999 and five in 2000–2002 (Vlies 2004). This trend has not been seen in Australia to date.

Table 34: Maternal deaths due to amniotic fluid embolism, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Comments	Parity[(a)]	Gestation (weeks)	Method of birth and birth outcome
Direct						
20–34	AFE	Haemorrhage, DIC	Placenta praevia, no labour	M	36	Emergency C/S, live birth
20–34	AFE	Haemorrhage, DIC	Spontaneous labour	P	n.s.	Vacuum extraction, live birth
20–34	AFE	Haemorrhage, DIC, possible air embolism	Previous caesarean	M	38	Elective C/S, live birth
20–34	AFE	Haemorrhage, DIC	Induced	P	n.s.	Spontaneous vaginal birth, live birth
20–34	AFE		Induced	P	41	C/S for fetal retrieval, neonatal death
20–34	AFE	Haemorrhage, DIC	APH	P	32	Emergency C/S, live birth x 2
20–34	AFE	Haemorrhage	Spontaneous labour	GM	36	Spontaneous vaginal birth, live birth
20–34	AFE	Haemorrhage, DIC	Spontaneous labour, artificial rupture of membranes	P	Term	Emergency C/S, live birth
≥ 35	AFE		Previous caesarean	M	40	Emergency C/S, live birth
≥ 35	AFE	Haemorrhage, DIC	Placenta praevia, no labour	P	≥ 34	Emergency C/S, live birth
≥ 35	Haemorrhage uterine rupture, PPH	AFE, DIC	Induced	M	40	Spontaneous vaginal birth, live birth

(a) P = primipara, M = multipara, GM = grand multipara.

n.s. Not stated.

Six of the 11 cases were complicated by DIC, with some women progressing to multi-system failure. Information was available on the timing of death in nine of the 11 cases. Seven women died within a day of birth, with five of these being less than five hours postpartum. One woman died 36 hours following birth and another died two days postpartum.

Illustrative cases

A woman had labour induced for post dates with prostaglandins. She collapsed in labour at 7–8 cm dilatation at the same time that her membranes ruptured. Her condition deteriorated rapidly with a cardiac arrest. An urgent caesarean section was performed for a live infant who later died. The postmortem examination showed (fetal) squames and fat in the capillary vessels in the lungs. No other cause of sudden death was identified. This woman died less than an hour from onset of symptoms, without bleeding or coagulopathy.

Cause of death: AFE, diagnosed at autopsy.

Maternal death classification: Direct.

A woman had artificial rupture of the membranes, in labour, at term. Immediately following she became agitated and fetal distress was diagnosed. She had an emergency caesarean section but developed severe PPH and coagulopathy. Despite return to theatre the bleeding could not be stopped and she died within 12 hours of onset of symptoms.

Cause of death: AFE, diagnosed at autopsy.

Contributing causes: PPH, coagulopathy.

Maternal death classification: Direct.

A woman with a history of previous caesarean section was scheduled for an elective caesarean section as she was reported to have a large baby. A caesarean section was performed at 38 weeks following spontaneous onset of labour. A cardiac arrest occurred after delivery of the placenta. She developed coagulopathy and was unable to be resuscitated, dying three-and-a-half hours after admission.

Cause of death: AFE, diagnosed at autopsy.

Contributing causes: PPH, coagulopathy, possible air embolism documented on autopsy report.

Maternal death classification: Direct.

Clinical comment

The AFE syndrome is a clinical diagnosis that must be made promptly, usually in the absence of confirmatory information, if the affected woman is to survive. Survival in a severe case relies on anticipation of major haemorrhage when the characteristic premonitory signs appear, preparation for and management of the bleeding, coagulopathy and hypoxia, and timely transfer to an intensive care unit where the cardio-respiratory consequences can be dealt with. Evidence from the UK AFE register shows that survival following a cardiac arrest can occur, with some women recovering quickly following the acute episode (Tuffnell 2005).

Successful resuscitation requires a swift response from the resuscitation team. Every maternity unit should have a 'disaster plan' to deal with such cases and drills should be held at regular intervals, with appropriate codes in place that minimise delay in managing this critical situation. Full resuscitation should be activated as soon as a woman collapses, which usually precedes the second phase of the syndrome (major bleeding) by a critical period of several minutes. During this time senior staff may be summoned, large cannulae inserted and blood ordered. Most of these cases arise unexpectedly, after a normal pregnancy and a normal, but often induced, labour (Clark et al. 1995).

A recent publication discusses the association between uterine stimulants and AFE (Wagner 2005). Wagner has critically reviewed studies that stated there was no direct association between the use of uterine stimulants and the incidence of AFE (Wagner 2005). He describes methodological errors in these studies, contesting the results and concluding that they do allow for a hypothesis of a causal relationship. He based this hypothesis on higher rates of uterine stimulant usage in women who experienced AFE, than the rates seen in the general population. Wagner (2005) urges caution and further research in the current climate of rising rates of inductions, increasing use of prostaglandins and maternal mortality rates which are not decreasing markedly in the developed world. Despite these comments, AFE is also seen after spontaneous labour, and may occur without uterine stimulation.

An autopsy diagnosis of AFE can be challenging, often requiring a pathologist with special training in obstetric pathology and the use of the appropriate stains to show the presence of mucin and fetal squames in the pulmonary arterioles. The following information was quoted in two of the autopsy reports received during the 2000–2002 triennium:

> *As noted by Attwood 'whenever there is uterine tear there is the possibility of amniotic fluid embolism. Paradoxically, it is the small incomplete lower uterine tears which are associated with lethal amniotic embolism'. It is the small incomplete tear in the lower uterine segment, which is bare of membranes at the time of crowning of the head, which is thought to be the most likely portal of entry of amniotic fluid into the maternal circulation. Such defects can be easily missed at autopsy. 'The diagnosis of AFE should always be considered as a possible cause of sudden unexpected death in late pregnancy, during parturition or in the early puerperium. Histologically, the components of amniotic fluid embolism are epithelial squames, lanugo hairs, fat derived from vernix caseosa, mucin and bile derived from meconium. Such findings can however, occur in cases of non-lethal amniotic fluid embolism and thus the autopsy findings must always be correlated with the clinical history' (Attwood 1972).*

Key learning points

- AFE must be suspected in any woman who becomes hypotensive, cyanosed, breathless, displays an altered mental state, loss of consciousness, coagulopathy or seizure in labour.
- All staff should be aware that any sudden serious change in maternal or fetal status may be an early warning sign of AFE. Early recognition and referral to senior staff is necessary, as is immediate resuscitation.
- Every maternity unit should have a 'disaster plan' to deal with emergencies. Drills should be held at regular intervals, with appropriate codes in place that allow no delay in managing critical situations.

The NPSU is developing a national database for women who have been diagnosed with an AFE, to monitor outcomes, predisposing factors and treatment.

Deaths from psychiatric causes

Introduction

A maternal death from psychiatric causes is one in which the psychiatric condition was a major contributor or cause of death and where the death would not have occurred if the woman had not been suffering from a psychiatric disorder (Oates 2002). Mortality from psychiatric causes in pregnancy, the puerperium and the 12-month period following the birth can encompass the wider issues of domestic violence and substance abuse.

In the past, deaths from psychiatric causes, apart from puerperal psychosis, were classified as incidental deaths. In line with recommendations from the CEMACH, Australia has classified these deaths as indirect since 1997. Because of this shift in classification from incidental to indirect, comparisons with triennia prior to 1997 must be undertaken with caution.

It is estimated that 15–25% of women will develop a significant mental health problem between conception and 12 months postnatally (Priest et al. 2005). Other epidemiological studies show similar rates of depressive symptoms or disorders both antenatally and postnatally (Evans et al. 2001), with the risk of severe mental illness more common during the three-month period following birth (Oates 2004).

In Australia, beyondblue the national depression initiative's National Postnatal Depression Program has been a four-year (2001–05) public health initiative that has involved multidisciplinary teams, the screening of 40,000 women antenatally and 12,000 women postnatally, and a large education and publicity campaign across the country (Buist & Bilszta 2005).

Key preliminary findings include:

- In Australia, rates of depression as recorded on a validated, perinatal specific screening tool, the Edinburgh Postnatal Depression Scale (EPDS), are high in the general postnatal population (15.7%), similar to rates in other Westernised countries.
- Antenatal rates of depression appear lower than postnatal rates (5.4–8.9% depending on cut-off score used) and, when trimester screened is considered, they are similar to other Westernised countries.
- For the first time, the importance of psychosocial risk factors is confirmed for the general Australian perinatal population. In particular, past history of abuse, prior history of depression, anxiety, lack of support, lower socioeconomic status and a stressful pregnancy are all key factors.
- Indigenous Australian women are at heightened risk of depression, and psychosocial factors are of particular importance in generating this elevated rate.
- These high rates of depression and identification of key psychosocial risk factors highlight the importance of universal perinatal depression screening.
- Provision of information and education of health professionals in depression identification and early intervention strategies must also take place (Buist & Bilszta 2005).

The risk of recurrence of severe postnatal depression, bipolar disorder and psychosis is very high, with between 20–40% of women with previous postnatal depression at risk of recurrence (Priest et al. 2005).

Data examining suicide in pregnancy suggest that although suicide rates are generally lower in pregnancy, there appear to be subgroups in whom there is an elevated risk of suicide by violent means, in particular women who are psychotically depressed (Appleby et al. 1998; Oates 2003).

In the UK, a data linkage study identified many more deaths with a psychiatric classification that had not been reported to the CEMACH. When these deaths, mostly late and indirect, were included in the mortality count, death from psychiatric causes became the leading cause of maternal mortality in the UK (Oates 2004). It is probable there is under-ascertainment of maternal deaths from psychiatric cause in Australia, particularly late maternal deaths.

Summary of the findings for 2000–2002

During the 2000–2002 triennium there were 10 deaths in which a psychiatric condition was determined to be the principal cause of death, with one being a late maternal death (Table 35). The median age of these women was 28 years (range 17–33 years), with six of the deaths occurring at 20 weeks gestation. Three deaths involved women who were reported as being Indigenous women.

Table 35: Maternal deaths from psychiatric causes, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death and comments	Gestation (weeks)	Method of birth and birth outcome
Indirect				
<20	Suicide by hanging	Alcohol and cannabis in blood and urine	7	Undelivered
<20	Suicide by hanging	Toxicology showed alcohol and toluene	7	Undelivered
20–34	Suicide by hanging	Domestic dispute	9	Undelivered
20–34	Suicide by hanging	High blood alcohol level, history of depression, substance abuse and self-harm	13	Threatened miscarriage at eight weeks, undelivered
20–34	Suicide by hanging	Recent pregnancy loss, difficult social circumstances	16	Miscarriage
20–34	Overdose of codeine and paracetamol	Interstitial pneumonitis	20	Undelivered
20–34	Suicide by poisoning, died nine days postpartum	Probable pre-existing undiagnosed depression— evident in last pregnancy	28	Emergency C/S, live birth
20–34	Suicide by hanging, 17 days postpartum	Previous postnatal depression, failure to detect severity of postnatal depression	Term	Elective C/S, live birth
20–34	Pedestrian in motor vehicle accident, six days postpartum	Known psychiatric illness, not taking medication and discharged against medical advice	Term	Live birth
Late indirect				
20–34	Drug overdose, 49 days postpartum	Severe pulmonary hypertension and heart failure	Term	Elective C/S, live birth

In the 1997–1999 triennium there were eight indirect deaths in which a psychiatric cause was determined to be the principal cause of death and five drug-related deaths which were classified as incidental deaths.

Illustrative cases

A woman committed suicide by poisoning at 28 weeks of pregnancy. In her previous pregnancy, six years prior, she had an 'emotional episode' that prompted her husband to seek help. This had not been followed up in this pregnancy.

Principal cause: Suicide by poisoning, ingestion.

Maternal death classification: Indirect.

A woman with a history of recurrent miscarriage had a miscarriage at 15 weeks gestation. She was seen by a social worker on the day of the miscarriage but declined counselling. She was prescribed on antidepressants by her general practitioner (GP) the following day. Side-effects, including insomnia, led to changes in medication. The autopsy revealed that she was not taking her medication when she committed suicide 15 days following the miscarriage.

Principal cause: Suicide by hanging.

Maternal death classification: Indirect.

A woman had a long history of mental illness (chronic schizophrenia), had required 13 admissions to hospital, some of which were the result of failed suicide attempts. Throughout the last six months of her pregnancy she had refused all medication. She had a history of domestic problems. She experienced no obstetric complications and delivered a live, healthy infant at term. She was discharged home at the insistence of her husband, and against the wishes of the hospital, on the day after delivery. Extended midwifery services were arranged, but no psychiatric follow-up was provided. Six days after delivery the woman died from shock and haemorrhage due to multiple injuries after being hit by a truck. The state report noted the lack of psychiatric follow-up as an important issue. Other comments included inadequate evaluation prior to discharge, unavailability of services during weekend discharge and non-compliance by the patient.

Principal cause: Pedestrian in motor vehicle accident.

Maternal death classification: Indirect.

State recommendations included:

- The need to reiterate to psychiatrists and obstetric clinicians that there is no need to cease taking psychotropic drugs during pregnancy, with the exception of lithium where an alternative drug should be sought. Women attending antenatal care who have established diagnoses of schizophrenia should be provided with information about psychotropic medications in pregnancy and during breastfeeding.
- Patient's psychosocial health and support needs are frequently more complex than traditional specialist services allow, and there is a need for close working relationships between obstetric psychiatric, primary care and social services. It is preferable for psychiatric assessment where possible to be available in routine antenatal services.
- Discharge planning should focus on the vulnerability of some patients if discharged late in the week or over the weekend when most community services are understaffed. In some instances discharge early the next week may be prudent.

Clinical comment

Seven of the nine deaths from psychiatric disorders in the 2000–2002 triennium occurred antenatally, highlighting that significant psychiatric morbidity can occur during pregnancy. Antenatal psychosocial screening may have helped identify some of these women as 'at risk' or even as being symptomatic. This should then lead to a mental health review where appropriate intervention, which can include case planning with social work, psychiatry and possibly government community service agencies, can be offered. Although some of these women declined help, in some cases a follow-up phone call or appointment would have been warranted given their complex circumstances. Follow-up may include actively engaging family members, monitoring of medication, and psychiatric follow-up throughout pregnancy and the immediate postnatal period.

Several of the cases reported here involved suicide where alcohol or cannabis was found in the bloodstream. Difficult social circumstances, undiagnosed depression, failure to recognise the severity of depression and co-existing illness were other factors identified in these cases.

The beyondblue National Depression Initiative recommends a psychosocial assessment process on the basis of its recent analysis of results of over 40,000 women screened across Australia during the National Postnatal Depression Program.

Seven states and territories have been involved in the beyondblue program and each have developed their own initiatives addressing similar concerns regarding maternal morbidity and mortality from psychiatric causes (Buist & Bilszta 2005). In Queensland, the project team has been involved in initiatives with Indigenous women across several sites. This report is due to be released in 2006. In NSW the initiatives include the use of two complementary questionnaires: the psychosocial questionnaire, which is part of the comprehensive interview undertaken by all women in early stages of pregnancy and the antenatal edinburgh depression scale (EDS), which enquires about the woman's level of emotional distress and anxiety in the past seven days. In addition, the NSW Families First Program has introduced universal psychosocial screening in the first few weeks postpartum.

Best practice

Women who have a past history of serious psychiatric disorder and are on, or have recently been on, psychotropes, should be assessed by a psychiatrist in the antenatal period in order to formulate a management plan that spans the entire perinatal period.

Universal antenatal screening for psychosocial risk and symptomatology should be considered where adequate psychosocial referral pathways are available.

The beyondblue recommendations suggest:

- depression screening as a part of routine antenatal and postnatal care
- use of the EDS as the best available and most practical screening tool
- use of additional key psychosocial questions to assess risk and planning perinatal care, in particular, level of support, history of anxiety and depression and current stressors
- antenatal screening in the third trimester
- postnatal screening 6–8 weeks after childbirth
- all pregnant women are provided with an information and resource booklet on emotional health in the perinatal period

- screening programs need to be accompanied by ongoing training and support of all relevant health professionals involved in perinatal care
- each obstetric/area health service needs to develop a local care-pathway including appropriate referral and allied health service links (Buist & Bilszta 2005).

Guidelines concerning the management of depression during pregnancy are soon to be published by the Royal Australian and New Zealand College of Psychiatrists.

Guidelines (due in 2006) for perinatal psychosocial screening are being prepared in the UK by the National Institute for Clinical Excellence.

Key learning points

- It is preferable for psychiatric assessment, where possible, to be incorporated into routine antenatal care. Early routine antenatal psychosocial screening and subsequent monitoring may bring women to the attention of psychiatric services earlier, or in a more sustained way.
- While women with psychiatric problems may be identified, many do not follow through with treatment. It is especially important in those cases to engage the assistance of family members to try to improve compliance and safety.
- Domestic violence often worsens in pregnancy and may be associated with an increased risk of maternal mental health problems and self-harm. If identified, women need referral to the appropriate agencies without delay.
- Where a woman is severely depressed or psychotic, the optimum, not the minimum, dosage of medication should be used. Cessation of and inadequate psychotropic dosage in pregnancy occurs when clinicians tend to use minimum dosage to reduce fetal exposure.
- Women should be given information about psychotropic medications in pregnancy and during breastfeeding as early as possible during the pregnancy. Ideally this should occur preconception.
- The risk of recurrence with severe postnatal depression, bipolar disorder and psychosis is very high, with between 20–40% of women with previous postnatal depression experiencing a recurrence.

Resources:

- The NSW Health Multicultural Communication Unit have many resources on their website including fact sheets, health professionals guides, and the antenatal and postnatal EDS, available in a number of different languages. The website is: <http://www.mhcs.health.nsw.gov.au/health-public-affairs/mhcs/publications/Pregnancy_and_Post_Natal.html>.
- The *beyondblue* PND Program developed a range of promotional, educational and resource materials which are available on its website. The site includes information in 19 languages other than English, as well as for Indigenous women, multiple birth mothers, depression management and postnatal EDS guides for health professionals. The website is: <http://www.beyondblue.org.au/postnataldepression>.

Hypertensive disorders in pregnancy

Introduction

Hypertensive disorders of pregnancy have been a leading cause of maternal mortality for many years, responsible for approximately 13% of maternal deaths across the world (Robson 2002). There are several different ways of classifying these disorders and the definitions used in this report are those recommended in the Consensus Statement of the Australasian Society for the Study of Hypertension in Pregnancy (Brown et al. 2000). Hypertension during pregnancy may develop as a result of the pregnancy (pre-eclampsia) or it may follow pre-existing hypertension. Hypertension in pregnancy is diagnosed when systolic blood pressure (BP) is ≥ 140mmHg and/or diastolic BP is ≥ 90mmHg.

Hypertension arising for the first time after 20 weeks gestation may be an isolated finding (gestational hypertension) or part of the multi-system disorder known as pre-eclampsia. A clinical diagnosis of pre-eclampsia will include gestational hypertension and one or more of the following: proteinuria, renal insufficiency, liver disease, neurological symptoms or signs, haematological disturbances or fetal growth restriction.

The major risk factors for pre-eclampsia include first pregnancy or a multigravida who has a new partner, maternal age greater than 35 years or under 20 years, obesity, multiple pregnancy, pre-eclampsia or eclampsia in a previous pregnancy, family history of pre-eclampsia or eclampsia, and pre-existing hypertension, renal disease, autoimmune disease or diabetes (American Academy of Family Physicians 2000; Brown et al. 2000).

Eclampsia is defined as the occurrence of convulsions, in association with pre-eclampsia, during pregnancy or in the first 10 days following birth (Robson 2002).

The most common cause of death among women who progress to eclampsia is intracranial haemorrhage followed by adult respiratory distress syndrome, with other causes including pulmonary or cerebral oedema, renal or hepatic failure and haematological abnormalities (Robson 2002).

Summary of the findings for 2000–2002

During the 2000–2002 triennium there were five deaths in which a hypertensive disorder was deemed to be the principal cause of death (Table 36). Four were direct deaths and one was an indirect death. Three deaths resulted from intracerebral haemorrhage and one from a ruptured subcapsular hepatic haematoma. The fifth case involved a woman with eclampsia who had a myocardial infarction. Treatment for Hodgkin's lymphoma had resulted in chemotherapy and radiotherapy-induced heart disease.

Hypertension was a contributing cause of death in a further two cases. One was a direct death where the principal cause of death was haemorrhage at caesarean section and the second was a late direct death where the principal cause of death was TTP (also presented in the *Haemorrhage – obstetric* chapter).

The women who died of hypertensive disease as a principal cause of death were aged between 17 and 41 years with a median age of 35 years. This was higher than the median for all deaths of 30 years. Four of the five women did not reach term, with information on gestation not available in one case. Four women died in hospital. The other woman was

found dead in her bed at home. In this case an autopsy revealed extensive bilateral cerebral and brain stem haemorrhage.

Table 36: Maternal deaths due to hypertensive disorders in pregnancy, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Parity[(a)]	Gestation (weeks)	Method of birth and birth outcome	Days post-partum
Direct						
<20	Hypertension	Extensive bilateral cerebral and brain stem haemorrhage	N	32	Died at home, undelivered	. .
≥ 35	Hypertension Pre-eclampsia	Haemorrhage from ruptured subcapsular hepatic haematoma, HELLP	P	32	Undelivered	. .
20–34	Hypertension Pre-eclampsia	Intracranial haemorrhage	P	27	Emergency C/S, live birth	5
≥ 35	Hypertension Eclampsia	Myocardial infarction, hyperlipidaemia, pulmonary oedema, Hodgkin's lymphoma	M	36	Emergency C/S, live birth	4
≥ 35	Haemorrhage (PPH, arterial bleeding at C/S)	Pre-eclampsia, fibroids present with bladder adherent to uterus	M	28	Preterm rupture of membranes, emergency C/S, live birth	0
Indirect						
≥ 35	Hypertension	Intracerebral haemorrhage, streptococcal pneumonia	GM	n.s.	Spontaneous vaginal birth, live birth	13
Late direct						
20–34	TTP	Pre-eclampsia Abruption, coagulopathy	P	34	Emergency C/S, live birth	49

(a) N = nullipara P = primipara, M = multipara, GM = grand multipara.

n.s. Not stated.

. . Not applicable.

In Australia, the number of deaths resulting from hypertensive disorders in pregnancy has decreased over the last three triennia. In 1997–1999 there were six deaths in which hypertensive disorders in pregnancy were the principal cause of death. For two additional deaths pre-eclampsia was a contributing factor (principal causes were PPH and AFE). All eight deaths were classified as direct maternal deaths.

Illustrative cases

A woman experienced an uneventful first pregnancy until 27 weeks gestation when she was referred to hospital by her GP, complaining of feeling unwell. Her BP was 140/100 mmHg, with 4+ proteinuria, a puffy face and slight oedema of hands and feet. The uterine fundus was noted to be smaller than expected (25-week uterine size). She was given celestone and diazoxide and commenced on methyldopa 250 mg and hydralazine 12.5 mg, each three times a day. She was transferred to a tertiary hospital. On admission her BP was 150/90 mmHg and continued 117–153/50–92 mmHg overnight. The next day the clinical team made a decision to perform a caesarean section. A magnesium sulphate infusion was commenced and a live born baby weighing 684 grams was admitted to the neonatal intensive care unit. The woman's BP ranged between 120–160/80–94 mmHg. On day three she complained of

blurred vision, initially without headache. Headache appeared overnight, requiring treatment with intra-muscular morphine in the early morning. She vomited shortly afterwards with a BP 160–170/80 mmHg and became drowsy. Three seizures occurred, followed by cardiac arrest. A computed tomography (CT) scan showed large left fronto-parietal haemorrhage with midline shift. Despite surgical evacuation of the intracranial haemorrhage, death followed soon after.

Principal cause: Pre-eclampsia.

Contributing cause: Intracranial haemorrhage.

Maternal death classification: Direct.

A woman had a precipitous birth at home. There was no evidence of pre-eclampsia before or after birth. She developed a severe headache six days postpartum and was admitted to hospital overnight with a provisional diagnosis of possible migraine. Her BP was 180/95 mmHg. The following day she became nauseated, developed hemiplegia and was transferred to a tertiary hospital where intracerebral haemorrhage was demonstrated on CT scan. She developed obstructive hydrocephalus with increasing intracranial pressure until death occurred 13 days after the birth. Hypertension was regarded as the cause of the haemorrhage.

Principal cause: Hypertension.

Contributing causes: Intracerebral haemorrhage, streptococcal pneumonia.

Maternal death classification: Indirect.

A woman died from hypovolaemic shock as a consequence of spontaneous rupture of a subcapsular hepatic haematoma. She had pre-eclampsia with thrombocytopenia and elevated liver enzymes.

Principal cause: Pre-eclampsia.

Contributing cause: Haemorrhage, ruptured hepatic haematoma.

Maternal death classification: Direct.

Clinical comment

The 2000–2002 triennium has seen a decrease in maternal mortality due to hypertensive disorders when compared with previous triennia. However, pre-eclampsia in its various permutations features prominently in this and other reports of maternal mortality. It is estimated that for every death, there are hundreds of cases of severe morbidity, with complications requiring intensive care. Among these are cerebral complications, in particular haemorrhage, causing permanent morbidity for the woman. For the baby, pre-eclampsia results in placental failure, growth restriction and sometimes extreme prematurity.

Contributing factors can include failure to appreciate the significance of hypertension, inadequate management of hypertension, lack of consultation and referral, and care at an institution that is not appropriate to the level of pregnancy risk.

Hepatic rupture is a well recognised, albeit extremely rare (estimated incidence 1:45,000) complication of pregnancy, almost always occurring in the context of pre-eclampsia or the HELLP (hemolysis, elevated liver enzymes, low platelets) variant of pre-eclampsia (Coelho et al. 2000; Abdi et al. 2001). Women present with severe right upper quadrant and epigastric

pain, pre-eclampsia (that occasionally does not appear severe on conventional criteria), elevated hepatic enzymes and thrombocytopenia. Haemolysis is present in up to 15% of these cases, usually transient, lasting no more than 24 hours. Hepatic rupture follows intrahepatic haemorrhage subsequent to hepatic infarction. This can accompany pre-eclampsia and occurs most commonly in the third trimester of pregnancy. As in the case described in this chapter, the rupture usually affects the right lobe of the liver. This condition always requires urgent delivery, but resuscitation with blood products and correction of the inevitable accompanying coagulopathy are pre-eminent elements in management. The disease demands continuing high-grade care postpartum as resolution is often delayed over several days, with continuing thrombocytopenia, liver abnormalities and hypertension, in addition to renal failure in many of the cases.

In summary, prevention of maternal death for high-risk women should begin with preconception counselling. Women with a history of recurrent severe early pre-eclampsia should be cautioned about the risks of further pregnancy, as should older, obese, or hypertensive women. Severe hypertension should always be treated vigorously. Every unit should have a standard protocol to deal with episodes of severe hypertension, defined as systolic BP ≥ 170 mmHg or diastolic BP ≥ 110 mmHg. Lower 'action levels' should be considered in young women and those with low BP at the booking visit. Thus a young woman whose normal BP is 100/60 mmHg may be at substantial risk when pre-eclampsia causes levels of 150–160 mmHg systolic or 100 mmHg diastolic.

Best practice

Two international multi-centred clinical trials have provided best practice recommendations for the prevention and treatment of eclampsia. Magnesium sulphate has been shown to be the anticonvulsant agent of choice in these trials (The Magpie Trial Collaborative Group 2002).

Key learning points

- Adequate treatment of hypertension should accompany a full clinical and laboratory assessment of the woman who presents with new hypertension after 20 weeks, with attention also to the fetal status (Brown et al. 2000).
- Inpatient care may be necessary when pre-eclampsia is diagnosed, as the progress of the condition is unpredictable.
- Early referral to a consultant obstetrician, and multidisciplinary involvement, should be considered for any woman presenting with signs of pre-eclampsia, and the birth should occur at a centre that is appropriately equipped to care for such women and babies (Brown et al. 2000).
- Continued careful postnatal observation of pre-eclampsia is necessary as the disease often does not immediately resolve following birth, and many complications occur in the postnatal period.
- All women who have hypertension during pregnancy should be reviewed some time in the months following birth. Some will require long-term follow-up with studies suggesting an increased risk of ischaemic heart disease and cardiovascular events in women who have had hypertension in pregnancy (Arnadottir et al. 2005).

Resources:

- A consensus statement on the detection, investigation and management of hypertension in pregnancy was published in 2000 by the Australasian Society for the Study of Hypertension in Pregnancy (Brown et al. 2000). The Society website and consensus statement can be found at: <http://www.racp.edu.au/asshp/asshp.pdf>.
- The Action on Pre-Eclampsia website has been established by a UK-based charity organisation and provides evidence-based information and support for women and practitioners on pre-eclampsia. The website is <http://www.apec.org.uk/home.htm>.

Thrombosis and thromboembolism

Introduction

Pulmonary thromboembolism (PTE) has been a major cause of maternal mortality in Australia and in the developed world. It arises from deep venous thrombosis (DVT), either in the leg or deep veins of the pelvis, which is often not recognised clinically. As well as the acute morbidity and mortality of venous thromboembolism, PTE carries a risk of subsequent pulmonary hypertension, of post-thrombosis syndrome; and of recurrent thromboembolism (Greer 2002).

The major risk factors for venous thromboembolism in pregnancy and the postpartum period include socio-demographic, clinical and operative factors. The socio-demographic factors include: advanced maternal age of 35 years or older, obesity defined as a body mass index (BMI) greater than 29, and immobility. The clinical and operative factors include: thrombophilia, a history of DVT or PTE, gross varicose veins, pre-eclampsia, nephrotic syndrome, current infection, or a significant current medical problem, caesarean section delivery particularly as an emergency in labour, operative vaginal delivery, and other recent surgery. (Obstetric Medicine Group of Australasia 2001; Greer 2002).

Summary of the findings for 2000–2002

There were three deaths in the 2000–2002 triennium in which PTE was judged to be the principal cause of death (Table 37). Two were classified as direct maternal deaths and one was classified as indirect. A further three deaths (one direct and two indirect) had infection as the principal cause of death and PTE as a contributing cause of death (Table 32). This is a decrease from the six deaths with PTE as a principal cause of death in the 1997–1999 triennium and eight in the 1994–1996 triennium.

All three women who died from PTE as a principal cause of death were aged 33 years and older. One woman had an unassisted vaginal delivery, the second had an emergency caesarean section and the third was undelivered. Maternal weight was recorded in two cases with one woman having a BMI of 36.

Of the six women who had PTE as either a principal or contributing factor in their death, all had at least one identified risk factor for thromboembolism and four had concurrent infections.

Table 37: Maternal deaths due to thrombosis and thromboembolism, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Parity[(a)]	Weight / BMI	Method of birth and birth outcome	Days post-partum
Direct						
≥ 35	Pulmonary thromboembolus Thrombosis of inferior vena cava	Old cerebral infarct and ischaemic heart disease on autopsy	GM	52 kg	Spontaneous vaginal birth, live birth	11
20–34	Pulmonary embolus	Deep vein thrombosis in the calf	M	BMI 36	Emergency C/S, live birth	2
20–34	Infection, septicaemia	Pulmonary thromboembolism, venous thromboembolism of the legs	GM	n.s.	Spontaneous vaginal birth, live birth	21
Indirect						
20–34	Pulmonary embolism	Infection, bilateral bronchopneumonia, cardiomyopathy	GM	n.s.	16/40 weeks, undelivered	. .
20–34	Infection, septicaemia	Pulmonary and pelvic thromboembolus	P	n.s.	Elective C/S, live birth	4
20–34	Infection, herpes simplex encephalitis	Pulmonary embolism	M	n.s.	C/S for fetal retrieval, stillbirth	4

(a) P = primipara, M = multipara, GM = grand multipara.

n.s. Not stated.

. . Not applicable.

Illustrative cases

A woman presented for antenatal care at 29 weeks gestation. Cardiac investigations included an echocardiogram at 32 weeks which revealed a left ventricle with function in the lower range of normal, and signs consistent with ischaemic heart disease and mitral regurgitation. She had extensive investigations for thrombophilia and autoimmune disorders with results in the normal range, apart from a slightly elevated antinuclear antibody titre at 40 (normal range <40).

The woman had an uneventful pregnancy with spontaneous onset of labour at 39 weeks gestation and delivery of a liveborn infant. Seven days postpartum she was brought to hospital by ambulance complaining of right-sided back pain and difficulty breathing. She was anxious to go home and discharged the same day, to be followed-up by her GP. Test results: a chest X-ray showed sub-segmental atelectasis in lower lobes, slight blunting of the lateral costophrenic angles in keeping with small effusions or pleural thickening.

The woman was found collapsed at home on day 11 postpartum, cyanosed and unresponsive. She was transferred to hospital by ambulance but resuscitation was unsuccessful. The autopsy found a large pulmonary thromboembolus occupying the left and right pulmonary arteries and thrombus (probably a couple of days old) lying within the inferior vena cava with another thrombus (1 to 4 weeks old) found within a tributary of the inferior vena cava.

Principal cause: Pulmonary thromboembolus and thrombosis of inferior vena cava.

Maternal death classification: Direct.

A woman with a history of pre-eclampsia in her first pregnancy commenced antenatal care in her second pregnancy at eight weeks gestation. Her booking BP was 140/100 mmHg and she was a non-smoker. The woman was cared for by a renal specialist and regularly attended a pre-eclampsia clinic, requiring antihypertensives during pregnancy. She required admission at 36 weeks for hypertension which settled overnight. She continued twice-weekly follow-up until 39 weeks gestation when she was admitted for induction of labour with Prostin E2 gel. She was given hydralazine 25 mg four times a day in addition to her usual clonidine.

Labour was augmented with syntocinon and required an epidural anaesthetic for pain relief. An emergency caesarean section was carried out for failure to progress after achieving full dilatation with the baby presenting in the posterior position. Her post-operative recovery was uneventful until 33 hours post delivery, when ambulation began. She collapsed suddenly and resuscitation was unsuccessful. She had been wearing compression stockings post caesarean section but was not receiving anticoagulants as thromboprophylaxis.

Principal cause: Pulmonary artery thromboembolus.

Contributing cause: Deep vein thrombosis in the calf.

Maternal death classification: Direct.

Clinical comment

Clinical guidelines regarding the investigation and management of women in whom venous thromboembolism is suspected were first published in the UK in 1995. Following this, the number of thrombosis deaths in the UK following caesarean section fell dramatically, and the number of antenatal deaths from thrombosis fell in the 2000–2002 triennium (Drife 2004).

These cases of pulmonary embolism are almost certainly not all avoidable. Routine prophylaxis prior to emergency caesarean section would involve unnecessary thromboprophylaxis of many women, in both of the women described above, thromboprophylaxis was not instituted, despite the presence of risk factors. These included age 35 or older, obesity, current infection and emergency caesarean section. The guidelines referred to below recommend thromboprophylaxis when risk factors exist. Increasing numbers of women are receiving prophylaxis, and it is likely that the mortality rate would be higher without this intervention. It is not known whether systematic application of the guidelines outlined by RCOG in the United Kingdom would further reduce mortality from PTE in Australia.

Best practice

A position statement published in 2001 on behalf of the Obstetric Medicine Group of Australasia entitled Anticoagulation in pregnancy and the puerperium has similar recommendations to the RCOG guidelines (Hague et al. 2001).

International guidelines support thromboprophylaxis when risk factors exist in pregnancy (Bates et al. 2004).

There has never been a reported episode of diagnosed neuraxial bleeding associated with thromboprophylaxis in pregnancy in Australia. Neuraxial analgesia is often used in labour and caesarean section. However concern is often expressed by anaesthetists, in view of the theoretical risk of a neuraxial bleed with the use of neuraxial analgesia.

Resources:

- A new guideline titled 'Thromboprophylaxis during pregnancy, labour and after normal vaginal delivery' was published in January 2004 by the RCOG (Lewis 2004). The website is:
  <http://www.cemach.org.uk/publications/WMD2000_2002/wmd-02aa.htm>.
- An updated guideline 'Prophylaxis against thromboembolism in caesarean section' is available from the RCOG (Lewis 2004). The website is:
  <http://www.cemach.org.uk/publications/WMD2000_2002/wmd-02ab.htm>.
- A working group on behalf of the Obstetric Medicine Group of Australasia published a position statement, Anticoagulation in pregnancy and the puerperium, 2001. The website is:
  <http://www.mja.com.au/public/issues/175_05_030901/omga/omga.html>.

Early pregnancy deaths

Introduction

For the purpose of this report, an early pregnancy death is defined as a maternal death during the first 14 weeks of pregnancy.

Ectopic pregnancy is a major cause of early pregnancy death and maternal morbidity across the world. In Australia, a NSW study using 1990–98 data concluded that the incidence of ectopic pregnancy was reaching a plateau (Boufous et al. 2001). However, since that time, the incidence of ectopic pregnancy has been increasing in some countries and this is thought mainly to be associated with the increasing incidence of pelvic inflammatory disease caused by chlamydia infection and smoking in women of reproductive age (Tay et al. 2000; Coste et al. 2004). Chlamydia is now the most common notifiable infection in Australia with a rate that has been rising by approximately 20% annually in recent years (Commonwealth of Australia 2005). Around 70% of notifications are in women of reproductive age of less than 25 years (Commonwealth of Australia 2005).

The CEMACH found mortality from ectopic pregnancy to be associated with substandard care, of which failure to suspect and diagnose the condition was a major factor (Lewis G (ed.) 2004).

Summary of the findings for 2000–2002

There were four early pregnancy deaths during the 2000–2002 triennium, however only one, a direct death resulting from a ruptured ectopic pregnancy, is reported in this chapter (Table 38). The other three deaths are reported in the cause specific chapters. One is reported in the *Infection* chapter and reports on a woman who died at six weeks gestation of septicaemia resulting from malaria. The second is reported in the *Haemorrhage* chapter and is of a woman who had a subarachnoid haemorrhage at six weeks gestation. The third is reported is of in a woman who had systemic lupus erythematosus and coronary artery disease and is detailed in the *Cardiac* chapter. She had a myocardial infarction at 10 weeks gestation.

The mortality from ectopic pregnancy in Australia has decreased from five deaths in the 1994–1996 triennium to one death in each of the 1997–1999 and 2000–2002 triennia.

Table 38: Maternal deaths, early pregnancy, Australia, 2000–2002

Age group (years)	Principal cause of death	Parity[(a)]	Gestation (weeks)
Direct			
20–34	Ruptured ectopic pregnancy	M	10
Indirect			
20–34	Subarachnoid haemorrhage	M	6
20–34	Cardiac, myocardial infarction	P	10
20–34	Infection, septicemia, malaria	N	6

(a) N = nullipara P = primipara, M = multipara.

Illustrative case

A woman attended a clinic at approximately 10 weeks gestation for a termination of pregnancy. After undergoing an ultrasound scan she was reportedly advised that the gestation was too early and asked to return the following week. Three days later she complained of severe pain in the hip and abdominal region. An ambulance was called to her home but she had a cardiac arrest in the ambulance en route to hospital. A provisional diagnosis of a ruptured ectopic pregnancy was made and an ultrasound examination showed a large amount of free fluid in the abdomen. The woman underwent a laparotomy where approximately 1.5 litres of blood was found in the peritoneal cavity and a left salpingectomy was performed. Extensive resuscitation continued however she died during the operation.

Principal cause: Ruptured ectopic pregnancy.

Maternal death classification: Direct.

Clinical comment

Clinicians must continue to be alert for ectopic pregnancy. With chlamydia notifications increasing rapidly in Australia, it is possible that there could be an increase in ectopic pregnancy in the future. In early pregnancy, unruptured ectopic pregnancy should be suspected when a gestational sac appears smaller than the dates suggest, or where there appears to be a sac without a fetus.

Key learning points

- While ectopic pregnancies are rare, they are still potentially fatal, and should always be part of the differential diagnosis in women of reproductive age who present with abdominal pain with or without vaginal bleeding.
- Atypical presentation of ectopic pregnancy includes those that mimic gastrointestinal or urinary tract conditions.
- Early diagnosis is critical in order to prevent serious morbidity or mortality.

Resources:

- Clinical practice guidelines for the management of suspected ectopic pregnancy are published by the Royal Women's Hospital (RWH) in Victoria (RWH 2002). The web site is: <http://www.wch.org.au/rwhcpg/womenshealth.cfm?doc_id=5081>.

Other indirect causes of maternal death

Introduction

In previous reports, Australia, as with other countries, has divided indirect deaths into deaths from cardiac disease, psychiatric causes and 'other' indirect deaths. In the 'other' category, the most common causes of death are deaths due to cerebrovascular disease, infection and deaths due to conditions such as asthma, epilepsy and diabetes. In this report the indirect deaths from cardiac disease (n=11), infection (n=10), psychiatric causes (n=9), haemorrhage–other (n=8), hypertension (n=1) and pulmonary embolism (n=1) were discussed together with direct deaths in the corresponding chapters. This chapter describes the remaining indirect deaths (n=13) including one late indirect death, that occurred in the 2000–2002 triennium.

Asthma is a common condition among Australians affecting 10–12% of adults (AIHW & ACAM 2005). Hospitalisation and death from asthma has decreased over the last 10 years. However, the rate of asthma deaths in Australia (0.3% of all deaths) is high in comparison to other countries. Asthma in pregnancy can be unpredictable with roughly one third of women experiencing an improvement in symptoms, around 50% experiencing minimal change and 20% experiencing a worsening of disease symptoms (McDonald & Burdon 1996).

Thrombotic thrombocytopenic purpura (TTP) is a rare condition which usually occurs in mid pregnancy and is characterised by microangiopathic haemolytic anaemia, thrombocytopenia, central neurological abnormalities, fever and renal dysfunction (Kam et al. 2004). Pregnancy is considered to be a predisposing factor for this disease. Plasma exchange can lead to remission in 75% of patients with TTP.

The incidence of cancer in pregnancy is lower than that of the non-pregnant population. However, some cancers, particularly those that are hormone dependent (cancer of the breast or cervix), are thought to accelerate in pregnancy (Lewis G (ed.) 2004). The Royal College of Obstetricians and Gynaecologists in the UK study group on cancer in pregnancy has identified certain cancers they believe are aggravated by pregnancy (Lewis G (ed.) 2004). The NACMM has followed the UK classifications for Australian deaths. Those cancers believed to be associated with pregnancy are classified as 'indirect' deaths, whilst other cancers not considered to be associated with pregnancy are classified as 'incidental' deaths.

Summary of the findings for 2000–2002

There were 14 indirect maternal deaths classified as 'other indirect' in the 2000–2002 triennium. Asthma was a principal cause of death in three cases and a contributing factor in the deaths of five other women. In the latter cases four women died from either septicaemia or adult respiratory distress syndrome resulting from pneumonia. In two cases the pneumonia was secondary to influenza A (*Infection* chapter). The fifth case was a late maternal death, 48 days postpartum. Another case involved a woman who died from cardiac failure, possibly an arrhythmia, where the autopsy showed evidence of asthma and pulmonary oedema.

Four women had TTP as either a principal (n=3) or contributing cause (n=1) of death during this triennium. Only three are described here as the fourth was a direct late maternal death (49 days following birth without discharge from hospital) which is presented in the

Haemorrhage – obstetric chapter. In the case where TTP was a contributing cause of death, systemic lupus erythematosus was the principal cause of death.

Cancer was principal cause of death in three women. In the 2000–2002 triennium in Australia the NACMM has followed the recommendations from the UK and classified these three deaths caused by cancers or tumours as indirect rather than incidental. Two death were due to cerebral tumours and one to metastatic carcinoma of the breast.

Other maternal deaths described in this chapter resulted from systemic lupus erythematosus, epilepsy, homicide, head injury (assault) and hyperammonaemic encephalopathy (Table 39). A death from assault was classified as indirect if the case report suggested the death was related to the pregnancy. Two deaths due to pulmonary embolism and hypertension are reported in the related chapter.

The women described in this chapter were aged between 26 and 40 years with gestational ages at the time of death of 9–36 weeks. There were four liveborn infants, with seven women dying undelivered, one infant was delivered stillborn by caesarean section and one pregnancy was electively terminated to facilitate active maternal treatment.

Table 39: Maternal deaths, other indirect causes[(a)], Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death	Gestation (weeks)	Method of birth, birth outcome and comment
Indirect				
20–34	Asthma		12	Undelivered
20–34	Asthma	Aspiration pneumonia	17	Undelivered
20–34	Asthma, severe bronchospasm		33	C/S, stillbirth
20–34	TTP	Multi-system failure	20	Undelivered
20–34	Systemic lupus erythematosus	TTP, renal failure	14	Therapeutic abortion, woman died a week later
20–34	Inoperable cerebral tumour, probable glioma		36	Elective C/S, live birth
≥ 35	Recurrence of previously treated cerebral tumour, glioma / astrocytoma		n.s.	Emergency C/S, live birth
≥ 35	Metastatic carcinoma of the breast, high-grade invasive ductal carcinoma		26	Spontaneous vaginal birth, live birth
≥ 35	Homicide	Stabbing to neck	26	Undelivered
20–34	Assault	Subdural haemorrhage	16	Undelivered
20–34	Epilepsy		16	Undelivered
20–34	Hyperammonaemic encephalopathy		9	Undelivered
Late indirect				
20–34	TTP	Haemorrhage from VasCath, ventricular dysfunction, pericardial effusion	n.s.	Woman presented 45 days postpartum and died 7 days later, live birth

(a) Two 'other' indirect maternal deaths with principal cause of death hypertension and pulmonary embolism are reported in Table 36 and Table 37 respectively.

n.s. Not stated.

Illustrative cases

A woman with generally well-controlled asthma, on budesonide and salbutamol inhalers, ceased her asthma medications at 12–14 weeks gestation owing to her concerns of the effect the medication could have on the pregnancy. She saw her GP two weeks later with worsening asthma, and was encouraged to recommence medication. At 17 weeks gestation she had chest tightness unrelieved by salbutamol inhaler or nebuliser. She had a cardio-respiratory arrest before arrival at hospital and was not able to be resuscitated.

Principal cause: Status asthmaticus.

Contributing cause: Aspiration pneumonia.

Maternal death classification: Indirect.

A woman developed TTP at 18 weeks gestation when she presented with significant proteinuria. She initially improved with antibiotics however, she was readmitted less than a week later with severe anaemia and thrombocytopenia. She was treated with platelet transfusion, corticosteroids, aspirin, plasmapheresis, intragam, vincristine, cyclophamide and dipyridamole. No significant improvement in platelet count occurred and neurological symptoms worsened. She progressed to renal failure, marked neurological deterioration and multi-system failure.

Principal cause: TTP.

Contributing cause: Multi-system failure.

Maternal death classification: Indirect.

A woman presented at 24 weeks gestation with confusion, unsteadiness and mild cough. She had bilateral chest X-ray infiltrates and was treated for suspected atypical pneumonia. A biopsy of a left breast mass was performed and a CT scan of her head was normal. Her condition deteriorated and the left breast mass showed high-grade invasive ductal carcinoma with further CT scans showing metastases to liver, spine, hips and sternum. Her condition deteriorated despite intensive treatment and she had a spontaneous birth of a liveborn infant at just over 26 weeks gestation. She then received palliative care until her death, three weeks postpartum.

Principal cause: Metastatic carcinoma of the breast.

Maternal death classification: Indirect.

Clinical comment

Asthma is a very common condition in Australia. Women with chronic health conditions such as asthma would benefit from regular visits to a general practitioner. The first case highlights the need for doctors to discuss the use of medications in patients with chronic conditions proactively. Pre-pregnancy discussions are of most benefit, as this may reduce the number of women self-ceasing medications that are essentially safe for use in early pregnancy.

Thrombotic thrombocytopenic purpura is a complex condition to accurately diagnose during pregnancy, as the initial presentation may mimic obstetric diseases like pre-eclampsia, including HELLP syndrome. Effective treatment by plasmapheresis is more likely to be successful when diagnosis is made promptly and treatment instituted without delay.

Plasmapheresis was provided for three of the women who diagnosed with but was unsuccessful. Information on plasmapheresis was not available for the fourth woman

Breast cancer is thought to accelerate in pregnancy as a result of the hormonal changes associated with the pregnancy (Lewis G (ed.) 2004). Women presenting with cancers or tumours in pregnancy require coordinated multidisciplinary care from midwives, obstetricians, oncologists, radiologists, palliative care teams, anaesthetists and paediatricians.

Best practice

Planned pregnancies should have medication and asthma plans discussed prior to pregnancy. Women with unplanned pregnancy should see their GP about their asthma management as soon as they know they are pregnant, even if they feel well and are asymptomatic at the time. A planned, proactive approach is in the best interests of both the mother and the fetus.

Women should be encouraged to continue breast self-examination regularly both antenatally and postnatally, reporting any breast lumps that do not resolve with breastfeeding.

Any woman who has experienced a previous cancer or tumour should receive preconception counselling from a specialist.

Key learning points

- Written asthma action plans have been shown to greatly improve the outcomes of asthma and should be a part of the management plan in pregnancy.
- The benefits of asthma control outweigh any potential for an adverse pregnancy outcome.
- Coordinated multidisciplinary care is often required in cases where pregnant women have chronic health conditions or present with an acute non-obstetric condition. Early involvement with other medical specialists, in planning and providing care to women with complex health needs, is warranted.

Resources:

- The American National Guideline Clearinghouse contains evidence-based clinical practice guidelines and related documents from all over the world. The website is: <http://www.guideline.gov/>.
- Australian Therapeutic Goods Administration. The website is: <http://www.tga.gov.au/docs/html/mip/medicine.htm>.
- British guideline on the management of asthma, 'A national clinical guideline', 2004. <http://www.guideline.gov/summary/summary.aspx?doc_id=5614&nbr=003784&string=asthma+AND+pregnancy#s24>.
- Canada Lung Association, <http://www.lung.ca/asthma/pregnancy/>.
- National Heart, Lung, and Blood Institute, 'Managing asthma during pregnancy: recommendations for pharmacologic treatment', 2005, <http://www.guideline.gov/summary/summary.aspx?doc_id=6259&nbr=004014&string=asthma+AND+pregnancy#s23>.

Section D: Other key areas

Maternal mortality in Aboriginal and Torres Strait Islander women

Introduction

Aboriginal and Torres Strait Islander people have significantly lower life expectancy (approximately 17–20 years less) than the non-Indigenous population (ABS & AIHW 2005). This is reflected in the maternal mortality ratio for Aboriginal and Torres Strait Islander women over the previous three triennia (1991–1999), estimated to be 21.4 per 100,000 women who gave birth which is approximately three times as high as that for non-Indigenous women (7.0 per 100,000 women who gave birth) (Slaytor et al. 2004). For 2003 the average perinatal mortality rate for babies of Indigenous mothers was estimated to be almost double that for babies of non-Indigenous mothers (17.5 vs 9.8 per 1,000 births) (Laws & Sullivan 2005). These higher mortality rates occur in a context of Aboriginal and Torres Strait Islander women having their first and subsequent babies at younger ages than non-Indigenous women. The average age of Aboriginal and Torres Strait Islander mothers in Australia in 2003 was 24.8 years with 22.7% of mothers aged less than 20 years; compared with 29.7 years for non-Indigenous mothers, with 3.9% aged less than 20 years (Laws & Sullivan 2005).

Aboriginal and Torres Strait Islanders experience lower levels of access to health services than non-Indigenous Australians, with only 30% living in major cities compared with 67% of non-Indigenous people (ABS & AIHW 2005). Aboriginal and Torres Strait Islander women have 1.5 times the rate of hospitalisations for complications of pregnancy, childbirth and the puerperium, when compared with age, sex and cause-specific rates for non-Indigenous Australians (ABS & AIHW 2005). Indigenous Australians are disadvantaged across a range of indicators including income, employment, educational status and housing, all of which influence health outcomes. Indigenous communities suffer high levels of grief and loss, due to high levels of mortality and morbidity; this impacts on all community members (Tatz 1999).

Indigenous status

Indigenous status is a key demographic variable in maternal mortality surveillance. An Aboriginal or Torres Strait Islander woman is a woman of Aboriginal or Torres Strait Islander descent who identifies as an Aboriginal and/or Torres Strait Islander, and is accepted as such by the community in which she lives (National Health Data Committee 2003).

The data domain for Indigenous status is:

- Aboriginal but not Torres Strait Islander origin
- Torres Strait Islander but not Aboriginal origin
- Both Aboriginal and Torres Strait Islander origin

- Neither Aboriginal nor Torres Strait Islander origin
- Not stated/Inadequately described

Reporting of Indigenous status

Reporting of Indigenous status in maternal death notifications is incomplete (Table 40). In the 2000–2002 triennium, Indigenous status was reported for 77 (89%) of the 87 maternal deaths (direct, indirect and incidental) and 82 (73%) of the 95 direct, indirect, incidental and late maternal deaths. Indigenous status has been collected for cases categorised as: direct maternal deaths since 1970; and for indirect and incidental deaths since 1991. Reporting of Indigenous status for late maternal deaths commenced with the introduction of national reporting of late maternal deaths in the 1997–1999 triennium. A priority for all future reporting should be to ensure that Indigenous status is identified and reported for all maternal deaths.

Table 40: Proportion of maternal deaths where Indigenous status was reported, Australia, 1970–2002

Triennium	Per cent
1970–1972	n.a.
1973–1975	n.a.
1976–1978[(a)]	50
1979–1981	48
1982–1984	51
1985–1987	84
1988–1990	91
1991–1993[(b)]	92
1994–1996	83
1997–1999	83
2000–2002	89

(a) Reporting of Indigenous status for direct maternal deaths commenced in 1970.

(b) Reporting of Indigenous status for indirect and incidental maternal deaths commenced in 1991.

n.a. Not available.

Reporting of the maternal mortality ratio

Reporting of an Indigenous-specific MMR commenced with the 1991–1993 triennium. This was the first triennium where denominator data were available from the National Perinatal Data Collection. The MMR published for Aboriginal and Torres Strait Islander women in the 1994–96 triennial report was 34.8 deaths per 100,000 women who gave birth (NHMRC & AIHW 2001). This included incidental deaths as was the reporting convention at the time. Since then Australia has adopted the international convention of including only direct and indirect deaths in the calculation of MMR. It has now been recalculated excluding incidental deaths with the resultant MMR being 17.4 per 100,000 women who gave birth in the

1994–1996 triennium. All separate calculations of maternal mortality by Indigenous status (for both Indigenous and non-Indigenous women) from 1997 onwards exclude deaths where Indigenous status is unknown (Table 41). Prior to 1997, deaths where Indigenous status was unknown were included in the numerator for non-Indigenous deaths (Table 41). An errata was published for the MMR for Aboriginal and Torres Strait Islander women in the 1997–1999 triennial report which gave a MMR of 23.5 deaths per 100,000 women who gave birth.

Maternal mortality ratio

The MMR for Aboriginal and Torres Strait Islander women for the 2000–2002 triennium was 45.9 per 100,000 women who gave birth. This was over five times as high as that for non-Indigenous women for the same period (45.9 per 100,000 women who gave birth compared to 8.7 per 100,000 women who gave birth). In 2000–2002 Aboriginal and Torres Strait Islander mothers comprised 3.5% of the total number of women who gave birth in Australia (ABS & AIHW 2005). Because the total number of Aboriginal and Torres Strait Islander women giving birth is relatively small in comparison with the total number of women giving birth in Australia for the period at 26,128 versus 753,901 women respectively, the markedly higher MMR for Aboriginal and Torres Strait Islander (45.9) has only a marginal impact on the overall Australian MMR (11.1). These figures reflect all cause, age-specific death rates for Aboriginal and Torres Strait Islander women of reproductive age, which are on average 3.3 to 5.3 times as high as non-Indigenous all cause, age-specific death rates (ABS & AIHW 2005). The higher MMR noted for the 2000–2002 triennium is a cause of significant concern and should be monitored closely at a national, state and territory level.

The higher MMR equates to one maternal death (direct and indirect) per 2,177 Aboriginal or Torres Strait Islander women who gave birth. These findings need to be interpreted with some caution owing to the very small number of deaths and the inconsistencies and lack of completeness of Indigenous identification.

The MMR for Aboriginal and Torres Strait islander women has been calculated for a six – year period to improve the stability of the estimate (Table 41). The rate ratio for the period 1997–2002 is 4.5 compared to a rate ratio of <3 for the periods 1991–1996 and 1994–1999.

Table 41: Maternal deaths by Indigenous status, Australia, 1991–2002

	Number of Indigenous maternal deaths		Number Indigenous women who gave birth	Maternal mortality ratio[a]		
Triennium	Direct	Indirect		Indigenous	Non-Indigenous[b]	Rate ratio[c]
2000–2002	4	8	26,128	45.9	8.7	5.3
Double Triennia						
1991–1996	4	5	44,535	20.2	7.1	2.8
1994–1999	4	6	48,526	20.6	8.0	2.6
1997–2002	5	13	51,658	34.8	7.7	4.5

(a) Ratio per 100,000 women who gave birth calculated using direct and indirect deaths only.

(b) For 1991 to 1996, includes deaths where Indigenous status was not reported. For 1997 to 2002, does not include deaths where Indigenous status was not reported.

(c) Indigenous maternal mortality rate divided by non-Indigenous maternal mortality rate.

Note: Excludes incidental deaths.

Summary of the findings for 2000–2002

Thirteen of the 95 deaths (13.7%) during the 2000–2002 triennium were Aboriginal or Torres Strait Islander women. There were 11 maternal deaths of Aboriginal women and two of Torres Strait Islander women. Four of the maternal deaths were direct, eight were indirect and one was an incidental death (Table 42). As with other chapters the incidental death is not presented here.

The 12 Aboriginal and Torres Strait Islander maternal deaths presented in this chapter were aged between 17 and 34 years with a median age of 27 years. This figure is lower than the median age of all maternal deaths in 2000–2002, which was 30 years. This reflects the younger age distribution seen in Aboriginal and Torres Strait Islander women giving birth with an average age of 24.7 years compared with 29.4 years in non-Indigenous women during the 2000–2002 triennium.

Table 42: Causes of maternal death among Aboriginal and Torres Strait Islander women, Australia, 2000–2002

Age group (years)	Principal cause of death	Contributing cause of death, comments	Parity[(a)]	Gestation (weeks)
Direct				
<20	Hypertension – eclampsia	Extensive bilateral cerebral and brain stem haemorrhage	N	32
20–34	Amniotic fluid embolism	Placenta praevia, ante and postpartum haemorrhage, DIC	M	36
20–34	Haemorrhage – genital tract	Placenta percreta – invasion to parametrial tissues, DIC	M	n.s.
20–34	Haemorrhage – genital tract	Placenta percreta – invasion to bladder and cervical tissues, APH	M	24
Indirect				
<20	Suicide – hanging		n.s.	7
20–34	Suicide – hanging		N	9
20–34	Suicide – hanging		N	13
20–34	Cardiac – ischaemic heart disease	Died 19 days postpartum	n.s.	n.s.
20–34	Subdural haemorrhage	Assaulted	M	16
20–34	Infection – pneumococcal meningitis	Cerebral oedema, diabetes insipidus, chronic left otitis media, streptococcus pneumoniae, sepsis	M	19
20–34	Cardiac – rheumatic heart disease, left ventricular failure, cardiogenic shock	Died in theatre during valve replacement, one day postpartum	M	28
20–34	Pulmonary embolism	Infection, bilateral bronchopneumonia, cardiomyopathy	GM	16

(a) N = nullipara, M = multipara, GM = grand multipara.

n.s. Not stated.

The distribution of Aboriginal and Torres Strait Islander death varied by remoteness area of usual residence with five (41.7%) women usually resident in the outer regional areas, four (33.4%) usually resident in remote and very remote areas and three (25.0%) women usually resident in the major city and inner regional areas. These data do not establish any

relationship between risk of death and place of residence and need to be interpreted with some caution owing to the very small number of deaths.

Illustrative cases

A woman presented to a remote generalist health centre with no on-site maternity service providers. The woman was not known to be pregnant. Her symptoms included chest pain, epigastric tenderness, marked hypertension and blurred vision. The woman was treated with antacids and analgesics and sent home with advice to return to the next medical clinic. She did not return for a medical review. This sequence of events was repeated several days later. The woman was discovered unconscious at home and was unable to be resuscitated.

Principal cause: Hypertension, eclampsia.

Contributory cause: Extensive bilateral cerebral and brain stem haemorrhage.

Maternal death classification: Direct.

A woman in her second pregnancy had a history of rheumatic fever, aortic valve stenosis, mitral valve stenosis, and mechanical aortic and mitral valve replacement. She was found to have severe aortic stenosis at antenatal booking. Severe left ventricular failure developed at 28 weeks gestation and she underwent a classical caesarean section. She subsequently developed refractory cardiac failure, requiring aortic and mitral valve repair 24 hours post delivery. She died in the operating theatre.

Cause of death: Rheumatic heart disease.

Maternal death classification: Indirect.

Clinical comment

The MMR for Aboriginal and Torres Strait Islander Australians has increased from 23.5 per 100,000 Aboriginal and Torres Strait Islander women who gave birth in the 1997–1999 triennium to 45.9 per 100,000 Aboriginal and Torres Strait Islander women who gave birth in the 2000–2002 triennium. This was an increase in the number of deaths from 7 to 12 deaths during the 2000–2002 triennium with four of the latter suffering violent deaths.

Multiple medical co-morbidities and less access to skilled practitioners and specialist services are factors evident in these cases. These factors, together with less antenatal visits and later presentation during pregnancy, are often seen in Aboriginal and Torres Strait Islander women compared with non-Indigenous women (Centre for Epidemiology and Research 2004; Stewart & Li 2005). Research suggests that shame and lack of confidence, together with care that does not meet the cultural needs of Aboriginal and Torres Strait Islander women, are contributing factors to these lower levels of antenatal care (Carter et al. 1987; Kildea 1999; Hurst 2005).

In both illustrative cases the woman had presented late or not at all for antenatal care; at 26 weeks in the woman with serious rheumatic heart disease and in the third trimester in the woman with undiagnosed eclampsia. The high prevalence of diabetes mellitus and renal disease in Aboriginal and Torres Strait Islander women increases the risk of developing pre-eclampsia. An emphasis on early antenatal care and detection and management of co-morbidities could significantly improve maternal and fetal outcomes. Ensuring this care is accessible and appropriate must be a priority for maternity service providers. All health

practitioners should be aware of the risk of pre-eclampsia in pregnant women and the key signs and symptoms of this potentially lethal disorder.

Three young (under 25 years) Aboriginal and Torres Strait Islander women committed suicide in the first trimester of pregnancy during the 2000–2002 triennium. Two of the three were in their first pregnancy with data on parity unavailable in the third case. One of these women had a history of depression and two had been drinking alcohol. Suicide at such an early stage of pregnancy is difficult to prevent with antenatal interventions, as antenatal care may not have commenced in many instances. Preconception care and other initiatives addressing the higher rates of suicide in the Aboriginal and Torres Strait Islander community are necessary to prevent such deaths.

Three Aboriginal and Torres Strait Islander women died as a result of complications of severe haemorrhage and in one case AFE, associated with placenta praevia and placenta percreta. Aboriginal and Torres Strait Islander women have more pregnancies complicated by medical co-morbidities such as preterm birth (14.1%) compared with non-Indigenous women (7.6%) (Laws & Sullivan 2005). These factors contribute to higher rates of caesarean section which in turn contributes to rising rates of placenta praevia and placenta percreta (Weerasekera 2000).

Aboriginal and Torres Strait Islander Australians have a high incidence and prevalence of acute rheumatic fever with subsequent rheumatic heart disease when compared with other Australians and many other populations across the world (AIHW 2004a). Social and economic disadvantage, overcrowding and poor sanitary conditions lead to proliferation of streptococcal infection and rheumatic fever (AIHW 2004a). There needs to be a high awareness of the risk of rheumatic heart disease, even in asymptomatic Aboriginal and Torres Strait Islander women.

Key learning points

- There should be an emphasis on early antenatal care for Aboriginal and Torres Strait Islander women to detect and appropriately manage co-morbidities.
- Aboriginal and Torres Strait Islander women of reproductive age with existing co-morbidities should receive preconception specialist review and advice if planning a pregnancy.
- Rheumatic heart disease remains a significant co-morbidity in Aboriginal and Torres Strait Islander women requiring preconception assessment and specialist involvement throughout the antenatal period.
- The development of new hypertension in a young person is always an indication for urgent investigation and management.
- All health professionals need to be aware of the clinical presentation of pre-eclampsia.
- Health practitioners need to be aware of the high rates of depression and suicide risk in Aboriginal and Torres Strait Islander women.
- Maternity services must strive to provide culturally appropriate maternity care, which can only be done in partnership with Aboriginal and Torres Strait Islander women.

Resources:

- Information on Aboriginal and Torres Strait Islander mental health and suicide prevention, including an Aboriginal-specific information leaflet on suicide prevention, can be found on the Indigenous HealthInfoNet. The website is: <http://www.healthinfonet.ecu.edu.au/frames.htm>.

Caesarean section

Introduction

The role of caesarean section in maternal mortality is unclear. The observational studies reported in the *Maternal deaths in Australia* and the UK *Why mothers die* series of reports are unable to clearly separate the maternal and fetal 'consequences of caesarean section from the indication for the operation' (Lewis G (ed.) 2004). Beginning with the 2000–2002 triennial report, the Confidential Enquiries into Maternal Deaths in the UK no longer includes a specific chapter on deaths after caesarean section. However, the report acknowledges the debate surrounding choice of method of birth and the inherent limitations of the data presented. The former is addressed by noting the 2004 guideline on caesarean section from the National Collaborating Centre for Women's and Children's Health on behalf of National Institute for Clinical Excellence (NICE) does not support planned caesarean sections without clear clinical indications (Lewis G (ed.) 2004). While the latter is concerned with the sensitivity and specificity of the data collected on caesarean section through the enquiries and the difficulty in interpretation of data presented, in particular a concern that 'undue weight might be placed upon the findings presented', the Enquiry has recommended that this is an area requiring research (Lewis G (ed.) 2004).

The chapter on caesarean section has been kept in this report to inform the debate on method of birth in Australia. There has been a marked increase in the proportion of births being delivered by caesarean section in Australia from 17% in 1988 (Lancaster & Pedisich 1993) to 28.5% in 2003 (Laws & Sullivan 2005). During the 2000–2002 triennium, the caesarean section rate increased from 23.3% of births in 2000 (AIHW 2003) to 25.4% in 2001 (Laws & Sullivan 2004a) and 27.0% in 2002 (Laws & Sullivan 2004b).

The national maternal death reporting form has a section titled: 'Type of delivery'. In relation to caesarean section the data fields are:

- elective caesarean section
- emergency caesarean section
- caesarean section for fetal retrieval in recently dead or moribund mother
- caesarean section (unspecified)
- other.

This chapter includes information of the women who died in the 2000–2002 triennium whose method of birth was caesarean section identified in one of the above categories. This includes deaths classified as direct, indirect, late and incidental.

Summary of findings for 2000–2002

Sixty-one (64.2%) women gave birth prior to their death. The type of delivery was not reported for one other woman although the notes suggested she was postpartum when she died. Forty-three of the 61 women who gave birth (70%) had a caesarean section (Table 43). Seven of the 43 caesarean sections were performed at perimortem for fetal retrieval. Of these 43 women, there were 19 direct deaths (one of which was late), 23 indirect deaths (four of which were late) and one incidental death. During the 2000–2002 triennium there were three cases where the caesarean section was thought to be a contributing cause of death. One case,

where maternal death was related to an elective caesarean section, is presented in the following chapter on *Deaths associated with anaesthesia.*

Table 43: Maternal deaths by type of death and type of caesarean section, Australia, 2000–2002

Type of caesarean section	Direct	Indirect	Incidental	Late	Total
Elective Caesarean section	4	6	—	3	13
Emergency caesarean section	12	8	1	2	23
Perimortem for fetal retrieval	2	5	—	—	7
Total	**18**	**19**	**1**	**5**	**43**

Illustrative cases

During the 2000–2002 triennium there were three cases where the caesarean section was thought to be a contributing cause of death. Two of the cases are presented below.

A woman with a twin pregnancy had a caesarean section at 34 weeks gestation for pre-eclampsia. The surgery was difficult as she had scar tissue from four previous laparotomies. Two of the laparotomies were for previous caesarean sections. Faecal peritonitis was diagnosed three days postoperatively, thought to be due to a small bowel perforation. Over a seven-day period she required four laparotomies and multiple transfusions for surgical wound washouts and abdominal bleeding. She developed septic shock and DIC and died 11 days postpartum.

Principal cause: Infection, septic shock secondary to faecal peritonitis.

Contributing causes: DIC, caesarean section associated.

Maternal death classification: Direct.

Caesarean section: Elective.

A woman was admitted with preterm rupture of the membranes at 27 weeks gestation. She had elevated BP and a caesarean section was performed. As the surgery was completed; bleeding from the left pelvic side wall was noted. A major artery was found to be bleeding with distortion of the pelvic anatomy, swollen tissues, fibroids and the bladder adherent to the lower segment of the uterus. A hysterectomy was performed; however, uncontrollable haemorrhage continued and she was unable to be resuscitated following a cardiac arrest.

Principal cause: Haemorrhage.

Contributing cause: Caesarean section associated.

Maternal death classification: Direct.

Caesarean section: Emergency.

Clinical comment

Examination of these data provides reassurance that there are few cases of maternal death in which either the operation of caesarean section, or the associated anaesthetic, has directly contributed to the death. However, this should not give rise to complacency about the possible impact of rising caesarean section rates on maternal death. The choice for caesarean section in a first birth may well lead to the inevitable choice of caesarean section in

subsequent births, removing the option of a potentially safer vaginal birth. In such cases, the contributor to the maternal death may not be the choice of method of birth in the index pregnancy, but a choice made in an earlier pregnancy when vaginal birth may have been a realistic option.

These cases highlight the surgical challenges faced by obstetricians, particularly in mothers who already have significant scarring, emphasising that this is not simple surgery but major abdominal surgery with the concomitant risks. Increasing rates of placenta accreta, described in the chapter on *Haemorrhage* also increase the risks associated with caesarean section.

Although the caesarean section in the first case was an elective caesarean section and believed to be a contributory cause of death, this woman had previously had two caesarean sections, making repeat elective caesarean section the accepted method of birth.

In the second case, the option of induction of labour with maternal hypertension was not a realistic choice at that gestation if the chance of neonatal survival was to be maximised. It is important to continue to monitor trends in caesarean-section-associated maternal deaths to better understand the relationship between obstetric decision-making and the consequences for future pregnancies.

Best practice

The National Collaborating Centre for Women's and Children's Health on behalf of National Institute for Clinical Excellence (NICE) released guidelines for the UK for caesarean section in 2004.

The NICE guidelines recommend that the following interventions be used to decrease morbidity from caesarean section:

- regional anaesthesia
- antibiotic prophylaxis
- thromboprophylaxis (following current guidelines)
- antacids
- anti-emetics.

Resources:

- National Collaborating Centre for Women's and Children's Health. Caesarean Section. Clinical Guideline. London: RCOG Press; 2004, The websites are: <htttp://www.nice.org.uk/> or <htttp://www.rcog.org.uk/>.

Deaths associated with anaesthesia

Introduction and definition

For the purpose of this report, deaths due to anaesthesia are defined as direct maternal deaths where the cause of death is directly attributable to the anaesthetic agent or procedure.

Summary of the findings for 2000–2002

During the 2000–2002 triennium 61 women died following giving birth to their baby. Of these, 43 had a caesarean section and thus an anaesthetic. The documentation pertaining to type of anaesthesia was incomplete for ten women, but showed that 22 (66.7%) of the 34 women where it was recorded had general anaesthesia, seven had epidural anaesthesia and four had spinal anaesthesia. This compared to the use of general anaesthetic for caesarean section in all mothers in Australia in 2003 of 8.4% (Laws & Sullivan 2005). There was one maternal death which was directly related to anaesthesia in the 2000–2002 triennium compared to three deaths in the previous triennium.

Illustrative case

A woman was living in a rural town and was in her third pregnancy. She was booked for an elective caesarean section at 38 weeks gestation in an urban centre. A healthy baby was delivered under epidural anaesthetic. The epidural catheter was removed on day four. After removal of the catheter she complained of low back pain and was noted to have tenderness over the sacroiliac joints. This settled over the next two days. She was discharged home on day eight postpartum.

Over the next 15 days she experienced increasing back pain, headache and dizziness. She became febrile at times and was seen by several doctors, receiving treatment for sacroiliitis. This included simple analgesics, non-steroidal anti-inflammatory drugs and antibiotics. The pain was thought to be referred from elsewhere and an appointment was made for her to see a neuro-physician.

On day 21 the woman again presented with back pain, headache and vomiting, and was noted to be restless, agitated and confused. She was transferred to a regional hospital where her condition quickly worsened. She required transfer to a tertiary metropolitan hospital, where on arrival she was acutely ill. She was in respiratory distress and unconscious with fixed, dilated pupils. Despite having burr holes to alleviate raised intracranial pressure, her clinical state did not improve and a decision was made for treatment to be withdrawn. Cultures taken from an epidural abscess and the base of the brain grew methicillin-resistant staphylococcus aureus.

Principal cause: Suppurative basal meningitis complicating epidural anaesthesia for caesarean section.

Maternal death classification: Direct.

Caesarean section: Elective.

Clinical comment

The number of women who have died from an anaesthetic-related procedure was less during the 2000–2002 triennium compared with the 1997–1999 triennium. Given the very small number of cases in recent triennial reports, this may reflect random variation or could be evidence of an actual decline in anaesthetic deaths. The CEMACH in the UK has more detailed information about a larger cohort of maternal deaths and showed that anaesthesia for caesarean section is 30 times safer than it was 40 years ago, with most deaths attributable to complications of general anaesthesia (Cooper & McClure 2004). This case illustrates that complications of epidural and spinal anaesthesia can also cause maternal death. Although rare, serious infection after regional anaesthesia will commonly present after discharge from hospital and failure to examine, diagnose and treat leads to poor outcome (Reynolds 2005).

Best practice

The Australian and New Zealand College of Anaesthetists (ANZCA) Guidelines for the management of major regional analgesia (ANZCA 2003) include specific principles for epidural analgesia in obstetrics, as follows,

- Epidural analgesia has the potential to change many of the normal physiological processes of labour and delivery. From the time that epidural analgesia is instituted, it is essential that the mother is under the care of a medical practitioner with obstetric training who can assess the mother as necessary, and rapidly effect birth of the baby by whatever technique is appropriate.
- The practitioner establishing regional analgesia must establish that the mother has consented to the procedure after having been informed about advantages, disadvantages and alternatives. This should normally be part of antenatal education.
- From commencement to completion of epidural analgesia in labour, there must be appropriately skilled staff and equipment available to monitor and care for both mother and fetus, and to manage any complications arising from the epidural analgesia or labour.

In every anaesthetising location, equipment for managing difficult intubations must be available (ANZCA 2000a, 2000b).

General principles of asepsis should be applied to the process of insertion and subsequent catheter management with epidural and spinal anaesthesia (ANZCA 2003; ANZCA 2005; Reynolds 2005).

Resources:

- The American Society of Anesthesiologists (ASA) difficult airway algorithm is the basis for management of the difficult airway in anaesthesia worldwide (ASA 2003). It can be found on their website, <http://www.asahq.org/publicationsAndServices/Difficult%20Airway.pdf>.
- ANZCA have the following professional documents available on their website, <http://www.anzca.edu.au/publications/profdocs/view.htm>:
  - Guidelines on Infection Control in Anaesthesia
  - Guidelines for the Conduct of Major Regional Analgesia in Obstetrics

- Recommendations on Minimum Facilities for Safe Anaesthesia Practice in Operating Suites
- Recommendations on Minimum Facilities for Safe Anaesthesia Practice outside Operating Suites
- Recommendations on Monitoring During Anaesthesia
- Guidelines for the Management of Major Regional Analgesia.

Section E: Method of review

State and Territory Maternal Mortality Committees

Most states and territories have expert committees that review maternal deaths (Appendix 3). The establishment and operation of these committees varies by their terms of reference, and the funding and legislative framework of each jurisdiction. These committees usually comprise some or all of the following experts: obstetricians, obstetric physicians, midwives, pathologists, general practitioners, epidemiologists, psychiatrists, anaesthetists, and Aboriginal and Torres Strait Islander and consumer representatives.

Each STMMC has developed different methods to maximise maternal death notifications; this may include notifications from health departments, attending practitioners, coroner's office, Registrar of Births, Deaths and Marriages, and review of the perinatal and hospital morbidity collections (Appendix 4). The STMMC review confidential information on each maternal death within their jurisdiction. The information reviewed may include hospital medical records, autopsy, toxicology, doctors', police and coroners' reports as well as other materials as directed by the individual Committee.

The method of enquiry differs by state and territory, with some jurisdictions using standard enquiry forms. Information collected varies but includes: usual place of residence of the woman, location at which death occurred, date of death, age at death, parity, gestational age at death, country of birth, date of delivery or abortion, number of days postpartum, Indigenous status, birth outcome, relevant medical history, interventions and procedures, admission to intensive care unit or coronary care unit, administration of blood transfusion, mechanical ventilation and / or anaesthesia, the terminal event, whether a post mortem or coronial inquest was conducted, pathology results, toxicology findings, principal and underlying causes of death, classification of death and any avoidable factors. This information is consolidated into a narrative as a case summary.

The STMMC consider each death individually to determine the cause of death where possible. Some of the committees also review the deaths for the presence of contributing factors. The STMMC categories each maternal death as direct, indirect or incidental. A number of the STMMC publish the findings of their maternal death review in annual state reports. Some publish case histories and jurisdictional MMRs.

Role of AIHW National Advisory Committee on Maternal Mortality

The role of the NACMM was to oversee the compilation of all reported deaths into a national report. Ethics approval was granted from the UNSW Human Research Ethics Committee for the review of the deaths and production of the report. All Committee members and contributing authors signed confidentiality undertakings as defined in section 29(1) of the AIHW Act 1987 prior to any access to the data.

Methods

The AIHW NPSU requests maternal death data from the states and territories. Data were provided to the NPSU for the 2000–2002 triennium using the national maternal death case report form (Appendix 5). The case report form was introduced in the 1997–1999 triennium, with the aim of standardising reporting to the NPSU. It is a summary form of key demographic, sociobehavioural factors, clinical, laboratory and death related information, with a text field for a detailed case summary. Names are not provided. Data were supplied in hard copy for all but one state which supplied it electronically.

This differs to the comprehensive audit process used by CEMACH in the UK, which allows for an evidence-based clinical review of each death and all surrounding issues that pertain to the death. Auditing and reporting is not as extensive in Australia when compared with the UK.

Completion of the form varied among the states and territories. Data elements such as Indigenous status, weight, country of birth and avoidable factors were not collected uniformly across Australia. Data validation and follow-up was undertaken by the NPSU to enhance reporting of key variables. Data were compiled to make a national maternal death database (NMDD).

A subcommittee of the NACMM, the Maternal Mortality Review Working Group (MMRWG) (Appendix 2) met three times to review all maternal deaths and classify them as direct, indirect or incidental, before allotting cases to specific chapters in the Report. Each maternal death was only counted once and assigned to one chapter. However, maternal deaths could be referenced in more than one chapter, for example a death from eclampsia where the method of birth was emergency caesarean section would be primarily reported in the *Hypertensive disorders in pregnancy* chapter but would also be referenced in the *Caesarean section* chapter.

In some cases the classification of death assigned by MMRWG differed to that reported to the NACMM on the maternal death form. The MMRWG objectives were to maintain consistency in the classification of deaths nationally, to be consistent with best practice using CEMACH as the gold standard, and to be inclusive rather than exclusive in categorising deaths (Lewis G (ed.) 2004). In 24% of the 99 reported cases for 2000–2002 (95 maternal deaths and four late incidental deaths) the classification of death was changed by the MMRWG. The changes in classification included reclassification of: two direct deaths to indirect (psychiatric and other haemorrhage); two indirect deaths to direct (pulmonary embolism and caesarean-section-associated infection); 14 incidental deaths to indirect (five psychiatric, three infection, two assaults, one each of cardiac, hypertension, haemorrhage and tumour); and four unascertained to indirect (one each of cardiac, TTP, psychiatric and malaria).

Case summaries

The purpose of including the case summaries or vignettes is stated by CEMACH to 'broadly describe the circumstances surrounding an individual woman's death and the lessons which could be drawn from this' (Lewis G (ed.) 2004). In the Australian reports the number of case summaries have been reduced to 1–3 per major cause of death, with only summary data included on the other deaths. The case summaries presented in this report were selected by members of the MMRWG and are summarised from antenatal records, hospital notes,

postmortem reports and coroners reports. The case summaries do not include all the information of any individual case and maybe the composite of two cases in some instances with the information presented illustrative of the cases. The cases have been selected to aid in the identification and dissemination of lessons learned from the overall review of cases: for the improvement of professional practice and the quality of overall service delivery; and for community awareness about the potential risks of pregnancy and the year following birth (Lewis G (ed.) 2004). The NACMM are cognisant of the responsibility and sensitivity of reporting maternal deaths. All efforts were taken to minimise the possibility of identification of the cases with only information relevant to the understanding of the causal pathway of the deaths included. However, due to the very small number of overall deaths and the limited number of cause–specific deaths there is a risk that spontaneous recognition of a particular death may occur and the NACMM regrets any distress this may cause family members and relatives. The NACMM judged that this small risk did not warrant the use of perturbation in the report. Furthermore, the NACCM was concerned that the integrity and utility of the report would be compromised by perturbation. A number of cases have already been published in the individual state and territory reports, and are reproduced in these reports. It should be noted that all deaths occurred 4–6 years prior to the compilation of the report. Also, in several instances, cases have already been reported widely in the electronic or print press.

Late maternal death reporting

Australia is now collecting and reporting on late maternal deaths in its triennial maternal death reports. Five late maternal deaths were reported in 1997–1999 and eight in 2000–2002. Four of the states and territories reported late maternal deaths. A further four incidental late deaths were notified to the NACMM. These deaths are not presented in this report as they do not satisfy the inclusion criteria. However, reporting of these deaths to NPSU is important as some of these deaths could be reclassified from incidental to indirect if definitions or criteria change and become more inclusive over time. It is anticipated that the process of reporting and investigating late maternal deaths may take some time to become established in the current maternal mortality surveillance systems. Caution should be used when interpreting the findings as under-ascertainment of these deaths is likely. It is anticipated that there will be more complete reporting of late maternal deaths in the future. Late maternal deaths will be included in the next triennium 2003–2005.

Validation of maternal deaths 2000–2002

The maternal death data received by the NPSU were validated with data from the Australian Bureau of Statistics (ABS) mortality data and National Hospital Morbidity Database (NHMD).

ABS Mortality Data

An extract of mortality data were prepared for women aged 11–54 years, by year of registration of death (2000–2003), with a date of death between January 1, 2000 and December 31, 2002 with the following underlying and or principal causes of death (ICD-10 codes: O00–O99 pregnancy, childbirth and puerperium chapter; F53 mental illness; A34

obstetrical tetanus; M83.0 puerperal osteomalacia; and E23.0 postpartum necrosis of pituitary).

Data from the National Maternal Mortality Database (NMMD) were validated against the ABS data. Maternal deaths identified in the ABS data, which were not in the NMMD, were notified to the relevant STMMC. There were 12 deaths notified to the STMMC. Of these, nine were confirmed as maternal deaths (one was confirmed as a late maternal death) by the relevant jurisdiction. These deaths have been included in this report. Three cases were found not to be maternal deaths by the relevant state or territory.

National Hospital Morbidity Database

The National Hospital Morbidity Database (NHMD) is a compilation of electronic, confidentialised summary records for admitted patients separated from almost all public and private hospitals in Australia. Data from the NMMD were validated against the NHMD data. Maternal deaths identified in the NHMD that were not in the NMMD were notified to the relevant state or territory health authority.

The parameters used to search the databases were: females, aged 10–54 years with a mode of separation from hospital of 'died' and one of the following ICD-10-AM diagnosis codes (O00–O99 Pregnancy, Childbirth and Puerperium chapter; F53.x Mental and behavioural disorders associated with the puerperium, not elsewhere classified; A34 Obstetrical tetanus; M83.0x Puerperal osteomalacia; E23.0 Hypopituitarism; Z37.x Outcome of delivery; Z34.x Supervision of normal pregnancy; Z35.x Supervision of high-risk pregnancy; and Z39 Postpartum care and examination).

The identification of maternal deaths in the NHMD using the above criteria and notification of them to the relevant state or territory health authority has been incorporated into the routine annual edit checks undertaken by the AIHW for the NHMD. The state and territory health authorities are asked to provide full details of these records to their STMMC for review. For the 2000–2002 triennium, 17 records were notified to the state or territory health authorities. Of these, nine were confirmed as maternal deaths and have been included in this report. Two records were identified as duplicates in the NHMD and the remaining six records were not identified as maternal deaths by the relevant state or territory health authority.

Ascertainment of maternal deaths

Death certificates

Australia now has pregnancy tick-box questions on all state and territory death certificates. There remains a lack of national consistency in the questions asked on the death certificates.

National Coroners Information System

The National Coroners Information System (NCIS) is a national Internet-based data storage and retrieval system for coronial cases in Australia. It provides an efficient and effective research tool to authorised users in the fields of death, injury surveillance, public health and

safety. The NCIS has recently incorporated ICD–10 codes which will allow for the identification of maternal deaths in the future.

Data development

The NMDD is an electronic data collection of maternal deaths for the six year period 1997–2002. During 2005, the NPSU finalised modifications to a pilot database which is to be trialled in one state to assess its feasibility for reporting of maternal mortality data to the NPSU.

A national workshop will look at future data development for monitoring maternal mortality. This will be a technical workshop which may comprise state and territory representatives together with representation from professional colleges, Coroners, ABS, AIHW, the National Centre for Classification in Health, the Australian Commission on Safety and Quality in Health Care, and the Australian Government Department of Health and Ageing. A key objective of the workshop is to examine the processes used to investigate deaths at the state, territory and national level. It will include investigating the development of a mechanism that would enable nationally consistent examination of maternal deaths. Methods for improving the ascertainment of late maternal deaths will also be presented and discussed.

Assessment of contributing factors

There is no agreed national definition for the term 'contributing factors' to a maternal death. The lack of a standardised definition or objective instrument may bias determination of contributing factors. The definition used by the NACMM is, 'some departure from the accepted standard of satisfactory care by the woman, practitioner or institution which may have contributed to the death' (Slaytor et al. 2004 p. 88). In an attempt to standardise contributing factors, five categories were included on the National Maternal Death Reporting Form. These are personal/family, logistical systems, facilities, health personnel and model of care.

The amount of information on contributing factors provided to the NPSU varied among states and territories. It depended upon privacy concerns and protocols used in examining the deaths. In some states where the deaths are examined, the Committee assigns contributing factors, based on collective expert opinion. Due to the lack of national standardisation of assignment of contributing factors, we have presented only general information on these factors.

In cases in which contributing factors were considered to be present, it is not suggested that death could certainly have been prevented. The presence of a contributing factor is regarded as an indication that the risk of death could have been lessened. This information is important to ongoing efforts to ensure safe motherhood through practice improvement. The utility of this measure needs to be investigated prior to inclusion in future reports.

Maternal deaths of non-residents transferred to Australia for medical care

In each triennium there is approximately one maternal death of a woman who has given birth in another country and subsequently arrived in Australia where the death has occurred. As the birth did not occur in Australia the death is not counted in Australian statistics. One such maternal death occurred in Australia during the 2000–2002 triennium.

A woman gave birth to her fifth baby in her usual place of residence, a country in the Pacific. In the three days following the birth, she developed a headache and was taken to one of the Torres Strait Islands and evacuated to Thursday Island. She experienced seizures and became unconscious. She tested negative for falciparum malaria and a clinical diagnosis of a cerebral vascular accident was made. Her condition rapidly deteriorated and she became decerebrate with fixed dilated pupils. She died on day 17 postpartum. A postmortem was not performed.

Principal cause: Intracranial haemorrhage.

Maternal death classification: Indirect.

Update of maternal deaths 1997–1999

Following publication of the 1997–1999 report, one maternal death was notified to the NPSU and a second was reclassified by the STMMC from an incidental death to an indirect death. The deaths have subsequently been included and reclassified in the NMDD and updated in the 2000–2002 report.

A woman died at home in her sleep at 32 weeks gestation. She had been diagnosed with diabetes in 1993 and had been commenced on insulin during the pregnancy. She complained of being tired during the day and went to bed in the mid afternoon. On checking six hours later, she could not be awakened. An ambulance was called but there were no signs of life. The postmortem could not find a cause of death and the coroner reported the cause of death to be diabetes.

Principal cause: Diabetes.

Maternal death classification: Indirect.

A woman presented at 31 weeks gestation with a flu-like illness and tonsillitis. Two weeks later she was admitted to the local hospital in a febrile condition with generalised aches. There was no meningism and she was normotensive. The woman was given intramuscular Penicillin for tonsillitis. She became disorientated; experienced a generalised seizure and the fetal heart could not be heard. She was intubated and transferred to a tertiary intensive care unit with a provisional diagnosis of eclampsia and HELLP Syndrome. She required a caesarean section for a stillborn infant and was commenced on a magnesium sulphate infusion. She developed DIC and multi-system failure and was unable to be resuscitated.

Bacteriological examination of postmortem tissue showed a light growth of *Escherichia coli* from the lung, uterus and liver. Histological examination showed marked and extensive intra-pulmonary haemorrhage and hepatic necrosis. The pathologist's comments were:

> The massive hepatic necrosis is non-specific and can be seen in patients dying of shock due to any cause. Hepatic features of pre-eclampsia/eclampsia such as

intra-sinusoidal fibrin deposition were not seen but the absence of this feature does not exclude pre-eclampsia/eclampsia as a contributing cause. Typically, acute fatty liver of pregnancy results in a reduced liver weight at autopsy with cholestatic changes but the massive necrosis and fatty change present does not exclude this either. In summary, the massive hepatic necrosis is attributed to hypoperfusion secondary to shock due to *Escherichia coli* sepsis, resulting in the irregular distribution of the necrosis.

Principal cause: Infection, *Escherichia coli* sepsis.

Contributing causes: DIC, hepatic necrosis.

Maternal death classification: Indirect.

Section F: Appendixes

Appendix 1: Membership of the AIHW National Advisory Committee on Maternal Mortality

Representative	Organisation / specialty
Associate Professor James King (Chair 2004–current)	State Mortality Committee Vic
Professor Michael Peek (Deputy Chair)	Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZOG)
Clinical Associate Professor Barry Walters	State Mortality Committee WA
Professor Michael Bennett	State Mortality Committee NSW
Dr Melwyn D'Mello	State Mortality Committee Tas
Professor David Ellwood	Territory Mortality Committee ACT
Dr Paul Devenish-Meares	State Mortality Committee Qld
Professor Jeffrey Robinson	State Mortality Committee SA
Ms Barbara Paterson	Territory Mortality Committee NT
Dr Elizabeth Sullivan	AIHW National Perinatal Statistics Unit
Professor Lesley Barclay	Australian Council for Safety and Quality in Health Care
Professor Michael Humphrey	RANZCOG: Specialist Outreach
Professor Michael Paech	Australian and New Zealand College of Anaesthetists (ANZCA)
Associate Professor Marie-Paule Austin	Royal Australian and New Zealand College of Psychiatrists
Dr Susan Arbuckle	Royal College of Pathologists of Australasia
Dr William Hague	Obstetric medicine physician
Ms Merryl Green (resigned, awaiting new appointment from Maternity Coalition)	Maternity Alliance
Associate Professor Deborah Black	School of Public Health & Community Medicine, UNSW
Ms Wendy Pollock	Australian College of Midwives

Appendix 2: Membership of AIHW National Advisory Committee on Maternal Mortality Review Working Group

The MMRWG met in December 2004 and in May 2005.

The members of the working group were:

Associate Professor James King (Chair)

Dr Elizabeth Sullivan (Co-chair)

Professor Michael Peek (present December 2004)

Ms Wendy Pollock

Dr Sue Kildea (present May 2005)

Appendix 3: Membership of the State and Territory Maternal Mortality Committees 2000–2002

The composition of the State and Territory Maternal Mortality Committees for the period 2000–2002 were as follows:

New South Wales Maternal and Perinatal Committee 2000–2002

Professor William Walters (Chair)

Dr Susan Arbuckle

Dr David Barclay (2000)

Ms Claire Bell

Ms Melinda Bell (2002)

Professor Michael Bennett

Dr Andrew Berry

Ms Pat Brodie

Dr Andrew Child

Ms Hannah Dahlen (2002)

Ms Jennifer Dawson (2002)

Dr John Danials (2000–2001)

Professor John Dwyer (2002)

Professor David Henderson-Smart

Dr Jane Hargood (2002)

Dr John Hobbs

Dr Ian Hoult

Ms Linda Jones

Dr Penelope Knowlden

Ms Judith Meppem

Ms Virginia Miltrup (2002)

Dr Des Mulcahy

Dr Elisabeth Murphy

Dr Louise Newman

Ms Margy Pym

Dr John Smoleniec

Ms Sue Stewart (2001–2002)

Dr Lee Taylor

Professor Brian Trudinger

Victorian Consultative Council on Obstetric and Paediatric Mortality and Morbidity, Maternal Mortality Subcommittee

Associate Professor James F King (Chair)

Dr Christine Bessell

Dr Virginia Billson

Professor Robert Burrows

Dr Methew Lynch

Professor Jeremy Oats

Ms Wendy Pollock

Professor Michael Permezel

Dr Andrew Ross

Dr Craig Walker

Queensland Council on Obstetric and Paediatric Morbidity and Mortality, Maternal Mortality and Morbidity Subcommittee

Dr Ifor Thomas

Dr Jeremy Oats

Ms Marie Barton

Ms Vicki Flenady

Dr Karen Lust

Dr Dianne Payton

Dr Ian Stephens

Dr Donald Cave

Professor Michael Humphrey

Dr Roy Hemsley

South Australian Maternal, Perinatal and Infant Mortality Committee, Maternal Subcommittee

Professor Jeffrey Robinson (Obstetrician, Chairperson)

Dr Scott Simmons (Obstetric anaesthetist)

Dr Brian Duffy (till 2002) (Obstetric anaesthetist)

Dr William Hague (Obstetric physician)

Dr James Harvey (Obstetrician)

Associate Professor T Yee Khong (Pathologist)

Mrs Elizabeth Wood (Midwife)

Dr George Kokar (General practitioner)

Dr Annabelle Chan (Public health physician, Medical Secretary)

Maternal Mortality Committee of Western Australia

Permanent Members

Professor Con Michael, Ex Officio (2000)

Dr Mark McKenna (Deputy), Ex Officio (2000)

Professor John Newnham (2001)

Dr Jan Dickinson (Deputy), ex officio (2001)

Dr Peter Hugo, Australian College of Obstetricians and Gynaecologists (2000–2002)

Dr Tim Jeffery, Commissioner of Health (2000–2002)

Dr Louise Farrell (Deputy), Commissioner of Health (2000–2002)

Provisional Members

Dr Michael Jones, Australian Medical Association (2000)

Dr Katrina Alexander, Australian Medical Association (2000)

Dr David Mildenhall, Australian Medical Association (2000)

Dr Robin Kirk, Australian Medical Association (2000)

Ms Julie Watson, Australian Nursing Federation (2000–2002)

Mr Terry Jongen, Australian Nursing Federation (2000–2002)

Investigators

Dr Christophor Nichols, Chair MMC (2000)

Ms Jane Whittaker, Chair MMC (2000)

Dr Everett Magann (2001)

Dr Virginia McLaughlin (2001)

Tasmanian Maternal Mortality and Morbidity Committee

The Committee did not meet during the 2000–2002 triennium, although there were three maternal deaths reported.

Northern Territory Maternal and Child Health Committee

The Committee did not meet in the 2000–2002 triennium. The NT representative for the NACMM was Dr Margaret O'Brien.

Australian Capital Territory Maternal, Perinatal Information Network

The data for the Australian Capital Territory maternal deaths 2000–2002 were collected and provided to the NPSU by the ACT Maternal Perinatal Information Network. The Committee was formed in 1998 and meets three or four times per year.

The Committee for 2000–2002 comprised of:

Professor David Ellwood, Associate Dean, Clinical School, The Canberra Hospital (TCH) (Chairperson)

Ms Maureen Bourne, Data Manager, Population Health Research Centre, ACT Health (Coordinator)

Ms Karen Lees, Population Health Research Centre, ACT Health

Associate Professor Graham Reynolds / Dr Alison Kent, Neonatologist, Centre for Newborn Care, The Canberra Hospital

Dr Jane Thompson, Clinical Health Improvement Program (CHIP), Women's & Children's Health (W&CH), TCH

Dr Sue Packer, Community Paediatrician, Child at Risk Assessment Unit, TCH

Dr Bish Mukerjee, Director of Obstetrics and Gynaecology, Maternity Unit, TCH

Ms Giovanna Richmond, Director, Child, Youth and Women's Health Program, ACT Community Care

Ms Rosemary O'Donnell / Ms Chris Bulters / Ms Rosemary Kennedy, Director of Nursing W&CH, Maternity Unit, TCH

Ms Sue Minter, Associate Director of Nursing, Maternity, Medical & Mental Health, Calvary Public and Private Hospitals

Ms Stephanie Ham, Clinical Nurse Consultant, Delivery Suite, John James Memorial Hospital

Mr Gary Kennedy / Mr Mike Clark, Manager, Data Management Unit, ACT Health

Ms Mary Kirk, Director of Nursing, Queen Elizabeth II Family Centre, ACT Health

Mr Geoff Bagnall, Consumer Representative, Health Care Consumers

Ms Emma Baldock, Homebirth Midwife

Appendix 4: Method of enquiry

State and Territory Maternal Mortality Committees

Each state or territory has a slightly different data collection methodology.

In New South Wales, the Director General of Health has issued a policy directing hospitals to notify maternal deaths to the NSW Maternal and Perinatal Committee. Information is also obtained from the NSW Midwives Data Collection, the Registry of Births, Deaths and Marriages via the Australian Bureau of Statistics (ABS), and there is an arrangement with the Coroners office to provide reports for all maternal deaths.

In Victoria, since 2005, the Registrar of Births, Deaths and Marriages is obliged to notify the Council on Obstetric and Paediatric Mortality and Morbidity of any maternal deaths. A variety of other sources are used to notify the Committee including the midwives' reports, the Coroners office and newspaper reports. Case histories are built up from postmortem reports, police reports and confidential medical reports.

In Queensland, all deaths, including maternal deaths, are reported via the Registrar General's Office to the ABS, where deaths are coded. Information about maternal deaths is also received via informal mechanisms and through information on the death certificate.

In Western Australia, maternal deaths are notified by the attending practitioner. The Department of Health, WA also receives death certificate data from the Registry of Births, Deaths and Marriages. Case histories are then gathered from hospitals, medical practitioner clinical case notes, and from coronial and postmortem reports.

In South Australia, maternal deaths may be notified through the SA perinatal data collection or advised by health professionals. The Secretariat of the Maternal, Perinatal and Infant Mortality Committee also actively seeks to identify maternal deaths through the following mechanisms: deaths from the Births, Deaths and Marriages Registration Division list; applying the search algorithm developed by the National Perinatal Statistics Unit to the South Australian hospital morbidity database; mandatory notifications of maternal deaths to the Department of Health by state-funded public health services under the Sentinel Event Reporting System; and newspaper reports and death notices. Case histories are prepared from confidential medical reports from medical practitioners, clinical case records, Coroner's Office and autopsy.

In the Northern Territory, information regarding maternal deaths comes primarily through hospital mortality data. Additional identification may occur through the NT Midwives Data Collection.

The small populations of Tasmania and the Australian Capital Territory increase the likelihood that detailed information is easily retrieved from hospitals, the Coroner's office and attending practitioners. The ACT also requests the Registrar General to provide a list of deaths of women where the women are reported to have a child or children that are less than one year of age at the time of death from the ACT death register. This process is particularly helpful for the identification of incidental maternal deaths. The ACT Maternal Perinatal Information Network coordinates the collection of the ACT maternal death data.

Appendix 5: National Maternal Death Reporting Form

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NATIONAL MATERNAL DEATH REPORTING FORM

1. This standardized reporting form is for the use of State and Territory Maternal Mortality Committees and hospitals (where indicated) in the review of all deaths in women while either pregnant or within 42 days of a pregnancy being delivered or terminated, irrespective of the duration and the site of the pregnancy, including abortions and ectopic pregnancies. Cases where the pregnancy was unlikely to contribute significantly to the death are also included.
2. All data will be treated confidentially and is covered under the protection of the AIHW Act.
3. This form was developed in conjunction with the National and State and Territory Maternal Mortality Committees. Some components have been developed from the Guidelines for completing the maternal death notification form (Second edition, 1999), Department of Health, South Africa.
4. Please return forms and direct enquiries to Dr Elizabeth Sullivan, Director, AIHW National Perinatal Statistics Unit, Level 2, McNevin Dixon Building, Randwick Hospital Campus, Avoca St, Randwick NSW 2031. Please telephone Elizabeth Sullivan on (02) 9382 1014 with any queries.

AIHW

For office use only: AIHW NPSU case number ☐☐☐☐☐☐☐

Details of deceased	
State/Country (of usual residence) **Postcode** (of usual residence) ☐☐☐☐	**State in which death occurred**
Date of death ☐☐/☐☐/☐☐☐☐	**State case number** ☐☐☐☐☐☐
Maternal date of birth ☐☐/☐☐/☐☐☐☐	**Maternal country of birth**
Torres Strait Islander or Aboriginal status ☐ Torres Strait Islander ☐ Aboriginal ☐ Both ☐ Non Indigenous ☐ Not specified	
Maternal age at death ☐☐/☐☐/☐☐☐☐	**Maternal weight at time of death**
Date of delivery/ abortion (if applicable) ☐☐/☐☐/☐☐☐☐	**If death occurred antepartum; gestational age at death** ☐☐ (weeks)
Plurality ☐ Single ☐ Multiple If multiple, no. ….	**If death occurred postpartum; number of days postpartum at death** ☐☐☐ (days)
Parity (including current pregnancy if delivered at or beyond 20 weeks gestation): ☐☐	**Did the mother smoke at all during pregnancy?** ☐ Yes ☐ No ☐ Unknown
Was this pregnancy the result of assisted reproductive technology? ☐ Yes ☐ No ☐ Unknown If yes, please specify ……	
Place of death	
Setting of death : ☐ Home ☐ Hospital ☐ Birth Centre ☐ Other (please specify) ……	**If hospital, was it:** ☐ The hospital in which she was booked to delivery ☐ Un-booked admission ☐ An emergency transfer from elsewhere ☐ Other (please specify) …… **Hospital level** ……
Date of last hospital admission (if applicable): ☐☐/☐☐/☐☐☐☐	**Date of last hospital discharge** (if applicable): ☐☐/☐☐/☐☐☐☐

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NATIONAL MATERNAL DEATH REPORTING FORM

Delivery and neonatal information

Type of labour: ☐ Spontaneous ☐ Augmented ☐ Induced ☐ No labour ☐ No specified

Type of delivery:

☐ Spontaneous vaginal
☐ Elective Caesarean section
☐ Forceps
☐ Vaginal breech
☐ Vacuum extraction
☐ Emergency Caesarean section
☐ Caesarean section for fetal retrieval in a recently dead or moribund mother
☐ Caesarean section (unspecified)
☐ Unspecified
☐ Other (please specify) .....

Number of previous Caesarean section deliveries: .....
Was the previous birth by Caesarean section? ☐ Yes ☐ No ☐ Unknown

Baby outcome (if applicable)
☐ Livebirth ☐ Stillbirth ☐ Neonatal death Birth weight: ..... (gms)

Maternal medical conditions

Pre-existing conditions

☐ Diabetes mellitus ☐ Cardiac disease ☐ Mental Illness
☐ Essential hypertension ☐ Epilepsy ☐ Other

Comments on pre-existing conditions:

Pregnancy related conditions

☐ Gestational diabetes ☐ Hypertensive disorder of pregnancy ☐ Ectopic pregnancy ☐ Other

Comments on pregnancy related conditions:

Interventions (tick appropriate boxes)

Early pregnancy		Antenatal		Intrapartum		Postpartum		Other	
Evacuation		Transfusion		Instrument Del.		Evacuation		Gen. Anaes.	
Laparotomy		Version		Symphysiotomy		Laparotomy		Epidural	
Hysterectomy				Caesarean		Hysterectomy		Spinal Anaes.	
Transfusion				Hysterectomy		Transfusion		Local Anaes.	
				Transfusion		Manual removal		ICU/CCU	
						Return to OT		Ventilation	

Other interventions: .....
Comments on interventions:

Anaesthesia: (related to terminal event)

☐ No ☐ Yes ....(please specify)

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NATIONAL MATERNAL DEATH REPORTING FORM

Birth attendant:

☐ Obstetrician ☐ Registrar/RMO ☐ Other Doctor ☐ Anaesthetist
☐ Midwife ☐ GP ☐ Other birth attendant .....

Cause of death – as specified by State or Territory Maternal Mortality Committee

Primary (underlying) cause of death:

Contributing (antecedent) causes of death:

Terminal event (description):

Classification of death

☐ Direct ☐ Indirect ☐ Incidental

Classification of death (into ICD-10 categories*)

☐ Pregnancy-related death – 0 to 42 days (any cause; O95 or other pregnancy, childbirth, puerperium code)
☐ Late maternal death – 42 to 365 days (direct or indirect; O96))
☐ Death from sequelae of direct causes – post 365 days (direct; O97)
☐ Other – incidental death occurring 42-365 days post-pregnancy or termination

Cause of Death – as specified on Medical Death Certificate

Direct cause of death (disease or condition directly leading to death)

.....

Antecedent causes of death:

Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last

(1) .....

(2) .....

(3) .....

Post-mortem information

Post-mortem conducted:	**Coronial inquest:**
☐ Yes ☐ No	☐ Yes ☐ No

Macroscopic/ microscopic findings: (please attach relevant documentation)

Toxicology findings: (please attach relevant documentation)

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NATIONAL MATERNAL DEATH REPORTING FORM

Avoidable factors as determined by State/Territory Maternal Mortality Committee

Avoidable factors ☐ Yes, avoidable factors present ☐ No, death was inevitable

System	Examples	✔
Personal/family	Delay in woman seeking help	
	Refusal of treatment or admission	
	Insufficient antenatal care	
Logistical systems	Lack of transport from home to health care facility	
	Lack of transport between health care facilities	
	Communication breakdown between health services	
Facilities	Lack of facilities, equipment or consumables	
	Lack/ delay in access to health services due to rural location	
Health personnel	Lack of human resources (e.g. lack of available staff)	
	Lack of antenatal care providers	
	Lack of expertise, training or education	
	Lack of access to interpreter services	
Model of care	Inappropriate early discharge	

Comments on avoidable factors:

Case summary:
(or please attach discharge summary, autopsy report and other relevant information)

Reported in State report: ☐ Yes ☐ No

If yes, which State or Territory:

.....

Name of person completing form:
.....

Signature: .....

Phone No. .....

Date: ☐☐/☐☐/☐☐☐☐

Please return forms to Dr Elizabeth Sullivan, Director, AIHW National Perinatal Statistics Unit, Level 2, McNevin Dixon Building, Randwick Hospital Campus, Avoca St, Randwick NSW 2031.

Appendix 6: Statistical methods and classifications

Age-specific and age-standardised rates

Age-specific rates

Age-specific mortality rates were calculated by dividing the average number of maternal deaths for the years 2000–2002 occurring in each specified age group by the corresponding population at risk in the same age group in the specified time period (i.e. the average of the Australian female population at 30 June for the years 2000–2002, for each age group), expressed as a rate per 100,000 women. These rates are used to compare differences in maternal mortality between age groups.

Age-standardised rates

Rates are adjusted for age to facilitate comparisons between populations that have different or changing age structures. This effectively removes the influence of age structure on the summary rate, described as the age-standardised rate. The method may be used for both incidence and mortality calculations. There are two different methods commonly used to adjust for age. In this report, direct standardisation was used in which the expected number of maternal deaths in each 5-year age group was calculated by multiplying the age-specific maternal mortality rates (see above) by the corresponding age group in the standard population (i.e. the Australian female population at 30 June 2001 for each age group) (Table 44) and dividing by 100,000. The age-standardised rate was calculated by summing the expected number of maternal deaths and dividing by the total of the standard population (the Australian female population aged 15–44 years at 30 June 2001) and multiplying by 100,000.

Table 44: The Australian female population as at 30 June 2001

Age group (years)	Females
0–4	624,858
5–9	657,874
10–14	660,094
15–19	662,077
20–24	641,636
25–29	706,171
30–34	739,696
35–39	750,770
40–44	744,821
45–49	683,539
50–54	648,237
55–59	495,911
60–64	408,042
65–69	346,923
70–74	334,826
75–79	292,000
80–84	201,800
≥ 85	183,313
Total	**9,782,588**
Total 15–44	*4,245,171*

Source: Australian Bureau of Statistics population estimates for 30 June 2003.

Glossary

Caesarean section (C/S): operative birth through an abdominal incision.

Epidural: injection of anaesthetic agent into the epidural space of the spinal cord.

Fetal death (stillbirth): death prior to the complete expulsion or extraction from its mother of a product of conception of 20 or more completed weeks of gestation or of 400 grams or more birth weight. The death is indicated by the fact that after such separation the fetus does not breathe or show any other evidence of life, such as beating of the heart, pulsation of the umbilical cord or definite movement of voluntary muscles.

Gestational age: the duration of pregnancy in completed weeks calculated from the date of the first day of a woman's last menstrual period and her baby's date of birth, or via ultrasound, or derived from clinical assessment during pregnancy or from examination of the baby after birth.

Grand multipara / multiparous: a woman who has had four or more previous pregnancies resulting in a live birth or stillbirth.

Induction of labour: intervention to stimulate the onset of labour.

Live birth: the complete expulsion or extraction from its mother of a product of conception, irrespective of the duration of the pregnancy, which, after such separation, breathes or shows any other evidence of life, such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles, whether or not the umbilical cord has been cut or the placenta is attached; each product of such a birth is considered liveborn (WHO definition).

Maternal age: mother's age at death.

Multipara / multiparous: a woman who has had at least one and less than four previous pregnancies resulting in a live birth or stillbirth.

Neonatal death: death of a liveborn baby within 28 days of birth.

Nullipara: a woman who has never given birth.

Parity: the number of previous pregnancies resulting in live births or stillbirths (of 20 weeks gestation or 400 gm birth weight), including current pregnancy if delivered at or beyond 20 weeks gestation.

Preterm birth: birth before 37 completed weeks of gestation.

Primipara/primiparous: a woman who has had no previous pregnancy resulting in a live *birth* or *stillbirth*.

Spontaneous vaginal birth: birth without intervention.

Stillbirth: see *Fetal death*.

Term: between 37–41 completed weeks of gestation.

Undelivered: a woman who has died while still pregnant.

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Maternal deaths in Australia 2000–2002 is the thirteenth report on women who die during pregnancy and childbirth. Maternal deaths are rare, catastrophic events and require monitoring and investigation. The report is an observational study of maternal deaths based on information provided by the states and territories which includes information about the women, pregnancy, clinical care and the deaths. Maternal deaths that occurred up to a year after the end of the pregnancy are included. Illustrative case summaries highlight key clinical and public health issues that may be causally related to maternal deaths. The report is produced by the AIHW National Perinatal Statistics Unit based at the University of New South Wales and will be particularly useful to maternity service planners and providers, consumers of maternity services, academics, students and those conducting research in maternity care.