Original Research
Radiation exposure from diagnostic imaging in trauma patients presenting to emergency department
Hilman H. Tjiang, Drew Richardson

A search for the high risk patient: a retrospective study of carotid endarterectomy
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Osteoporosis: the forgotten diagnosis?
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We are proud to present Volume 3 Issue 2 of the Medical Student Journal of Australia (MSJA). It has been a privilege to work on producing such an exciting and interesting issue of the journal.

Producing this issue of the MSJA has given the editorial team a unique view at the quality of research and opinion produced by the medical student cohort. It is obvious that the Australian National University Medical School’s (ANUMS) association with world class research institutions such as the John Curtin School of Medical Research (JCSMR) and the National Centre for Epidemiology and Population Health (NCEPH) have enhanced the research skills of the student population.

This edition has been characterised by a surge in research articles, which reflects the growing reputation of the journal, with increasing numbers of researchers choosing to submit to the journal and allowing the editorial team to select truly high quality research. It is research that can help change the future practice of medicine, and it is exciting to see how students are contributing so positively to improving patient care.

However, research is not the only focus of this issue; we have a variety of other articles ranging from the humorous to the stressful which all together help to create a picture of the varied and interesting experience that a medical student can expect to see throughout their study and hopefully, their career.

The editorial committee has worked cohesively and effectively in producing this journal and would like to thank previous editorial teams for providing an excellent framework in which to produce this issue. It has been a pleasure and a privilege to read the work of our peers and we hope you also enjoy reading this edition of the MSJA.

MSJA Editorial Board
Alexander Dillon, Jeffrey Lai, Mimi Chiu, Abby Gnanendran, Morgan Edwards, Philip Chia, Mitchell Blake, Ragu Krishnamoorthy
It is my pleasure to welcome you to Volume 3, Issue 2 of the Medical Student Journal of Australia, developed and coordinated by final year medical students at the Australian National University (ANU). Congratulations to the team for producing another edition of the journal, and to all those who have contributed.

In this issue there are reports on a number of research projects completed as part of the ANU Medical School MBBS program. The ANU is a leading research intensive university, and given this, the production of these reports is particularly pleasing. The practice of medicine is the professional application of medical science and population health science. It is essential that doctors are fully conversant with research principles and practice. Congratulations to all who have had their projects accepted for publication - it is a real achievement. It strengthens the research ethos of the Medical School.

In this edition there are research articles on some of the more prevalent diseases such as diabetes and osteoporosis as well as less common but important aspects of medicine such as carotid endarterectomy and radiation exposure in trauma patients. Also included are some more reflective pieces of students’ elective experiences at the end of their third year of study as well as an interesting article on the history and gender bias of medical eponyms.

I look forward to reading future issues.

Professor Nicholas Glasgow

Professor Nicholas Glasgow
MBChB, MD, FRNZGP, FRACGP, FACHPM
Dean, Medicine & Health Sciences
Dean, ANU Medical School
ANU College of Medicine, Biology and Environment
INTRODUCTION

Diagnostic radiology studies are used extensively in the emergency departments. They provide rapid and accurate diagnosis for the attending emergency physicians for the assessment of life-threatening injuries in emergency patients, especially blunt trauma patients where the injuries are not easily-visible diagnosed. Current technological advancement has pushed the technique to produce higher sensitivity and accuracy than ever before. It has become an inseparable element in modern medicine in the diagnostic process. However, the ionising radiation produced by the radiologic studies (plain X-ray radiographs and Computer Tomography (CT) scans) has been associated with specific risks.

In general, risks associated with ionising radiation can be categorised into two effects which can be measured in doses: radiation dose for acute exposure effect (unit: Gray/Gy) and effective dose for chronic exposure effect (unit: Sievert/Sv). The acute exposure effect, commonly termed ‘radiation sickness,’ includes damages to skin, GI tract, lung, CNS, bone marrow and can potentially lead to death at a high enough dose (1). Radiologic studies are not associated with acute exposure effect because it does not generate harmful level of radiation dose (typically <10mGy)/(1). However, the more concerning effect of diagnostic imaging is its chronic exposure effect or the so-called stochastic effects, which include carcinogenesis and hereditary effects. The association of exposure to ionising radiation with development of cancer has been extensively studied from 1945 atomic bomb survivors in Japan. These studies have shown strong evidence linking increased risk of developing both leukaemia and solid neoplasia to exposure of high doses (>150 mSv) of radiation from the atomic bombs (2-5).

ABSTRACT

Objective: Trauma patients presenting to emergency department require extensive radiologic investigations which are associated with high-radiation doses. The objective of this study is to determine the amount of cumulative effective dose received by adult trauma patients presenting to emergency department during the first 24 hours of their care.

Method: Emergency department records for trauma patients presenting to the Canberra hospital (ACT, Australia) between 1st January and 31st December 2008 were retrospectively reviewed for all diagnostic (plain radiographs and Computed Tomography (CT) scans) imaging performed on adult (>18 years old) trauma patients who arrived directly from the scene of injury within the first 24 hours from arrival. Estimated radiation dose was used to calculate the total radiation dose for each individual.

Results: A total of 118 patients met the inclusion criteria and were assessed for radiation dose. The mean effective dose received by trauma patients was 11.3 mSv; with CT–scan contributing the majority (94%) of the total radiation dose. 42% (50 patients) of the patients received less or equal to 5 mSv from their initial 24 hours assessment in the emergency department while around 26% (31 patients) received between 25 mSv to 30 mSv radiation dose from diagnostic imaging.

Conclusions: Trauma patients presenting to emergency department receive significant effective dose from diagnostic imaging during their first 24 hours assessment. One in three patients received 25 mSv to 30 mSv effective dose, ten times higher than the background radiation of 3 mSv. This is a small but assessable excess cancer risk considering this is only the first 24 hours stay in the emergency department. The benefits of diagnostic radiologic investigations should be weighed with the radiation risk associated. Unnecessary imaging, especially CT scan, should be avoided.
the level given out by diagnostic radiologic studies, is by linear non-threshold (LNT) extrapolation of cancer risk from high-dose data. This model is endorsed by the Biological Effects of Ionising Radiation (BEIR) VII reports of the US National Academy of Sciences (10) and the International Commission on Radiological Protection (ICRP) (11). According to the LNT model, effective dose of more than 100 mSv is linearly proportional to the risk of developing cancer. This has been proven by substantial evidences from epidemiology studies (12). However for doses lower than 100 mSv, there are no direct measures but rather conclusions of the risk and radiation exposure and the risk in this level is extrapolated from the risk at higher doses (see Figure 1). This model implies that there is no safe level of radiation exposure that corresponds to nil increased in risk (12). However LNT model is not without controversy, it has been argued to be under-protective (meaning exposure to low level ionising radiation could increase the risk of developing cancer above the extrapolated risk derived from the model) or over-protective (exposure to low level ionising radiation may confer some health benefits) as suggested by some studies (13–15). Despite these arguments, LNT model is still the most widely accepted model in estimating risk of low level radiation dose until more studies have been done.

Due to the severity of injuries, trauma patients are subjected to multiple diagnostic imaging investigations even during their initial 24 hours of assessment upon presentation to the emergency department. This has raised concerns amongst...
emergency physicians and radiologists that perhaps these patients have been over-radiated which increase their risks of developing cancer associated with ionising radiation. Estimating effective dose from diagnostic imaging would provide emergency physicians with objective data which would facilitate comparison among individuals and groups of patients, and would provide a foundation for further investigation.

While studies to evaluate effective dose from radiologic studies received by trauma patients in the emergency department in other countries have been conducted previously (20–22), there seems to be a distinct lack of a similar study conducted here in Australia. In this context, the objective of this study is to determine the amount of cumulative effective dose from diagnostic imaging received by adult trauma patients presenting to the emergency department during their first 24 hours of care. Additionally, this study also aims to investigate which specific mechanisms of injury subject trauma patients to high cumulative effective radiation dose and which diagnostic imaging modality exposed the patient with the highest effective radiation dose.

**METHODS**

Emergency department records for trauma patients presenting to the Canberra hospital (ACT, Australia) between 1st January and 31st December 2008 were retrospectively reviewed for all diagnostic radiologic investigations performed on adult trauma patients (>18 years old) who arrived directly from the scene of injury within the first 24 hours upon presentation. The types and quantity of diagnostic radiologic investigations (plain radiographs and Computed Tomography (CT) scan) performed to the patients in their first 24 hours of assessment and the mechanism of injury of each patient were also recorded. A typical effective dose for each type of plain radiographs (X-rays) and CT-scans were obtained from references in the literatures (23–26). Table 1 shows the effective dose for each procedure of diagnostic imaging assessed in this study.

Microsoft Excel 2007 for Windows was used for statistical analysis of the data. The cumulative effective dose for each patient was calculated by summing up the effective dose of each procedure performed to that patient. The data was stratified to mechanism of injury and was analysed to identify the most common mechanism of injury for trauma patients presenting to the

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Table 2: Diagnostic imaging conducted on trauma patients in the emergency department in the initial 24 hours of assessment

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<tr>
<th>Number of X-rays</th>
<th>Frequency</th>
<th>%</th>
<th>Number of CT scans</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
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<tr>
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<td>27</td>
<td>22.9</td>
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<td>16.1</td>
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<td>Total</td>
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<tr>
<td>Mean</td>
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<td>Mode</td>
<td>1</td>
<td></td>
<td>Mode</td>
<td>0</td>
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[MVA – Motor Vehicle Accident; Auto-ped – Pedestrian struck by motor vehicles.]
emergency department. Additionally, the average number of diagnostic imaging performed for each mechanism was analysed from the data. The data was also stratified to diagnostic imaging modality and was analysed for the contribution of each modality to the total effective dose received by trauma patients.

Ethics approval was obtained from both the Australian National University (ANU) ethics committee and Australian Capital Territory (ACT) health research ethics committee as part of an ongoing study.

RESULTS
During the one year study period, a total of 118 patients met the study inclusion criteria (adult trauma patient, presenting direct from the scene of injury) and included in the study with age ranging from 19 to 92 years old (mean age 42 years). It was noted that ten patients of this group died in the hospital after their presentation to the emergency department, this gives this group of patients with a mortality rate of 8%. The mechanisms of injury were classified as the followings: “motor vehicle accident” (MVA), “fall”, “laceration”, “auto-ped” (pedestrian struck by motor vehicle) and “others” — which include burn injuries and assaults. The most common mechanism of injury for trauma patients presenting to the emergency department was found to be MVA with 78 cases (66%), followed by fall (17 cases; 15%), laceration (12 cases; 10%), auto-ped (5 cases; 4%) and other mechanisms with 6 cases (5%). This is presented in the pie-chart in Figure 2.

In total, there were 260 X-rays and 199 CT-scans recorded for the whole group. The majority of the trauma patients received one or two X-ray studies performed in the initial 24 hours of their assessment in the emergency department (23% each). On average, the trauma patients received 1.7 CT-scan study in the initial 24 hours assessment of their care, while the majority of the patients (45 patients; 38.1%) had no CT-scan done in this time frame. This is presented in table 2.

Based on the mechanism of injury, “fall” trauma patients had the highest average diagnostic imaging performed for both X-rays and CT-scan: 2.5 and 1.9 studies per person respectively. “MVA” trauma patients also had high number of diagnostic imaging studies conducted with 2.4 X-rays per person and 1.9 CT-scan studies per person on average.

While “laceration” and “others” trauma patients had the least performed diagnostic imaging studies (laceration: 1.0 X-rays and 0.3 CT-scans on average; Others: 0.8 X-rays and 1.6 CT-scan on average). This is shown in table 3.

The total cumulative effective dose received by trauma patients in this study was 1330.93 mSv. Most of the effective dose was from CT-scan studies which contributed 94% to the total effective dose (1259.42 mSv) while plain X-rays contributed a much smaller proportion (74.81 mSv; 6%) (Figure 3).

Out of the total effective dose received from CT-scans (1259.42 mSv), 33.8% (450 mSv) is from CT scan series of the chest, abdominal and pelvis (CT-chest/abdominal/pelvis) and 21.6% (288 mSv) was from CT-scans of the cervical spine (CT-cervical spine). CT-scan of the pelvis (12.4 mSv; 1.0%) contributed the least effective dose to the total effective dose by CT-scan. The contribution of other CT-scan modalities to the total effective dose from CT-scan is given in table 4. On the other hand, X-ray of the pelvis (38.5 mSv, 51.5%) was shown to be the major contributor to the total effective dose of plain X-rays (74.79 mSv). The second highest

Figure 3. Overall radiation dose by radiologic modalities.
contributor to total effective dose of plain X-ray was X-ray of the lumbar spine (10.8 mSv; 14.4%) (table 5). In total, it was apparent that CT-scans of chest/abdominal/pelvis and CT-scan of cervical spine were the major contributors to the total cumulative effective dose to the trauma patients (29% and 20.2% respectively) (table 4 & 5).

Out of 118 trauma patients included in this study, majority (50 patients; 42.4%) received less than or equal to 5 mSv cumulative effective dose from diagnostic imaging during their initial 24 hours of assessment in the emergency department, while 26.3% of the patients (31 patients) received between 25.01 mSv to 30.00 mSv cumulative effective dose. Smaller proportion of the patients received between 5.00 mSv and 25.00 mSv cumulative effective doses (17% for 5.01 – 10.00 mSv; 4.2% for 10.01 – 15.00 mSv; 6% for 15.01 – 20.00 mSv; 2.5% for 20.01 – 25.00 mSv). There were two patients (1.7%) recorded who received greater than 30 mSv cumulative effective dose. This is presented in Figure 4. On average, the cumulative effective dose of this patient group from diagnostic imaging was found to be 11.3 mSv and the median effective dose was 7.8 mSv.

By mechanism of injury, "MVA" trauma patients had the highest median cumulative effective dose (10.77 mSv) followed by “fall” trauma patients (7.99 mSv). Trauma patients with “laceration” as the mechanism of injury had the lowest median cumulative effective dose (0.02 mSv). This is presented in the box plot on Figure 5.

**DISCUSSION**

Trauma patients are subjected to many diagnostic radiology studies during their initial 24 hours of management in the emergency department due to the nature of their injuries. They normally present with multi blunt injuries which required extensive radiologic imaging studies. These studies expose the patients to ionising radiation, which at higher doses have been associated with development of cancer through studies of atomic bombs survivors in Japan in 1945 (2-5). Association of development of cancer with low level ionising radiation is much less evident but using the currently accepted approach to estimate the risk of low level ionising radiation, the Linear Non-Threshold (LNT) model, there is no safe level of radiation exposure. The main objective of this study is to measure the cumulative effective dose received by trauma patients in the emergency department during the initial 24 hours of their assessment. Another aim of this study is to observe which radiologic imaging modalities expose the patients with high effective dose.

Estimating effective dose from diagnostic radiology studies would provide emergency physicians with objective data which would facilitate comparison among individuals and group of patients and would provide a foundation for further investigation. Retrospective review of the emergency medical records in one year period (2008) was conducted, data of emergency trauma patients who met the study inclusion criteria; adult (age > 18 years old) and primary patients direct from the scene of incidents; were included in the study. The mechanism of injury and radiologic
modalities performed to the patients in the first 24 hours in the emergency department were recorded.

This study discovered that the average cumulative effective dose exposed to the emergency trauma patients during their initial 24 hours of assessment was 11.3 mSv and the median effective dose was 7.8 mSv. According to the Linear Non-Threshold (LNT) model (18), the mean cumulative effective dose of 11.3 mSv would give an additional 89 cancer cases per 100,000 individuals exposed or approximately 1 in 1100 persons exposed (18). This is almost four-times higher than the yearly natural radiation of 3 mSv (3). But one must keep in mind, even though LNT model is the currently accepted approach to assess risk in exposure to low level of ionising radiation, there are no direct evidences that show diagnostic radiologic imaging leads to increased risk in development of cancer. And there are studies that show the LNT model itself might be over-protective or under-protective as explained in the introduction (see Figure 1).

However, the finding of this study is significantly different from another study conducted previously by Winslow et al (22). They found that the median cumulative effective dose of emergency trauma patients to be 40.2 mSv, almost five-times higher than the finding of this study (22). Winslow et al and this study are similar in terms of design and sample population; both measured the effective dose of radiation exposed to adult trauma patients during their initial 24 hours of assessment. Both studies were done in level 1 trauma centres and were both looking at similar number of patients (86 patients in Winslow study) (22). However, the Winslow study only included blunt trauma patients and not “laceration” trauma patients which is not the case with this study as it included all trauma patients (22). “Laceration” trauma patients are only 10% of the total sample population in this study and do not have a large impact on the result (see Figure 2). Another factor that might contribute to the difference in value is that the effective dose in the Winslow study was calculated by the CT scanner for each scan while in this study it was obtained from references value in the literatures (Table 1) (22). The effective dose in the Winslow study might be a more accurate representative of effective dose exposed from CT-scan to trauma patients. This difference could be attributed to different protocol utilised by the centres where the studies were done.

This study also found that most trauma patients presenting to emergency department exposed to less than 5 mSv (42.4%) or between 25 to 30 mSv (26.3%) effective doses from radiologic imaging. And by mechanism of injury, motor vehicle accident (MVA) was the main mechanism of injury for trauma patients presenting to emergency department (66%) (see Figure 2). Motor vehicular accident; “MVA” and “auto-ped”; and “fall” patients have higher median cumulative effective dose (10.77 mSv, 7.99 mSv and 5 mSv respectively) than “laceration” and “others” (see Figure 5). Possible explanation for this could be attributed to the severity and the “blunt” nature of the injuries which required more thorough investigations.

There are limitations of this study that must be taken into consideration. Firstly, this was a retrospective study, which indicates there are limitations with missing data and the possibility of errors in data collection. Since this study only

<table>
<thead>
<tr>
<th>CT-scan modalities</th>
<th>Frequency</th>
<th>Effective dose given (mSv)</th>
<th>Proportion to total effective dose of CT-scan</th>
<th>Proportion to total effective dose of diagnostic imaging (CT-scan + X-ray)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT-chest</td>
<td>17</td>
<td>158.1</td>
<td>12.6%</td>
<td>11.9%</td>
</tr>
<tr>
<td>CT-abdominal</td>
<td>19</td>
<td>172.9</td>
<td>13.7%</td>
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</tr>
<tr>
<td>CT-pelvis</td>
<td>2</td>
<td>12.4</td>
<td>1.0%</td>
<td>0.9%</td>
</tr>
<tr>
<td>CT-chest/abdo/pelvis</td>
<td>25</td>
<td>450</td>
<td>35.7%</td>
<td>33.8%</td>
</tr>
<tr>
<td>CT-cervical spine</td>
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<td>288</td>
<td>22.9%</td>
<td>21.6%</td>
</tr>
<tr>
<td>CT-lumbar spine</td>
<td>3</td>
<td>27.3</td>
<td>2.2%</td>
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</tr>
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<td>CT-brain</td>
<td>59</td>
<td>129.8</td>
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<td>16.8</td>
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<td>CT-extremities</td>
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</tr>
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<td>CT-pulmonary angiogram</td>
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<td>Total</td>
<td>192</td>
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in the emergency department within diagnostic radiology imaging in trauma patients are exposed to higher exposure of effective dose. In conclusion, it is apparent that practice patterns at other institutions. Another additional limitation is that the effective dose for each procedure was estimated from other studies and was not measured directly. This might under or over estimate the effective dose of each procedure since there are many variables that can affect radiation exposure, including beam current, which can change between machines and centres. Direct measurement with dosimetre could be done in future studies to directly measure the effective dose. Though there is a study done previously which directly measured effective dose with dosimetre, but it was not specifically looking at the effective dose exposed in the initial 24 hours of assessment in the ED (21). Lastly, this study might underestimate the effective dose exposed to emergency trauma patients since it does not include effective dose received from fluoroscopy which have higher exposure of effective dose.

In conclusion, it is apparent that trauma patients are exposed to significant effective dose from diagnostic radiology imaging in the emergency department within 24 hours upon presentation. In this study, the mortality rate of the sample population was 8% and the mean effective dose from diagnostic radiologic investigations was 11.3 mSv, which is four times higher than the yearly natural radiation of 3 mSv. As any other modes of investigation in medicine, the benefits and risks of diagnostic radiology imaging should be considered by emergency physicians, including interns, before exposing patients to ionising radiation.

REFERENCES

<p>| Table 5: Effective radiation dose from each X-ray modality conducted on trauma patients in the emergency department in the initial 24 hours of assessment |</p>
<table>
<thead>
<tr>
<th>X-ray modalities</th>
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<th>Proportion to total effective dose of diagnostic imaging (CT-scan + X-ray)</th>
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Figure 5: Median cumulative effective radiation dose from diagnostic imaging by mechanism of injury. MVA – Motor Vehicle Accident. Auto-ped = pedestrian struck by motor vehicle.

INTRODUCTION

It has been estimated that the cost associated with osteoporotic fractures on the Australian Health Care system was 1.9 billion dollars during 2001, with someone being admitted with a minimal trauma fracture every 8.1 minutes (1,2). In 2007, the incidence and burden of these fractures increased, with the admittance rate every five to six minutes, with a prediction for every 3.7 minutes in 2021 (1,2). It is now estimated that the total cost of osteoporotic care is 7.4 billion dollars per annum, with approximately 2 million people being affected by the disease within Australia alone (3).

Current guidelines recommend that anyone who suffers a minimal trauma fracture be investigated for osteoporosis (4-7). However, investigation and commencement of therapy for osteoporosis following a minimal trauma fracture is notoriously poor (4,6,8,9). A study by Teede et al found only 13% of patients admitted for a minimal trauma fracture has risk factors identified during 2003-2005 at 16 major hospitals in Australia (10). Other Australian studies found similar rates of investigation (11). Studies overseas indicate that osteoporosis care is similar to that in Australian Hospitals, with American studies finding 6% of women, and less than 1% of men undergo osteoporosis investigation after sustaining a fragility fracture (12). Previous fracture greatly increases the risk for future fractures (8,13,14). Fracture risk is approximately doubled in presence of a prior fracture and a prior vertebral fracture increases the fracture risk four-fold (8,13,14). Early identification of osteoporosis and subsequent treatment has been shown to prevent further bone mineral density loss and reduce both future fracture risk and mortality by up to 90% (1,8,15,16). Furthermore, up to 33% of patients sustaining a hip fracture will die within the following 12 months, and prevention can significantly decrease the burden of the disease (3). With as many as 45% of fracture patients having signs of osteoporosis, early intervention can prove valuable in decreasing the burden on the healthcare system (16). Furthermore, if patients with falling bone density are identified early there may be more chance of intervening before bone density falls below the ‘fracture threshold’ (17). Earlier intervention is better for fracture risk (2).

The accepted first line therapy for osteoporosis involves both

ABSTRACT

Objectives: The objectives of this study were to determine:
(i) The incidence of minimal trauma fractures in patients over 40 years of age presenting to The Canberra Hospital (TCH) Fracture Clinic,
(ii) The incidence of osteoporosis screening and intervention in patients presenting to TCH with a minimal trauma fracture, and
(iii) The incidence of repeat fractures in patients suffering a minimal trauma fracture

Design: Retrospective medical records audit of a random selection of 200 records

Setting: Acute care teaching hospital.

Participants: 200 from the 736 patients presenting to TCH Fracture Clinic between March and August 2008, over the age of 40 years. 109 had minimal trauma, with 76% females and 24% males.

Main Outcome Measure: Evidence of osteoporosis screening and intervention in medical records.

Results: Of the 109 patients who sustained a minimal trauma fracture, 10% were on anti-osteoporotic therapy prior to presentation, which included anti-resorptives, calcium and vitamin D supplements. Nine (8.3%) patients had osteoporosis investigations following admission to TCH, 23 (21.2%) patients suffering a minimal trauma fracture had appropriate anti-osteoporotic therapy initiated (anti-osteoporotic therapy included alendronate, calcium carbonate and cholecalciferol). 17 (17.43%) patients were found to have sustained repeat fractures, however only one patient re-fractured between March 2008 and April 2009.

Conclusions: The likelihood of investigation into and treatment for osteoporosis is low in patients presenting to TCH following a minimal trauma fracture. Implementing a strategy to identify patients with low bone mineral density attending TCH Fracture clinic will ensure that there is adequate intervention to reduce the risk of future fractures.
Medical and non-medical interventions. Systematic reviews have shown that the bisphosphonates alendronate and risendronate reduce the risk of osteoporotic spinal and non-spinal fractures (1). Vertebral fracture risk is approximately reduced by 50% within 12-18 months with both of these drugs (5). Other medical interventions include increasing calcium intake with dietary increases and/or supplements, and addition of a vitamin D supplement (cholecalciferol) (5). Weight bearing exercise can increase bone mineral density, and reduce the risk of falls by improving balance, co-ordination and strength. Both of these reduce the risk of osteoporotic fracture (5).

Previous studies at the Canberra Hospital (TCH) in osteoporosis evaluation and management following hip fracture resulted in an increase from 31.7% to 63.9% of discharged patients being treated for osteoporosis (18). However, as this study only focused on osteoporosis care for inpatients following hip fracture, the incidence of evaluation and management of osteoporosis for ambulatory care patients with other fragility fractures is unknown.

This study investigated the incidence of osteoporosis screening and treatment of patients presenting to TCH with a minimal trauma fracture over 40 years of age, and investigated the incidence of repeat fractures for these patients. A retrospective medical records audit conducted to determine these. This study will provide the basis for a concurrent prospective study at TCH which will identify patients over 40 years with fractures, and provide them with information on osteoporosis, a referral for DEXA (Dual Energy X-ray Absorptiometry) bone density scanning, and a blood test. Readmissions for fracture will be counted over the following five years within this patient group. Together, these studies will aim to determine the incidence of second fractures in patients presenting to TCH, and to prevent second fractures from occurring.

**METHODS**
We obtained the records for all patients attending fracture clinic at TCH over the age of 40 years between March 2008 and August 2008 with 736 patients matching this criteria. A random sample of 200 from the 736 available records was created by using a random number generator in Excel, and choosing the first 200 matching files. Figure 1 demonstrates the process for selecting the medical records for audit.

A number of factors were looked for within the records. The first was evidence of a minimal trauma fracture. We defined minimal trauma fractures as falls from less than one metre, and excluded any trauma involving large forces, such as motor vehicle accidents, falls from ladders and roofs and bicycle accidents (3,4,10). Once these patients had been identified, further analysis of these records was conducted,

**WHAT IS ALREADY KNOWN ON THIS TOPIC**
- Osteoporosis represents a significant burden on Australia’s Health
- The incidence of osteoporotic fracture is predicted to increase
- Management of osteoporosis following a minimal trauma fracture is poor

**WHAT THIS STUDY ADDS**
- The management of osteoporosis following a minimal trauma fracture at TCH is similarly poor as other Australian hospitals
- The incidence of osteoporosis screening and initiation of anti-osteoporotic therapy was 8.3% in an outpatient fracture clinic setting, prior to commencing a new program.
- Implementing a program within TCH Fracture Clinic could be effective in increasing osteoporosis screening and treatment initiation, reducing the risk of future fractures.
including evidence of any advice, investigation and treatment for osteoporosis. This information was analysed in order to determine whether the patient had appropriate osteoporosis management following a minimal trauma fracture.

Finally, evidence of repeat admissions for these patients was recorded, and the reason for admission examined in order to calculate the incidence of second fractures within this group of patients.

To determine the required sample size for this study, an online statistical power calculator was used (19). To evaluate the power of the study, we calculated our sample size as 200, the proportion of people screened for osteoporosis was 4.5% and we wanted a 95% confidence interval. This produced a margin of error of 0.09297 and required sample size of n = 72. Therefore our sample size of 200 is sufficient.

RESULTS
Population Characteristics
We collected data from 200 patients that attended the fracture clinic at TCH between March and August 2008, and were over the age of 40. Of these, 109 had a minimal trauma fracture and 76% were females, and 24% were males.

Among the 61 patients with a minimal trauma fracture requiring admission, 82% were female and 18% were male. The mean age of males presenting with a minimal trauma fracture was younger at 59y±13.2, which was statistically different to the mean age for females which was 66.9y±14.7 (p = 0.01). Figure 2 demonstrates the age and sex distribution of patients sustaining a minimal trauma fracture.

Of the 109 patients sustaining a minimal trauma fracture, 11 (10%) were on anti-osteoporotic medications upon presentation to TCH. Among them were seven (6.4%) patients on bisphosphonates (alendronate and risendronate), six (5.5%) on calcium (calcium carbonate) and seven (6.4%) on vitamin D (cholecalciferol).

Investigations relevant to osteoporosis
Two patients had documented evidence for being advised to seek osteoporosis investigation by their general practitioner. Nine (8.3%) patients had documented evidence of investigations for osteoporosis, with eight having calcium, magnesium and phosphate (CMP) blood levels investigated, and one having their vitamin D levels checked (Figure 3). DEXA scan orders or results were not recorded for any patients. These nine patients who were investigated for osteoporosis were admitted for treatment for their fracture. There were no patients followed up as outpatients for osteoporosis investigations through fracture clinic.

Treatment Initiation
Of the 109 patients, 21.2% experiencing a minimal trauma fracture were started on anti-osteoporotic medications, 11% were started on bisphosphonates (alendronate), 16.5% were started on calcium (calcium carbonate), and 17.4% were started on vitamin D (cholecalciferol). All patients who had been started on anti-osteoporotic therapy had been commenced while inpatients of the hospital. No patients were commenced on anti-osteoporotic therapy as outpatients.

Figure 2: Age and sex distribution of minimal trauma fractures
Repeat Fractures

17 (15.6%) patients had attended TCH for repeat fractures, with 16 of these patients having sustained fractures prior to our data collection (March to August 2008). Of the 109 patients sustaining a minimal trauma fracture, one patient sustained a fracture after their presentation to the CH fracture clinic between March and August 2008. With only one patient re-fracturing in our study period, a 5-year fracture risk of 6.9% applies to our population.

Figure 4 demonstrates the osteoporotic management received by patients presenting to TCH with minimal trauma fractures.

DISCUSSION

This study sought to document the incidence of minimal trauma fractures in persons over 40 years of age presenting to the CH fracture clinic between March and August 2008 and determine the incidence of repeat fractures, and evidence of any osteoporosis screening and intervention.

The management of osteoporosis in patients sustaining a minimal trauma fracture at TCH is inadequate, with the screening rates for osteoporosis very low in patients presenting with a minimal trauma fracture. We found that only 8.3% of patients sustaining a minimal trauma fracture had osteoporosis screening, with all investigations done on inpatients, mostly under the care of an orthogeriatric team. No patients had investigations conducted through the fracture clinic. Investigations for osteoporosis involved biochemical testing only (calcium, magnesium and phosphate), with no patients having a DEXA scan for assessing bone mineral density. Likewise, commencement of anti-osteoporotic treatment was infrequent at 21.2% and only started in patients that were admitted for their minimal trauma fracture. We found that of the patients attending the fracture clinic between March and August 2008, one patient had re-fractured within the study period. With only one patient re-fracturing since August 2008, a 5-year fracture risk of 6.9% applies to our population. Osteoporosis Australia regards a 5-year fracture risk of less than 8% as having a low risk of repeat fractures (20). From this we can conclude that of the patients attending TCH fracture clinic between March and August 2008, they have a low risk of repeat fracture, however the 5-year fracture risk obtained from our study may be inaccurate due to reasons discussed below.

The limitations of this study lie in its reliance on documentation within medical records, which are often very general and incomplete in their detail. Advice or suggestions for patients to see their general practitioner about osteoporosis screening is not routinely documented in the patient notes, and the discharge summary for inpatients is often very brief, and only contained information about what management was completed during the patient’s stay at the hospital. Therefore, this study may have underestimated the osteoporosis care of patients following a minimal trauma fracture, as we did not explore investigations carried out by practitioners outside of the hospital. As we only audited medical records from TCH, the incidence of repeat fractures could be underestimated, as only fractures presenting to TCH were identified. Repeat fractures that occurred may have been treated at other hospitals within the area but not included in our study. TCH treats the majority of fractures in this area, however a small number of GPs will manage fractures, and a small number of patients will be followed up by private orthopaedic surgeons off campus. A small number of people have repeat fractures treated at a local rural hospital also. This study did not attempt to measure compliance with bisphosphonates. Patients may have been commenced on bisphosphonates, however due to the low compliance associated with these drugs patients may have stopped taking their medication, affecting the results of the study (21).

Studies conducted at other major Australian hospitals have identified similar weaknesses in managing osteoporosis in patients sustaining a minimal trauma fracture. However screening for osteoporosis at TCH was conducted less frequently than at similar Australian hospitals, with reported levels of 64% being screened for osteoporosis after sustaining a minimal trauma fracture, compared to only 8.3% having evidence of any screening conducted while under the care of TCH (10). Concordant with findings from studies at both Australian and International hospitals, which found only 7-20% of patients receive anti-osteoporotic therapy following a minimal trauma fracture, 21.2% of patients presenting to the CH Fracture Clinic with a minimal trauma fracture were started on anti-osteoporotic therapy (22). Two Australian studies identified that only 5% and 13% of patients sustaining a minimal trauma fracture were commenced on anti-osteoporotic therapy (3,10). The commencement of anti-osteoporotic therapy in patients with minimal trauma fractures at TCH is being implemented more frequently than other major Australian teaching hospitals (3,10).
All patients screened and commenced on treatment were admitted suggesting that there is a difference in standard of practice between TCH Fracture Clinic, and inpatients, as no outpatients seen in the fracture clinic were investigated or commenced on treatment. Despite current guidelines which recommend osteoporosis investigation for anyone that suffers a minimal trauma fracture, most patients presenting to TCH with a minimal trauma fracture received no osteoporosis screening at all (4). This represents a significant gap in the care of patients sustaining a minimal trauma fracture attending TCH outpatients.

The repeat fracture rate identified in patients presenting to TCH with a minimal trauma fracture was much lower than elsewhere in Australia, at 17.43%. It is well known that osteoporosis greatly increases the risk for fractures, and previous fracture increases future fracture risk at least two-fold (13, 14, 23). Previous studies at major Australian teaching hospitals identified that 40-49% of patients presenting to the hospital with a minimal trauma fracture had sustained a fracture prior to this event (3, 24). This may suggest that osteoporosis care in general practice within the ACT is being conducted appropriately, however studies in primary care settings have suggested that anti-osteoporotic therapy is not being prescribed to patients sustaining minimal trauma fracture (10). More likely is that patients are presenting to other hospitals or practitioners within the region, as patients are referred to TCH Fracture Clinic from many hospitals and general practitioners within the ACT and NSW. As most patients attending TCH for fractures are referred for follow up in the Fracture Clinic, implementing strategies to screen for osteoporosis in patients with minimal trauma fractures attending the clinic will ensure that there is effective coverage to identify those with low bone mineral density and implement anti-osteoporotic therapy to reduce the risk of further fractures in these patients.

This study provides background data for a prospective study conducted at the CH Fracture Clinic. This prospective study will identify patients presenting with minimal trauma fractures, and implement an osteoporosis investigation and treatment program. It will then follow up these patients at three months, twelve months, and five years to determine if the fracture rates were reduced following anti-osteoporotic therapy.

CONCLUSION
Osteoporosis care in at risk patients is poor, with very low rates of screening and treatment being implemented. However, osteoporosis management for inpatients at TCH is better than that for outpatients. A program to effectively investigate and initiate therapy could significantly reduce the future fracture risk in patients sustaining a minimal trauma fracture and consequent burden of health for these patients.

REFERENCES

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INTRODUCTION

Type 1 diabetes mellitus is an autoimmune disease with destruction of pancreatic islet beta cells and a subsequent loss of insulin production with patients prone to ketoacidosis (1). Type 1 diabetes accounts for over 90% of childhood and adolescent diabetes in Australia. Incidence of type 1 diabetes in Australian children and adolescents is 24 cases per 100,000 population per year and there were over 8000 new cases of type 1 diabetes in Australia between 2000 and 2008 (2). Staines et al. (3) found a bimodal distribution of age of onset, with a small peak at age 5 – 6 years and a larger peak at age 10 – 12 years.

Children diagnosed with type 1 diabetes are managed by a multi-disciplinary team which includes the patient and his/her family, a paediatric endocrinologist, a diabetes educator, a dietitian and a psychologist or social worker (4). Suboptimal management of childhood diabetes leads to poor control and may impair growth, delay puberty and accelerate long-term diabetic microvascular and macrovascular complications (5).

With medical advances, patients with ‘paediatric diseases’ such as type 1 diabetes mellitus are surviving for longer and reaching adulthood. The options for these patients include transition to adult services, long term retention under paediatric care, or discharge from hospital supervision (6). The concept of ‘transition’ from paediatric to adult management has increased in importance as a means of improving the outcome for paediatric patients with chronic disease. Transition has been defined as the ‘planned, purposeful movement of the adolescent or young adult with a chronic disease from a child (and family)-centred to an adult-orientated health care system’ (7-8).

The transition age and success of transition are important factors in the management of diabetes patients, which have been shown to result in fewer hospital admissions and fewer days in hospital, higher self-care levels, lower HbA1c levels and delayed development of complications (9,10). Failed transitions result in a lack of continuity of care and reliance on crisis treatment rather than management and crisis prevention (11).

Abstract

Objective: To determine the age of transition of type 1 diabetes patients from the paediatric to adult service at The Canberra Hospital and to compare the clinical attendance rates between adult and paediatric services.

Research Design and Methods: Using the paediatric diabetes database, 176 patients born before 1993 were analysed and their last three paediatric consultations and first three adult consultations recorded. Patients were designated as having been successfully transitioned, unsuccessfully transitioned (stopped), not transitioned but still in paediatric care, and dropped out from paediatric care. The age of transition and paediatric and adult clinic attendance rates were measured.

Results: 123 patients were successfully transitioned, 20 patients stopped, seven patients dropped out and 26 are still in paediatric care. The mean age for transition was 16.9 ± 2.3 years (range 10.7 – 25.4 years). The mean age of the ‘not yet transitioned’ patients was 18.1 ± 1.1. The mean age of the last paediatric visit for patients who dropped out was 15.9 ± 3.6 years. The clinical attendance rates were visits every 6.2 ± 3.7 months and 5.1 ± 4.2 months for paediatrics and adults respectively. The difference was statistically significant (P < 0.05).

Conclusions: The Canberra Hospital’s transition program is consistent with the majority of the Australasian Paediatric Endocrine Group’s clinical practice guidelines, works to address barriers to transition through advanced planning of transition and provision of the adolescent clinic and transitions patients at an age consistent with the literature.
The recommended principles of successful transition include:

- Flexible services to suit the needs of the young person;
- No set age for transition, with the patient’s developmental readiness, health status, completion of puberty and school level being considered;
- Transition occurring only after the young person is able to manage his or her illness sufficiently independently – preparation should be provided by paediatric services;
- Services appropriate for both chronological age and developmental attainment;
- Coordinated transition, including an outline of the process, a visit to the adult clinic, and one or more joint paediatric-adult clinic visits;
- Participation of all parties involved i.e. the paediatric and adult endocrinologist, the patient, the patient’s family and all supporting staff; and
- Management of common concerns of young people, including development, sexuality, mood, smoking, drug and alcohol use and sexual activity (6-7,12-13).

This clinical audit was designed to determine the age of transition of paediatric type 1 diabetes patients from the paediatric clinic to the adolescent/adult clinic at The Canberra Hospital (TCH). Rates of attendance before and after transition were also sought and compared. The purpose of this audit was to ascertain whether patients are being transitioned appropriately.

METHODS
A list of 367 patient numbers recorded on TCH paediatric diabetes database was provided. The patient list was refined so as to only include patients born before 1993 with type 1 diabetes. Patients with type 2 diabetes, or diabetes attributable to other causes, for example, cystic fibrosis, were removed. Patients who were born after 1993 were removed as that would make them less than seventeen years at the time of analysis and would not likely show a transition age. Patients who were seen by adult endocrinologists from diagnosis were not included.

Patients were analysed on the basis of specialist letters – only letters signed by an endocrinologist were counted as a consultation. Letters signed by diabetic educators, nurses or dieticians were not included as a consultation. The last three paediatric visits and first three adult visits were recorded.

The date of transition was defined as the first consultation by an adult endocrinologist. A successful transition was defined as three consecutive consultations with an adult endocrinologist since in this study only three adult visit dates were recorded. Clinical attendance rates were determined by averaging the time between visits and defined as the ‘paediatric’ and ‘adult’ visiting rate. The paediatric and adult clinical attendance rates were compared using a Student paired T-Test.

RESULTS
Of the 367 numbers provided, 176 patients were used for the study. 191 patients were not used for reasons such as insufficient data, not having type 1 diabetes, moving

![Figure 1: Histogram showing the age of transition of type 1 diabetes patients from paediatric to adult services at TCH.](image)
Of the 176 patients, 143 patients (81.25%) had a transition date as defined above. The mean age for transition was 16.9 ± 2.3 years (range 10.7 – 25.4 years). Figure 1 shows the distribution of ages of transition.

There were 20 (11.4%) patients who were considered to have transition ages as per the above definition but who could not be considered as having successfully transitioned. These patients had less than three visits with the adult endocrinologists with no information of ongoing care with private or interstate/overseas physicians. These patients were classified as ‘stopped’.

For the patients who were not transitioned, 26 (14.75%) had not been transitioned to an adult endocrinologist and had seen a paediatrician within the last 12 months. These patients were categorised as ‘not yet transitioned’. The mean age of the ‘not yet transitioned’ patients was 18.1 ± 1.1 years. Of these patients, 14 were over 18. Figure 2 shows the distribution in age of these patients who have not yet been transitioned.

The remaining seven patients (4%) who were not transitioned have been classified as ‘dropped out’ as they had not seen a paediatrician in over 12 months and there were no letters of referral to interstate or private practice to indicate their progression. The mean age of the last paediatric visit was 15.9 ± 3.6 years, with a range of 8.6 – 19.1 years. Removing the patient aged 8.6 at their last paediatric visit, the remaining six patients were aged from 17.2 ± 1.6 with a range of 14.6 – 19.1 years.

109 patients were used for the clinical attendance rate calculation, as only patients who had recordable paediatric and adult visits were used so as to keep the statistical population the same. For the paediatric group, the range of clinical attendance was 1.5 – 19.8 monthly. The mean rate between consultations was 6.2 ± 3.7 months. The distribution of paediatric attendance rates can be seen in Figure 3. For the adult rate, the range of clinical attendance was 0.6 to 27.0 months between visits. The mean rate was 5.1 ± 4.2 months between visits. The distribution of adult attendance rates can be seen in Figure 4. Using a paired Student t-test to compare the means, a p value of 0.04 was calculated at the 95% confidence interval. Therefore the visit rates are statistically different between the adult and paediatric population, with the rate lower for the adult visits than for the paediatric visits.

DISCUSSION

In this study, 123 patients were successfully transitioned, 26 had not yet transitioned and were still in paediatric care, 20 had unsuccessful transitions and seven patients dropped out from paediatric care and were never transitioned.

In a literature review of transition age, it was found that the mean age of transition ranges from 15.9 to 19.1 (14-20). Table 1 summarises the ages of transition from different studies. The mean transition age of 16.9 ± 2.3 at TCH falls within those values, suggesting that the age of transition at TCH is comparable to other centres. The 26 patients (14%) not yet transferred ranged from 16.7 to 21.7. Of these, 14 (8%) were over the age of 18. This is a higher rate than the 3.9% non-transitioned patients over 18 in the Kipps et al. study (18).

For this study, a number of patients were referred to the adolescent clinic at an early age due to two paediatricians...
leaving and retiring. These changes happened around the years 2003-2004 and meant that some patients as young as 13 and 14 were referred to the adolescent/adult diabetes services for continuation of their care. The presence of these patients in the data set may skew the data to the left and underestimate the true value.

It was expected that the paediatric clinical attendance rate would be lower than the adult rate, due to the large demand of adult diabetic patients (both type 1 and 2) in the adult clinic and the increased waiting times for appointments. However, from the data it was found that the time between consultations was statistically lower in the adult patients than the paediatric patients. This can be accounted for in a number of ways. A number of patients were referred to the adult services due to life events such as pregnancy, or mental health issues. These patients are not expected to have impacted on the age of transition; however their conditions lead to an increased number of visits to the adult clinic which may impact upon the visitation rates. Also, patients referred from interstate or those with poor compliance were started off in the adult clinic with increased attendance rates when transferred. Therefore the adult visitation rate is likely to be inaccurate with the mean skewed so that the rate appears less than the true value.

There are significant differences between the nature of diabetes care from the paediatric to the adult services. Paediatric services tend to be family focused and socially oriented while adult services are more individual and disease focused (11). Adult care units are more apt at treating diabetes complications and are reported to be less adept at dealing with patients’ global problems (14).

For young patients, the transition to different health services can be a major life event as they are forced to leave respected carers and are thrust upon new, unknown ones (21). There are many barriers in the way of successful transition. For some patients, they feel ‘why fix what is not broken’ and cannot understand the need to move from a service that has served them well for many years. For other patients, moving to adult services is seen as a step towards disease complication and even death. Furthermore, life events such as graduation, moving away from home, pursuing new educational goals and beginning work all provide distraction from chronic illness management such as diabetes (22). Family may also impact on transition, as the movement to the individual-focused adult clinic can cause anguish and upset for the patient’s family who are suddenly pushed out of the process and are not included in decision making or planning. This can lead families to sabotage the transition procedure (11,23).

The establishment of a ‘joint clinic’ staffers by both paediatric and adult physicians and run as an intermediate between paediatric and adult care can be used to reduce the barriers to transition (18). Joint clinics provide a medium for the family to meet and form rapport with the future adult physician as well as promote independence and reduce the anxiety felt from a rushed transition (11).

The International society for paediatric and adolescent diabetes (ISPAD) Clinical Practice consensus guidelines 2006-2007 advise that transition should include both paediatric and adult teams and be an organised process (24). Furthermore they advise that the age for transition varies according to maturity and service availability.

The Australasian Paediatric Endocrine Group (APEG) clinical practice guidelines advise that transition should occur at a time of relative stability in the adolescent’s health, and usually at a time such as when the adolescent finishes school or enters the workforce (25). However, the admission and patient policies of the individual hospitals, the wishes of the adolescent as well as his or her emotional and physical maturity and the presence of any co-morbidities must be taken into account. The ideal transfer to an adult service should involve:

- A ‘preparation phase’ (from 12 years) which should be planned and organised by a transition service as well as openly discussed with written information for patient and their family over several years leading up to the transition.
- A ‘formal transition phase’ (16-18 years) in which the patient is directed to an adult diabetes specialist or a clinic with a special interest in young adults with type 1 diabetes. This should be facilitated by a visit to the adult service or by the adult physician attending the adolescent clinic and should be formally arranged with appropriate letters or referral and medical history information.
- An ‘evaluation phase’ (18-19 years) to confirm that transition has taken place and that continuing care has been arranged.

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<td>Orr, Fineberg and Gray (1996)</td>
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<td>Pacaud et al. (2005)</td>
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<td>Holmes-Walker, Llewellyn and Farrell (2007)</td>
<td>18.9 ± 2.53</td>
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TCH provides in- and out-patient care for paediatric, adolescent and adult diabetes by a small paediatric and adult team. The transition of patients is not controlled by any set guidelines or procedures, but is done in a way to follow APEG guidelines as best as possible. As per the APEG clinical practice guidelines, TCH ensures that transition is planned well in advance and is discussed with the patient and his/her family. There is no specific transition service or written guidelines, instead transition is at the paediatrician's discretion at a time when the patient is deemed physically and mentally mature and is done through direct referral to adult endocrinologists. The formal transition phase is arranged with appropriate referral letters to adult endocrinologists with interest in adolescent health and the patient is able to meet adult endocrinologists in an adolescent clinic. The adolescent clinic is designed as a 'diabetes one stop shop', being run by adult endocrinologists and paediatric diabetes educators and dieticians between 4-6pm so as to fit in easily after school and provide an opportunity for patients to meet adult specialists before transition. The formal transition procedure is also run in a two step manner – with the transition to the adult endocrinologist happening first and then the transition to an adult educator happening later. This provides a level of familiarity and continuity to the patient. There is currently no formal evaluation of transition at TCH.

CONCLUSION
Transition from paediatric to adult care for type 1 diabetes patients is a significant time in the patient’s care and an important indicator for their future health. Of 176 patients born before 1993, 123 were successfully transitioned, 26 had not been transitioned yet and were still in paediatric care, 20 had unsuccessful transitions and seven patients dropped out from paediatric care and were never transitioned. It was found that TCH's paediatric and adult services were able to follow most of Australasian Paediatric Endocrine Group's clinical practice guidelines and transition patients at an age comparable to the literature by a small team with no formal procedures. Furthermore, TCH works to address barriers to transition through advanced planning of transition and provision of an adolescent clinic and two tiered transition program.

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7. Rosen DS, Blum RW, Britto M, Sawyer SM, Siegal DM. Transition to


ABSTRACT

Objective: To compare the clinician gender performing Pap smears in general practice and determine whether there is a difference in rural versus urban areas.

Design and Setting: This is a retrospective cross sectional study as part of a Clinical Audit Project (CAP) undertaken by the Australian National University (ANU) Medical School. Data is collected on patient presentations while students are on clinical placements which may be in general practice, community health or hospital settings, in both rural and urban locations. Patients give written consent to the collection and use of the data.

Main Outcome Measure: This paper examined females presenting for Pap smears, and clinician gender performing each Pap smear. Odds ratios of female clinicians performing Pap smears in rural and urban general practice were calculated.

Patients, Participants: The 2006 - 2010 CAP data set contained information on 7786 patients.

Results: Overall, female GPs have higher odds of performing a Pap smear than male GPs, across rural, urban and combined samples (odds ratio 4.4, 3.3 and 3.9 respectively). This finding is statistically significant at the 5% level in each case. Female GPs in rural areas appear to have higher odds of performing a Pap smear than their female counterparts in urban areas, but this result is not statistically significant at the 5% level.

Conclusions: While there was no statistically significant difference between urban and rural locations in the proportion of Pap smears performed by female clinicians, this study found that female clinicians consistently perform more Pap smears than male clinicians in both locations.

INTRODUCTION

Over 3.5 million women participated in the National Cervical Screening Program in Australia in 2006-07, which is equivalent to 61.5% of the eligible population (1). In general practice, 1 in 50 encounters will be for the purpose of a female genital check-up/Papnicolau (Pap) smear (2). Pap smears are a useful screening tool for cervical cancer and it is recommended that all sexually active women between the ages of 20-69 be screened every two years. It is therefore important that women feel comfortable with their general practitioners who perform the majority of these examinations. Clinician gender is a major factor in creating a comfortable setting that encourages women to undertake regular screening.

Generally, female patients have a preference for a female doctor, especially when consulting about gynaecological problems (3-6). This preference is even more pronounced with young and nulliparous women. Women are in a vulnerable position during gynaecological examination and the experience can trigger negative feelings such as pain, embarrassment and awkwardness (4). It is thought that less embarrassment and anxiety is felt when discussing these problems with a woman (3). It is worth noting that male patients also prefer a clinician of the same gender for genital examinations (6,7).

This preference is affected by the availability of a female doctor which is particularly relevant in rural areas. There are nearly twice as many male GPs as female GPs working in Australia and this differential is even greater in rural areas (2). The participation rates for cervical screening in Australia for major cities, regional areas and remote areas respectively in 2007-08 were 61.4, 61.1 and 56.1 percent of eligible women (8). While we recognise that the lower level of participation outside major cities is multifactorial, it is likely that clinician gender plays a significant role.

This study aims to examine the clinician gender performing Pap smears in general practice and to determine whether there is a difference in rural versus urban areas. We hypothesised that Pap smears are more likely to be performed by female clinicians but that this trend may be...
affected by the predominance of male clinicians in rural areas.

METHODS
Data on patient encounters in clinical settings have been collected by successive third year medical student cohorts at the Australian National University since 2006. This is part of the Clinical Audit Project, which until 2009 was known as the Health Information Project. While on clinical placements, students were required to collect data on 30 consecutive patients, until 2008 when the requirement changed to a minimum of 15 consecutive patients. Clinical placements may be in general practice, community health, or hospital settings, in both rural and urban locations. Information recorded during the patient encounter includes patient demographics, up to three symptoms or reasons for the encounter, up to three diagnoses at the end of the encounter, and up to three pre-existing diagnoses. Also included are a list of previous medications used by the patient and a list of procedures, investigations or medications initiated as a result of the encounter. The gender of the student and, as of 2007, the clinician is also recorded.

The collection and use of the data associated with CAP has been reviewed and approved by the Australian National University Human Research Ethics Committee, Australian Capital Territory Health Ethics Committee and New South Wales Greater Southern Area Health Ethics Committee. Written consent is required from all patients.

RESULTS
Figure 1 shows that 68 out of a total of 5,624 patient encounters involved a Pap smear and the majority of these (88%) were in general practice (Table 1). Female general practitioners (GPs) performed 65% of pap smears, despite conducting only 33% of all consultations (Table 2). This trend was consistent across rural and urban settings (Tables 3 and 4). The 2006-2010 CAP data set contained information on 7,786 patients. Fifty-one of these patients were excluded for a variety of reasons, including patient non-consent and inability to understand English.

For this project, Microsoft Excel software was used to clean and recode the data set. Patient data from 2006 was excluded, as clinician gender was not recorded that year. To extract all clinical encounters involving a Pap smear, ICPC-2 codes and descriptors, as well as free text descriptors, were used to search the data. Examination of unique patient identifiers ensured no duplication of patients. This data set was then refined with the following exclusions; encounters were excluded if they had a symptom or diagnosis indicating a Pap smear, but no corresponding Pap smear or cervical cytology included in the procedures/investigations section, as in such cases it was not certain whether a Pap smear was done. Patients were also excluded when their encounter related to follow-up for previously abnormal or unsatisfactory Pap smear results, and there was no indication of a repeat Pap smear being performed. With such exclusions, our final data set included 68 patients. The Epi Info™ Version 3.5.1 software package was used for statistical analysis of the results from general practice settings only. Statistical significance was determined by calculating odds ratios and using the chi-squared test.

DISCUSSION
Consistent with other studies (3-6) our results show a strong tendency for female GPs to perform more Pap smears than male GPs, whether in urban or rural locations. In addition, this study found no significant difference in the proportion of male practitioners performing Pap smears in urban versus rural centres, despite male GPs comprising a greater

<table>
<thead>
<tr>
<th>Consultation Location</th>
<th>Total Patient Presentations</th>
<th>Pap smears (% of total)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GP</td>
<td>2299</td>
<td>60 (2.6)</td>
</tr>
<tr>
<td>ED</td>
<td>553</td>
<td>1 (0.9)</td>
</tr>
<tr>
<td>Community Health</td>
<td>101</td>
<td>7 (6.9)</td>
</tr>
<tr>
<td>Other</td>
<td>2,671</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Total</td>
<td>5,624</td>
<td>68 (1.2)</td>
</tr>
</tbody>
</table>
Given lower participation rates of females living in rural and remote areas in the National Cervical Screening Program (1) the finding that male GPs in rural areas perform no more Pap smears than urban GPs suggests that a proportion of women, if unable to pursue their preference for a female practitioner to perform a Pap smear, do not then automatically choose a male practitioner. Rather, they may choose not to have a Pap smear performed at all. This has significant implications for the overall efficacy of the cervical screening program in rural areas. Further, this study found a trend towards rural female GPs performing a greater proportion of Pap smears than urban female GPs, which may be a reflection on the posited unmet demand for screening services to be performed by female practitioners in rural areas. While the finding was not statistically significant in the present study, and would require more data to confirm or refute, labour force statistics tend to support the hypothesis.

By way of illustration, in 2007 there were 95 full time equivalent primary care clinicians per 100,000 head of population (39.1% female) in major cities versus 84 full time equivalent primary care clinicians in outer regional areas (33.2% female) (9). Given the strong preference of patients for female practitioners to perform Pap smears, the greater proportion of male GPs in rural centres further exacerbates the overall problem of realistic access to cervical screening in rural areas. However, this may be offset slightly by a greater number of nurses, the majority of whom are female, in outer regional areas compared to major cities (1,191 vs 1,086 per 100,000) (10).

A potential complicating factor in this analysis relates to demographic variation in rural versus urban settings, given that women over the age of 25 are less distressed by the idea of male practitioners performing Pap smears than those in the under 25 age group (4), and there are a higher proportion of women in the older age group living in rural centres relative to women aged 20-24 (Figure 2).

While this factor could impact on screening participation in rural versus urban areas, it is likely that the absolute medical workforce shortages in outer regional areas is a more significant factor overall given the participation rates recorded by the Australian Institute of Health and Welfare (9).

There are several limitations to our study. Firstly, the relatively small number of Pap smear procedures recorded thus far in the CAP data set. Although there is sufficient power in our results to confirm that female GPs perform more Pap smears than their male counterparts, the second trend we observed of rural female GPs performing more Pap smears than their urban colleagues, the second trend we observed of rural male GPs do not perform more Pap smears than their urban colleagues, the second trend we observed of rural female GPs performing more Pap smears than their urban female counterparts did not reach significance. If this apparent trend had reached significance it would have confirmed the second part of our hypothesis, that the preponderance of male GPs in rural areas affects the gender of clinicians most often performing Pap smears. As more encounters are added to the CAP data set, it may be possible to determine whether this rural/urban difference is a genuine one, and revisit the second part of our hypothesis.

Secondly, a potential sampling bias exists due to the sensitive nature of a Pap smear consultation and the way in which consent is gained from patients for student participation. Anecdotal reports from students indicate women are often asked by practitioners, receptionists or practice nurses in the student’s absence, and would require more data to confirm or refute, labour force statistics tend to support the hypothesis.

Table 2. Frequency of Pap smear presentations in General Practice, and whether these are performed by female or male clinicians.

<table>
<thead>
<tr>
<th>General Practice Nature of Consultation</th>
<th>Clinician Gender</th>
<th>Female (%)</th>
<th>Male (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pap smear consult</td>
<td>Female</td>
<td>39 (5.1)</td>
<td>21 (1.4)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non pap smear consult</td>
<td>Female</td>
<td>721 (94.1)</td>
<td>1518 (98.6)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>Female</td>
<td>760 (100.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>1,539 (100.0)</td>
<td></td>
</tr>
</tbody>
</table>

*One general practice encounter included in this total does not appear in either the rural or urban stratifications in Tables 3 and 4, as its location was unspecified.

Table 3. Frequency of Pap smear presentations in Rural General Practice as performed by female and male clinicians.

<table>
<thead>
<tr>
<th>Rural General Practice Nature of Consultation</th>
<th>Clinician Gender</th>
<th>Female (%)</th>
<th>Male (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pap smear consult</td>
<td>Female</td>
<td>23 (5.5)</td>
<td>11 (1.3)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non pap smear consult</td>
<td>Female</td>
<td>399 (94.5)</td>
<td>848 (98.7)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>Female</td>
<td>422 (100.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>859 (100.0)</td>
<td></td>
</tr>
</tbody>
</table>

Table 4. Frequency of Pap smear presentations as in Urban General Practice, as performed by female and male clinicians.

<table>
<thead>
<tr>
<th>Urban General Practice Nature of Consultation</th>
<th>Clinician Gender</th>
<th>Female (%)</th>
<th>Male (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pap smear consult</td>
<td>Female</td>
<td>16 (4.7)</td>
<td>10 (1.5)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non pap smear consult</td>
<td>Female</td>
<td>322 (95.3)</td>
<td>669 (98.5)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>Female</td>
<td>338 (100.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>679 (100.0)</td>
<td></td>
</tr>
</tbody>
</table>
and the student informed only when a woman is willing for them to be present. Therefore it is possible a significant number of Pap smear encounters where the student's presence is refused by the patient may not be recorded at all in the CAP data set. This could potentially influence our results if clinician gender affects a woman's likelihood of assenting to a student's presence (for example if women are more comfortable to have students present with a female clinician, or vice versa).

A third limitation to this study is that the CAP data set reflects differences between Canberra and rural south-eastern New South Wales, where ANU students conduct their clinical placements. These differences may not be representative of urban versus rural differences Australia wide.

Our results may partly explain lower participation rates in the National Cervical Screening Program in rural and remote areas. Initiatives to increase the availability of female practitioner Pap smear services in rural areas may therefore reduce the city-country gap in cervical screening participation rates. Such initiatives might include addressing barriers to female GPs relocating to rural areas, increased provision of Pap smear services by female practice nurses in practices without female GPs, or increased provision of (female) nurse-based community health Pap smear programs in rural areas.

The hypothesis that Pap smears are more likely to be performed by female practitioners was borne out by the results of this study. The hypothesis that this trend would be affected by the greater proportion of male doctors in rural areas was not supported by our results but neither was this study of sufficient size to rule out an effect, nor was it able to determine if this factor impacted on other trends such as the overall lower participation rate in cervical screening in rural areas. Should the trend we observed, that female GPs perform a greater number of Pap smears in rural areas, be supported by further data collection this hypothesis may still prove correct.

REFERENCES


Table 5. Summary of statistical analysis showing odds of female GPs performing Pap smears compared to male GPs, across rural, urban and combined general practice settings. OR = odds ratio, CI = 95% Confidence interval.

<table>
<thead>
<tr>
<th>Consultation Location</th>
<th>Statistical Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Practice (Rural + Urban)</td>
<td>OR: 3.9 (CI: 2.3, 6.7) x² test: p &lt; 0.05</td>
</tr>
<tr>
<td>Rural General Practice</td>
<td>OR: 4.4 (CI: 2.1, 9.2) x² test: p &lt; 0.05</td>
</tr>
<tr>
<td>Urban General Practice</td>
<td>OR: 3.3 (CI: 1.5, 7.4) x² test: p &lt; 0.05</td>
</tr>
</tbody>
</table>

Figure 2: ABS 2001 Census of Population and Housing (Reproduced from (11))
A search for the high risk patient: A retrospective study of carotid endarterectomy

Priyanka Dhillon*, David Hardman**
*Medical Student, The Australian National University
**Vascular Surgeon, The Canberra Hospital; Professor, The Australian National University

INTRODUCTION

Since Dr Felix Eastcott performed the first carotid endarterectomy at St Thomas’ Hospital London in 1954, carotid endarterectomy (CEA) has remained the ‘gold standard’ in the treatment of carotid artery stenosis. Despite the durable track record, the role of CEA has been continually questioned. The 1990s saw the role of carotid endarterectomy clarified by a series of randomised trials. This last decade has seen improvements in medical management and the plethora of new technologies rechallenge the role of CEA. Although several trials including the North American Symptomatic Carotid Endarterectomy Trial (NASCEAT), European Carotid Surgery Trial (ECST), Endarterectomy versus Angioplasty in Patients with Symptomatic Severe Carotid Stenosis trial (EVA-3S), Stent-Protected Angioplasty versus Carotid Endarterectomy Collaborators trial (SPACE) and Asymptomatic Carotid Atherosclerosis Study (ACAS), have demonstrated the value and role of CEA, there remains continued confusion over the supposed role of carotid artery stents (CAS) (1-6). In an attempt to develop a market for new technology, a subset of patients was identified as being better treated with CAS. This identification was based on the applicability of CEA to all patients considered as a homogenous cohort. A subgroup of ‘high risk’ patients were thus identified as having at least one risk factor for CEA.

Proponents of CAS suggest, mostly on the basis of exclusion criteria of older trials (NASCEAT and ACAS), that a group of specific ‘high-risk’ patients may benefit

ABSTRACT

Background: Despite the numerous studies comparing carotid endarterectomy (CEA) and carotid artery stents (CAS), the question of superiority remains ambiguous largely due to the concept of the ‘high-risk’ patient. Industry sponsored trials have suggested that CAS is a more suitable treatment for carotid artery stenosis in patients who have certain medical and anatomical risk factors for CEA. This study attempted to identify a subpopulation of patients who underwent CEA and responded poorly then perhaps the concept of the ‘high-risk’ patient for CEA is warranted and should not be discredited as an agenda driven attempt by industry to create its own niche market and supply the needs of that market.

Methods: 50 patients were examined in a retrospective study to determine the perioperative, 3-6 months and 12 month clinical outcomes of CEA. The primary endpoint being studied was non-fatal stroke (ipsilateral) or death. Secondary outcomes being analysed included cardiovascular complication, cranial nerve damage, neurovascular complication (other than stroke) and restenosis requiring reintervention. Information was collected from medical records at The Canberra Hospital (TCH) and follow-up information from private rooms of surgeons involved. ‘High-risk’ patients were identified as having at least one risk factor for CEA.

Results: Of the 50 patients, 47 were ‘high-risk’. One ‘high-risk’ patient had a fatal myocardial infarction perioperatively and one ‘high-risk’ patient had an ipsilateral stroke requiring reintervention at 3-6 month follow-up. The most frequently occurring secondary endpoint perioperatively was labile blood pressure, however, there was no association between high risk patients and poor blood pressure control perioperatively. There was also no association between number of risk factors and labile blood pressure perioperatively (non-significant).

Conclusions: The results of CEA performed at The Canberra Hospital do not reveal a population of patients who have increased morbidity or mortality after CEA, despite the fact they were categorised by definition as ‘high-risk’.
from CAS in comparison to CEA (1, 7). Such patients primarily included octogenarians, patients with contralateral occlusion or stenosis, medical co-morbidities (pulmonary dysfunction, angina, diabetes and hypertension) and anatomically hostile lesions (7-9). Table 1 summarises the patient categories considered to identify the candidates for CAS as the preferred alternative to CEA on the basis of perceived operative risk.

The identification of the ‘high-risk’ patient is necessary not only in establishing the role of CAS but also identifying patients who may not be receiving optimum treatment under current practise guidelines (10). However, it is prudent, to acknowledge that the myriad of studies and ‘high-risk’ registries which aimed to reveal the superiority of CAS, receive significant industry funding (11). Some intellectual caution should be exercised in attempting to determine a patient profile for the ‘high-risk’ patient. If such a niche group did not exist the role of CAS would be largely redundant as CEA has been found to be effective not only procedurally (perioperative outcomes and rate of restenosis) but also preventing recurrent disease (reduced incidence of ipsilateral stroke) (1-6). Underlying this is the reality that CAS is a significantly more expensive procedure than CEA which in turn raises the economic and moral issues of widespread applicability of CAS in a public health setting facing financial constraints (12, 13).

The aim of this study was to identify the cohort of patients in whom the use of CEA did not produce clinically desirable outcomes as a result of concomitant medical risk factors and anatomical risk factors.

METHODS
This small sized single-centre retrospective study was conducted at The Canberra Hospital (TCH), a University teaching hospital. 50 consecutive patients who had undergone CEA during the period 2005 -2007 were included in the trial. Both symptomatic and asymptomatic patients were included. No patient was excluded from the study due to decreased life expectancy, comorbid conditions, or severe neurological deficits after stroke that may have precluded the benefit of CEA. Permission to access medical records was granted by the General Manager of the TCH.

A definition of ‘high-risk’ patient was identified from trials and current literature (1, 7-9). For the purposes of this study a ‘high-risk’ patient included patients who had at least one of the following criteria: ≥ 80 yrs of age; contralateral carotid stenosis; medical co-morbidities or anatomically hostile neck lesion.

The specific medical co-morbidities assessed in this study included: pulmonary disease, renal disease, diabetes, hypertension and cardiovascular disease (CVD) (previous myocardial infarction (MI), angina, coronary artery bypass surgery (CABG)). Pulmonary disease included patients with chronic obstructive pulmonary disease (COPD) or chronic obstructive Airways disease (COAD). Renal disease was broadly applied to encompass mild, moderate and severe renal insufficiency, including dialysis dependent patients. Anatomically hostile neck lesions included previous tracheostomy, radiation therapy and location of lesion.

The degree and location of stenosis was obtained from Doppler studies undertaken on all patients. The primary endpoint being assessed was the incidence of neurovascular event (ipsilateral stroke or transient ischemic attack (TIA)) or death periperooperatively (up to 30 days post procedure). Secondary endpoints assessed included cranial nerve injury/damage, neurological complications (other than ipsilateral stroke), cardiovascular complication (particularly non-fatal MI and labile blood pressure (BP)) and restenosis. All outcomes were assessed at 3-6 months and 12 month follow-up.

Data was analysed using Gen Stat software. The chi-squared test was used to compare perioperative labile blood pressure in ‘high-risk’ patients and perioperative labile blood pressure and number of risk factors per ‘high-risk’ patient. It was also used to compare contralateral stenosis and perioperative labile blood pressure. P < .05 was considered statistically significant for all analyses. If patients underwent more than one CEA, only the outcomes of the first CEA were included in this study. Logistic regression models for risk factors were not analysed due to low event rates.

RESULTS
Of the 50 patients examined, 47 patients had at least one risk factor, categorising them as a ‘high risk’ patient. The majority of patients (>50%) had three risk factors or more for CEA (Figure 1). The most frequent risk factor was hypertension (84%) followed by cardiovascular disease (52%) (Table 1).

The mean age of patients involved was 74 ± 8 years of age in males and 72 ± 10.1 years in females, with 26 % of pa-

<table>
<thead>
<tr>
<th>Table 1. High risk patient for CEA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anatomic high-risk for CEA</strong></td>
</tr>
<tr>
<td>Radical neck surgery (for cancer)</td>
</tr>
<tr>
<td>Neck radiation</td>
</tr>
<tr>
<td>Tracheostomy</td>
</tr>
<tr>
<td>Inaccessible lesion</td>
</tr>
<tr>
<td><strong>Medically high-risk for CEA</strong></td>
</tr>
<tr>
<td>Age (≥ 80 yrs)</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Angina</td>
</tr>
<tr>
<td>Coronary artery disease/IHD (active)</td>
</tr>
<tr>
<td>Pulmonary disease</td>
</tr>
<tr>
<td>Renal insufficiency</td>
</tr>
<tr>
<td>Severe neurological disability</td>
</tr>
</tbody>
</table>
tients greater than or equal to 80 years of age. The majority of patients (86%) were symptomatic patients (Table 1, Table 2). Most common location of lesion was Proximal ICA.

Of the primary and secondary endpoints examined, difficulty controlling perioperative blood pressure amongst all patients was the most significant finding (38% in all patients and 36% amongst ‘high-risk’ patients). The results show no association between ‘high-risk’ patients and labile blood pressure perioperatively ($\chi^2$ statistic = 1.11, df = 1, $p = 0.29$). There was also no association between the number of risk factors and labile blood pressure perioperatively ($\chi^2$ statistics = 2.03, df = 5, $p = 0.845$). Results may not be significant due to the limited sample size. Of the three non ‘high-risk’ patients, two patients experienced complications involving uncontrolled blood pressure perioperatively.

One asymptomatic ‘high-risk’ patient, with 5 risk factors, died perioperatively as a result of a fatal MI (primary endpoint). Another ‘high-risk’ patient had a Non ST Segment MI (NSTEMI) preoperatively with symptoms resolving by day three of intensive care until (ICU) admission post-procedure.

At 3-6 month follow-up, one patient had a recurrent TIA in the same hemisphere requiring reintervention with CAS. The occurrence of a contralateral TIA in one high risk patient was not thought to be related to the carotid endarterectomy.

No patient in this study had an anatomically hostile neck lesion due to tracheostomy or location of the lesion. Two patients had had previous radiation to the cervical area but did not experience reduced morbidity or mortality perioperatively or at 3-6 month follow-up.

**DISCUSSION**

No therapeutic procedure would have withstood the pressures of scrutiny as those faced by CEA if it were anything short of the most effective treatment for carotid stenosis producing an acceptable stroke risk reduction whilst limiting perioperative stroke and death rate, in a difficult clinical group of patients. Since the advent of endovascular therapy, CAS has been identified, primarily by industry, as an alternative therapy to CEA, particularly in ‘high-risk’ patients. The results of this paper, however, indicate that there is no identifiable group of patients who respond poorly to CEA.

In this study, 47 of the 50 patients examined were by definition ‘high-risk’. This fact alone supports the view that the majority of patients who undergo this procedure can be expected to have a range of significant medical comorbidities. Of the 47 ‘high-risk’ patients, one asymptomatic patient (unstable lesion) did suffer a fatal MI perioperatively. This patient had five risk factors one of which was cardiovascular disease (recent MI, unstable angina and left ventricular dysfunction). Active coronary artery disease is perhaps one of the most significant risk factors for cardiac complications post-procedure (14, 15). The incidence of death or non-fatal stroke perioperatively in this study (primary endpoint: 2.1%) amongst ‘high-risk’ patients is significantly lower than CAS in a ‘high-risk’ population (15, 16).

The low incidence of any ipsilateral stroke or death in this study, perioperatively (2%, n=50) and at 3-6 month (2%, n=50), confirm the safety and short term benefits of CEA especially in comparison to CAS (9.6% at 30 days, 11.7% at 6 months, from EVA-3S) (3). The SAPPHIRE study, which provided the stimulus for CAS proponents, had a 30-day incidence of stroke after stenting of 3.6% (16). However, these results are unreliable as the majority of patients (>70%) were in fact asymptomatic patients who inherently carry a lower stroke rate than do symptomatic patients (3, 16).

One symptomatic ‘high-risk’ patient had an ipsilateral stroke due to restenosis requiring reintervention using CAS at 3-6 months. The use of CAS may be justified in this situation given evidence suggesting that surgical treatment of a secondary lesion originally treated with CEA is associated with a higher risk of adverse
In this case the secondary lesion is most likely due to fibro-intimal hyperplasia (within 36 months of CEA) and thus less likely to embolise during CAS (18). However, further studies are required to identify the short and long term outcomes of CAS after restenosis.

Interestingly, 38% of all patients had labile blood pressure perioperatively. Although there is no association between ‘high-risk’ patient or the number of risk factors and perioperative labile blood pressure, the reason for this result could be due the nature of the procedure. Carotid arteries at the level of the carotid bifurcation and internal carotid artery contain baroreceptors which are important in maintaining blood pressure. CEA could thus result in compromised baroreflex possibly due to the removal of sensory afferent fibres from the lumen at the time of atheroma removal (19). Not all patients had hemodynamic instability perioperatively (20). Although this study failed to reveal a significant statistical association between contralateral stenosis and poor blood pressure control perioperatively ($\chi^2$ statistic = 0.33, df = 1, p = 0.56), studies have demonstrated a significant relationship. For example, Nouraei et al demonstrated CEA resulted in significant acute elevation in blood pressure and that this change was more pronounced in patients with contralateral disease (relative risk 4.0, 95% CI 1.8 to 8.9, p<0.0002) (20).

One of the suggested benefits of CAS is its non-invasiveness and thus reduced capacity to produce haemodynamic instability. However CAS is not simply an innocuous procedure but rather has its own spectrum of complications including, hemodynamic compromise, cerebral embolization, stroke and death (11, 21). In a case-control study comparing CAS and CEA in which patients were matched for age, symptoms and coronary disease, poor post-operative blood pres-
sure control was observed in 34% of CAS patients despite the use of atropine (22). Although baroreflex dysfunction seems to be a risk inherent to the site of disease and procedure, there is some evidence to suggest that baroreflex dysfunction is associated with a 3.3 times higher statistically significant risk of developing further cardiovascular complications and an eightfold increased risk of cardiovascular mortality in the 5 years following procedure (20, 23). The analysis of this association is, however, outside the scope of this study and would be better examined in a prospective study.

It must be noted from the outset that this study is limited in power (n = 50) which in turn limits the significance of its results. Furthermore, there were 47 ‘high-risk’ and only 3 ‘non high-risk’ patients thus preventing any significant comparative analysis. In addition to this, the study defined ‘high-risk’ to include purely the presence or absence of factors such as MI, hypertension or angina. Recent evidence suggests that recent MI (within past 3 months), unstable angina and uncontrolled hypertension (particularly systolic hypertension) as opposed to simply the presence of disease is more relevant to the outcome of CEA (14, 15).

Despite the limitations of this study it would appear that the concept of the ‘high-risk’ patient as advocated by CAS proponents is not supported by this study. The role of industry in marketing new technology continues to be problematic. The US Food and Drug Association (FDA) approved the use of CAS in ‘high-risk’ patients on the basis of the SAPPHIRE study (11). Since the release of that study, its veracity in terms of data collection, analysis and clinical applicability have been undermined by serious allegations of bias. the lead author of the report was removed from the trial's final presentation due to a ‘conflict of financial interests’ as he was the inventor of the Angioguard embolic protection device which was used in the SAPPHIRE Trial, later purchased by Johnson & Johnson in 1999, for $40 million (24, 25). The lead author was later removed from the Cleveland Clinic amid FDA investigations into “irregularities of study protocol” (25). Perhaps even more worrying is the fact that two more recent studies, EVA-3S and SPACE, produced distinctly contradictory results to the SAPPHIRE study. These difficulties are of real concern as the results from the SAPPHIRE trial had been used as the basis for public policy.

Concerns for patient safety resulted in the premature cessation of the French EVA-3S study which compared CEA and CAS in patients with symptomatic severe carotid stenosis in a multicentre, randomized non-inferiority study (3). Concerns regarding equipoise were warranted given the 30-day incidence of any stroke or death was 3.9% after CEA and 9.6% after CAS with a relative risk of 2.5 (95% CI, 1.2 to 5.1; p<0.01) (3). At 6 months the incidence of any stroke or death was 6.1% after CEA and 11.7% after CAS (P=0.02) (3). Similarly the industry sponsored Wallstent trial, which was the first randomized multicentre trial designed to compare CEA and CAS, was also stopped prematurely after early analysis revealed the CAS arm to have a combined risk of stroke or death at 30 days of 12.1% in comparison to 4.5% in the CEA group (26). The results of this trial can only be found in abstract form and one can only speculate why the results have not been published. Although the results of the SPACE study were not quite so pronounced the study failed to demonstrate the non-inferiority of CAS (5). Thus without the presence of a ‘high-risk’ population that can be shown to not benefit from CEA, the role of CAS appears to be limited as its results do not appear to be either equal or better than CEA.

The role of CAS is not only undermined in the lack of the ‘high-risk’ patient group, identified by CAS marketers, but also its...
Industry is entirely without agenda in the
It would be naive to suggest that in-
olved in health care to ensure medicine
vigilance is required on the part of all in-
affect public policy indicates that more

some of which relate directly to arthro-
to be affected by more than one disease,
with carotid artery stenosis are very likely
risk' indicates that patients who present
94% of patients were by definition 'high-
just another example of clever market-
ing. That is, there are no pa-
the stent and cerebral protection device)
any

Numbers given in brackets are a percentage of the total number of 'high-risk' patients (47)
‘number of patients = 15

CONCLUSION
It would be naive to suggest that in-
the realms of medical advancement. The no-
tion of the ‘high-risk’ patient is perhaps
just another example of clever market-
ing. The FDA has currently approved the
use of CAS for symptomatic patients with
>50% stenosis and for asymptomatic pa-
tients with >80% stenosis who are also
‘high-risk.’

The results gathered from this study, how-
However, indicate there is no ‘high-risk’
patient for CEA. That is, there are no pa-
tients on the basis of medical co-morbid-
ities who should be considered for CAS
as opposed to CEA. The very fact that
94% of patients were by definition ‘high-
risk’ indicates that patients who present
with carotid artery stenosis are very likely
to be affected by more than one disease,
some of which relate directly to athro-
sclerosis. Fundamentally, the fact a sin-
gle study (SAPPHIRE study) was able to
affect public policy indicates that more
vigilance is required on the part of all in-
volved in health care to ensure medicine
is not commercialised for the benefit of
industry. For CAS to have a viable role
in the treatment of carotid stenosis, it
needs to prove its cost-effectiveness by
producing clinically superior results to
CEA. It has currently failed to achieve this
in all patients including the elusive ‘high-
risk’ patient.

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Gender and Suicide
A Case Report

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During a GP placement, I sat in on a consult with a year 12 high school student and her boyfriend. Her usual GP was away and as such she had never met this GP nor me before. The consult centred on the patient’s increasing anxiety. She described having a low mood and that her anxiety had worsened since being prescribed an anxiolytic. After initial questioning, the GP asked if the patient “... ever had thoughts about suicide or ending it all?” At this point, the patient asked her boyfriend to leave the room. As the consult progressed it was evident the patient did have suicidal ideation, had plans for committing suicide and had previously attempted suicide. This was a challenging consult; the patient left with advice to cease the current medication (was prescribed an antidepressant instead) and asked to come back for review with her usual GP in a few weeks time. This consult left me with many questions, particularly related to gender and suicide.

Internationally, suicide is the third leading cause of death among adolescents (1). In Australia, suicide accounted for 20% of all Australian deaths in the 16-24 age group in 2004 – 2006 (2), and was the leading cause of death for both males and females in the 25-34 year age group in 2007 (3).

Help-seeking is recognised as a protective factor against suicide and is vital for early treatment and prevention of mental health problems during adolescence (4). A recent study of Australian high school students examined the association between suicidal ideation and intentions to seek help from a GP for suicidal thoughts (4). Higher levels of suicidal ideation were related to lower intentions to seek help from a GP for suicidal problems (4).

I was interested to note that the gender of the doctor does not impact on suicide inquiry (5). Factors positively associated with addressing suicide included having a personal or vicarious experience with depression, asking more depression-related questions and working in academic medical settings (5).

Asking about suicidal ideation is important as it is a proxy measure for suicide (6) and an independent estimate of risk for suicide completion (4). Young females are twice as likely as males to report suicidal ideation and suicide attempt behaviour (7). However, males are three to fourfold more likely to die by suicide than females (7). This large gender difference is not attributable to artefact to data collection and has been described as the “gender paradox of suicidal behaviour” (8). Multiple reasons have been proposed for this gender paradox including biological differences between sexes, social norms and cultural expectations that differ amongst the genders and even iron levels, with observation of a gender-dependent association between poor iron status and the history of attempted suicide (9, 10).

It is difficult to apply the current understanding of gender differences in suicide to predict which females (or males) with suicidal ideation will complete fatal suicide. I find it feasible that had the patient in the above case study been male, a referral to the mental health crisis team would have been the immediate outcome of the consult. Despite all of the current knowledge and research into the gender paradox of suicide perhaps it is beneficial to treat each patient independently of gender such that any at-risk patient (with suicidal ideation and plans) should be referred to the mental health crisis team or other such support.

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What’s in a name? Gender and the eponym

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The human desire to be remembered has ironically conjured that which many medical students tend to forget – the medical eponym. In fact, our medical forefathers (and mothers) have been so busy naming things that we have ended up with over 8000 medical eponyms – enough to make you develop Magnan’s sign. Thankfully, these eponyms can be attributed to only 3383 people, so if you remember Charcot and his triad, you can be merciful that he also coined a syndrome, joint, zones, a type of vertigo and aneurysms to name but a few.

Other than the indefatigable Charcot, it is the male of the medical world that has been most responsible for embarrassing us on the ward round, when the esteemed professor asks: “what is Ganser-Dämmerzustand syndrome?”†

Like explorers charged with conquering new lands, our testosterone-fuelled medical pioneers have been quick to immortalize their name in every landmark of the human body, from Head’s zones § to Goldstein’s toe ‡. On the other hand, of these 3383 eponym-givers, only 129 women (4%) have bequeathed their name to some disease, sign or symptom. Over the next few paragraphs, we will look at the sizable contribution of these women to medicine through their eponyms. We will also look at some of the men who have given their name to the female form or attempted (often falsely) to describe the female psyche.

It is likely we have all benefited from Virginia Apgar’s work. The anaesthetist, born in New Jersey in 1909, gave her name to her score, which plays an important part in evaluation in the newborn child. In fact, the beauty of this eponym is that it is also an acronym, which is a boon to every student and junior doctor when faced with a screaming neonate. Apgar initially had to give up a surgical career after intense gender discrimination, however she made the role of anaesthesia her own; she was one of only two anaesthetists to feature on a United States stamp. She was also an accomplished builder of musical instruments, in particular building a string quartet.

Yvonne Barr is fondly recalled by every doctor faced with a teenager with fatigue and cervical lymphadenopathy, having discovered Epstein-Barr virus. Infectious disease eponyms are particularly prevalent amongst women, for example Rebecca Lancefield is considered the enemy of every streptococcus after her discovery of the bacteria’s surface antigens. Margaret Dix, an English physician, sent the world spinning when, along with Hallpike, she devised a test for benign positional vertigo. Jacqueline Noonan was able to define the case where a pseudo-Turner’s syndrome can occur with a normal array of chromosomes – this paediatrician was later recognized as one of America’s best doctors. Such is the beauty of the eponym. With a single word one can describe a complex scientific or clinical entity whilst paying homage to the hard work and dedication of the name bearer. Also, a good eponym is actually a memory aid, rather than a hindrance. It is often a useful and efficient way of communicating clinical data to a colleague. Therefore, by documenting an Apgar score for example, not only do we give a relevant and efficient piece of medical data; we honour the memory of our esteemed colleagues.

Not all eponyms, however, present such a prototype. Below are a few examples of eponyms, incidentally bearing the name of men, which have reinforced a gendered knowledge base of medicine. Firstly, let’s deal with our old friend Charcot. Jean-Martin Charcot was a French neurologist who had a long and distinguished career, busily naming lots of things that he saw. He even has an island in Antarctica named after him. He was fascinated by the concept of hysteria, that is, conversion of psychological stress into physical symptoms, emotional volatility and overdramatic or attention-seeking behaviour. This term has now been replaced in the modern era, particularly because it was originally thought to be only a disease of women (hystera is the Greek word for uterus). Charcot thought that he had discovered ‘hysterogenic’ zones across the body (Charcot’s zones). Thankfully Charcot’s zones, like hysteria, have fallen out of favour in modern times; Charcot will have to be content with his 15 other eponymous syndromes.

Clérambault’s syndrome is another example of an eponym that embodies sexism. Gaétan Henri Alfred Edouard Léon Marie Gatian de Clérambault described ‘A condition in which a woman becomes deluded that a certain man is in love with her. The man, with whom only a brief acquaintance exists, is usually older and of higher social status. The man, who has done nothing to stimulate or encourage such a belief, is at first unaware of it but is later likely to be embarrassed by telephone calls, letters and amorous advances.’ It appears monsieur Clérambault had a little trouble with unrequited...
love and subsequently sought to medicalise it. There is no equivalent syndrome for one of male gender and no scientific reason this behaviour would be a peculiarity to one of the female sex. This is an example of an eponym that deserves to be left to the history books.

Finally, in all of man’s immortalisation in the human body, female anatomy has been popular amongst the males keen to attach their name to a structure. Even the word vagina has its connotations with the stereotypical warrior male, the word coming from the Latin for sheath or scabbard. A German, Gabriel Fallopius, was responsible for popularising the word vagina and also gave his name to the Fallopian tubes in the 17th century. Since then, many men have dedicated their names to female anatomy, from Pawlik’s triangle†, to Frankenhauser’s ganglion** and Skene’s glands††. Caspar Bartholin the Younger clearly took more interest in the female form than Bartholin the Elder, both anatomists, as it was the younger Dane who named the greater vestibular glands. Finally, Ernst Gräfenberg can be credited with discovering what many in the medical profession consider not to exist (rather than cannot be found) – the G spot. Needless to say, this would probably be one of the more popular eponyms (although only in its abridged form).

This is not an argument against the use of eponyms to describe anatomical structures. Poor eponyms generally fall out of use anyway. For example when a functional name gives a far better clue to a structure’s location (compare paraurethral glands with its eponym Skene’s glands). It is more to point out that our knowledge base of medicine is skewed towards men, given the near exclusion of women from academic medicine until only the last century.

So from Alzheimer’s disease to Zollinger-Ellison syndrome‡‡, we have a considerably rich bank of eponyms to draw upon to enhance the clinical environment. We need only to be aware that with their use comes acknowledgement of their origins and in some cases, a subtle reminder of the gendered knowledge base in medicine.

† An area on the anterior wall of the vagina in contact with the base of the bladder and distinguished by the absence of vaginal rugae.
** Uterovaginal plexus of nerves
†† The paraurethral glands of the female

‡‡ Triad of 1) hypersecretion of gastrin, 2) multiple, atypically located, often recurrent peptic ulcers, and 3) a noninsulin producing islet cell tumor of the pancreas
Man made miracle

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Organ transplantation has revolutionised modern medicine. It is one of the most effective treatment options for many otherwise fatal conditions. Australia, with one of the most sophisticated healthcare systems in the world, is currently ranked 17th globally for the number of transplants performed per annum (1). This equates to only 200 transplants per year, which is one of the lowest rates in the developed world. A challenge for future medical professionals is to make the shift to making organ transplantation a routine procedure. An increase in the rate of these procedures is needed as they show definite benefits over alternative treatments. Furthermore, organ transplantation is financially beneficial for the Australian healthcare system as it is often more expensive to provide alternative treatments. It would not only be beneficial to the patient, but it also goes hand in hand with a doctor’s legal and ethical duty to offer the best medical care. The first step to improving the organ donor and transplant rate is to identify the reasons for Australia’s poor ranking. Some of these reasons are due to legislations, while others are due to public misconceptions. There are ways that doctors and medical students can help overcome these reasons and improve donor numbers in the future.

There are many reasons why the organ transplant rate is so low in Australia. Firstly, Australian legislation does not have ‘presumed consent’, like many of the top ranking developed nations. Presumed consent means that everyone is considered to be an organ donor unless they specify otherwise. According to the International Registry of Organ Donation and Transplantation, Spain, Belgium, France and Italy all follow this rule and constitute four out of the top five nations in the International Donor Statistics (2007). Australia, on the other hand, has an ‘opt in’ policy. This means that a person needs to register with the Australian Organ Donor Registry (AODR) to make their decision known. In addition to registering, it is necessary to inform family members of the decision to donate as it is the next of kin that will ultimately give consent (1). There are certain misconceptions that the public have which may contribute to Australia’s status in organ transplantation. They include the following:

- doctors will not work as hard to save the lives of donors. This is not true as doctors have a legal and ethical obligation to uphold the values of beneficence (do good) and non-maleficence (do no harm). The Australian Medical Association code of ethics is an example of a document that entails the above responsibilities (2).
- organ donation is against their religion. While this may be true for a minority of religions, it is not the case for the most commonly represented religions in Australia (1).
- they cannot have an open casket funeral. This is also incorrect as there are no visible scars when people are clothed, and appearance is completely maintained (1).
- rich and famous people receive organs first even if they are not at the top of the transplant list. Like the other misconceptions, this is false. There is no difference in allocation of organs between people (1).
- minorities should not donate due to race. This lies untrue because matching donors with recipients is not determined solely by race. The criteria that do determine donor-recipient matching include such things as blood and tissue typing (1).
- if they have been listed as a donor on their licence, their organs will be donated. However, this is only one step in assuring organ donation. In addition to registering with the AODR, it is important that the donor talk to their family and the next of kin about their wishes for donation. The next of kin ultimately decides in the event of death if organ donation is a possibility (1).

The organ donation rate in Australia needs to and can be improved. The Universal Declaration of Human Rights (UDHR), that Australia follows, states that all individuals have a right to life (3). An organ donation is often the best option to consider in managing many patients. Organ donations have a multitude of benefits in addition to aiding the recipient. For example, families and friends of the recipient also benefit from knowing that their loved one’s life is saved or made better. Also, a donor’s family often find it easier to say goodbye to their loved one when they know that they have committed such a generous and selfless act. Additionally, a donation can be a means to an end of continuing other treatment options. Thus there is financial benefit for the health system, tax payers, and also for patients. For example, in renal failure, the costs of dialysis run at approximately $84,000 per year. A kidney transplant of approximately $75,000 with $11,000 annual costs could have a drastic financial benefit (1). Lastly, the opportunities are endless for recovered transplant recipients, with many of them going into professions that benefit the community (e.g. paramedics, police officers etc.).

It should be the goal of current medical students and practitioners to improve upon these rates given the benefits they exemplify. One possible way is to partake in an educative and encouraging role and inform acquaintances and patients about the benefits of organ donation. Another possible way is to encourage patients and people we
encounter to register with the AODR and inform their loved ones of their decision to donate. Students in particular can establish organisations within universities and host events that promote awareness of organ donation. Anyone from twelve months to ninety years could be eligible for donation, so we should all become organ donors ourselves.

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Image courtesy of Professor Amanda Barnard
Deaths from Rheumatic Heart Disease
Deaths from rheumatic heart disease in Australia have declined dramatically since the discovery and widespread distribution of antibiotics during the 1950s, as well as the economic development and improved living conditions in most parts of Australia over the last century (6).

Figure 1 demonstrates the downward trend in death rates from rheumatic heart disease in Australia. However, the prevalence of rheumatic heart disease may, in reality, be a lot higher. According to data collected through the National Aboriginal and Torres Strait Islander Health Survey, approximately 0.7% of Indigenous Australians have rheumatic heart disease as a long term condition, which represents about 3,500 people (4).

Indigenous Australians have significantly higher rates of rheumatic heart disease compared to non-Indigenous Australians. This includes incidence, prevalence, and number of deaths from the disease. A complex interplay of infectious, environmental and social factors is responsible for increased exposure to group A streptococcus in Aboriginal communities, which can lead to rheumatic fever and subsequent rheumatic heart disease. These factors include inadequate housing and sanitation, poor access to medical care and social exclusion. Prevention strategies should focus on vaccination (when it becomes available), improving living conditions in Indigenous communities, and maintenance of registries of people who already have the disease to ensure follow up care.

Abstract
Indigenous Australians have significantly higher rates of rheumatic heart disease compared to non-Indigenous Australians. This includes incidence, prevalence, and number of deaths from the disease. A complex interplay of infectious, environmental and social factors is responsible for increased exposure to group A streptococcus in Aboriginal communities, which can lead to rheumatic fever and subsequent rheumatic heart disease. These factors include inadequate housing and sanitation, poor access to medical care and social exclusion. Prevention strategies should focus on vaccination (when it becomes available), improving living conditions in Indigenous communities, and maintenance of registries of people who already have the disease to ensure follow up care.

INTRODUCTION
This paper outlines the ways in which infectious, environmental and social factors interact to give rise to rheumatic heart disease. In some remote Indigenous communities, the rates of rheumatic heart disease have been well documented as being among the highest in the world (1).

DEFINITIONS
Rheumatic Fever:
Acute rheumatic fever is a delayed, inflammatory complication of an untreated throat or skin infection from group A Streptococcus bacteria, also known as Streptococcus pyogenes (2).

Rheumatic Heart Disease:
Rheumatic heart disease is caused by damage to the heart resulting from acute rheumatic fever. Rheumatic heart disease can affect the heart valves, the heart muscle and its lining, and the connective tissue throughout the body (2).

Indigenous Australians:
Indigenous Australians are Australians who are of Aboriginal and/or Torres Strait Islander origin. Although the majority of Indigenous Australians live in urban towns and cities (about 75%), the term ‘Indigenous Australians’ is used in this paper to indicate those people living in the remote communities of northern and central Australia where most cases of rheumatic heart disease occur (3).

PATTERNS AND BURDEN OF DISEASE
Incidence and Prevalence of Rheumatic Heart Disease
Prevalence
Table 1 outlines the prevalence of recorded acute rheumatic fever and chronic rheumatic heart disease in the Australian population in 2006.

Incidence
From 2002-2006 there were 350 new reported cases of rheumatic fever and rheumatic heart disease. All of the cases were Indigenous Australians (2).

Difference in prevalence between Indigenous and non-Indigenous Australians
Aboriginal Australians and Torres Strait Islanders who live in remote geographical locations are most affected by rheumatic heart disease (5).

Deaths from Rheumatic Heart Disease
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rheumatic heart disease is extended and/or repeated occurrences of acute rheumatic fever. The pathogenesis of rheumatic fever involves the triad of a genetically susceptible individual, a group A Streptococcus infection and an abnormal host immune response (8).

Whilst rheumatic fever has traditionally been associated with Streptococcus infection of the pharynx, recent studies suggest that pyoderma may be a more significant risk factor for the disease in Indigenous communities in tropical areas (9-12). Pyoderma is often a secondary infection to scabies, which is also extremely common in Indigenous communities (13).

Another important risk factor is compliance with prophylactic antibiotic regimes. It is clinically indicated that individuals with a history of rheumatic fever have injections of benzathine penicillin every 28 days for a minimum of 10 years after diagnosis (14). Reduced compliance with this routine increases the likelihood of recurrent infection and subsequent rheumatic heart disease.

Table 1. Rheumatic fever and rheumatic heart disease in the Australian population (2006)

| Acute Rheumatic Fever and Chronic Rheumatic Heart Disease (as defined by ICD-10) |
|----------------------------------|----------------------------------|
| Number of Australians with the disease 2006 (prevalence) | 1,402 |
| Number of new cases 2002 – 2006 (incidence) | 350 (all were Indigenous Australians) |
| Hospitalisations 2006 - 2007 | 2,561 |
| Deaths 2006 | 285 |

However, not all individuals with a history of rheumatic fever will necessarily develop the condition again upon re-exposure to Streptococcus pyogenes (8). Extensive searches for genetic susceptibility factors have been undertaken, including human leukocyte antigens, B-cell alloantigens, and cytokine genes, but more evidence is required before the genes can be conclusively linked with the disease (8).

Environmental Risk Factors
The high rates of rheumatic heart disease among Indigenous Australians are largely due to an increased exposure to Streptococcus pyogenes, related to overcrowding and poor living conditions, combined with a lack of access to adequate medical care (5,16,17).

It has been well established since the 1950s that a strong correlation exists between incidence of rheumatic fever and poor housing (18). Many Aboriginal and Torres Strait Islander people living in remote communities do not have access to adequate medical care. The high rates of rheumatic heart disease among Indigenous Australians are largely due to an increased exposure to Streptococcus pyogenes, related to overcrowding and poor living conditions, combined with a lack of access to adequate medical care (5,16,17).

Figure 1 – Trends in death rates for chronic rheumatic heart diseases, Australia 1931 – 2005, from Australian Institute of Health and Welfare’s Deaths from Chronic Rheumatic Heart Diseases(7)
have access to clean water, adequate sanitation, safe housing or sufficient rubbish disposal (19). Communal toilets, overcrowding, and pests are also significant problems (20,21).

These conditions expose residents more frequently to pathogenic strains of *S. pyogenes* than their non-Indigenous counterparts. When residents are provided with new housing and appropriate sewage removal systems, the number of skin infections in the community declines (22).

Lack of access to medical care is another key environmental risk factor, as early diagnosis and treatment of pharyngitis and pyoderma is critical in the prevention of rheumatic heart disease (16). In rural and remote Indigenous communities, primary care is often provided through community health centres where staff turnover is high and resources are overwhelmed by a large number of acutely ill patients (23).

While community health centres are located in most remote Indigenous communities (13), studies have demonstrated that residents only feel safe to attend a clinic if the service provided is culturally appropriate and a trust relationship has been established with the health care provider (14).

Other obstacles to accessing health care include lack of transportation and lack of an active recall and reminder system to help patients remember treatment times (14).

**Social Risk Factors**

As outlined by Marmot in *The social determinants of health inequality*, Aboriginal and Torres Strait Islander peoples are a socially excluded minority within their own country (24).

Many Indigenous Australians are still struggling to overcome the cultural dispossession and disconnection with land enacted by governmental policies throughout Australian history (25). This has a considerable impact on individual, family and community understandings of health and well-being, health-seeking behaviour and compliance with prescribed treatment regimes (26).

Other social risk factors for Aboriginal people and Torres Strait Islanders in terms of poor health outcomes are lower rates of education, employment and income than non-Indigenous Australians (27). These issues are inherently linked to broader issues of social exclusion, discrimination, marginalisation from mainstream labour, and economic deprivation (26).

**PREVENTION STRATEGIES**

A group A streptococcal vaccine is currently being developed and will be trialled in Pacific nations before being rolled out in Australian Indigenous communities for the prevention of rheumatic heart disease (29,30). Until the vaccine is available, the primary prevention strategy should be focused on reducing the reservoir of circulating group A streptococcus bacteria by improving the living conditions of Indigenous Australians in rural and remote communities, and improving their access to appropriate medical care (5). In addition, maintenance of the registries in the Top End of the Northern Territory is essential in ensuring follow up care for those already suffering from rheumatic heart disease (5).

**CONCLUSION**

Indigenous health has come to the fore of the Australian cultural consciousness in recent years with the national apology, the Close the Gap campaign by non-Government organisations and the Australian Government’s subsequent Close the Gap strategy. These are positive steps in the right direction, but any Indigenous health policy must be careful to consider the underlying social and environmental factors that give rise to more proximal infectious factors.

The unacceptable burden of rheumatic heart disease carried by remote Indigenous communities in northern and central Australia will only be remedied by adequately addressing the social and environmental factors at the root of the problem, such as poor housing, lack of access to adequate medical care, and social exclusion.

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INTRODUCTION

In 2001, Russia was described as having the fastest growing population of Human Immunodeficiency Virus (HIV) internationally (1). By the end of 2007, it was estimated that the number of individuals living with HIV in the Russian federation was between 560,000 – 1.6 million, representing approximately 3.5% of the world’s HIV population (2). This paper uses poverty as a foundation to exemplify how the causative and often intertwined sociocultural-political factors have contributed to this HIV epidemic in the Russian federation.

BACKGROUND TO THE EPIDEMIC

Over the past century, Eastern Europe has witnessed great social and political change, from the era of Communism, Nazism and the Cold War, to the fall of the Soviet Union in 1989 and the armed conflict in the former Yugoslavia. This social and political change has lead to increasing levels of poverty within the Russian Federation. The current HIV epidemic emerged in Russia in 1996, relatively late by comparison with HIV epidemics in other regions. Russia is considered to have one of the fastest growing populations of HIV in the world, with the United Nations (UN) estimating that up to 1.6 million individuals are infected with HIV (3). However, unlike the epidemic in Sub-Saharan Africa, HIV is being spread predominantly by intravenous drug use (IDU). Russia has the highest rate of IDU in the world. The current epidemic is further compounded by the Russian Government’s ‘zero tolerance’ policy approach that illegalised needle exchange programs.

POVERTY, ECONOMICS AND HIV

To suggest that poverty alone causes HIV oversimplifies the complex web of biphasic and causative relationships that intertwine poverty and health outcomes. For example, in Sub-Saharan Africa, South Africa and Botswana are two of Africa’s wealthiest nations and have the highest population and prevalence respectively of HIV worldwide (4). The contribution of socioeconomic-cultural-political contexts to the HIV epidemic, the impact of government policy and the heterogeneous dissemination of wealth and access to health care in affected societies need to be acknowledged.

Health outcomes follow socioeconomic status (SES), with those of higher SES enjoying better health outcomes than those of lower SES (5). South Africa and Russia are comparable as they have two of the greatest discrepancies in SES. The ratio of wealth/consumption of the richest ten percent of the Russian and South African populations is 70 times greater than that of the poorest ten percent (1). Therefore, while the necessary health and education infrastructure may exist within these countries, individuals living in poverty may not have access to them.

Poverty also results in individual’s engaging in ‘desperate practices’, such as paid sex work, in order to obtain an income (1,6). This increases their potential exposure to HIV, and also to STI’s that further increase their chances of developing HIV (7). Also, individuals who engage in such behaviour are less likely to have been educated about HIV and be taking the appropriate precautions (8). Furthermore, those who contract HIV have fewer resources to cope with the consequences of the illness, and have less access to the necessary health infrastructure. Individuals of a lower SES, who are more likely to develop HIV, are most often employed in labour intensive industries. HIV makes people too weak to perform physical labour. Often the patients and their families cannot afford for the patient to be out of work, nor can a family member be a fulltime caregiver and not work and many of these patients cannot afford the treatment (7).
INTRAVENOUS DRUG USE AND THE CONSEQUENCE OF ‘ZERO TOLERANCE’ POLICIES

The fall of the Soviet Union has led to increased levels of poverty in Russia and has also created a sense of despondency among many. This has resulted in increased self-gratifying risk-taking behaviour, including sexual promiscuity and drug abuse (1). This combined with increasing production of heroin in neighbouring Central Asian countries and consolidation of Russia as a major trafficking route, has made heroin cheaper and intravenous use of it more frequent (9,10). Russia has one of the highest rates of intravenous drug use in the world, with approximately 2-3% of 15-64 year olds using (three times as many as in Western European countries) mainly intravenous heroin (6).

The Russian Government has responded to increasing heroin levels with a ‘zero tolerance’ policy approach. Such policies have criminalised the distribution of sterile injecting equipment and methadone. The HIV prevalence in intravenous drug users is between 8-64% throughout the country, with IDU being responsible for 70% of HIV cases (2). A meta-analysis of 63 studies conducted in Russia between 1998-2000 put syringe sharing within the range of 40-60% (11). Another study has found that in Togliatti (southern Russia), 84% of injecting drug users reported sharing syringes (8). Furthermore, in Ekaterinburg (central Russia) 86% of drug users had shared syringes in the past month (12), while in Moscow, 35-41% of injecting drug users reported sharing syringes (10). Previous research has shown that needle exchange programmes reduce syringe sharing, reduce HIV prevalence, engage marginalised individuals with health programmes, all without increasing drug use (13,14). In cities without needle exchange programmes, HIV infection rates increase at 6% a year, in comparison to cities that do, where infection rates decrease by 6% a year (14,15). Furthermore, many IDU fund their habits through sex-work, with between 30-39% of users having engaged in paid sex work (16,17).

The criminalisation of drug use and sex work within Russia has also lead to increasing levels of incarceration. Russia has the second highest prevalence of incarceration in the world (611 per 100,000), behind the United States (702 per 100,000). Prison overcrowding in Russia is a serious issue, with the average space per prisoner being 2m² often resulting in prisoners sleeping in shifts (10). Punitive drug laws have resulted in an increase number of drug users and sex workers in prison, many of whom are HIV positive. Prison overcrowding presents a great opportunity for infectious pathogens, such as tuberculosis and STIs. HIV is also a significant problem in under-resourced Russian prisons. While drug use is illegal, it is still practiced in prisons, mostly without clean equipment for reasons discussed already. The incidence of disease within the prison population, and the sharing of equipment and unsafe sex have lead to one author to refer to Russian prisons as “functioning as incubators for HIV and other infectious diseases” (10). Estimates put the prevalence of HIV in the Russian prison population at 6%, though it is suggested that this figure grossly underestimates the real prevalence (2).

Another factor that has been implicated in the HIV epidemic in Eastern Europe is the lack of access to anti-retroviral treatment. Not only does effective treatment reduce plasma viral load and therefore transmission rates, effective management of HIV reduces the burden upon the health sector. Brazil is a country comparable to Russia, with a per-capita GDP of $7,360 in 2001 (Russia’s was $7100) (14). In 1996, the Brazilian Government pressured pharmaceutical companies to make anti-retroviral treatment more affordable, otherwise the Government would no longer recognise pharmaceutical patents and would use generic substitutes. It is now estimated that between 70-95% of Brazilians have access to effective HIV medication, in comparison to 10-25% of Russians (18,19). The success of the program in Brazil has lead to a large decline in the incidence of HIV in Brazil and has forced Joint United Nations Programme on HIV/AIDS (UNAIDS) to revise the scale of the epidemic (14). It has also saved Brazil an estimated $1 billion in hospital resources through reduced admissions (14).

A ROLE FOR DOCTORS

The HIV epidemic in Russia gives several opportunities for doctors to intervene. As discussed, HIV is being spread throughout Russia by intravenous drug users sharing injecting equipment and also through unprotected sex. Many of these individuals are often ignorant of safe practices and also of HIV itself. Also, the zero tolerance approach of the Russian Government is helping the epidemic spread amongst users. Furthermore, success of the Brazilian approach needs to be highlighted to Russian policy makers for reducing the burden of HIV at both a population and economic level. Advocacy and lobbying for such development could be performed by doctors, who are informed of the facts and can promote safe and effective prevention and treatment methods for containing the epidemic.

CONCLUSION

In conclusion, the relationship between poverty and HIV is mediated by a complex web of interrelated factors. Poverty is not just a lack of wealth, but a lack of access to social infrastructure such as education and effective healthcare. The HIV epidemic in Russia is occurring due to high rates of IDU, fuelled by increasing levels of poverty since the fall of the Soviet Union and increased production and access to cheap Afghan heroin. This is perpetuated by a government that has responded with a zero tolerance approach that results in users sharing equipment. Finally a comparison is made with Brazil, briefly highlighting the impact of an evidence-based response to the HIV epidemic. Given the context in which HIV has arisen and developed in Russia, a strong and effective response will have effects beyond HIV itself. Without successful intervention, it is estimated that between 5.4 – 14.5 million Rus-
sians will have HIV by 2025 (19).

REFERENCES


INTRODUCTION

Sexually transmissible infections (STIs) pose a serious challenge to Australia, particularly for the Aboriginal and Torres Strait Islander communities who are disproportionately afflicted.

The National Centre in HIV Epidemiology and Clinical Research (NCEHR) released two reports in 2008, STI AASR ’08 (1) and STI ATSISR ’08 (2) that detail the patterns of STIs in Australia as at 31 December 2007. Of note was that HIV/AIDS, gonorrhoea, and syphilis remained significant problems, disproportionately affecting the Aboriginal and Torres Strait Islander communities (1, 2).

Two STIs for which prevention may be working effectively include hepatitis B, with vaccination in effect (3) and donovanosis for which the number of cases in the Aboriginal and Torres Strait Islander communities dropped from 16 cases in 2003 to three in 2007; this may be attributable to a successful National Donovanosis Eradication Project (1, 2).

Abstract

In spite of an increase in the understanding of the pathogenesis, manifestation, and sequelae of the many sexually transmissible infections (STIs), they remain a major health problem. Chlamydia has evaded prevention strategies of the Department of Health and Ageing and increased in incidence in Australia. The disease is simple in its transmission, and may be silent in manifestation, but can lead serious consequences such as infertility in women. The Aboriginal and Torres Strait Islander communities are affected more than non-Indigenous communities and this paper presents a pilot strategy to address chlamydia: THATS-C. The strategy looks to use a communal centre such as an Aboriginal arts centre, to increase the rates of rational screening for, and consequent treatment of chlamydia. If effective, this could be applied to other small communities that have community centres with the hope that it could then be widely used in the Northern Territory (NT) to prevent the continued spread of chlamydia.

Table 1. Data sourced from STI AASR ’08(1) and relevant for new cases in a period of 1 year ending December 31 2007. Rates are age-standardised per 100,000.

<table>
<thead>
<tr>
<th>Infection</th>
<th>Aboriginal &amp; Torres Strait Islander NT</th>
<th>Non-Indigenous NT</th>
<th>Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of cases</td>
<td>Rate</td>
<td>Number of cases</td>
</tr>
<tr>
<td>HIV</td>
<td>0</td>
<td>-</td>
<td>5*</td>
</tr>
<tr>
<td>Hepatitis B**</td>
<td>2</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Hepatitis C***</td>
<td>26</td>
<td>-</td>
<td>119</td>
</tr>
<tr>
<td>Chlamydia</td>
<td>1,313</td>
<td>1,988</td>
<td>867</td>
</tr>
<tr>
<td>Gonorrhoea</td>
<td>1,398</td>
<td>2,130</td>
<td>202</td>
</tr>
<tr>
<td>Syphilis</td>
<td>103</td>
<td>163</td>
<td>16</td>
</tr>
<tr>
<td>Donovanosis</td>
<td>1</td>
<td>-</td>
<td>0</td>
</tr>
</tbody>
</table>

*estimate; **There were 9 cases in total in the NT however 4 did not report; ***There were 227 cases in total in the NT however 82 did not report.
Chlamydia stands out as an easily treatable, but otherwise serious infection, for which a control strategy is presented: THATS-C.

**CHLAMYDIA**

Chlamydia diagnosis rates for Australia as a whole have continued to rise (by 7% between 2006 and 2007) to 245 per 100,000 and was the most frequently reported notifiable condition in 2007 with 51,867 cases reported (1). The rates of diagnosis are highest in females (293 versus 199 per 100,000) and in the 20-29 year old age group (1). When comparing the Aboriginal and Torres Strait Islander and non-Indigenous communities there is a markedly higher rate of diagnosis in the Aboriginal and Torres Strait Islander communities (1,241 versus 264 per 100,000 in the non-Indigenous community in 2007) (1). There were 2,825 Aboriginal and Torres Strait Islander patients with the infection of which 1,766 were female patients (62.5%) and 1,059 male patients (37.5%) (1).

The Northern Territory (NT) has consistently had the highest chlamydia rates (Figure 1) (1); a rate consistent with large Aboriginal and Torres Strait Islander communities (Figure 2) (1). The NT had 1,313 cases in the Aboriginal and Torres Strait Islander communities out of the 2,825 Aboriginal and Torres Strait Islander Australian total (46% of cases from all states and territories except NSW and QLD) whereas the NT had 867 cases in the non-Indigenous community out of the 22,827 non-Indigenous Australian total (3.7% of cases from all states and territories except NSW and QLD) (1).

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### Table 2. Burden of STIs for the Aboriginal and Torres Strait Islander females compared to the Australian female population as a whole in 2003 (source: Vos, T. et. al. 2007(7))

<table>
<thead>
<tr>
<th>STIs (exl. HIV/AIDS)</th>
<th>Female Aboriginal and Torres Strait Islander</th>
<th>Female Australian</th>
</tr>
</thead>
<tbody>
<tr>
<td>DALY</td>
<td>1%</td>
<td></td>
</tr>
<tr>
<td>DALY Rank</td>
<td>20th</td>
<td>86th</td>
</tr>
<tr>
<td>Rate of DALYs per 1,000</td>
<td>1.9</td>
<td>0.2</td>
</tr>
<tr>
<td>DALYs RR</td>
<td>9.2 (the third highest DALY RR)</td>
<td></td>
</tr>
<tr>
<td>YLD</td>
<td>1.6%</td>
<td></td>
</tr>
<tr>
<td>Rate of YLDs per 1,000</td>
<td>1.6</td>
<td>0.2</td>
</tr>
<tr>
<td>YLDs RR</td>
<td>8.2 (The second highest YLD RR)</td>
<td></td>
</tr>
</tbody>
</table>
Although the rate of diagnosis for chlamydia in Australia is greatest in the 20-29 year age group, the rate is highest in the female 13-19 year age group in the Aboriginal and Torres Strait Islander communities whereas males too are highest in the 20-29 year age group (2); rates are highest in Aboriginal and Torres Strait Islander women, as they are in women of the non-Indigenous communities (2). Strikingly high rates were noted in the Aboriginal and Torres Strait Islander communities living in very remote regions and also in Alice Springs (2, 4) (Figure 3).

**BURDEN OF STIS**
The eight national health priorities do not include chlamydia or STIs as a group (5). Chlamydia and STIs fall into the infectious & parasitic diseases category accounting for 1.7% of the total DALY (6) burden in the Australian population as a whole (5). STIs (excluding HIV/AIDS) accounted for 1.0% (450) of DALYs in Aboriginal and Torres Strait Islander females in 2003 and were ranked as the 20th leading cause of DALYs, as compared to the 86th cause for the whole Australian population (7) (Tables 1, 2).

**PREVENTION STRATEGIES**
Given that gonorrhoea and chlamydia are disproportionately affecting the Aboriginal and Torres Strait Islander communities in the NT (Table 1) a thorough exploration of prevention and management strategies in the NT focusing on chlamydia in the female Aboriginal and Torres Strait Islander communities of the NT is essential.

Novel prevention approaches should reflect on those that have already been pursued, such as the broad strategy formulated in 2005, National Aboriginal and Torres Strait Islander Sexual Health and Blood Borne Virus Strategy (NATSI SHBBVS) (8), which was complementary to, National HIV/AIDS State-

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**Table 3.** The nine principles fundamental to health strategies for Aboriginal and Torres Strait Islander communities from the National Strategic Framework for Aboriginal and Torres Strait Islander Health 2003-2013 (8)

| 1. Cultural respect |
| 2. A holistic approach |
| 3. Health sector responsibility |
| 4. Community control of primary health care services |
| 5. Working together |
| 6. Localised-decision making |
| 7. Promoting good health |
| 8. Building the capacity of health services and communities |
| 9. Accounting for health outcomes |
gy (NHAS), National Hepatitis C Strategy (NHCS), and National Sexually Transmissible Infections Strategy (NSTIS). Briefly, the key objectives of the strategies relevant to chlamydia were to improve access to diagnosis, treatment, testing, surveillance, research, and improve awareness of STIs (8). To achieve these ends, the strategies aimed to partner effectively the mainstream health services and Aboriginal Community Controlled Health Services (ACCHS), as well as with drug and alcohol services, the education sector, the correctional sector, and the community sector (8). Successful prevention required screening for STIs, access to prevention tools (condoms), early detection strategies (client recall; reminder systems), contact tracing, education programs, and reduction in structural and bureaucratic barriers to health care (8).

However, given that these strategies were introduced in 2005, and a reduction in chlamydia has not been observed, it can be assumed that either the access to diagnosis increased, thereby illuminating many cases that had previously been missed whilst concealing any actual reductions due to the strategy, or simply that the strategy failed in its entirety, or that an otherwise exponential increase in cases has been dampened. Ultimately, more efforts than those outlined in 2005 need to be taken to reduce the number of cases.

Recent additions have included the NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care Setting (9), which provide guidelines that GPs can follow when taking a history, examining patients, and ordering investigations in order to maximise detection and appropriate treatment for STIs (9). In addition “Safe Sex. No Regrets” educational campaign began running in August 2008 (10).

A STRATEGY AND ITS EVALUATION
It may be too early to assess the effectiveness of the new guidelines on the prevention of chlamydia and this report introduces a complementary strategy to those that are in place. The strategy, a targeted and highly accessible treatment and screening chlamydia campaign (THATS-C), assimilates knowledge that has been garnered from approaches trialled over the recent years (Tables 3-5), and keeps with the principles of cultural awareness, sensitivity, and safety (11, 12).

THATS-C
The strategy should be trialled in a
community that had a community focal point, such as an Arts Centre. As Alice Springs has the highest number of chlamydia cases (Figure 3) a community in that region would be an ideal location to trial a prevention strategy. The interface for the THATS-C should be an Aboriginal owned and operated facility that has been a part of its community for at least ten years. The longevity of this facility would be an indicator that it was stable and would last for the duration of a trial. Its artists and elders who would be ideal ambassadors for the trial should link the facility to the community.

The women of the arts centre would be invited to become involved in the program, for which they would be remunerated. The role of these ambassadors would be to distribute a brief questionnaire to the younger women in the communities. These questionnaires would attempt to identify and prompt those women that are at high risk of carrying chlamydia to complete a urine test. The questionnaires would focus on behaviour and the presence of symptoms. The questionnaires would have a conclusion computed by the person that had filled in that questionnaire. The conclusion would suggest the individual complete a urine test for chlamydia, or not to. No other party would need to see or evaluate the questionnaire. It should be designed with expert consultation from Infectious Diseases specialists, NCHCR and with the women of community so that questions are of a culturally sensitivity language.

The arts centre would be fitted out with a room where women could collect urine samples and equipped with refrigeration facilities to store the samples. Between the community members of the Arts centre, the samples would then be ferried daily, to a Remote Aboriginal Health Network outpost, and from there, to an Alice Springs pathology lab. Confidential results in sealed envelopes would be disseminated back to the women that had completed the urine samples via the ambassadors. A female healthcare worker could then visit the Arts centre and dispense antibiotics once a week.

CONCLUSION
Chlamydia cases have increased in Australia, with Aboriginal and Torres Strait Islander women in the NT being disproportionately affected. A novel prevention campaign, THATS-C, recognises cultural lessons, and could be a convenient and acceptable approach to detecting and treating chlamydia. THATS-C could be trialled in a community.

Table 4. Recommendations in The link between primary health care and health outcomes for Aboriginal and Torres Strait Islander Australians (Griew, R. 2008): Local evidence and lessons(13)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>“Genuine local Indigenous community engagement to maximise participation, up to and including full community control”</td>
</tr>
<tr>
<td>2.</td>
<td>“A collaborative approach to working with other service providers”</td>
</tr>
<tr>
<td>3.</td>
<td>“Delivery of core primary health care programs such as material and child health and/or chronic disease prevention, detection and management”</td>
</tr>
<tr>
<td>4.</td>
<td>“Evidence-based approaches adapted to local conditions”</td>
</tr>
<tr>
<td>5.</td>
<td>“A multidisciplinary team approach employing local community members”</td>
</tr>
<tr>
<td>6.</td>
<td>“Service delivery that harmonises with local Aboriginal and Torres Strait Islander ways of life”</td>
</tr>
<tr>
<td>7.</td>
<td>“Adequate and secure resourcing”</td>
</tr>
</tbody>
</table>
nity focal point, such as an Arts centre, where ambassadors would be targeting those most at risk and prompting them, via a questionnaire, to be screened for chlamydia. This would both circumvent the asymptomatic nature of chlamydia and bypass access issues of remote communities. Chlamydia rates would be monitored and compared to regional and national data in order to indicate success or failure.

REFERENCES


Table 5. Obstacles to counter when creating prevention strategies for the Aboriginal and Torres Strait Islander communities; based on lessons from the Inala Health Centre General Practice.

<table>
<thead>
<tr>
<th>Obstacle Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. “No Indigenous person working within the centre”</td>
</tr>
<tr>
<td>2. “Staff perceived as unfriendly and uncaring”</td>
</tr>
<tr>
<td>3. “Staff talk down to you, ‘make you feel ashamed’”</td>
</tr>
<tr>
<td>4. “Staff body language, as interpreted by Indigenous people, suggested they were not wanted at the centre”</td>
</tr>
<tr>
<td>5. “Treated poorly at reception”</td>
</tr>
<tr>
<td>6. “Showed low self-tolerance to Indigenous child behaviour”</td>
</tr>
<tr>
<td>7. “Long wait to see doctor”</td>
</tr>
<tr>
<td>8. “There is ‘nothing’ at the centre that Indigenous people can identify with”</td>
</tr>
</tbody>
</table>
INTRODUCTION

A medical student, following an intern, asks how he knows to give the litre of fluid over eight hours, rather than ten.

“Ahh…” stammers the intern, “there’s no hard science to it.”

Prescribing fluids is an area where junior doctors are currently allowed a lot of freedom. The prevailing belief is that little harm can be done to a patient, as long as basic principles are followed and certain simple parameters such as blood pressure, electrolytes and development of oedema, are duly monitored. Using intravenous [IV] fluid therapy [FT] to keep patients hydrated is therefore viewed as like giving a drink of water, straight into the vein - more an act of hospitality than medical management. Prophylaxis against thirst perhaps.

However earlier this year, the British Medical Journal published a letter sent from a concerned clinical researcher and a senior professor of surgery from Edinburgh (1). They surveyed 33 Foundation Year 1 doctors (interns) during their first hospital rotation as doctors, testing their knowledge of fluid and electrolyte balance, and ability to prescribe fluids correctly. They described the results as “alarmingly poor”,

Table 1: Composition of normal saline, 5% dextrose, 4.5% albumin, Hartmann’s solution and gelofusine. Source: Ambrose et al 1997, Anaesthesia UK 2004 (3,4)

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Electrolytes mmol/litre</th>
<th>Tonicity</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9% saline</td>
<td>154 sodium, 154 chloride</td>
<td>Isotonic</td>
<td>5.0</td>
</tr>
<tr>
<td>5% dextrose</td>
<td>275 glucose (rapidly absorbed)</td>
<td>Hypotonic after absorption</td>
<td>4.0</td>
</tr>
<tr>
<td>4% dextrose + 0.18% saline</td>
<td>30 sodium, 30 chloride, glucose 40 grams (not mmol/L)</td>
<td>Hypotonic</td>
<td>4.0</td>
</tr>
<tr>
<td>4.5% albumin</td>
<td>40-50 grams (not mmol/L), &lt;160 sodium, 136 chloride</td>
<td>Isotonic</td>
<td>7.4</td>
</tr>
<tr>
<td>Hartmann’s solution</td>
<td>131 sodium, 5 potassium, 2 calcium, 111 chloride, 29 lactate</td>
<td>Isotonic</td>
<td>6.5</td>
</tr>
<tr>
<td>Gelofusine</td>
<td>154 sodium, 125 chloride, trace potassium and calcium gelatin 40 grams (not mmol/L)</td>
<td>Isotonic</td>
<td>7.4</td>
</tr>
</tbody>
</table>
and called for better and more formalised teaching of fluid administration in medical schools.

Another English study looked at post-operative fluid prescribing (24-hours after abdominal surgery) and found that of 71 patients, 17% had an adverse outcome directly related to incorrect IV FT, mostly from sodium or potassium overload (2). Moreover, the fluid prescriptions often did not correspond to measured clinical parameters. The authors concluded that junior medical staff were not using available information to prescribe IV FT correctly.

The evidence base for selection of correct IV FT in critical care, paediatric and peri-operative settings is considerable, and growing. For patients who fall into none of these categories, such as the adult inpatient receiving medical or supportive care, there are no landmark studies to guide good practice, and we are referred instead to our physiology textbooks for best practice on maintenance and replacement fluid therapy.

The purpose of this article is to identify literature that can help us prescribe fluids correctly. Guidance on choice of fluid in the setting of maintenance therapy and non-urgent replacement therapy was particularly sought, but was lacking. Not covered here is the physiology of fluid and electrolyte balance, nor electrolyte disturbance, although this is the foundation of good fluid prescribing and readers are urged to maximise familiarity with this subject. For convenience, Table 1 shows the electrolyte composition of some commonly used fluids, and Table 2 presents normal electrolyte requirements.

SEARCH STRATEGY
The articles were identified through online search of UpToDate and of the biomedical abstract database Pubmed using “fluid therapy”, “fluid maintenance” and “intravenous fluids” as search terms, and by examining the reference lists from articles identified in this manner. Three of the most interesting and relevant were selected, and will be summarised below. Suggestions for further reading are provided at the end of this article.

ARTICLE TITLE: “HOW TO SELECT OPTIMAL MAINTENANCE INTRAVENOUS FLUID THERAPY”(7)
This irresistibly-titled article provides a lively discussion on FT rationale and fluid requirement calculations. We will restrict ourselves to a discussion of two major issues that they raise: firstly, defining the rationale for FT, and secondly, a minor point of physiology to do with vasopressin, which may have important implications for FT.

Precisely because it is so obvious, it must be important: intravenous fluid therapy should always have a purpose, and the better defined that purpose, the easier it will be to make correct decisions. Five possible reasons for IV FT are defined, beyond the simple “s/he’s not eating or drinking”:

- “Defend the normal blood pressure
- Return the intra-cellular fluid (ICF) to normal
- Replacing ongoing renal losses
- Giving maintenance fluids to match insensible losses
- The need for glucose as fuel for the brain (with regard to glucose composition of FT)”

For each of these, there are potential pitfalls. In hypotensive patients who are already volume overloaded, consider what the use is of endless IV fluid in defending their blood pressure. When trying to resuscitate the ICF compartment, be mindful of the speed with which this should be accomplished (avoid rapid electrolyte shifts). Replacing renal losses is cruel to the kidneys if they are combating an excess water load, only to have it all pour back in again!

The article discusses the calculation of insensible losses in some detail, emphasising that the calculations may err, especially in the elderly, the obese and those with infectious disease. Although brain glucose requirement can be supported with IV dextrose, recall that the brain is not the only organ that competes for glucose. Importantly, if the glucose given IV stimulates insulin sufficient to suppress lipolysis, then this will impair glucose utilisation, as fatty acid oxidation and glucose oxidation are interdependent processes. Therefore, having insight into the purpose of the IV FT helps to determine the amount, tonicity and composition that should be given.

As alluded to above, the article is critical of tradition methods of fluid calculation, which may overestimate water requirement. While not necessarily causative, this compounds the problem of iatrogenic hyponatraemia, the most common electrolyte disturbance in hospitals. Their discussion of hyponatraemia offers a simple model of its pathophysiology:

- hyponatraemia is more often due to water excess rather than sodium depletion, and
- water excess will persist where the ability to generate a compensatory water output is diminished, such as in increased vasopressin production.

<table>
<thead>
<tr>
<th>Requirement</th>
<th>Usual (Western) dietary intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>Minimum of 1600mL/day(5)</td>
</tr>
<tr>
<td>Sodium</td>
<td>1-2 mmol/kg/day (3)</td>
</tr>
<tr>
<td>Potassium</td>
<td>0.5-1 mmol/kg/day (3)</td>
</tr>
<tr>
<td>Chloride</td>
<td>1-2 mmol/kg/day (3)</td>
</tr>
</tbody>
</table>

Table 2: Daily water, sodium, potassium and chloride requirements in normal adult Source: Ambrose et al 1997, Guyton and Hall 2006, Jamison 2003 (3,5,6)
Vasopressin production may be increased for many reasons, not necessarily related to volume status, and this phenomenon is common in hospitalised patients. Examples of stimuli for increased vasopressin are summarised in Table 3. The implication for our practice is that factors not related to the patient’s disease, and in the presence of otherwise normal fluid physiology, the patient’s ability to excrete excess water may be impaired.

A limitation to this article is that it was written in Canada, and therefore reflects Canadian practice which may or may not necessarily correlate with Australian practice.

**ARTICLE TITLE:** “THE HISTORY OF 0.9% SALINE” (8)

Why saline? Had you ever considered where fluid therapy comes from? If these questions stir your curiosity, have a read of this article, which describes how Dr. William Brooke O’Shaughnessy, a medical graduate aged 22, came to think of injecting salt solution for the treatment of cholera during an outbreak in England in 1831. His theory was that the oxygenation properties of the salt would revive the blood. It was not for some time that the usefulness of fluid itself was appreciated, but as understanding developed over the next century and more, fluids of varying toxicity and composition were trialled, in an attempt to create a fluid that would successfully mimic human serum. Ringer’s (1883) and Hartmann’s (1932) solutions are still in common use, not so much Lattas (1832) or Churton’s (1888).

The “invention” of 0.9% saline is attributed to Hartog Jakob Hamburger, a Dutch chemist who in 1896 compared the freezing points of human and animal blood serum with varying concentrations of salt solution, and determined that 0.9% saline is isotonic – previously 0.6% saline was thought to be isotonic. It is unclear exactly how his experiments led to 0.9% saline being adopted in clinical settings as so-called “normal” saline, as its administration leads to a large sodium load. Moreover, we are reminded that excessive sodium chloride (and undesirable effects of sodium can manifest above just 100mmol daily intake, or two thirds of one litre of normal saline (8)) is associated with renal vasoconstriction, immune cell dysfunction, and delayed return of gastric function after abdominal surgery.

So just why is 0.9% saline so popular? Since 1831, different electrolyte compositions of fluids have been tried, trialled and rejected; or used, sometimes with great effect and sometimes because nothing better was available. Mass production and time constraints have killed creativity, and created a tendency in us to just prescribe whatever was prescribed last time, but our goal should be the same as the early experimenters’ – to restore the patient’s physiological electrolyte balance as faithfully as possible.

**ARTICLE TITLE:** “PROBLEMS WITH SOLUTIONS: DROWN-ING IN THE BRINE OF AN INADEQUATE KNOWLEDGE BASE” (9)

Ten years ago, one of the authors of the previous article, with assistance from colleagues, undertook a telephone survey of junior doctors to determine their knowledge of and confidence in good fluid prescribing practices. 200 junior doctors (100 interns within the first ten days of their first intern job, 50 interns who had completed six-eight weeks as interns, and 50 PGY2s on surgical wards) were asked fifteen questions regarding fluid prescription.

By now we are not surprised that their findings were that inadequate knowledge of fluid and electrolyte requirements and poor prescribing in FT were common, and for our purposes, the exact proportion of which-groups-answered-what is not relevant. Instead, the same questions administered in the study are offered to readers as a challenge, see Box 1. The answers are given in the article itself as part of the discussion of findings.

**CONCLUSION**

If only one recommendation were allowed, it would be this: know the purpose for which the IV fluids are being prescribed. If this condition could be fulfilled 100% of the time, then our fluid prescribing practice would become tighter, and better tailored to individual patients’ needs. Other aspects of individualised FT are to be aware of the patient’s usual weight, existing volume status and most recent serum electrolyte readings. Apart from these parameters, and even apart from their disease process, anxiety states, pain and some commonly used drugs (see Table 3) can decrease vasopressin production and diminish the effectiveness of a person’s water balance system.

The early experimenters strove to find the most physiologically fluid possible that would rehydrate a person effectively,
without having prior knowledge of the normal composition of serum. Now that this information is readily available, we should use it to nourish our patients correctly.

**FURTHER READING**


Hilton AK, Pellegrino VA, Scheinkestel CD. Avoiding common problems associated with intravenous fluid therapy. MJA 2008; 189(9): 509-13

**REFERENCES**


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**Box 1: Questions asked of junior doctors in telephone survey by Lobo et. al 2001 (9)**

<table>
<thead>
<tr>
<th>Question</th>
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<tbody>
<tr>
<td>Who on your ward is responsible for fluid prescription?</td>
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<tr>
<td>How confident are you prescribing fluids?</td>
</tr>
<tr>
<td>Was the training provided to you satisfactory?</td>
</tr>
<tr>
<td>Were guidelines provided?</td>
</tr>
<tr>
<td>When are the fluid prescriptions checked?</td>
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<tr>
<td>What are normal, daily sodium and potassium requirements?</td>
</tr>
<tr>
<td>What is minimum urine output?</td>
</tr>
<tr>
<td>What are the sodium and potassium contents of normal saline, dextrose, Hartmann's solution and gelofusine?</td>
</tr>
<tr>
<td>What is desired post-operative urine output?</td>
</tr>
<tr>
<td>How best is post-operative fluid requirement calculated?</td>
</tr>
<tr>
<td>What is the best measure of fluid balance?</td>
</tr>
<tr>
<td>How do urinary sodium and osmolality change post-operatively as compared to pre-operatively?</td>
</tr>
<tr>
<td>How often should serum electrolytes be checked in post-operative patients who are receiving fluids?</td>
</tr>
<tr>
<td>How much potassium does a patient require, one day after right hemicolecotomy?</td>
</tr>
<tr>
<td>What fluids would you prescribe for a 70kg male patient three days after right hemicolecotomy?</td>
</tr>
</tbody>
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Arterial blood gases

Mimi Chiu*

*Medical Student, The Australian National University

The aim of sampling arterial blood gas (ABG) is to assess a patient’s acid/base balance, hypoxia and carbon dioxide retention and hence, allow for the initiation of appropriate treatment. This article explains the importance of arterial blood sampling, what the indications, contraindication and risks are as well as some useful tips when performing one.

Arterial blood sampling is defined as the sampling and measurement of arterial blood levels of oxygen and carbon dioxide and typically, pH.

<table>
<thead>
<tr>
<th>Indication</th>
<th>1. Evaluation of adequacy of ventilation, oxygen carrying capacity of blood and acid base levels</th>
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<tbody>
<tr>
<td></td>
<td>2. Establish diagnosis of, and severity of respiratory failure</td>
</tr>
<tr>
<td></td>
<td>3. Guide therapy</td>
</tr>
<tr>
<td></td>
<td>• oxygen administration</td>
</tr>
<tr>
<td></td>
<td>• mechanical ventilation</td>
</tr>
<tr>
<td></td>
<td>• treatment of acid base imbalance</td>
</tr>
<tr>
<td></td>
<td>4. Patient management – critically unwell patients</td>
</tr>
<tr>
<td></td>
<td>5. Arterial cannulation</td>
</tr>
<tr>
<td></td>
<td>• continuous pressure monitoring</td>
</tr>
<tr>
<td></td>
<td>• frequent blood sampling</td>
</tr>
<tr>
<td></td>
<td>• diagnostic angiography</td>
</tr>
<tr>
<td></td>
<td>• therapeutic embolisation</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Contraindications</th>
<th>Absolute</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Positive Allen’s test</td>
</tr>
<tr>
<td></td>
<td>Absent pulse at site of insertion</td>
</tr>
<tr>
<td></td>
<td>Infection at site</td>
</tr>
<tr>
<td></td>
<td>Evidence of vascular disease involving selected limb</td>
</tr>
<tr>
<td></td>
<td>Presence of arteriovenous fistula</td>
</tr>
<tr>
<td>Relative</td>
<td>Severe coagulopathy</td>
</tr>
<tr>
<td></td>
<td>Anticoagulants</td>
</tr>
</tbody>
</table>

| Adverse effects | 1. Mostly related to arterial cannula insertion: |
|                | • haematoma                                       |
|                | • sepsis                                          |
|                | • arterial thrombosis                              |
|                | • arterial ischaemia                              |
|                | 2. Arteriospasm                                    |
|                | 3. Vasovagal                                       |
|                | 4. Pain                                           |

| Useful tips    | 1. Site selection |
|                | • radial artery has the benefit of availability of collateral circulation and is relatively close to the surface and easy to palpate and stabilize |
|                | • brachial artery is large and easy to palpate but used only when radial is unsuccessful as collateral circulation is not as abundant as radial and is in close proximity to nerves |
|                | • femoral artery is good for low output states, large and easy to palpate, however associated with greater infection rate and is in close proximity to femoral vein which can be easily punctured. Only used when radial is unsuccessful. |
|                | 2. Documentation |
|                | • result of Allen's test                           |
|                | • collection time                                   |
|                | • puncture site used                                |
|                | • patient's inspired oxygen concentration           |
|                | • site condition (if any complication)              |
|                | • results of sample                                 |

REFERENCE
Twenty pairs of eyes were staring at me as my quivering hands held up a model of the clavicle. The stakes were high – my reputation as a final-year medical student was on the line. Clearing my throat and summoning all the confidence I had, I declared in a loud voice, “This is a scapula.” Needless to say I quickly corrected myself, but this was just one of the many memories I will be taking away from my specialty rotation in Medical Education over at Monash University.

Medical teaching has always been a passion of mine, so when the opportunity to take up a specialty rotation in Medical Education came along, I jumped at the chance. On the first day of the rotation, I met up with my supervisor – the lovely Professor Christine McMenamin, Director of the MBBS curriculum at Monash University. We had a productive discussion on what I could do to get the most out of the rotation. Besides being eager to take on a research project in medical education, the budding Orthopaedic surgeon in me was looking forward to teaching the Year 1 medical students anatomy of the upper limbs, among other topics. Professor McMenamin was more than happy to have me help out with this and before the discussion ended, she left me with a few very encouraging words – ‘Teaching can be very rewarding.’

So yes, I was officially a “medical educator”, but to be utterly honest, I had no idea how to deliver a structured tutorial for medical students. However, after a few chats with various tutors later, I had a three-step structure all sorted out:

**STEP 1: PREPARATION**
The first thing I was advised to do was to prepare for my classes. I picked up my copy of the tutor’s manual and went over the relevant sections thoroughly. The manual was an absolute lifesaver – it clearly listed out the topics that had to be covered during each tutorial. I poured over various anatomy textbooks, gathering and jotting down key points (and witty “did-you-know” facts) in the manual. While structuring the tutorials, I found it very helpful to throw myself back into the shoes of a Year 1 student – What did I like/ not like about my Year 1 tutorials? How can I improve them? At the same time, I also found it useful to rehearse in front of a mirror. Rehearsing allowed me to pick out weaknesses in my delivery, thus enabling me to correct them.

**STEP 2: DELIVERY**
I was very nervous about delivering the anatomy tutorial. I had no idea how the students would react to the session and I was extremely worried that I would make a huge fool of myself. Doubts aside I gathered my courage, swept up my manuals and went off to class.

I had twenty students in my tutorial – a much larger group than I expected. I had originally planned for the students to lead the discussion and teach one another, but I quickly realised that the plan was doomed to fail. These students were all relatively new to anatomy, having just learned the basic principles, with most having close to no knowledge of the upper limbs. I had to make impromptu changes to my approach! I decided to direct the discussion with a descriptive exercise – I would show them a bone model, identify it (which was how the clavicle-scapula mix-up came about) and then get one of the students to describe it. For example, I would hand the clavicle to one of the students saying, “Look at the clavicle, run your finger along its surfaces and tell me as much about it as possible.” The students loved the exercise! They picked out various structures on the bones (i.e. ‘This part is rough so a ligament or a muscle probably attaches here!’) and through that I managed to get an interesting discussion going. Occasionally, I would interrupt the discussion to deliver a mini-lecture, or to provide the students with tips, but otherwise I was happy to play the role as the facilitator.

At the end of the session, I gave the students some topics to prepare for the next session so as to guide their self-study.

**STEP 3: EVALUATION**
Before the students left, I asked them for feedback. The number of students who enjoyed the tutorial pleasantly surprised me. Most of them liked the amount of visual aids I used – bone models, wet specimens and badly self-drawn pictures. They also found the descriptive exercise very useful, as it allowed them to visualise structures and their associations. However, some students would have preferred if I had broken the group into smaller groups, so that more students could be engaged. This feedback helped me a lot in my planning and preparation of subsequent sessions – I took all comments into consideration and made modifications to the way I taught.

So with that, I concluded my first anatomy tutorial. It went much better than I had expected. While there were definitely some areas for improvement, there were also many positives. Importantly, as nervous as I had been at the start of the tutorial, I was having a lot of fun by the time the session was in full swing.
In the weeks that came, I took the group through several more anatomy tutorials. I went from strength to strength with each session, progressively fine-tuning the way I taught. I also developed a close bond with my students, and took much pride as I saw them gradually develop confidence and competence with their upper limb anatomy knowledge.

In reflection, my teaching experience was indeed, as Professor McMenamin had predicted, very rewarding. Given the chance, I would definitely love to return to medical education in the future. After all, as one of my fellow anatomy tutors once said, “Teaching’s enjoyable and easy – it’s just about making students believe you have known all your life what you learnt this morning.”

I hope my story will encourage more senior medical students and junior doctors to take up teaching.

ACKNOWLEDGEMENTS

I am heartily thankful to my supervisors, Professor Christine McMenamin and Dr Nicole Koehler, as well as my fellow tutors, whose encouragement, guidance and support throughout the rotation fuelled my passion for teaching.
Medical school and the enculturation of self-neglect

Jared Michael Stephenson* and Michael Li*
*Medical Student, The Australian National University

My unshaven cheek rolls away from its safe resting place in my palm, jerking me back into a state I can best describe as ‘awake’. I rub what I now know to be my proximal interphalangeal joints around what I now know to be my orbital cavities and quietly panic as I try to recite the muscles and nerves I’m using. I glance briefly at the clock behind me, taking a moment to focus as the fuzzy red blotches become legible numbers. 2:14. In front of me, a copy of Harrison’s lies open, accompanied by a half empty mug of lukewarm coffee and a small pool of what I now know came mostly from my submandibular salivary glands.

Much criticism has been leveled at the working conditions that doctors are exposed to. Self-neglect through sleep deprivation or excessive stress is of increasing concern within the medical profession. Justifiably so, given their potential to impair doctors’ personal wellbeing and compromise patient care. Despite these potentially catastrophic outcomes, self-neglect remains entrenched within the profession. The adaptive responses to the academic demands of medical school may help account for this, as self-neglect becomes habitual within this ‘safe’ context where patients’ wellbeing is not immediately threatened. Indeed, through sleep deprivation, exposure to emotional exhaustion, and risky alcohol use, medical school can be seen to normalise self-neglect, entwining it throughout the medical profession.

THE MODERN DOCTOR

The stereotypical modern doctor is a goal-oriented figure who absolutely prioritises their patients and their career to the exclusion of all else, including their personal wellbeing. One American study indicated that doctors routinely work between 60 to 130 hours per week during their residency, including periods without sleep exceeding 30 hours (1). Burnout has also been identified as a common theme within the early years of practice, a consequence of intolerable stress due to emotional exhaustion and a low sense of personal accomplishment (2). Whether these are viewed as causative or symptomatic of self-neglect, they both reflect its established presence within the medical profession.

The potential for self-neglect to compromise the physical and emotional safety of both doctors and their patients makes it problematic. The sequelae of delaying or foregoing sleep are well researched, with sleep deprivation linked to an obvious decline in cognitive function and mental acuity (1,3), with implications for occupational health and safety. For example, doctors are placed at a greater risk of needle-stick injuries (3) while patients are threatened by an increased likelihood of medical errors. Further, as health professionals become ‘worn down’, their degree of cynicism increases, along with a decay in humanitarian attitudes and empathy, all of which have downstream implications on the effective and professional delivery of care (4). With such evidence of the personal and professional consequences of self-neglect clearly laid out, it can be difficult to comprehend why health professionals would ‘choose’ to de-prioritise their personal wellbeing.

MEDICAL SCHOOL AND STRESS

And so we return to me. Disheveled, bleary eyed, and almost certain my bed is whispering my name as I try to commit ganglia, Latin adjectives and metabolic pathways to memory. The workload almost demands a lack of sleep, with any semblance of wakefulness driven by stress. And while research suggests that stress and its sequelae are all at their highest in the more advanced years of medical training (5), I can feel them seeping in my pylorus. Ball and Bax (5) studied a cohort of medical students asking them to complete questionnaires elucidating their health habits, alcohol consumption, degree of depression and their life satisfaction across a semester of study. It was revealed that as the semester progressed, students tended to adjust their health habits for the worse. Sleep patterns deteriorated, alcohol consumption increased, and physical activity and socialisation with peers declined. In other words, students compromised their personal wellbeing as academic demands necessitated.

The intrinsic relationship between medical school and stress is reported by students worldwide (4). In Australia, a study by Leahy et al. found that 44% of undergraduate medical students could be classified as ‘psychologically distressed’ (6). With accumulating stress comes an accumulation of depressive symptoms and dissatisfaction with life, the latter two increasing from the beginning to the end of the semester (5). Further, there is a higher prevalence of depression and anxiety amongst medical students than within the general population (4). Such psychological morbidity appears to have an adverse impact on academic performance, whilst also leading to cynicism, an unwillingness to care for the chronically ill and decreased empathy (2). However, the normalisation of stress is an often overlooked yet critical negative outcome. Stress, when experienced on a constant or regular basis, may
become a familiar state and a ‘typical’ part of life. Unfortunately, excessive stress can then potentially become nigh unrecognisable, by medical students and doctors alike, until the effects of professional burnout become all too apparent.

“You’re feeling sleepy. On the count of three, I’ll snap my fingers and you’ll... keep studying’

In nine months of medical school we have learned how to identify an aortic stenosis with our ears, renal failure with our eyes, and a host of pathologies simply by asking the right questions. But medical knowledge is not all that has been imparted. To some degree, the hidden curriculum has also taught us that eight hours of sleep in the evening is an ill-afforded luxury and two hours less sleep at night can be compensated for by a ten minute siesta in the afternoon. Ball and Bax (5) noted that by the end of the semester, over 50% of their participants reported staying up late and waking up early to study. Further, 47% of students obtained six hours or less of sleep per night which decreased towards the end of the semester as they accommodated the demands of study, demonstrating a formation of poor sleeping habits early in their study.

Ultimately, such a practice is maladaptive. At the university level, being awake and alert is conducive to the learning process, while mental fatigue simply is not. Mulligan (7) indicates that individuals deprived of sleep for 36 hours exhibited a 40% reduction of memory retention of new material compared to those who maintained a healthy sleeping pattern. A study pattern that forgoes sleep, then, is inefficient and is likely to coherently add to any burden of stress. However, once full-time study becomes full-time work in which cognitive function is imperative for patient care, the consequences of sleep deprivation can be catastrophic. Research has shown that 24 hours without sleep produces a similar reduction in performance to a blood alcohol concentration of 0.1% (3). Most alarmingly, a consistent reduction of sleep to six hours or less per night caused cognitive deficits as severe as those caused by two nights of acute sleep deprivation (7). It is evident that maladaptive sleeping patterns are trialled and accepted in the context of university where the negative impacts may be deemed acceptable compromises. However, once they are transferred into the clinical setting the potential risk may outweigh the benefit and as they have become normalised, such habits are challenging to discourage.

WORK HARD, PARTY HARD

In our new discourse of risk factors and causal pathways a discussion of self-neglect must address alcohol use, misuse and abuse. Risky drinking behaviours are an established cultural norm for medical students and practitioners alike (8). In the context of an excessive workload, there is little time to relieve stress, so when the opportunity arises it is taken to excess. Ball and Bax (5) found that over 20% of the students they surveyed could be classified as partaking in ‘problematic drinking’, while 18% of female and 11% of male medical students reported an increase of alcohol consumption during medical school. Additionally, the relationship between stress, anxiety, distress and high alcohol consumption in medical students appears to be reciprocated (4). That is, stressed students turn to alcohol as a means of emotional

Box 1. Self-care advice for medical students from medical students

- Form a study group. Studying with and teaching one another is an excellent way to consolidate information while remaining social.
- Maintain a close group of friends. These are the people who can provide you with psychosocial support, debrief with you after confronting experiences and generally share any burdens or triumphs.
- Know your limit and set your own goals. Everyone learns differently, so try not to compulsively compare yourself to your peers.
- Maintain social activities outside of medicine. Keep in touch with friends outside of your medical peer groups. These people are able to keep your study stresses in perspective, and remind you that there are aspects of your life outside of medicine.
- Be part of a mentorship program. Medicine is a tough degree so having a mentor to show you how to get through as smoothly as possible makes life a lot easier. Mentor a student yourself once you have settled down to help others through a tough period of transition.
- Organise clinical placements with your mentor or with any health professional that is willing to show you around their workplace. These reinforce and apply theoretical knowledge and are a good opportunity to ensure you are committing yourself to the career you want to be in.
- Volunteer to run extracurricular events promoting healthcare and support to the community. These provide a sense of personal satisfaction, look good on your resume and allow you to meet people from other years and different professions.
- Apply for scholarships. There are scholarships around for people of all educational backgrounds to apply for. Having a scholarship helps alleviate the stress of financially supporting yourself while studying.
- Stay healthy. When things get busy, don’t neglect yourself! Sleep and eat well, stay fit and active. This keeps you performing at your peak.
- Know when to seek help and where to get it. Friends, family, counsellors and doctors are readily available to help out when the times get tough.
release, while high alcohol use leads to increased stress. At best, this failure of homeostasis solves nothing. At worst, it is textbook decompensation, one which can potentially transcend the boundary between medical school and medical practice to complicate co-morbid stressors.

CONCLUSION
By the time medical students have become practitioners, they have participated in a medical school culture that espouses pushing oneself to the limits of mental and emotional strength and endurance, a culture where self-neglect is normalised out of perceived necessity. Much like the clichéd ‘frog in boiling water’, small compromises in self-care accumulate to a degree in which the potential for physical or emotional harm to both patient and medical practitioner is actualised. Consequently, it is imperative that medical schools accentuate the importance of self-care by integrating strategies for dealing with the volume of material into orientation programs (5), and that they place an ongoing emphasis upon student well-being. It is equally important for medical students to maintain healthy study habits as part of a balanced lifestyle. However challenging the process may be, the benefits of curbing the culture of self-neglect within the medical profession are too great to ignore.

REFERENCES
Scrubs and ties

Tim Lovell∗
∗Medical Student, The Australian National University

Scrubs; introduced to the ANU medical students in 2011. Scrubs; taken up with rapture by many from the cohort. OK... maybe ‘many’ is overstating it. Adopted by ‘some’ or ‘several’ may be more accurate. And ‘rapture’, that’s just not the right word. Taken up with due consideration, pause... and caution seems closer to the truth. Or how about simply rejected?

There was tentative interest when the prospect of wearing scrubs was announced. But this was soon followed by apathy. Over the last several months, it has become apparent that students have a number of reasons for not wearing scrubs.

Thus far I’ve heard that some students don’t feel that they should pay for the scrubs. And one set of scrubs would not be enough for a week; as a clinician told me ‘one to wear, one for tomorrow, and one for the wash’. Other students say that they don’t want to wash the scrubs if they are smeared with blood or any other type of human discharge and the intention of scrubs to reduce the risk of infection. Wouldn’t scrubs warrant a ‘hospital grade’ wash? Further to this, there are those that feel that scrubs will not be accepted by surgeons. In line with this, some wonder if scrubs may be appropriate only in a few clinical contexts.

To successfully introduce scrubs to the Canberra medical community, it has been suggested that the consultants lead by example. One student felt that it would take 15 years, others say if they are to be a success they should be a mandated uniform.

Are all of these reasons for not wearing scrubs genuine, or is it simply that most do not like the image of wearing scrubs?

The cost of scrubs is an issue. However, the cost of a pair of suit pants, or some dresses, as well as several shirts for the week can very quickly exceed the cost of a set of scrubs at $88. Added to this, the dry cleaning required for some items compounds their cost, particularly over four years. Scrubs can be washed at home and drip dry on a coat hanger over night. Even as an initial investment, and certainly in the long term, they would work out to be a cheaper alternative to conservative dress. On the other hand, conventional ‘clinical’ attire can be worn to a wide variety of occasions and therefore an economical investment, whereas scrubs can really only be used in a clinical setting.

With respect to those students who do not want to wash their scrubs, I find it interesting that in contrast, they don’t demand that the hospital clean their clinical attire. Why is it accepted that one would clean their ‘clinical’ clothes that see the same clinical situations as scrubs, and not the latter?

Being accepted by the medical community as a whole is important. We work as a team, and kinks in the dynamic interactions between the specialties and students should be avoided if at all possible. One might ask if the introduction of scrubs was discussed with all disciplines, including surgeons, or was the decision made with little consultation? Indeed, if the primary basis for the introduction of scrubs is an infectious diseases one, then perhaps it falls within the domain of the Infectious Diseases specialists alone.

Regarding the appropriate clinical placement for a student to wear scrubs, I feel that there are several. The acute care rotation is the archetypal rotation that most students I have spoken to feel scrubs are appropriate for. This is where identification made possible by scrubs, particularly in emergency situations, comes to the fore. The name tags that are often on our belts are rarely scrutinised, and this is difficult in in-
tense situations. But beyond the acute care rotation, I feel that scrubs are more than appropriate in the Senior Medicine and Surgery (SMS) and Foundations of Internal Medicine and Surgery (FIMS) rotations. It is on these rotations that I would want to be clearly identifiable, and to be seen as someone keen to be involved with my ‘sleeves rolled up’;
dressed ready to develop my practical clinical skills.

Bearing these reasons for and against scrubs in mind and their effect on infection control, there is another element of crucial importance - professional appearance. How we appear to patients can affect their perceptions of us, their level of comfort with us and the history they give us. Thus, it is important. In the vein of professional appearance, there are surgeons that require those working on their team to wear ties. By wearing a tie, you can overtly demonstrate a type of respect for the clinical situation and the patient.

But in this day and age, with widening socioeconomic gaps and rising health care costs is the tie really a more appropriate professional uniform for health-carers? Or does it in fact create distance between the practitioner and the patient? I do not think it appropriate that doctors dress as stock brokers yet some of the watches and accessories worn by some are more appropriate to Wall Street. Being a doctor is not about making money and yet with the expense of medical care combined with a doctor appearing aloof, distanced and behind the desk wearing a tie and speaking in a language unfamiliar to the patient, it would be easy to create this counterproductive image. Students may subconsciously mould themselves to this image of their consultant.

The option to wear scrubs has not been widely adopted by medical students, and it will be interesting to see what place they hold in the years to come. Ultimately however, I value what knowledge a doctor has, the morals and ethics by which they use it, their teamwork and an unprejudiced approach to all patients far more than whether or not they wear a set of scrubs or a tie.
A hacking cough stirs me as I sleep, my ears become attuned, ringing as the cold dark night sits over the room. Nothing, eyes drift closed.

Cough.

No it’s not a cough, it’s a gasp, a hack, a desperate breath, a child trying to breathe as their throat closes in. It continues, another gasp, another cry, another hack.

My eyes flick open – they mirror my wife’s. We lay there silent, hoping she goes quite, wishing her breathing to settle. If we don’t move, no decisions need to be made. But it worsens, constant, she tries to cry but it is stifled as if she needs to breathe more.

We pick her up in the dark cold merciless night and try to settle her. Her breathing almost taunts us, it quietsens and our tense muscles begin to unwind. We lay her down with a silent beseeching atheist’s prayer to any god who’ll listen. She hacks and screams in desperation again – we tense, pick her up and the cycle begins again and again.

Give her medicine – how old is she, how much can we give. Can we give her more? Hands tremble, I read the warnings, don’t exceed this, see a doctor if this, no more than that.

We snap at each other, a combination of tiredness, desperation and futility.

She opens her mouth and swallows the syringe of sticky orange medicine, her eyes look so trusting – her parents are here, she feels safe.

Like a child and its parent, I put my complete trust in the medicine.

Inside I don’t know what to do. I’m no parent – I passed no exams to get to this stage in my life, there is no algorithm I’ve learnt that I can follow. Why hasn’t someone taught me what to do?

I sit down to think. I look to the computer. I check a text book. I fortify my confidence with knowledge. I tune out the cries, my mum’s hints, I try to assess dispassionately.

It must be croup.

The Royal Children’s website says not to take kids with croup into the emergency department (ED) unless worried. What does that mean – of course I’m worried, what parent wouldn’t be. Is it suggesting I should be, or indeed could be, more worried. Stupid statement.

Time passes as I gather my evidence, solidify my resolve, formulate my decision. I tell Claire what I think. We agree, looking at our child.

But with the time it’s taken me to be confident with my own knowledge, she seems to have improved. When she is snuggled into Claire’s shoulder, upright and warm, she breathes easily. She closes her eyes and with seeming effortlessness, drifts asleep.

We sit and watch her chest rise smoothly and regularly. Grandma disappears back upstairs with some last advice that falls unheeded around us.

We sit and watch.

Claire leans back into the pillows and closes her eyes.

I sit and watch.

My child breathes softly.

I go into the spare room and lie down.

The dark night again settles down around me, inexorably draping me alone with my choices.
Glasgow, a Tale of Two Cities

James McCracken

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It was the best of times, it was the worst of times, it was the age of wisdom, it was the age of foolishness, it was the epoch of belief, it was the epoch of incredulity, it was the season of Light, it was the season of Darkness, it was the Spring of hope, it was the Winter of despair, we had everything before us, we had nothing before us, we were all going direct to Heaven we were all going direct the other way: A Tale of Two Cities, Charles Dickens.

While Dickens’ A Tale of Two Cities is a juxtaposition of London and Paris prior to the French Revolution, it could very well refer to the juxtaposition of the two Glasgows. During the great Victorian and Edwardian era, the tea and tobacco trade brought great ships up the River Clyde into Glasgow and with it, great prosperity. Glasgow was referred to as the second city of Empire. This period was celebrated with the Glasgow International Exhibition in 1901 and is remembered in Glasgow’s wealth of inner city Victorian and Edwardian architecture, including the Kelvingrove Art Gallery and Museum, and the City Chambers.

However, after the First World War, Glasgow was at the epicentre of the Great Depression. The River Clyde was barren of her ships, while the city also lost many of her buildings to the German Luftwaffe in World War II. While the gritty wee lass tried to revive her ailing economy, progressively many of her industries moved offshore and many of her ships set sail, never to return, leading to a period of rapid economic decline. This was also an era marked by Glasgow’s development of the infamous ‘Tenements’ and ‘Overflow housing’ to accommodate the poor souls who had migrated to Glasgow, in search of employment. The 1960’s architecture is a brutal contrast to the rich Victorian & Edwardian tapestries that flow throughout Glasgow.

My grandparents, Lily and James McCracken departed Glasgow to the promise of Australia as newly married, and expectant, ‘10 pound Scots’ in 1960. They finally settled in Sunshine, Melbourne’s working class inner-west. Jim found work as a builder and Lily raised three kids – Virginia (my mother), James & Penelope. After my parents separated when I was three, we lived with my grandparents. Sadly, Jim died of stomach cancer in 1986 at the age of 46. He'd never returned to Scotland, nor remained in contact with the McCracken family.

In December 2010, after 34 hours of transit, I’d finally arrived in Glasgow. I collected my luggage and wandered out into the chill of the Glasgow night, and headed to my residence in the West End. The next day I started my elective in Orthopaedics and Trauma at The Southern General Hospital, a 900 bed teaching hospital in Govan, Glasgow’s answer to the Bronx. After spending a morning being cleared to work in her Majesty’s National Health Service (NHS), I met my supervisor, Mr Marc Bransby-Zachary and my two leading lights, Orthopaedic Registrars Evan Crane and Andrew Wilkinson. I was warmly welcomed with a cup of tea, an offer of an afternoon in theatre and a lift home at the end of the day.

Each day began with a team meeting at 8am. We would all arrive in the darkness, arm ourselves with a cup of tea, go through the overnight presentations and discuss the operations for the day. Typically by then, it was time for the briefest of ward rounds and another cup of tea, before clinic or theatre at 9am, depending on what team you were in. I was warmly welcomed into the teams and given the opportunity to do a great deal, from assisting in operations and using the tools, through to inserting the odd screw, nail or suture. I also sat in on various clinics, examined patients and elicited pathology. I was also invited to clinics and lists at other hospitals, including a day of Orthopaedic Oncology at the Royal Infirmary and a night assisting Mr Colin Walker as the Glasgow Rangers club doctor. Andrew and Evan used the time between operations to exponentially increase my surgical knowledge: “Have you heard of torque suspension and the carpals? What about risk factors for a patella dislocation?”

After Hogmanay in Edinburgh, and a few days spent discovering Glasgow, I returned to the Southern General Hospital to further immerse myself in my elective. The NHS is said to be one of the premiere public health care systems, and the Glasgow and Greater Clyde NHS is a fine example. Glasgow is a city of 1.3 million people (serving a region of 2.3 million – 41% of Scotland’s population) served by five major hospitals (The Southern General Hospital, The Royal Infirmary, The Victoria Infirmary, Gartnavel & Western Infirmary), and one specialist paediatric hospital. The patients in clinic on my first day back were patients who had injured themselves over the New Year weekend, many of whom we would operate on later that week (including a man who fractured his scaphoid while curling). The waits were minimal to see a surgeon or operation due to the three dedicated orthopaedic theatres.

It was minus two degrees Celsius and snowing on my first Saturday night in Glasgow. I was in a taxi on my way to a McCracken family party. Prior to departing Australia, and through the medium of Facebook, I’d met a second cousin. I had been invited to her mother’s 60th birthday in Clydebank. I gazed through the foggy taxi windows at the city that went
past, unaware of the overwhelming Scottish hospitality that awaited and that this would be the beginning of a series of family dinners and parties. I’d spend the early parts of each night being toured around, wiping lipstick from my cheek, being reminded of how much I remind people of Jim, and how much they missed Lily. One such occasion was a dinner at the Singer, an old factory turned restaurant in Clydebank. Ironically, this was the very place my grandparents had met, as young adults, working at the Singer Sewing Factory. The orange mantle clock, that was a wedding present to my grandparents from the Singer girls, still sits on Lily’s mantle to remind her of those days.

After many years of hearing the tales of life in Scotland, and being so far away, there was a certain warmth of embracing and reconnecting with the family. However, I came to understand why Lily and Jim had left. They were from Clydebank, the part of Glasgow that accommodated the overflow. There were no bluestone roads where they were from. They had left for the greater opportunities and the sunshine (as my grandma would remind you) that Australia had promised. It became evident, as I got to know my family, that we’d made the most of these opportunities. I was visiting Scotland as a medical student, when no one from either side of my family had been to university. It was also evident in the other Glasgow that I came to embrace, the West End not Clydebank. The cafes, the cask ales, the architecture. It was a different Glasgow to the one that Jim and Lily had left behind, but one I’m sure they’d be glad I loved.
I had a very unique and fun experience on my medical elective in Kilimanjaro Christian Medical Centre (KCMC) in Moshi, Tanzania, but after I returned home I asked myself why that was. As most medical students undertaking electives in developing countries, I had this optimistic and possibly naive belief that I was going to make a real difference. For the past three years I had been the recipient of excellent teaching from inspirational health professionals at the Australian National University and The Canberra Hospital. However, although I was learning a considerable amount, I never felt I was a truly indispensible member of the team. I was a passenger, a spectator, a sponge. But this was all about to change. I was about to work in an understaffed and under-resourced hospital that was going to need my skills and knowledge. How wrong I was...

Prior to my first day, I talked to some international students who had been working at KCMC for some weeks. After the introductions, I quickly turned the conversation to the hospital and what the experience was like. After I had expressed my enthusiasm to practice as much medicine as possible and maybe make some management decisions, I was laughed at. I also expressed my interest in developing my procedural skills. “You won’t get to do that much,” replied one of the international students quashing my hopes, “you’ll be lucky to take blood.” Indeed, venepuncture was the only procedural skill I practiced on my elective.

My expectations were dented but not crushed. I had travelled over 11000 kilometres to get a unique medical experience and I was determined to get it. However, it only took until the first day of my elective for me to realise why the international students laughed at my enthusiasm. I arrived at morning handover early and I was the only person there. Slowly people began to trickle into the room, and then more people, and then more still. The room was overflowing with doctors, administrators and most of all, students, both local and international. I quickly realised that I was not special, I was not going to be making the decisions and I was in competition with every other student to see and do the interesting things that were difficult to experience at home.

The sheer number of students made day-to-day business cumbersome and time consuming. I usually found myself about three or four rows back in the ward rounds, and sometimes I could not even fit in the room. The ward rounds were usually great learning opportunities, with the senior doctors spending lots of time teaching, but it was difficult to learn when I struggled to see or hear. Once again, I felt like a spectator, except this time, I had a much poorer view.

The language barrier was another major obstacle to learn and practice medicine. Although the local students and staff spoke English, very few patients were able to. I spent a few weeks before my elective learning Swahili from videos on the internet and phrasebooks but this did little to help me in the clinical setting. The only things my simple language skills were capable of doing were to introduce myself and ask permission to perform a clinical examination (this did serve me well as I performed many examinations and elicited some amazing signs that I thought I would never see outside of a textbook). However, history taking, the foundation of any clinical consultation was virtually impossible without an interpreter. Although it was possible to find a local student to help they usually had other things to do, which meant that I did not have time to take comprehensive histories. I felt even more dependent on other members of the team than as a student back home.

The afternoons on the ward were quite different from the action of the morning ward rounds. The wards were almost empty. The few consultants and the plethora of students usually vanished, which left one or two (or sometimes none) of the junior medical staff, a few nurses, and a large number of very sick patients. I usually spent this time going back over interesting patients from the morning round, reading some notes and performing some physical examinations. There were so few local people to act as interpreters (and they were all incredibly busy) that history taking was usually not an option. I often found myself wandering around the ward trying to find something to do or someone to follow, and often giving up and taking an early mark. This lack of structure is by no means unique to my elective experience. It has been identified as a common reason for electives failing to meet the optimal educational opportunities that overseas placements can potentially offer (1).

So with the combination of the large number of students and relatively few senior medical staff, the language barrier, and often a lack of structure, why did I have a unique and fun elective experience? In the end, I still saw some amazing pathology, met some amazing people, experienced a health care system in a developing country and saw some things that I will never forget. It may appear that I am bitter towards my elective experience but this is not the case. I understand that the factors that contributed to a subopti-
mal elective experience were a product of the health system I was in. In retrospect, it would have been wiser to choose an elective at a more remote location, where I may have been more “needed” and afforded more responsibility. I know that in my future years I will have many more opportunities, both in Australia and overseas, to take on extra responsibility and who knows, even make a real difference.

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still wonder what I should do when I grow up. I’ve ended up in Canberra with my wife Robyn, two daughters, Sarah and Julia, and our dog and cat, Rosie and Jack, respectively. And sometimes I wonder how this has happened.

I completed an honours degree in zoology at the University of Queensland in 1982, and set off for London, with the intention of completing a PhD in invertebrate biology. But for various reasons ultimately decided a little way into this course of study that I should follow in my mother’s footsteps and enter the profession of medicine. With time on my hands I worked as a laboratory technician in the in vitro fertilisation laboratory of the Cromwell Private Hospital in London, with the obstetrician, Ian Craft. While I didn’t make it onto the front page of The Times (having left the UK by that time), I was part of the team responsible for Britain’s first “test tube” triplets.

Following this I returned to The University of Queensland for my MBBS, which I completed in 1989 (and before ANU Medical School existed I came to Canberra as a student, for a medicine term at The Royal Canberra and Woden Valley Hospitals in my final year). On completion of my medical degree I left Brisbane for Cairns, with the intention of staying an indeterminate (although I thought probably short) period. Ultimately I stayed for 12 years and still miss the Cairns “winters” during my sub-zero Canberra rides to work. I spent the first five of these in various medical roles including intern, resident and principal house officer (the last in obstetrics and neonatology) at the Cairns Base Hospital, with stints in small hospitals including Babinda, Yarrabah, Weipa, Normanton, Thursday Island and Gordonvale. I also spent time as a medical officer at an Indigenous Community Controlled Health Organisation, Wuchopperen. North Queensland was medically interesting and I learnt a lot. The stark differences in health between Indigenous and non-Indigenous Australians made me think about the health of populations, rather than individuals. There were many examples, but I suppose one that struck me was that it was not uncommon to manage labour in Indigenous women with rheumatic endocarditis, whereas I never saw a case in a non-Indigenous woman. I then completed my PhD (on Ross River virus) and specialist training in Public Health Medicine. I also got married and both my daughters were born at The Cairns Base Hospital, Sarah in 1998 and Julia in 2000.

I returned to Brisbane in 2002 where I lived until early 2008. I spent this period in general practice, mostly half-time. From mid-2002 until 2007, I also provided a clinical service in The Queensland Centre for Intellectual and Developmental Disability (QCIDD), a University of Queensland Medical School Centre located at The Mater Hospital. The centre “aims to improve the health and well-being of the adults with intellectual and developmental disabilities who live in Queensland. This is achieved through clinical practice, education, research and advocacy”. The work was totally new to me, challenging and difficult, particularly from an ethical perspective, and exposed me to the diverse causes of intellectual disability spanning infectious, traumatic, toxic, genetic and chromosomal aetiologies. I learnt about and cared for people with rare syndromes (which I may have heard about in medical school, but mostly forgotten) such as CHARGE, Smith-Magenis and Prader-Willi. As was the case in my work with Indigenous Australians in North Queensland, I was struck by the poor health of my patients relative to other Australians, but also by the contribution of medicine to this poor health status.

The point of writing this is not to offer a dull chronological outline of parts of one’s life, as I’ve done above. Rather it is to tell you what I’ve learned and how my understanding has changed through my experiences.

Life in and out of medicine

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The point of writing this is not to offer a dull chronological outline of parts of one’s life, as I’ve done above. Rather it is to tell you what I’ve learned and how my understanding has changed through my experiences.
My early education in biology has influenced my view of the world, and perhaps has led me often to think about systems (and not just human systems) rather than individuals – perhaps at least part of the reason for my entering public health medicine. My PhD research on Ross River virus, as well as allowing me the chance to travel around to various North Queensland swamps trapping and being bitten by mosquitoes (and trying to stay reasonably clear of the crocodile slides) gave me the opportunity to wonder about the interplay between pathology of the body and mind. The human need for an explanation often leads people to rationalise vague and chronic symptoms as being due to previous Ross River virus infection – and there is an analogy to many other diagnoses. For those of you who have the stamina and the opportunity, a doctorate is a stimulating and rewarding (though also sometimes lonely and soul destroying) experience which will (as a renal physician colleague from Cairns, who’d taken time out from his clinical work for a PhD, told me) teach you how to think.

The time I spent in adult developmental disability medicine at QCIDD also had a profound influence on my thinking. Early on it became obvious to me that my patients with intellectual disability were commonly managed with psychotropic medications as a chemical straightjacket. Through my work at QCIDD a randomised control trial (RCT) of risperidone, haloperidol and placebo for aggression in adults with intellectual disability (the Neuroleptics in the treatment of aggressive challenging behaviour for people with intellectual disabilities (NACHBID) trial) (1) was performed, and I led the Australian arm of the trial. The trial demonstrated no benefit for the antipsychotic medication over placebo (and the latter has, of course, fewer adverse effects and lower cost). The clinical work which led me to the study and associated research made me acutely aware of the power of the medical profession, particularly when treating disempowered groups such as people with intellectual disability. Just how central diagnosis is to medicine was also brought home to me by this work – implicitly, “challenging behaviour” has been accorded the status of a diagnosis in developmental disability medicine. Despite an absence of evidence, antipsychotic medications have been (and undoubtedly still are) used to manage this “diagnosis” to the great detriment of the patients who I cared for at QCIDD. But perhaps the NACHBID trial will go some way to changing this.

I initially saw the work I did on disability as something of a detour, as I’d seen myself as an infectious diseases epidemiologist, or perhaps a public health physician in communicable disease control. So far I’ve never really combined my interests in disability and infectious diseases (other than a patient with intellectual disability who I managed, and who I felt fairly sure, on the basis of history and serology, was disabled as a consequence of Murray Valley encephalitis), and perhaps I never will. But it’s given me variety in my life and career and I’ve learned a lot from it. And I’ve remained interested and involved in infectious diseases epidemiology. I’ve never lost the early fascination I felt for my work on Ross River virus. The biology of vector-borne diseases still amazes me. And my expertise in this area has resulted in collaboration with colleagues in various countries and travel to work with them, hence visits to La Reunion and Paris (Chikungunya, not a virus of chickens but an Alphavirus closely related to Ross River virus), Henan Province in China (a tick-borne Bunyavirus), New Caledonia and Cambodia within the last five years. Via a PhD student, Kazi Rahman, I’m vicariously involved in research on visceral leishmaniasis in Bangladesh, and another student, Yanni Sun, is researching multi-drug resistant tuberculosis in Henan province, China. Vicky Ng, who submitted her PhD this year, worked on Ross River virus and climate, rekindling my interest in this Australian Alphavirus (and teaching me more about kangaroo and wallaby reproductive biology than I ever thought possible).

In my medical life various individuals have influenced my thinking. The clinical skills of many of the consultants with whom I worked in North Queensland were truly inspirational. Among these were the physicians Clive Hadfield and John Thompson and the obstetricians Michael Humphrey and Paul Howat. My PhD supervisor, Adrian Sleigh (now Professor at National Center for Epidemiology and Population Health (NCEPH), but at the time at The Australian Centre for International and Tropical Health and Nutrition at The University of Queensland), originally trained as a physician, inspired my interest in epidemiology and population health and led me through the rigours of my PhD. He demonstrates how clinical knowledge and training can enrich and illuminate research and population health action in global health. A little more recently Ernest Hunter, now regional psychiatrist for Cape York and the Torres Strait, with whom I worked as a public health medicine registrar in 2001-2 provided me with an example of how a clinical role can be combined with population health through his tireless work on alcohol in Indigenous communities. Another psychiatrist whose ideas have recently influenced me, though whom I’ve never met, is Robert Lifton. Lifton’s book “The Nazi Doctors” demonstrates starkly just how fragile civilisation and civility are. And perhaps the central role of doctors in the prison camps should be no surprise given the power and status of the medical profession. Humility is not the only quality required of a great, rather than merely competent, doctor, but it is one of the most important. Lifton’s book demonstrates just how dangerous the opposite of humility, arrogance can be. It also shows, admittedly in a rather extreme case, the extent to which doctors reflect the society in which they live and work.

Sue Cotterell, whose practice I worked in for five years in Bris-

† My only criticism of this remarkable book, which I’d recommend to all ANUMS staff and students, is the rather obvious title (reminding me of the publication in America of Primo Levi’s account of his time in Auschwitz as “Survival in Auschwitz” rather than “If this is a man” – the title used for publication elsewhere).
bane, showed me the best of general practice. Her involvement with families, her hard work and her dedication really made a difference to the health of her practice population. I think through her example and my work in her practice I came to fully appreciate the value of continuity of care in general practice, at least as important, if not more so, than a general practitioner’s knowledge and skills. Nick Lennox, a general practitioner and director of QCIDD, combines clinical medicine with advocacy and world class research in adult developmental disability medicine. Nick provided me with the stimulating environment and freedom that I needed when working with him at QCIDD to develop my research interests in developmental disability medicine.

I’ll close by mentioning another doctor who has been an inspiration to me in the last decade. Helen Beange AM (2), now in her 80s, has worked tirelessly as a clinician and advocate with intellectually disabled people. I’ve had the privilege of working as deputy chair, with Helen as chair, on a working party producing a position paper on disability for the RACP during 2010-11. Her dedication to this task, despite her worsening blindness due to macular degeneration, has amazed me. And she has certainly maintained the rage, reminding me often that disabled people, as well as the Jews in the German prison camps, have suffered and died at the hands of doctors. Doctors have the power to heal, but also to harm, so knowledge and skill, while important, are of little or no value without humility and compassion.

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The role of ethics committees

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Since the atrocities committed by NAZI researchers in the second World War, human research has undergone increased scrutiny and the role of ethics committees in protecting humans has taken on great importance which has been reinforced by the subsequent formulation of human rights declarations (1).

The role of ethics committees is still unquestionably important as rogue, corrupt or overzealous researchers still need to be reigned in (with the recent example of Dr Tonmoy Sharma who repeatedly falsified ethics approval, failed to gain informed consent and failed to declare conflicts of interest). However many researchers will also be familiar with the often trivial rejections of research applications by ethics committees. It is not uncommon, but very frustrating, for articles to be rejected on the basis of formatting or disagreement on statistical methods.

It could be argued that poor formatting shows disrespect and lack of care or attention to detail, unwanted traits which might flow on to poor patient care or data protection in the research process (2), and that inappropriate statistical methods could result in poor study outcomes, thereby unnecessarily involving patients in research. However, it could also be argued that formatting is a matter of opinion and many research groups have their statistical methods designed by professional statisticians.

Rejections of research submissions for trivial reasons are not isolated incidents. Anecdotal and empirical reports suggest difficulties in obtaining ethics approval after the first submission. Sentiments that gaining ethics approval is time consuming and occasionally inconsistent have existed since the 1990s. A British study estimated that each ethics application costs the ethics committee the equivalent of £800 and the researcher £850 as well as an average of 44 man hours (3,4).

Ethics committees are not only rejecting applications on trivial and non-ethical considerations, Australian data from the 1990s suggest that 1.1% of applications were rejected outright (4), while more recent data from the United Kingdom reported that 9% were rejected on ethical grounds (5). It is clear ethics committees are still essential, but it is also apparent that the integrity of an ethics committee may be undermined if it continuously rejects applications on frivolous grounds. It is most valuable for researchers and patients to know that approval for research was given because the ethical considerations in the application are sound, and this should be the focus of the committee.

Ethics committees may feel they also need to monitor non-ethical considerations to justify their existence. But ethics committees need not be concerned with these aspects as the committees themselves are indispensable. When things are going well it feels as though we don’t need them, which makes it seem harder to justify their existence. However, their existence in and of itself may encourage potential researchers to thoroughly consider the ethics of their research pursuits. It is somewhat paradoxical, but the value of an ethics committee may be commensurate with the degree to which they are seen to be superfluous.

REFERENCES

Drug companies pushing the boundaries with direct-to-consumer advertising

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SO WHEN DOES THE AD BREAK BEGIN?

Drug companies are becoming more innovative in the ways they sell products to consumers. Traditionally in Australia, drug companies have targeted doctors by offering them free stationary, medical equipment and paying for conferences in exotic locations. It was up to the doctor to take on this burden, decide whether to accept such gifts and to decide what was best for their patients. These methods are waning with stricter laws recently introduced, but drug companies have also developed methods for getting their product directly to the consumers.

Australia has laws restricting direct-to-consumer advertising of prescription drugs as this may undermine the doctor-patient relationship. However, this hasn’t stopped drug companies from directly reaching consumers as they have become more inventive in their methods. An example of this is the recent Eli Lilly “Ready Anytime” advertisement campaign, which encourages men with erectile problems to see their doctor. The loophole is that the advertisement does not mention Cialis, the Eli Lilly erectile dysfunction medication. Instead, the Eli Lilly logo appears at the end of the commercial.

This is not the only method Eli Lilly have used to directly target consumers. In 2008, Cialis was promoted in a ‘news story’ by National Nine News. The story was ostensibly an informative piece on a medication that can re-introduce spontaneity into the sex lives of men with erection problems. However, it is difficult to believe this when almost all of the footage presented in the story was provided by Eli Lilly (1).

Eli Lilly are not alone in the use of these methods. In 2009 Pfizer ran a similar television advertising campaign targeting men with erection problems and another targeting people who are trying to quit smoking, the message of both adverts being to visit your GP to get “real help”.

One may argue that these advertisements are not bad since they raise awareness of genuine health issues that appropriate medical intervention may improve. For example, if an advertisement encourages men to take action on a problem that has broad ranging negative effects, then is it such an issue that a drug company raises this awareness? Ultimately, the burden comes back to the doctor to deal with the repercussions of drug advertising. So even though direct-to-consumer drug advertising has many negative consequences, these can be nullified by competent patient assessment and management. This should ultimately encourage us to be better clinicians.

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