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- Problems in paradise
- History of plague
- Army Malaria Institute

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Journal of Military and Veterans' Health

Australian Military Medicine Association

STATEMENT OF OBJECTIVES

The Australian Military Association is an independent, professional scientific organisation of health professionals with the objectives of:

- Promoting the study of military medicine
- Bringing together those with an interest in military medicine
- Disseminating knowledge of military medicine
- Publishing and distributing a journal in military medicine
- Promoting research in military medicine

Membership of the Association is open to doctors, dentists, nurses, pharmacists, paramedics and anyone with a professional interest in any of the disciplines of military medicine. The Association is totally independent of the Australian Defence Force.

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Inside this edition

The theme for this Edition is the history of developments in military medicine. As I was preparing for this Edition, I was reminded of some letters that I had from my great-uncle, Captain Gordon O. Robertson, who was the Regimental Medical Officer for the 57th Battalion, A.I.F. in 1918. On the 10th July 1918, he wrote the following to his Commanding Officer:

"The health of the men in the Battalion is fairly good at present except for this epidemic of influenza which has left all of those affected with it very weak. This combined with the continue strain of line work is gradually undermining their health.

I would advise that the Battalion, if possible, should be given shorter spells in the line in order to give the men every chance by rest and cleanliness to keep in health. If they are kept in the line for long spells now there is every reason to expect large evacuations with sickness and also a large number of evacuations with nervous instability if they have to undergo a bombardment"

Despite the passing of nearly 94 years, our military health staff continue to manage the challenges of infectious diseases, mental health issues and communication with senior officers. As many have postulated before, we ignore the lessons of history at our peril.

We are fortunate to have three excellent new, and one reprinted, historical military medical articles on the First World War ("Problems in Paradise"), Second World War ("Pioneer Aviation and a Medical Legacy"), and the challenges of infectious diseases ("History of plague", "Australian Malaria Institute") in this Edition. These are all introduced with a guest Editorial by Dr Keith Horsley. In addition, we have a range of new articles on methods for cooling during operations, perspectives on traumatic limb amputations and new Naval medical capabilities.

I am pleased to see more and more articles being submitted to the Journal and I would encourage all our readers to consider submitting an article. We have recently been indexed in Scopus and CINAHL and continue to work towards indexing in PubMed, which should make the Journal more attractive for our authors. Our next themed issues are on 'Mental Health' (July 2012) and 'Veterans' Health' (October 2012), so if you have relevant papers, please get them in soon. All other articles are also welcome.

Dr Andy Robertson

Editor-in-Chief

President's message

Welcome to the ANZAC edition of JMVH and, as we reflect on what ANZAC day means to us and the nation as a whole, spare a thought for those serving members on deployment overseas.

I, as were many within the health community, was saddened to hear that Rear Admiral Graeme Shirtley AM has passed away after 3 years fighting cancer in early April. I know many of you were there at his funeral as a mark of respect. Graeme was a former patron and strong supporter of the Association; he attended all our conferences and could be relied on to give some forthright opinions. He often said that it was amazing what a person could get away with when you don't have a frontal lobe.

As a mark of AMMA's deep appreciation of Graeme's support over the years, there will be a Graeme Shirtley oration starting at this year's AMMA conference; he will be sadly missed.

The AMMA conference organisation continues with some exiting keynote speakers and this year we have Dame Carol Black (Rehabilitation), Colonel Bob Hale US Army (Head and Neck Trauma) and, Professor Michael Reade (The inaugural chair of Military Medicine and Surgery) presenting. The new convention facility at the Brisbane Convention and Exhibition Centre has been dusted off and is ready to provide us with a memorable three days in October. This year we are also trying to "value add" for members and delegates by conducting workshops prior to the conference. The conference website will have update information on these and the program.

Recently many would be aware that the ADF Health Journal has ceased publication, however our Journal remains a fundamental function of the Association and the committee is committed to its publication and improvement. With this in mind we are looking to fill the space that ADF Health held.

Looking forward to catching up with you all at the 21st AMMA Conference.

Greg Mahoney

President

History Lessons Learned

Keith Horsley, Guest Editor

The theme of this edition of JMVH is historical; the raison d'être for such an edition is the belief that there are lessons for today from the experiences of yesterday.

This view has not been universally accepted throughout history. The American car-maker and entrepreneur Henry Ford was one who did not see the study of history as useful, famously noting that "history is all bunk".

This journal is one that would disagree, and disagree strenuously, from Ford's view. The study of the history of military and veterans' health reveals that there are common themes that occur again and again, and that we ignore them at our peril.

A few examples can provide substance to this argument.

Any survey of military history reveals that infectious disease is a vital element to any military campaign. From the defeat of Napoleon's Grande Armee in Russia in 1812 (defeated more by typhus than by Alexander or the Russian Winter), the loss of life meticulously documented by Florence Nightingale in the Crimean War (where she showed that poor hygiene killed many more than Russian bullets did) and the horrendous toll that malaria took in the Japanese Imperial Army of World War Two, there are many examples of infectious disease being a vital factor in military conflicts. We learn this truth from history.

Similarly, we know from history that there are many examples of the malicious environment of war giving rise to post-conflict syndromes. These range from da Costa's syndrome after the American Civil War, the Shell Shock of World War One, Combat Fatigue of World War Two and PTSD following the Vietnam War. Other examples abound; they are not countless, but their number is very large. A study of history also shows a predilection to assigning physical causes to these syndromes. A concern here is that our current interest in Minimal Brain Injury might not be, at least in part, a further re-iteration of this recurring pattern from history. As with infectious disease, we gain wisdom from our study of history in this area.

Since the introduction of gunpowder into war some half a millennium ago, war has become a noisy place. Since that time, we have noted that military personnel and veterans share a loss of hearing. Indeed, the characteristic pattern of audiometric loss following noise exposure is now known as "the gunner's notch". Napoleon himself, a former artillery man before he was an Emperor, complained that the church bells no longer sounded the same – he could not hear the high notes. Our study of history shows us that sensori-neural hearing loss is a near-universal feature of being in combat.

We have also seen again and again from history that the provision of surgical care for the wounded as soon as is possible after injury greatly reduces mortality and morbidity. Again and again history shows us that forward care, rapid evacuation of the wounded and the provision of effective first-aid is vital in the treatment of the war injured.

"History is all bunk" said Ford. "Ford is all bunk" says History.

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Problems in paradise: medical aspects of the New Zealand occupation of Western Samoa, 1914 -1918

Michael Tyquin PhD, EB (Hons), BEc

Abstract

Western Samoa was an imperial German possession until occupied in August 1914 by 1,500 New Zealand troops. The force, which landed unopposed, was accompanied by almost 90 health staff of the New Zealand Army Medical Corps. They worked to ensure the health of two relief forces from 1914 to 1918. The German health administration they inherited became an added burden with respect to civilian health and sanitation matters on the island. Many health challenges and problems were faced by the Corps during its deployment in Western Samoa.

Keywords: Western Samoa, New Zealand Expeditionary Force, World War One, Spanish Influenza

Before looking at the medical problems faced by the New Zealand garrison through its long occupation of Western Samoa from 1914 to 1918, it is necessary to briefly examine the reasons for its being sent there in the first place

On the evening of Thursday, 6 August 1914, the New Zealand Governor received a cable from the Secretary of State in London. It was believed the Dominion Government could make a valuable contribution to the war if its forces seized the German garrison and its wireless station at Apia, Western Samoa.

Almost immediately men were enrolled and a force was assembled by 11 August. Numbering almost 1,500 troops, it consisted of the 5th (Wellington) Regiment and its band, the 6th (Auckland) Regiment, an artillery battery, a company of New Zealand Field Engineers, a section of signallers, men of the New Zealand Railway Engineers and small groups from the Army Service Corps, a Post & Telegraphic detachment and an 11-man contingent from Fiji. The force was accompanied by 72 officers and men of the New Zealand Medical Corps, together with a matron and six nurses, two dentists, a 'compounder' (or pharmacist's assistant) and a bugler.[1]

The first the medical staff in New Zealand knew anything about an expedition was on Friday 7 August 1914, when it received a request for one section of a field ambulance to accompany an advance party. Instructions were then issued to the corps to supply a unit which could act both as a small base hospital and as a field ambulance. Medical supplies included 12 lbs. chloroform, 2 lbs. boric acid, 2 lbs. petroleum jelly, 15 yards of waterproof sheeting, 300 first field dressings, 1,000 safety pins; and a quantity of the ubiquitous Condy's crystals.

The troops were crammed on board two dirty and badly ventilated transport ships - the *Moeraki* and the *Monowai.* Accompanied by three light cruisers, the convoy left Wellington on Saturday 15 and proceeded towards New Caledonia.

There was little in the way of acclimatising the soldiers who had left a New Zealand winter for the tropics. En route the soldiers were very sea-sick, but apart from a successful appendectomy, intense training and lectures, the voyage was otherwise uneventful. At Noumea the French heavy cruiser *Montcalm* and the cruisers *HMAS Australia* and *HMAS Melbourne* joined the convoy as escorts. Shortly thereafter it left for Fiji, where the chief medical officer gave his New Zealand colleagues a briefing on tropical diseases.

Despite rumours of a German fleet in the vicinity, the Occupation Force landed unopposed on the morning of 29 August.

The rather sedate landing was a far cry from Gallipoli. A boat put ashore with a white flag and the terms of surrender were accepted by the German Governor's deputy. On Sunday 30 the British Union Jack was hoisted formally at the Apia court house.

Almost immediately after the proclamation was read the Samoan officials were reinstated in their former positions and German administrators were allowed, for the time being, to remain in office. A bungalow on the waterfront, owned by the main German firm in Apia, was occupied as a make-shift temporary hospital by the field ambulance and centres for sick

Figure 1. Apia Hospital, with some NZ military patients Courtesy Alexander Turnbull Library, Wellington, New Zealand

Figure 2. Members of the NZ Force landing in Apia harbour, 29 August 1914 Courtesy Alexander Turnbull Library, Wellington, New Zealand

parades were established elsewhere. The remainder of the Medical Corps camped at the race course. Medical orders soon became frequent and warnings were issued against 'drinking unboiled water, against sustaining sunstroke and sunburn, bathing more than twice daily and against walking bare foot.

The Apia Hospital, originally the gift of a German philanthropist, was commandeered almost immediately. It was a large, airy bungalow building, the cool wards and living quarters opening onto wide verandahs. However the Principal Medical Officer (PMO) soon reported adversely on its operating theatre which had insufficient light during periods of cloudy weather.

Medical officers, nursing sisters and orderlies of the NZMC were busy with both military and civilian patients at the hospital.

Barely a month passed when, on Friday 11 September, the German Chief Health Officer informed Lieutenant Colonel Matthew Holmes, the PMO, that his entire hospital staff of 15 had decided to cease work and that they would vacate the hospital next morning. Holmes therefore had 16 hours notice 'to take on the whole of the civil as well as the military medical work' in Apia and surrounding districts.[2] This placed a considerable additional load on the army medical and nursing staff until a German doctor (an officer in the Imperial German Naval Reserve) was authorised to deal with plantation workers and the Chinese community of indentured labourers.

Issues

There were accidents normally associated with garrison life. Some were fatal, such as the death of a nurse from meningitis, an accidental shooting and a soldier who fell from a hotel balcony and died of a fractured skull. There were also the 'occupational' hazards of soldiering in the tropics. In January 1915 there was an epidemic of ringworm and, at the end of that year, many men presented with the irritating complaint, 'dhobie itch' (or *Tinea cruris*). There was also a constant stream of soldiers who presented with sores resulting from mosquito bites and skin abrasions, which, unless treated early, became septic and difficult to cure.[2]

An indication of the hospital work is provided by a report for the period 15 January to 1 February 1915 when an average of 22 patients were seen daily. During the month there were two cases of appendicitis (one surgical), five cases of hernia, one of varicocele and one of varicose leg veins, which were treated to render the patients fit for active service. Ongoing medical problems for which soldiers were treated included synovitis, 'septic eye', filariasis, 'debility', dysentery, 'ear trouble' and soft tissue injuries.[2] This profile changed according to season.

In the wet periods medical officers reported an increase in the incidence of 'catarrhal jaundice, "colds" and rheumatism.' The increasing demand on the medical service was offset by a reduction in infectious eye diseases during the cooler months (June and July) due to the smaller fly population. The garrison had succumbed to eye problems almost immediately after landing at Apia. There was a high rate of an extremely contagious disease, described by a contemporary authority as being caused by 'a special microbe, resembling in its effects *Gonorrhea opthalmia*, the offending carrier being the fly, which soon became a dreadful pest.' This condition responded well to the local application of silver nitrate. There were more than 100 cases of ophthalmia among soldiers in 1915.

Ear problems among the troops was a direct result of swimming in the sea and the local rivers. These conditions were almost always an infection of the ear canal. Consequently medical officers tried to restrict bathing, but with little success. The commonest form of ear complaint with which soldiers presented was

reported to be meatitis which 'caused such intense pain that in many instances the patient was totally unfit for duty.' In addition, numerous cases were caused by insect bites and not infrequently insects had to be washed out of ears with syringing.

It is interesting that medical diaries observe that many of the younger men of the garrison were 'breaking down'. Between 18 December 1914 and 14 January 1915, 14 men had been returned medically unfit to New Zealand. It was hoped that the relief force, which consisted of older men, would be more resilient. Less than two months later the transport *Moeraki* left Apia with a considerable number of men who were showing 'signs of being unfavourably influenced by the climate.' These men were used to supplement the home force. By late October 1916, doctors recommended that troops do no work between 10 am and 3 pm, but this regulation was not always observed.

Alcoholism

Alcoholism has long been associated with the tedium of garrison duty. In a private letter the PMO wrote to the Director of Medical Services in New Zealand, Surgeon General R.S.F. Henderson and admitted that his attempt to place the island's hotels 'out of bounds' had been unsuccessful. He advised that: 'There is about 25% of the men who are boozers & they have created a very bad impression among the Samoans…'

Alcoholism was not confined to the ranks. A civilian doctor brought from New Zealand on contract as a Government medical officer had arrived (with one other doctor) on 17 January 1916. Less than a month later the Provost Marshal in Samoa wrote letters to all the publicans in Apria forbidding them to supply the medico with intoxicating liquor of any kind.

Disease

The garrison had its first case of dengue fever within a month of landing in Samoa. Subsequently there were sporadic outbreaks in the following year, particularly in April and May when the fever was reported to be more severe. The analgesic and antipyretic properties of aspirin were most effective, while quinine was a good tonic during convalescence. The only prophylactic measure available was to take precautions to avoid mosquito bite.

In June 1915 the whole force was inoculated for the second time against typhoid as two cases had been identified. It was then decided to inoculate a third time, due to the success reported by the United States military of giving a course of three injections.

The first case of dysentery (caused by *Shigella*) occurred on 6 September and debilitated many soldiers throughout the following month. The average stay in hospital was about 10 days and, in the absence of adequate supplies of suitable foods, the outbreak had to be dealt with by the supply of arrowroot from the field ambulance. At the various posts small temporary clinics were established by the Medical Corps. These had a supply of drugs and dressings and sick parades were held twice daily, under the supervision of a medical officer. Arrangements were made so that temporary and minor cases of sickness could be admitted locally to avoid overcrowding the base hospital.

The main disease vectors in Western Samoa at that time were mosquitoes (dengue fever and filariasis), flies (typhoid, opthalmia, framboesia) and contaminated water (typhoid and dysentery). Typhoid (*Salmonella typhi*) was endemic to parts of the main island of Western Samoa.

Dental problems

Not long into the occupation, a total of 439 garrison troops presented over a three week period in 1914 with dental problems ranging from salivary calculus to periostitis. Steady progress was made repairing the ravages of decay in their teeth, with a consequent marked improvement in general health.

From the force's mobilisation in August 1914 to January 1915, the total number of attendances for dental treatment was 1,719. Thirty per cent required 'extreme work'. 'A large number of badly decayed teeth (1,204) have been extracted, practically all being done with local anaesthetic and 735 fillings have been inserted.' The PMO noted that the condition of a number of men's teeth was so bad that they had to be repatriated to New Zealand.

It is noteworthy that the authorities included dentists among those who deployed, a lesson not learned by the Australian army until early 1916.

Public Health and Sanitation

The occupation saw many lost opportunities, due mainly to the lack of resources. There was a dearth of basic equipment, including pumps for mosquito spraying (which had to be done by means of watering cans). Regulations were periodically promulgated forbidding 'the dumping of rubbish on the foreshore' and to clear land of 'tins, coconut shells, or other receptacles that might catch and retain water.'

Throughout August 1915 there was a conscious effort on the part of the medical authorities to contribute to public health. The fences around the Market Hall were moved to provide more ground space and two

large shelter trees were transplanted in front of the market. Particular attention was also given to latrines which were constructed on a pan system and designed to prevent flies becoming a nuisance. Several improvements were made to the Government Prison as well as repairs to the hospital's water supply and drainage.

Despite these measures, the battle to institute public hygiene measures was not going well, for by mid 1916 the PMO wrote to the Commander-in-Chief in Samoa (Colonel Robert Logan): 'It is a matter of urgency that something should be done soon so that we can get a start up to improve the sanitary conditions.' He suggested that Fiji's health regulations should be adopted in their entirety 'for consideration at the termination of the Occupation...'[2]

Among the civilian population, the most common problems were cataracts and the 'follicular' variety of conjunctivitis. Medical officers discovered a novel method of treating the 'so-called Samoa Trachoma' which consisted of dusting the infected area with subchloride of mercury. This treatment was 'inexpensive and painless.' Among children yaws (or *framboesia*) was especially predominant, while, in the general population filariasis and, to a much lesser extent, Hansen's Disease were also present.

Climate and poor sanitary conditions together made life difficult for the medical staff. In June 1916 the PMO reported that sanitary facilities at the various army establishments on the island were a disgrace, particularly with regard to cooking and messing arrangements. He similarly condemned the Quartermaster's store three weeks later. The floor underneath the building was so filled with rubbish preventing ventilation that temperature and humidity in the building were excessively high, leading to large scale wastage. Among a large number of examples cited were the destruction of 625 lbs potatoes and 2,296 lbs tinned fruit in just two months.

Diet

The climate played havoc with bulk food and large quantities became infested with weevils or went mouldy. It was soon discovered that rice did not keep well in bags and arrangements were made to have it shipped to the garrison in tins. Twelve months later it was found that bread was also causing digestive troubles and Australian flour, which had better keeping qualities, was substituted for the New Zealand flour then in use at the garrison. Ironically, there were shortages of fruit and vegetables because the Samoan subsistence economy still had not caught up with the greatly increased demands.

Medical officers unanimously agreed that the standard ration provided the calorie requirements of the force, but that it was bland and monotonous. They suggested the inclusion of curries and pawpaw, together with beer and mineral water in the messes.

As the small Apia freezer and ice works could not cope with demand it could not provide sufficient storage for the garrison's frozen meat (from New Zealand) and much of it went bad. Local supplies weren't favourably viewed either. Of a sample of three carcasses tested in 1916, two were 'condemned and burnt', while a third was described as 'tubercular.'

Dress

Upon their arrival in Western Samoa soldiers wore the standard New Zealand army issue of heavy woollen uniforms.

Not surprisingly, local stores did a 'roaring' trade in light singlets and shirts, while every possible article of heavy army apparel was discarded. Shorts became shorter and shorter until orders were issued to regulate their length. With hard use and rough marching, army boots became a problem and the lack of suitable equipment soon became a pressing logistics issue. In addition, the dye in army issue shirts was found to be defective. Two pairs of boots and one uniform per man were not sufficient to stand up to the strain.

Medical Routine

The training of all members of the Medical Corps continued throughout the occupation. Routine sick parades were discontinued within months of the force landing, to allow more time for the training of the ambulance unit. It now had company drill 'four mornings a week, lectures on three mornings a week; and practical work five afternoons a week.' The nursing orderlies in the hospital rotated with medical staff in the field, so that 'every member of the unit had personal experience in nursing duties before leaving Samoa.'[2]

Psychological Problems

The routine and tedium of garrison life, even in beautiful surrounds, eventually told on the health and state of the troops stationed there throughout the occupation. This picture was drawn by one who was there:

'The weather had broken in September and for days the rain fell in torrential downpour. The rivers rose and the low-lying flats on which the racecourse camp was situated became a sea of mud, which added to the discomfort of the already harassed guards and picquets and swamped the tents and surroundings. With no bunks to keep the men off the soaking

ground, the poor quality of the food, the pests and ever-present mosquito, which here abounded, the detachments for return to New Zealand daily grew in numbers.'[1]

By the middle of 1916, the PMO's diaries reflect a certain bewilderment about ongoing illness among the diminishing garrison. He noted:

'There is no serious sickness in the Garrison but for the size of it there are a lot of minor ailments which it appears should not exist. On looking for the cause of this it seems to me to be in a great measure due to the "mental unrest" of many of the Garrison who after about 18 months residence in Samoa find life very irksome and monotonous, the younger ones expressing a keen desire for a more active life.'[4]

They were also eager to join their colleagues at home in preparing to set off for the Western Front in France. Indeed many of them did find their way to the Somme to join the general slaughter there.

Apart from continual training and route marches, amusements were few and consisted in swimming, boxing, blind-fold boxing and cockfighting. This routine was rarely interrupted. However, on 14 September 1914 the German heavy cruisers Scharnhorst and Gneisenau appeared in Apia harbour at day-break and an attack was momentarily expected. A mild panic ensued and several hospital patients effected miraculous recoveries. But, on seeing the Union Jack flying on the Government buildings, the German ships slowly turned about and left without firing a shot. The German intent had been to surprise allied warships in Apia harbour. But all these vessels had since left the area. Also, shelling the largely undefended capital would have been a needless waste of ammunition . So the two enemy ships sailed off.

By 15 January 1918, 77 men had served in Samoa non-stop for three years. Doctors had long recommended rotating tours and relief after long service, but such a system was introduced only in May 1918. Even then it applied to only small groups of men, so that it was not until September that all the 'old timers' had been sent home.

The Spanish 'flu

Throughout the occupation there were minor epidemics of measles and colds whose effects were more severe among the civilian, rather than the AngloCeltic garrison population. However these epidemics were brought to the island by New Zealand troops. Measles, particularly, was characterised as a 'raging epidemic, with children being worst affected.' There were apparently some deaths, although the number is not recorded. But the most notorious outbreak the authorities had to deal with (unsuccessfully) was the influenza pandemic of 1918.

On 7 November 1918, the transport *Talune* which had left New Zealand via Fiji, moored at Apia without quarantine restrictions. Several people on board were suffering from influenza and some later died. At the time influenza was a notifiable illness, yet the New Zealand authorities failed to notify its administration in Apia of the progress of the disease. The New Zealand Administrator, Colonel Robert Logan, also refused offers of assistance from nearby American Samoa. As the PMO later recorded in his diary: '...it was not until we opened the papers received by the mail brought by the *Talune* that we learnt there had been an outbreak of a serious nature in Auckland and Suva. The disease spread with alarming rapidity, all the barracks were infected within a week, ninety per cent of the men being affected.' Shortly after, he wrote that since his last report 'we have experienced a severe epidemic of influenza, which unfortunately has proved fatal to seven men of the Garrison.'[3]

However there was no mention that 22% percent of Western Samoa's population (almost 9,000 victims, aged mainly between 18 and 35) perished in a few months.[5] The huge mortality rate was hard to explain to angry Samoans, as American Samoa, a little over 100 kilometres to the east, escaped the pandemic unscathed because of standard quarantine procedures which were stringently policed by the United States Navy.

As noted this was the year in which the influenza epidemic, commonly known as "Spanish 'flu" swept the world. On 19 November the military governor in the capital of Apia telegraphed Wellington for help, but his request was refused on the grounds that all doctors were needed in New Zealand. Australia offered the only alternative source of aid. In response, on the following day, the Australian Commonwealth Naval Board began forming a joint relief expedition from available military and naval medical personnel under Surgeon Lieutenant Frank Temple Grey.

HMAS *Encounter* was ordered to deploy almost immediately from Sydney with a cargo of medical and humanitarian supplies. Commanded by Captain Hugh Thring, she departed on 24 November 1918, ten minutes after completing loading.[6] As a precaution, all 450 members of *Encounter's* crew were doubly inoculated as the ship had suffered 74 cases earlier in the year at Fremantle. *Encounter* arrived in Suva on 30 November and took on coal and 39 tonnes of water. Later that day she departed and arrived off Apia (where there were now 50 deaths

reported a day) on 3 December. Within a few hours, six surgeons, 18 medical orderlies and three naval sick berth ratings and their stores were ashore.[7] This deployment is regarded as Australia's first overseas relief expedition.

Conclusion

It is important to remember that unlike the Australian Army Medical Service, the New Zealand Medical Corps was formed only in 1908, barely six years before it was deployed in Western Samoa. Nevertheless, the garrison duty there gave its members unusual opportunities for training and administering to the medical needs of both its soldiers and to a wider civilian population.

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The History of Plague – Part 1. The Three Great Pandemics

John Frith

Plague is an acute infectious disease caused by the bacillus *Yersinia pestis* and is still endemic in indigenous rodent populations of South and North America, Africa and Central Asia. In epidemics plague is transmitted to humans by the bite of the Oriental or Indian rat flea and the human flea. The primary hosts of the fleas are the black urban rat and the brown sewer rat. Plague is also transmissible person to person when in its pneumonic form. *Yersinia pestis* is a very pathogenic organism to both humans and animals and before antibiotics had a very high mortality rate. Bubonic plague also has military significance and is listed by the Centers for Disease Control and Prevention as a Category A bioterrorism agent.[1]

There have been three great world pandemics of plague recorded, in 541, 1347, and 1894 CE, each time causing devastating mortality of people and animals across nations and continents. On more than one occasion plague irrevocably changed the social and economic fabric of society.

In most human plague epidemics, infection initially took the form of large purulent abscesses of lymph nodes, the bubo (L. bubo = 'groin', Gr. boubon = 'swelling in the groin'), this was bubonic plague. When bacteraemia followed, it caused haemorrhaging and necrosis of the skin rapidly followed by septicaemic shock and death, *septicaemic* plague. If the disease spread to the lung through the blood, it caused an invariably fatal pneumonia, pneumonic plague, and in that form plague was directly transmissible from person to person.

The three great plague pandemics had different geographic origins and paths of spread. The Justinian Plague of 541 started in central Africa and spread to Egypt and the Mediterranean. The Black Death of 1347 originated in Asia and spread to the Crimea then Europe and Russia. The third pandemic, that of 1894, originated in Yunnan, China, and spread to Hong Kong and India, then to the rest of the world.[2]

The causative organism, Yersinia pestis, was not discovered until the 1894 pandemic and was discovered in Hong Kong by a French Pastorien bacteriologist, Alexandre Yersin. Four years later in 1898 his successor, Paul-Louis Simond, a fellow Pastorien and a French naval doctor, demonstrated that the Oriental rat flea was the vector for the bacillus and the sources of the bacillus were the sewer rats.[3,4]

The germ, the rats, and the fleas

Yersinia pestis is most likely a clone that evolved some 1,500 to 20,000 years ago from a common ancestor with *Yersinia pseudotuberculosis*, an animal enteropathogen which does not cause blood infection or plague. The bacillus probably arose in Asia, although it had been present in east-central Africa for the past two millennia and probably longer.[2, 5] There are three biovars of the bacillus based on phenotypic differences, Antiqua, Medievalis, and Orientalis, and it has been postulated that they were responsible for the first, second and third pandemics respectively. A paleomicrobiological analysis by Drancourt et al in 2004 however indicated that all three pandemics were most likely caused by the Orientalis biovar.[6, 7]

There had also been debate for some time whether the Justinian and the Black Death plagues were actually due to *Yersinia pestis* and were not instead epidemic haemorrhagic fevers[3,8]. Analyses by Drancourt and others have found evidence of the plague bacillus in specimens of persons known to have died in the first two pandemics. It is also very difficult to discount the vivid and accurate descriptions of the bubonic plague that were given by historians such as Procopius of Caesarea, Gabriel de Mussis and Giovanni Boccaccio.

The vector host in the Black Death pandemic was *Rattus rattus* - the 'old English' or black rat. The black rat originated in India and migrated along the Silk Road from Asia to the Middle East, and then travelled in ships on the sea trade routes, settling in most areas of the world and cohabitating comfortably with humans in their houses, factories, and dockyards. The black rat had probably acquired the disease from contact with infected wild rodents from the steppes of Central Asia and Russia, and in fact the disease may have been transported in infested marmot hides taken by Tartar hunters from Manchuria. The brown or sewer rat, Rattus norvegicus, didn't become prevalent in the Western world until the 18th century. It originated in Central

Asia and migrated westward on the sea routes from China and India. The brown rat flourished in Europe where there were open sewers and ample breeding grounds and food and in the 18th and 19th centuries replaced the black rat as the main disease host.[4, 9]

The primary vectors for transmission of the disease from rats to humans were the Oriental or Indian rat flea, *Xenopsylla cheopis*, and the Northern or European rat flea, *Nosopsyllus fasciatus*. The human flea, *Pulex irritans*, and the dog and cat fleas, *Ctenocephalides canis* and *felis,* were secondary vectors. In the pandemics, the infected fleas were able to spread the plague over long distances as they were carried by rats and by humans travelling along trade routes at sea and overland, and also by infesting rice and wheat grain, clothing, and trade merchandise. When infected, the proventriculus of the flea becomes blocked by a mass of bacteria. The flea continues to feed, biting with increasing frequency and agitation, and in an attempt to relieve the obstruction the flea regurgitates the accumulated blood together with a mass of *Yersinia pestis* bacilli directly into the bloodstream of the host. The fleas multiply prolifically on their host and when the host dies they leave immediately, infesting new hosts and thus creating the foundations for an epidemic.[10, 11]

The Justinian Plague of 541-544

The first great pandemic of bubonic plague where people were recorded as suffering from the characteristic buboes and septicaemia was the *Justinian Plague* of 541 CE, named after Justinian I, the Roman emperor of the Byzantine Empire at the time. The epidemic originated in Ethiopia in Africa and spread to Pelusium in Egypt in 540. It then spread west to Alexandria and east to Gaza, Jerusalem and Antioch, then was carried on ships on the sea trading routes to both sides of the Mediterranean, arriving in Constantinople (now Istanbul) in the autumn of 541.[12, 13]

The Byzantine court historian, Procopius of Caesarea, in his work History of the Wars, described people with fever, delirium and buboes He wrote that the epidemic was one 'by which the whole human race came near to be annihilated'. Procopius wrote of the symptoms of the disease :

> " ... with the majority it came about that they were seized by the disease without becoming aware of what was coming either through a waking vision or a dream. ... They had a sudden fever, some when just roused from sleep, others while walking about, and others while otherwise engaged, without any regard to what they were doing. And the body showed no change from its previous colour, nor was it hot as might be expected when attacked by a fever, nor indeed did any inflammation set in, but the fever was of such a languid sort from its commencement and up till evening that neither to the sick themselves nor to a physician who touched them would it afford any suspicion of danger. It was natural, therefore, that not one of those who had contracted the disease expected to die from it. But on the same day in some cases, in others on the following day, and in the rest not many days later, a bubonic swelling developed; and this took place not only in the particular part of the body which is called boubon, that is, "below the abdomen," but also inside the armpit, and in some cases also beside the ears, and at different points on the thighs."[14]

The focus of the Justinian pandemic was Constantinople, reaching a peak in the spring of 542 with 5,000 deaths per day in the city, although some estimates vary to 10,000 per day, and it went on to kill over a third of the city's population. Victims were too numerous to be buried and were stacked high in the city's churches and city wall towers, their Christian doctrine preventing their disposal by cremation. Over the next three years plague raged through Italy, southern France, the Rhine valley and Iberia. The disease spread as far north as Denmark and west to Ireland, then further to Africa, the Middle East and Asia Minor. Between the years 542 and 546 epidemics in Asia, Africa and Europe killed nearly 100 million people.[15, 16]

The pandemic had a drastic effect and permanently changed the social fabric of the Western world. It contributed to the demise of Justinian's reign. Food production was severely disrupted and an eight year famine followed. The agrarian system of the empire was restructured to eventually become the three field feudal system. The social and economic disruption caused by the pandemic marked the end of Roman rule and led to the birth of culturally distinctive societal groups that later formed the nations of medieval Europe.[12]

Further major outbreaks occurred throughout Europe and the Middle East over the next 200 years - in Constantinople in the years 573, 600, 698 and 747, in Iraq, Egypt and Syria in the years 669, 683, 698, 713, 732 and 750 and Mesopotamia in 686 and 704. In 664 plague laid waste to Ireland,

and in England it came to be known as the Plague of Cadwaladyr's Time, after a Welsh king who contracted plague but survived it in 682. The plague continued in intermittent cycles in Europe into the mid-8th century and did not re-emerge as a major epidemic until the 14th century.

The 'Black Death' of Europe in 1347 to 1352

The Black Death of 1347 was the first major European outbreak of the second great plague pandemic that occurred over the 14th to 18th centuries. In 1346 it was known in the European seaports that a plague epidemic was present in the East. In 1347 the plague was brought to the Crimea from Asia Minor by the Tartar armies of Khan Janibeg, who had laid siege to the town of Kaffa (now Feodosya in Ukraine), a Genoese trading town on the shores of the Black Sea. The siege of the Tartars was unsuccessful and before they left, from a description by Gabriel de Mussis from Piacenza, in revenge they catapulted over the walls of Kaffa corpses of people who had died from the Black Death. In panic the Genoese traders fled in galleys with 'sickness clinging to their bones' to Constantinople and across the Mediterranean to Messina, Sicily, where the great pandemic of Europe started. By 1348 it had reached Marseille, Paris and Germany, then Spain, England and Norway in 1349, and eastern Europe in 1350. The Tartars left Kaffa and carried the plague away with them spreading it further to Russia and India.[17]

A description of symptoms of the plague was given by Giovanni Boccaccio in 1348 in his book Decameron, a set of tales of a group of Florentines who secluded themselves in the country to escape the plague :

> ".. in men and women alike it first betrayed itself by the emergence of certain tumours in the groin or armpits. Some of which grew as large as a common apple, others as an egg, some more, some less, which the common folk called gavocciolo. From the two said parts of the body this deadly gavocciolo soon began to propagate and spread itself in all directions indifferently; after which the form of the malady began to change, black spots or livid making their appearance in many cases on the arm or the thigh or elsewhere,.."[17]

The term "Black Death" was not used until much later in history and in 1347 was simply known as "*the pestilence*" or "*pestilentia*", and there are various explanations of the origin of the term. Butler [11] states the term refers to the haemorrhagic purpura and ischaemic gangrene of the limbs that sometimes ensued from the septicaemia. Ziegler[17] states it derives from the translation of the Latin *pestis atra* or *atra mors*, 'atra' meaning 'terrible' or 'dreadful', the connotation of which was 'black', and 'mors' meaning 'death', and so 'atra mors' was translated as meaning 'black death'.

The social impacts of the Black Death in Europe during the 14th century

The overall mortality rate varied from city to city, but in places such as Florence as observed by Boccaccio up to half the population died, the Italians calling the epidemic the *mortalega grande,* 'the great mortality'. [18] People died with such rapidity that proper burial or cremation could not occur, corpses were thrown into large pits and putrefying bodies lay in their homes and in the streets. People were as much afraid they would suffer a spiritual death as they were a physical death since there were no clergy to perform burial rites:

> "Shrift there was none; churches and chapels were open, but neither priest nor penitents entered – all went to the charnelhouse. The sexton and the physician were cast into the same deep and wide grave; the testator and his heirs and executors were hurled from the same cart into the same hole together."[18]

Transmission of the disease was thought to be by miasmas, disease carrying vapours emanating from corpses and putrescent matter or from the breath of an infected or sick person. Others thought the Black Death was punishment from God for their sins and immoral behaviour, or was due to astrological and natural phenomena such as earthquakes, comets, and conjunctions of the planets. People turned to patron saints such as St Roch and St Sebastian or to the Virgin Mary, or joined processions of flagellants whipping themselves with nail embedded scourges and incanting hymns and prayers as they passed from town to town.[17, 19, 20]

> "When the flagellants – they were also called cross brethren and cross bearers – entered a town, a borough or a village in a procession their entry was accompanied by the pealing of bells, singing, and a huge crowd of people. As they always marched two abreast, the procession of the numerous penitents reached farther than the eye could see." [20]

The only remedies were inhalation of aromatic vapours from flowers and herbs such as rose,

theriaca, aloe, thyme and camphor. Soon there was a shortage of doctors which led to a proliferation of quacks selling useless cures and amulets and other adornments that claimed to offer magical protection.

In this second pandemic, plague again caused great social and economic upheaval. Often whole families were wiped out and villages abandoned. Crops could not be harvested, travelling and trade became curtailed, and food and manufactured goods became short. As there was a shortage of labour, surviving villager labourers, the 'villeins', extorted exorbitant wages from the remaining aristocratic landowners. The villeins prospered and acquired land and property. The plague broke down the normal divisions between the upper and lower classes and led to the emergence of a new middle class.[17, 9] The plague lead to a preoccupation with death as evident from macabre artworks such as the '*Triumph of Death*' by Pieter Breughel the Elder in 1562, which depicted in a panoramic landscape armies of skeletons killing people of all social orders from peasants to kings and cardinals in a variety of macabre and cruel ways.

In the period 1347 to 1350 the Black Death killed a quarter of the population in Europe, over 25 million people, and another 25 million in Asia and Africa.[15] Mortality was even higher in cities such as Florence, Venice and Paris where more than half succumbed to the plague. A second major epidemic occurred in 1361, the *pestis secunda*, in which 10 to 20% of Europe's population died.[13] Other virulent infectious disease epidemics with high mortalities occurred during this time such as smallpox, infantile diarrhoea and dysentery. By 1430, Europe's population was lower than it had been in 1290 and would not recover the pre-pandemic level until the 16th century.[13, 21]

Quarantine

In 1374 when another epidemic of the Black Death re-emerged in Europe, Venice instituted various public health controls such as isolating victims from healthy people and preventing ships with disease from landing at port. In 1377 the republic of Ragusa on the Adriatic Sea (now Dubrovnik in Yugoslavia) established a ships' landing station far from the city and harbour in which travellers suspected to have the plague had to spend thirty days, the *trentena*, to see whether they became ill and died or whether they remained healthy and could leave. The *trentena* was found to be too short and in 1403 in Venice, travellers from the Levant in the eastern Mediterranean were isolated in a hospital for forty days, the *quarantena* or *quaranta giorni*, from which we derive the term quarantine.[8,18] This change to forty days may have also been related to other biblical and historical references such as the Christian observance of Lent, the period for which Christ fasted in the desert, or the ancient Greek doctrine of "critical days" which held that contagious disease will develop within 40 days after exposure.[22] In the 14th and 15th centuries following, most countries in Europe had established quarantine, and in the 18th century Habsburg established a *cordon sanitaire*, a line between infected and clean parts of the continent which ran from the Danube to the Balkans. It was manned by local peasants with checkpoints and quarantine stations to prevent infected people from crossing from eastern to western Europe.[8]

The leather costume of the plague doctors at Nijmegen

In the 15th and 16th centuries doctors wore a peculiar costume to protect themselves from the plague when they attended infected patients, first illustrated in a drawing by Paulus Furst in 1656 and later Jean-Jacques described a similar costume worn by the plague doctors at Nijmegen, an old Dutch town in Gelderland, in his 1721 work Treatise on the Plague. They wore a protective garb head to foot with leather or oil cloth robes, leggings, gloves and hood, a wide brimmed hat which denoted their medical profession, and a beak like mask with glass eyes and two breathing nostrils which was filled with aromatic herbs and flowers to ward off the miasmas. They avoided contact with patients by taking their pulse with a stick or issued prescriptions for aromatic herb inhalations passing them through the door, and buboes were lanced with knives several feet long.[19]

The Great Plague of London of 1665 to 1666

Plague continued to occur in small epidemics throughout the world but a major outbreak of the pneumonic plague occurred in Europe and England in 1665 to 1666. The epidemic was described by Samuel Pepys in his diaries in 1665 and by Daniel Defoe in 1722 in his A Journal of a Plague Year. People were incarcerated in their homes, doors painted with a cross. The epidemic reached a peak in September 1665 when 7,000 people per week were dying in London alone. Between 1665 and 1666 a fifth of London's population died, some 100,000 people.[17] The Great Fire of London in 1666 and the subsequent rebuilding of timber and thatch houses with brick and tile disturbed the rats' normal habitat and led to a reduction in their numbers, and may have been a contributing factor to the end of the epidemic.[9]

An old English nursery rhyme published in Kate Greenaway's book Mother Goose 1881 reminds us of the symptoms of the plague :

'Ring, a-ring, o'rosies, (a red blistery rash)

A pocket full of posies (fragrant herbs and flowers to ward off the 'miasmas')

Atishoo, atishoo (the sneeze and the cough heralding pneumonia)

We all fall down.' (all dead)

Plague waxed and waned in Europe until the late 18th century, but not with the virulence and mortality of the 14th century European Black Death.

The Third Pandemic of 1894

The plague re-emerged from its wild rodent reservoir in the remote Chinese province of Yunnan in 1855. From there the disease advanced along the tin and opium routes and reached the provincial capital of K'unming in 1866, the Gulf of Tonkin in 1867, and the Kwangtung province port of Pakhoi (now Pei-hai) in 1882. In 1894 it had reached Canton and then spread to Hong Kong. It had spread to Bombay by 1896 and by 1900 had reached ports on every continent, carried by infected rats travelling the international trade routes on the new steamships.[3,23] It was in Hong Kong in 1894 that Alexandre Yersin discovered the bacillus now known as *Yersinia pestis*, and in Karachi in 1898 that Paul-Louis Simond discovered the brown rat was the primary host and the rat flea the vector of the disease.[3, 4, 24, 25]

In 1900 the plague came to Australia where the first major outbreak occurred in Sydney, its epicentre at the Darling Harbour wharves, spreading to the city, Surry Hills, Glebe, Leichhardt, Redfern, and The Rocks, and causing 100 deaths. John Ashburton-Thompson, the chief medical officer, recorded the epidemic and confirmed that rats were the source and their fleas were the vectors in the epidemic. There were 12 major outbreaks of plague in Australia from 1900 to 1925 with 1371 cases and 535 deaths, most cases occurring in Sydney.[26]

The third pandemic waxed and waned throughout the world for the next five decades and did not end until 1959, in that time plague had caused over 15 million deaths, the majority of which were in India. There have been outbreaks of plague since, such as in China and Tanzania in 1983, Zaire in 1992, and India, Mozambique and Zimbabwe in 1994[15, 27.] In Madagascar in the mid-1990's, a multi-drug resistant strain of the bacillus was identified[15, 28.] Currently around 2,000 cases occur annually, mostly in Africa, Asia and South America, with a global case fatality rate of 5% to 15%.[28]

Bubonic plague is a virulent disease with a significant mortality rate, transmitted primarily by the bite of the rat flea or through person-to-person when in its pneumonic form. There have been innumerable epidemics of plague throughout history, but it was the pandemics of the 6th, 14th and 20th centuries that have had the most impact on human society, not only in terms of the great mortalities, but also the social, economic and cultural consequences that resulted. The course of development of communities and nations was altered several times. Much has changed to prevent the recurrence of pandemic plague, such as the development of the germ theory and the science of bacteriology, public health measures such as quarantine, and antibiotics such as streptomycin, but plague today is still an important and potentially serious threat to the health of people and animals.

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Army Malaria Institute: its Evolution and Achievements. First Decade: 1965-1975

Karl H. Rieckmann and Anthony W. Sweeney

Abstract

This article describes the resumption of malaria research activities by the Australian Army during the mid-1960s - about 20 years after they were discontinued at the end of World War II . At the start of the decade, some malaria infections were no longer being suppressed adequately by proguanil or chloroquine, whereas the addition of dapsone to the prophylactic regimen was effective in preventing falciparum malaria during military operations in Vietnam. However, severe toxicity observed in a few individuals following the use of this drug combination emphasized the need to develop alternative prophylactic regimens. The small malaria research unit was able to demonstrate potentiation of antimalarial activity between proguanil and dapsone in a rodent malaria model, raising the possibility that a drug combination using lower (and non-toxic) doses of dapsone might be effective in protecting soldiers against drug-resistant malaria. This synergistic drug activity was only able to be determined in mice inoculated with blood from other infected mice because routine malaria transmission via infected mosquitoes reared in the insectary proved difficult. Although anopheline mosquito colonies could not be used successfully to determine the causal prophylactic activity of drugs against sporozoite-induced infections, they were becoming useful for investigating the biological properties of a novel fungus discovered to attack mosquito larvae in the insectary. Initial plans to evaluate the effectiveness of new antimalarial drugs in soldier volunteers were shelved in favour of taking appropriate measures to procure Aotus monkeys in which drug activity against human malarial parasites could be assessed.

Background

Malaria has been recognized for a very long time to be a major threat during combat operations in malarious areas. During the First World War, personal protection and mosquito reduction, mainly by drainage and oiling of mosquito breeding sites, were the main measures used to prevent malaria. The only antimalarial drug available was quinine and, although it was partially effective in treating patients with malaria, toxicity precluded the prolonged use of this drug for malaria prevention. Later on, in the 1920s and 30s, German scientists discovered the first two synthetic antimalarials - pamaquine and atebrine. Chloroquine, which came to be the most widely used antimalarial in the latter half of the 20th century, was not synthesized by them until the early 1940s. At about the same time, British scientists discovered proguanil (Paludrine). During the Pacific War in the 1940s, malaria became a serious problem for Australian and other troops during combat operations. This prompted General Douglas MacArthur to comment that "this will be a long war, if for every Division I have facing the enemy, I must count on another Division in hospital with malaria, and a third Division convalescing from this debilitating disease". Because of the gravity of the situation, the US Army embarked on an intensive program to evaluate various drug regimens for their effectiveness in the prevention and treatment of malaria.

Australian Army contribution to malaria prevention. There was an alarmingly high rate of malaria cases among Australian troops during the first year of the campaigns in Milne Bay and Kokoda. In February 1943 analysis of sickness casualties from the disease showed that if malarial infections continued at these levels there would be insufficient available manpower in Australia to maintain the required force in New Guinea.[1] This was a key stimulus to the formation of the Land Headquarters Malaria Research Unit (LHQ-MRU) which was established at Cairns, North Queensland by Brigadier N.H. Fairley in June 1943. Early investigations carried out by this Unit revealed that malaria could be controlled by taking one tablet of atebrine (100 mg) every day.[2] Enforcement of this drug regimen by Unit Commanders ensured that thousands of soldiers in New Guinea were protected against malaria. In fact, strict discipline reduced malaria in the Australian Army to a level previously unknown in a force operating in a highly malarious area. Nevertheless, despite its efficacy, the drug did have some disagreeable side-effects, such as a yellowish discolouration of the skin, and it was not widely used after the war. Treatment of established vivax infections with pamaquine was

useful in preventing malaria relapses, whereas administration of the drug to a small number of volunteers during the pre-erythrocytic incubation period did not prevent subsequent primary attacks of vivax malaria but did prevent relapses.[3] Proguanil was not useful for treatment, but this well-tolerated drug was very effective in preventing falciparum malaria when administered during the incubation period.[4] Although parasite resistance to proguanil started to appear in a few places as early as 1948, it remained effective in protecting most Australian soldiers for another two decades.

The malaria studies by Brigadier Fairley's group at the LHQ Malaria Research Unit at Cairns not only carried out meticulous studies to evaluate the effectiveness of antimalarial drugs, but they also obtained considerable new information about the life cycle of the human malaria parasite and the pharmacokinetics of various drugs. All these accomplishments over a period of just 3 years (1943-1946) could not have been possible without the participation of hundreds of soldiers and a very dedicated group of investigators. The remarkable saga of this effort to control malaria and its outstanding achievements have been documented in considerable detail in Tony Sweeney's book - Frontline Malaria.[5]

Antimalarial drugs in the 1950s. In the years after WWII chloroquine became the main drug used for the treatment and prevention of malaria, although the Australian Army continued to use proguanil for prevention where parasite resistance to this drug had not yet developed. Two other drugs were also introduced – pyrimethamine, an antifolate similar to proguanil, and amodiaquine, a 4-aminoquinoline similar to chloroquine.[6] In the meantime, an intensive program had been initiated in the USA to synthesize 8-aminoquinoline compounds that might be less toxic and more effective than plasmoquine which had its origin in the 1920s. Primaquine emerged as the best and least toxic of these new 8-aminoquinolines for eradicating the residual liver stages of *Plasmodium vivax*[6] and was first used very effectively against vivax malaria during the Korean conflict in the 1950s. However, some vivax infections, especially from the southwest Pacific area, had a reduced susceptibility to this drug.[7]

Global malaria eradication program. Anti-mosquito measures are of course an extremely important component of any antimalarial control activities. Following the discovery of DDT, 6-monthly indoor residual spraying (IRS) to control adult anopheline mosquitoes was introduced as a supplementary measure to ground control of larval breeding sites. In 1955 the WHO launched a global malaria eradication program based on IRS combined with active and passive surveillance to detect and treat malaria infections. Although a few countries achieved malaria eradication, most countries struggled to interrupt malaria transmission.[6] Certainly, in unstable environments with a breakdown in public health activities, such as military conflicts, protection of troops continued to rely heavily on ground control measures and rigorous adherence to personal protection against mosquito bites, in addition to chemoprophylaxis.

Proguanil resistance. During the early 1960s, daily proguanil prophylaxis, coupled with anti-mosquito measures, seemed to control malaria quite well during limited deployments overseas. However, in 1962, there was a major malaria outbreak among Australian and New Zealand troops serving with the 28 COMWEL Brigade in northern Malaya. Although antimalarial discipline may not have been as good as it should have been, limited investigations by the malaria research unit in Kuala Lumpur indicated that some strains of malaria in the area had developed resistance to proguanil.

Chloroquine resistance. By the early 1960's, there was also evidence of emerging chloroquine resistance in Southeast Asia and South America. It was soon observed that chloroquine could no longer provide adequate protection against falciparum malaria for American troops in Vietnam. Most of these infections were also resistant to pyrimethamine and, to a lesser extent, proguanil. This prompted the US Army to embark on a major malaria research program which included the synthesis of thousands of chemical compounds and screening them for their antimalarial activity using the *Plasmodium berghei* malaria mouse model.[8] The most promising compounds were then screened for their activity against chloroquine- and antifolate-resistant isolates of *Plasmodium falciparum* using a newly-developed in vitro test.[9] Some of them then underwent further evaluation in *Aotus* monkeys and human volunteers.[10] Based on some early findings, American troops were treated with various combinations of quinine, pyrimethamine, dapsone, and various sulphonamides. In some US units, daily dapsone was added to the weekly chloroquine-primaquine prophylactic regimen.

Establishment of Malaria Research Laboratory

The emergence of chloroquine resistance in the early 1960s, coupled with the demonstration that some Australian soldiers in Malaya were not being adequately protected by daily proguanil prophylaxis, prompted Professor Robert Black, Army Consultant in Tropical Medicine, to recommend the establishment of a Malaria Research Laboratory (MRL). As an Army Captain serving at the LHQ Malaria Research

Unit at Cairns, Professor Black had experienced first hand the devastating impact of malaria on combat operations in the 1940s. With the growing commitment of Australian troops to conflicts in Southeast Asia, Professor Black proposed, in 1965, that the MRL be established under his supervision at the University of Sydney's School of Public Health and Tropical Medicine (SPHTM). In June 1966, approval was given for the MRL to be established, staffed initially by a Major Medical Officer, two Captain Scientific Officers and three non-commissioned (NCO) Laboratory Technicians. Professor Black hoped that human malaria studies, similar to those at Cairns, could be conducted in Army volunteers to counter the growing drug resistance problem.

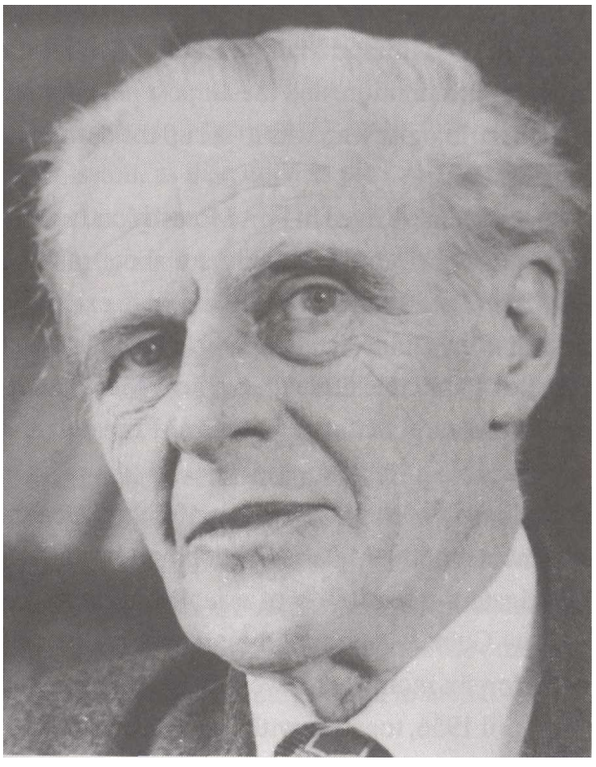

Figure 1. Professor Robert Hughes Black, Army Consultant in Tropical Medicine

In the meantime, the importance of identifying effective drug regimens for malaria prophylaxis was highlighted by Australian troops deployed to Vietnam. Towards the end of 1966 and 1967 (after the wet season), Australian troops experienced peak monthly malaria rates of 140 and 190 per thousand per year. Despite very intensive efforts that all personnel took 200mg proguanil daily (double the normal dose) and took every precaution to avoid mosquito bites, the rate rose to 455 in October 1968. In November, a study was initiated to assess the value of adding 25mg dapsone to the daily suppressive dose of 200mg proguanil. The suspicion that parasites had developed a marked degree of resistance to proguanil was confirmed when dapsone/proguanil prophylaxis resulted in a dramatic reduction in the incidence of falciparum malaria. Furthermore, this synergistic drug combination provided better protection than alternative regimens used by other forces in the area.[11] Unfortunately, a number of soldiers – mainly in US units taking daily dapsone and weekly chloroquine/primaquine developed agranulocytosis following daily administration of 25mg dapsone.[12,13] Consequently, the use of a daily dose of dapsone was no longer included in malaria prophylactic regimens.

Facilities and Staff

Following the establishment of the MRL in 1966, a biochemist/parasitologist (Captain George Michael Galvin), an entomologist (Lieutenant Elizabeth Kalucy), and some technicians commenced their work in small quarters located at the SPHTM. Although some basic malaria research procedures were established, laboratory studies were hampered by inadequate accommodation and the lack of qualified staff. Michael Galvin was transferred in 1968 and Elizabeth Kalucy resigned the following year for family reasons, with the result that the mosquito colony which was set up to transmit rodent malaria could no longer be maintained.

The need for enhanced malaria research activities was stressed following a statement by the Adjutant-General in 1969 that "events in Vietnam had shown the Australian Army to be extremely vulnerable to the ravages of Malaria". He also commented on the "meagre, ineffective and totally inadequate research effort".[14] In the same year, a medical officer (Major Ian Saint-Yves) and an entomologist (Captain Anthony Sweeney) were recruited, both having been involved previously in malaria control/eradication activities in PNG. An Army Malaria Research Advisory Board (AMRAB) was also established to serve as an expert committee and to give professional guidance and authority to the subject of malaria research within the Australian Army. The first meeting of the Board, convened under the auspices of the Adjutant-General, was held on 9 Dec 69 under the chairmanship of the Director General of Medical Services, Major General C.M. Gurner. Subsequent meetings during 1970 considered various ways of meeting the challenges posed by the growing malaria problem, including the possible use of Army volunteers to develop effective antimalarial drug regimens.

Figure 2. Unit staff January 1970. From L to R: CAPT A.W. Sweeney, MAJ I. Saint-Yves, CPL N. Tinney, CPL R. Green and CPL C. Gulley

In July 1970, the MRL was re-designated as the 1st Malaria Research Unit (1MRU) (Raising/ Reorganization Instruction 31/70 dated 17 Jun 70). The staff was increased from 6 to 8, comprising a Colonel Director, three Major Scientific Officers, three NCO Laboratory Technicians and one Clerk. It was hoped that a full-time Director would provide the necessary leadership and expertise to tackle the growing malaria problem. It was also planned to move AMRU from the University to be co-located in more appropriate facilities at 2 Military Hospital, Ingleburn, located southwest of Sydney. Unfortunately, adequate staffing of the unit remained a problem, with many technical staff being at the unit for less than a year. An exception to this was Corporal David Cowdrey who was assigned to the Unit in 1971 and remained actively involved in malaria research activities (including 14 overseas deployments) until his retirement in 1989. In an effort to fill the vacant Director's position, the option of civilianising the position was accepted in June 1972. Following the resignation of Major Saint-Yves in June 1973, 4 staff members remained, with Captain Sweeney being the only one of the original 1MRU group.

In July 1973 Dr AP Ray was appointed as the first director of the unit and work proceeded to accommodate the Unit on the grounds of 2 Military Hospital at Ingleburn. When AMRAB was convened during the opening ceremonies in April 1974, Dr Ray and Professor Black envisaged that the scope of work at the unit could now be expanded with a view to progressive assessment of drugs in infected rodents, then in *P. falciparum*-infected *Aotus* monkeys and, finally, in infected Army volunteers at the adjacent Field Hospital. As 8 of the 9 establishment positions were being filled, there was a feeling of optimism that, with some additional staff, these proposed research activities could be carried out over the ensuing years.

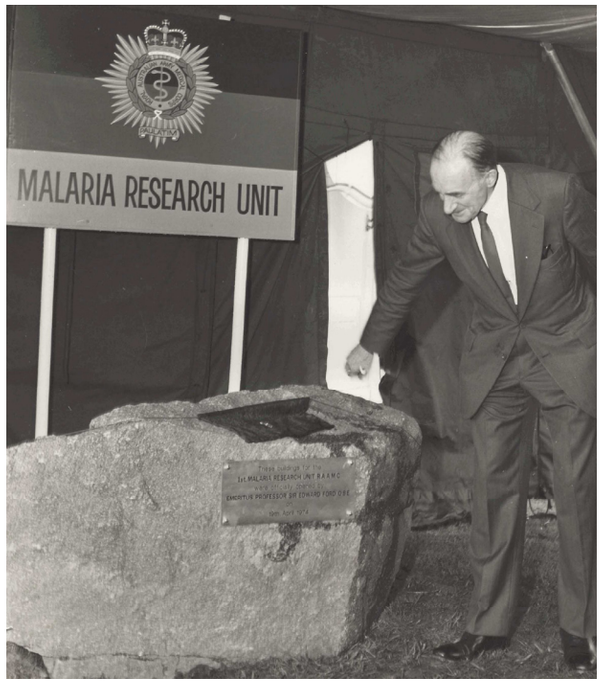

Figure 3. Emeritus Professor Sir Edward Ford opening Unit at Ingleburn on 19 April 1974. He played a dominant role in the New Guinea Force (1942-1943) "in defeating malaria before it defeated us".

Activities

The role of the MRL was to carry out malaria research activities to protect soldiers adequately and safely against drug-resistant malaria spreading southward from its original focus along the Thai-Cambodian border. By the late 1960s, there were rumours that chloroquine-resistant malaria may even have reached PNG, although this could not be confirmed when Major Saint-Yves investigated this possibility in the Milne Bay area following a request from PNG authorities.[15] In addition to the malaria research efforts by the US Army and some other overseas organizations, the Australian Army's participation was considered important because malaria and malaria vectors have certain characteristics unique to the Pacific region. Although investigations with human malaria parasites cannot be initiated, the following studies were performed following the establishment of a rodent malaria model and an insectary.

Antimalarial activity against rodent parasites

Rodent malaria models have been used since the 1950s to obtain preliminary information about the potential antimalarial activity of various drugs and drug combinations.[16] Such studies usually constituted the first step in developing new drug regimens for the prophylaxis and treatment of

malaria. Studies with rodent malaria were initiated at the Unit before the move to Ingleburn. Although attempts to do this were not entirely successful in the beginning, it was soon possible to routinely transmit rodent malaria parasites from mouse to mouse through intra-peritoneal inoculation of P. *berghei*-infected red cells. This provided the means for assessing the efficacy of various drugs and drug combinations in mice infected with chloroquine-resistant strains of *P. berghei*.

Proguanil and dapsone were among the first drugs to be evaluated in the rodent model, including the assessment of urinary drug concentrations by spectrofluorimetry. Results showed potentiation of antimalarial activity between various combinations of dapsone and proguanil. Early observations of synergistic activity, even at low doses of dapsone, suggested that the drug combinations might retain their efficacy at dapsone doses lower than those used by Australian soldiers during the Vietnam conflict. If further studies were to confirm these preliminary findings, lowering the dose of dapsone might avoid the rare case of agranulocytosis observed during proguanil/dapsone prophylaxis in Vietnam.

Transmission of rodent malaria via anopheline mosquitoes

The rearing of anopheline mosquitoes in an insectary commenced soon after the establishment of MRL. Its primary purpose was to transmit *P. berghei* from one rodent to another via susceptible anopheline mosquitoes, thereby enabling the causal prophylactic activity to be determined against pre-erythrocytic liver stages. As far as malaria protection was concerned, the ability of a drug (e.g. proguanil) to exert its activity against the liver stages (before release of parasites into the blood circulation) had distinct advantages over a drug that acted only against the blood stages.

The first mosquito colony of *Anopheles annulipes* was established by Elizabeth Kalucy in 1968 from specimens collected at Castle Hill in Sydney. The colony was maintained by a labour-intensive, induced mating technique because natural mating in cages was not possible. During May 1969 cyclical transmission of rodent malaria was achieved by feeding mosquitoes from this colony on mice infected with a gametocyte producing NK65 strain of *P. berghei*[17]. Dissections of mosquitoes two or more weeks after feeding showed oocysts in about half the stomachs and heavy sporozoite infections in the salivary glands of a few mosquitoes. Parasitaemia developed in five albino rats bitten by mosquitoes which had previously taken infective blood meals from mice. These encouraging results suggested that this mosquito/parasite combination might lead to the establishment of an experimental transmission model of rodent malaria. Unfortunately further experiments with this strain of *An. annulipes* were not possible because of the lack of technical staff to maintain the mosquito colony after Kalucy's departure in 1969.

After Tony Sweeney's arrival at the unit in 1970, a new colony of *An. annulipes* was established from larvae collected at Nattai River near Mittagong. In January 1971 *An. hilli* larvae collected during a field survey at Gove in the Northern Territory were transported back to the insectary.[18] This species adapted very well to the laboratory as it could mate in the confined space of small laboratory cages and could mature the first egg batch without the need for a blood meal.

Figure 4. CAPT A.W. Sweeney feeding Anopheles mosquitoes.

A second series of *P. berghei* transmission experiments using the two new colonies of anopheline mosquitoes were made with the NK65 strain and also with the ANKA strain.[19] Both species were almost completely refractory to the NK65 strain as only around 1% of mosquitoes fed on infected mice developed oocysts and sporozoites. Results with the ANKA strain were not much better - *An. annulipes* could not be infected and *An. hilli*, despite its ease of maintenance, experienced a 90% mortality during the 2-3 week holding period necessary for the completion of the mosquito cycle of the parasite. Although a few mice were able to be infected by inoculation of sporozoites from surviving mosquitoes, it became obvious that a local mosquito/rodent malaria model for causal prophylactic studies was not a feasible proposition with the available mosquito colonies

Antimalarial activity against human parasites.

Although the rodent malaria model, even without mosquito transmission, was useful for obtaining

preliminary information about the activity of potential antimalarial drugs, it could not reliably predict the efficacy of such drugs against human malaria. An animal model that can be infected with human malaria parasites would obviously be a very important asset as an intermediary step in assessing the potential value of drugs before their administration to human volunteers. After the discovery in the early 1960s that human malaria infections could be experimentally induced in *Aotus* monkeys (Owl monkeys) from South America, it became possible to assess the potential value of drugs against human malaria parasites before their evaluation in human volunteers.[10] With the extension of chloroquine-resistant malaria in Southeast Asia and a greater urgency to identify effective alternative drugs, AMRAB agreed in 1974 that steps be taken to acquire this monkey model for research activities at the Unit. Importation restrictions, construction of climate-controlled primate accommodation, and training of animal handlers delayed the arrival of the first shipment of 12 monkeys until 1982.

Since inception of the Unit in the mid-1960s, human studies with Army volunteers at the Military Hospital, modelled on those carried out at the LHQ unit during the mid-1940s, were considered to be an important component of the project. During 1973 and 1974 the feasibility of such studies received considerable attention by various members of AMRAB. After careful consideration, it was decided that such studies could not be instituted then for a variety of different reasons. Instead, efforts were made to approach military and public health authorities in the Asia-Pacific region to explore the possibility of carrying out collaborative field studies to evaluate new drug regimens in malarious areas of their country. Early attempts to do so were not particularly successful. The prime example was the demise of attempts to develop collaborative field studies in assessing the prophylactic effectiveness of a proguanil/dapsone combination in a highly malarious area of India. Despite progressive approval by relevant health authorities in the Indian and Australian armies over a period of 2 years, this potentially very important project never got off the ground.

Discovery and laboratory evaluation of the fungus Culicinomyces as a biological control agent of mosquitoes.

During February 1972, a dramatic larval mortality was noted in the *An. hilli* colony. Within a period of three days more than 90% of larvae were dead or moribund and the extinction of the colony seemed imminent. The suspicion that a fungus was responsible was confirmed after inoculating trays of healthy larvae with dead specimens, isolating the fungus on a nutrient agar medium, and killing larvae by exposing them to spores (conidia) produced in artificial culture.[20] The taxonomic status of the fungus was clarified in 1973 after the same organism was discovered infecting mosquito larvae in North Carolina, USA. It was subsequently described as a new taxon, *Culicinomyces clavisporus*.[21]

Initial studies indicated that *Culicinomyes* was a very efficient larval pathogen that might have potential as a biological control agent of mosquitoes. Accordingly, it was subjected to systematic laboratory investigation to evaluate this possibility. The mode of infection was via the alimentary tract. The conidia were ingested during feeding and adhere to the cuticle lining the mosquito foregut and hindgut, where they germinated and penetrated into the body cavity. This subsequently became filled with a dense interior mycelium. After death, a sporulating layer formed on the exterior cuticle which produced conidia that were infectious to other larvae.[22] It implied that the fungus could be able to recycle in the aquatic environment after its original application and infect successive generations of mosquito larvae. The additional finding that infective conidia was readily obtained in aerated broth cultures of peptone and yeast extract heightened the possibility of industrial mass production.

By the end of 1975 the initial promise of *Culicinomyces* as a mosquito larvicide was reinforced by further favourable laboratory results. The host range was expanded to include *Anopheles, Culex* and *Aedes,* three medically important genera of mosquitoes, as well as larvae of *Chironomidae* (midges) and biting midges of the family *Ceratapogonidae*.[23] Additional investigations revealed that *Psychodidae* (moth flies), other aquatic insect larvae, freshwater shrimps, and the mosquito fish *Gambusia* were not affected by long term exposure to infective conidia, indicating that this fungus only acted against certain families of the Order Diptera.

Conclusion

The small malaria research unit, established in the mid-1960s in response to the growing threat of drug-resistant malaria to Australian troops, was hampered in the scope and extent of its activities by limited or inadequate staff and facilities during the first decade of its operation. Although significant studies with human malaria parasites could not be carried out, useful information was obtained during investigations involving mice and mosquitoes. The main achievements of the unit were : 1) establishment of a rodent malaria model and an insectary to rear various species of anopheline mosquitoes; 2) preliminary data indicating that low doses of dapsone could act synergistically with proguanil to potentiate

the activity against rodent malaria parasites; 3) discovery that the fungus *Culicinomyces clavisporus* attacked mosquito larvae and might eventually be used as a biological control agent.

Acknowledgement

We would like to thank MAJOR S. Frances for his efforts in retrieving the archived photographs and providing them to us.

Highlights

Early 1960's	Chloroquine resistance develops in Southeast Asia and proguanil resistance is observed among Australian soldiers in Malaya.
1965	Professor Robert Black, Army Consultant in Tropical Medicine, recommends establishment of a Malaria Research Laboratory (MRL), with a view to future assessment of the effectiveness of new drug regimens in Army volunteers.
1966	High prevalence of malaria among Australian troops in Vietnam is controlled when dapsone is added to proguanil prophylaxis, although a few soldiers develop agranulocytosis while taking dapsone. MRL is established at the University of Sydney, but establishment of basic malaria research procedures are hampered by limited staff and inadequate laboratory facilities.
1969/1970	Adjutant-General stresses importance of enhancing malaria research activities and establishes the Army Malaria Research Advisory Board (AMRAB). MRL re-designated 1st Malaria Research Unit (1MRU). Research activities strengthened, leading to routine blood transmission of rodent malaria parasites in mice and occasional transmission of rodent malaria by anopheline mosquitoes reared in the insectary.
1972	Discovery of a new fungus, *Culicinomyces clavisporus*, killing larvae in mosquito colony.
1973	With only 4 staff members still at the unit, adequate staffing remains a problem.
1974	Following appointment of Dr A P Ray as director, 1MRU is re-located from University of Sydney to grounds of 2 Military Hospital at Ingleburn, southwest of Sydney. Eight of the 9 establishment positions are filled. AMRAB recommends that Aotus monkeys be procured to assess the potential value of new drugs against human malaria parasites. Earlier plans to evaluate the efficacy of new antimalarial drugs in Army volunteers at the military hospital are shelved in favour of collaborative field studies in malarious countries.
1975	Transmission of rodent malaria parasites via blood inoculation is well established. Although various species of anopheline mosquitoes have been colonised by now, routine malaria transmission via mosquitoes remains problematical, thereby preventing assessment of drug activity against parasite liver stages and gametocytes. Preliminary findings indicate potentiation of activity between low doses of proguanil and dapsone against rodent malaria parasites, suggesting that dapsone dosage may be able to be reduced to prevent possible agranulocytosis during proguanil/dapsone prophylaxis. Progressive studies with *Culicinomyces* continue to produce favourable laboratory results and encourage further investigations to determine its potential value as a biological control agent for mosquitoes.

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Crushed ice ingestion – a practical strategy for lowering core body temperature

Matt Brearley PhD

Abstract

Exercise together with environmentally induced heat stroke continue to pose a problem for military operations in hot climates. A variety of cooling strategies are required by the military to mitigate the risk of heat stroke due to the variety of climates and physical workloads encountered by defence personnel, combined with their individual physical characteristics and uniforms/protective attire. This paper highlights that cooling is traditionally applied as a treatment for heat stroke rather than used to prevent its onset. Recent evidence from the field of sport science demonstrated that cold fluid consumption can act as a heat sink to blunt the rise of core body temperature. Furthermore, the addition of crushed ice to beverages substantially improves its heat storage potential, resulting in decreased core body temperature and enhanced endurance performance. While crushed ice will not be universally available in defence settings, it is a strategy that requires minimal equipment, is relatively quick to prepare, is not labour intensive and does not require the removal of a soldier's uniform. The military should therefore consider the use of crushed ice ingestion as a preventative measure against heat stroke.

Key words: crushed ice ingestion, hyperthermia, cooling, heat stroke

Exercise and Environmentally Induced Heat Stroke

Exercise and environmentally induced heat stroke (EEHS) describes inadequate heat loss and/or excessive endogenous heat production during exercise[1], and is diagnosed by a core body temperature greater than 41°C and altered cerebral function. Given the variety of climates and physical workloads encountered by military personnel, combined with their individual physical characteristics and uniforms/protective attire, it is not surprising that EEHS and other heat related illnesses threaten the health of defence force personnel. The risk and incidence of EEHS in military settings is well described[2-10], with the duration and degree of core body temperature elevation considered as the primary predictors of patient outcome[11]. That 37 US Army soldiers died and a further 5248 required hospitalisation for heat illness from 1980 to 2002 highlights this point.[12] Within an Australian context, 65 cases of heat related illnesses were reported over a 15 week period (2003/4) during training courses conducted in tropical field conditions, inclusive of one death[13]. A more recent report illustrates that hot climates continue to influence global military operations, with 311 cases of EEHS reported among a total of 2887 heat related injuries within the US Armed Forces during 20102.

A wide variety of strategies are required to combat the development of, and to treat, EEHS. Strategies preparing defence force personnel for hot conditions include heat acclimatisation[4] and improved physical fitness[14]. Heat acclimatisation is a key strategy to improve tolerance of hot conditions as the majority of heat related illnesses occur within the initial days of deployment to the new environment[15]. Maintaining an adequate hydration status [16], pacing of effort and work to rest guidelines[17] seek to limit heat storage to manageable levels and can be broadly classified with heat acclimatisation and physical fitness as preventive measures for development of EEHS. In contrast, cooling is generally classified as a treatment for EEHS, with the objective of rapidly lowering core body temperature to minimise the degree and duration of hyperthermia. The majority of military and occupational cooling research has therefore been dedicated to lowering the core body temperature of symptomatic patients or athletic subjects with exercise induced hyperthermia, with cold water immersion demonstrating the fastest cooling rates and therefore the preferred cooling method to counter EEHS[18,19]. The limitations of relying on cold water immersion in the Australian military setting have been highlighted by McKenzie[20], with limited access to adequate volumes of ice in the field the primary constraint. Alternatives to cold water immersion include enhancing evaporation and/or convection

by fanning[21], spraying/dousing with water[22], resting in the shade[22], resting in an air conditioned area[23], personal cooling systems[24] and the administration of cool intravenous fluids[25]. Conduction based cooling methods include the application of ice packs[26] cooling blankets[27], limb water immersion[28], temperate whole body water immersion[23], and ice-wet towels[29]. Use of the aforementioned methods and a combination thereof generally achieves protracted cooling times when referenced against cold water immersion[27], resulting in the recognition of cold water immersion as the gold standard for treatment of exercise and environmental heat stroke[30]. Regardless, the alternate strategies to cold water immersion may be the most appropriate cooling treatment for EEHS where access to adequate volumes of cold water is not possible. Management of core body temperature may also be aided by the aforementioned modalities prior to the classification of EEHS. The aim of cooling provided during scheduled rest periods is to prolong exposure time by limiting the development of high core body temperatures. Such an 'intermittent cooling' approach is considered proactive, seeking to regulate an individual's physiological state to prevent EEHS, rather than to treat it. The field of sport science has utilised intermittent cooling and cooling applied prior to competition (pre-cooling) to improve physiological responses and in most cases physical performance of athletes competing in hot conditions[31]. The constraints of most athletic competitions dictate that prerequisites for cooling techniques are minimal equipment, time efficiency, ability to be deployed in the field and not labour intensive. Similar constraints may coexist in a variety of military settings. With this in mind, practical field deployable cooling strategies are required to improve tolerance and decrease the incidence of EEHS.

Cooling by Cold Fluid Consumption

A potential intermittent cooling mode for select military settings is the ingestion of cold water. It is well established that beverage consumption benefits physiological, perceptual and performance outcomes during endurance activities in the heat, compared to fluid abstinence[32,33]. Fluid consumption can also directly influence core body temperature as a result of the heat transfer between the beverage and the gastrointestinal tract. The specific heat capacity of water dictates that ~4.2 kJ of energy is required to heat 1 kg of water by 1°C. Researchers have utilised the heat capacity of ingested beverages to lower core temperature following ingestion of cold fluids in comparison to beverages served at near core body temperature (37-38°C). Wimer et al.[34] demonstrated a small core temperature benefit when consuming ~1.35 L of fluid served at 0.5°C compared to 38°C during the latter stages of moderate physical activity in warm conditions. This study provided fluids following the initial hour of exercise by which time substantial heat storage had occurred. The cold fluids blunted the rise of core temperature by ~0.14°C. The outcomes of this study were insufficient to warrant cold fluid ingestion as the benefits were small and the timing of fluid consumption was vastly different to practices in the field. Lee et al.,[35] utilised a more common approach of pre-loading with 0.9 L of fluid during the 30 minutes preceding physical activity, combined with periodic consumption (0.1 L/10 minutes) during submaximal exercise until exhaustion, to compare responses to 4°C and 37°C fluid.

Consumption of the cold beverage resulted in a 0.5°C core body temperature decrease prior to exercise, with core body temperature remaining significantly cooler until the 45th minute of the performance trial. Perceptual ratings and time to exhaustion (~64 v ~52 minutes) also benefited, highlighting the potential of cold fluid consumption. However, the reported benefits are tempered by both experimental designs using warm fluids served at similar temperatures to that of deep tissue temperature and much warmer than the preferred beverage temperature of 15-20°C[36]. When ad libitum consumption was compared between cold (1.3 L at 4°C) and a more common beverage temperature (1 L at 19°C) during submaximal cycling to exhaustion, the core body temperature benefit was reduced to 0.25°C at the cessation of cycling. in spite of the greater cold fluid consumption[37]. The small benefit reported by Mündel et al.,[37] may be slightly underestimated as subjects cycled for an extra seven minutes (~62 v ~55 minutes) during the cold fluid trial. However, the results are more likely explained by the limited cooling power of the ingested drink. With less than half the difference (15°C) between the experimental beverage referenced to other studies[34,35], the ingested heat capacity was ~63 kJ compared to ~140 kJ and ~158 kJ for Wimer et al.[34] and Lee et al.[35] respectively. While preferable over warm fluids, the small benefit for a relatively large volume of fluid suggests that cold fluids cannot be independently relied upon to substantially improve the heat storage capacity of military personnel in hot settings.

Cooling by Ice Ingestion

The limited cooling capacity of cold fluids could be enhanced by the addition of ice to beverages as the conversion of ice to water utilises the latent heat of melting to theoretically absorb more heat than an equivalent volume of cold fluid. One litre (L) of ice requires ~334 kJ to melt, and once in a liquid

form the heat storage capacity mirrors that of a cold ingested beverage. Hence, the potential heat storage conferred by 1 L of crushed ice is ~489 kJ to melt and warm to 37°C, compared to ~155 kJ for cold water (0°C) to reach 37°C. Despite being recommended as a potential cooling option in athletic settings,[38] few published reports of the physiological responses to crushed ice ingestion are available. A small field of research has demonstrated the potential of crushed ice ingestion (CII), commonly known as 'slushies' as a practical cooling modality. When compared to the ~0.2°C core body temperature decrease observed from rest for temperate water consumption (26°C), CII resulted in a significantly greater (~1.1°C) core temperature improvement prior to physical activity[39]. In that study, 150-200 g of crushed ice was consumed every 8-10 minutes over a 30 minute period, resulting in subjects ingesting ~553 g on average, or 6.8 g/kg body mass. Although the true core temperature benefit is likely to be overestimated due to the susceptibility of ingestible core body temperature sensors to local cooling of the gastrointestinal tract[40], 40 km cycling time trial performance improved in the hot conditions with mean power output 6.9% higher following ice ingestion with similar core body temperatures observed during the trial. Siegel et al.[41] used a similar ice ingestion schedule (1.25g/kg-1 body mass every 5 minutes) to deliver an average ~600 g of crushed ice, the equivalent of 7.5 g/kg body mass over 30 minutes. Importantly, the comparison beverage was served at 4°C and demonstrated a 0.25°C decrease in core body temperature prior to exercise compared to the 0.66°C observed following CII. Core body temperature remained cooler pursuant to CII for the initial 30 minutes of the running trial. The significance of this study is that it demonstrated a worthwhile benefit for CII over cold water consumption, which in turn, is superior to drinking warm fluids[35]. The moderately trained subjects also improved running time to exhaustion in hot conditions from 40.7 (cold water) to 50.2 minutes following the slushie. With a quantified benefit for CII, the strategy was combined with the use of ice wet towels to compare against ad libitum cold fluid consumption and whole body cold water immersion[42]. Dual boluses of 7g/kg body mass were consumed across 30 minutes (14g/kg body mass) by well trained athletes with their torso and legs draped in ice-wet towels. Prior to performance, cold water immersion demonstrated the greatest core body temperature cooling effect (~0.6°C), significantly cooler than CII (~0.3°C), which in turn, was significantly cooler than ad libitum cold fluid consumption (~0.0°C). These results were reversed during the subsequent 46.4km cycling time trial in hot conditions, with no statistical difference in the core body temperature response, notwithstanding the higher power output sustained following CII (~3% higher than cold fluid and ~2% highe than cold water immersion). Despite these studies demonstrating lower core body temperatures and improved endurance performance in the heat, performance is not universally improved following CII.

To test the effectiveness of crushed ice ingestion following substantial heat storage (core body temperature 38.9°C), moderately trained cyclists ingested 1 L of slushie or cool fluid (~18°C) during a 50 minute recovery period[43]. CII resulted in mean core body temperature of ~37.0°C compared to ~37.4°C following the cool beverage. Time to complete a set amount of work did not differ between trials, despite the cyclists lower core temperature during the initial stages of the performance trial. While endurance performance did not alter, this study demonstrated that intermittent cooling via crushed ice ingestion is an effective modality to lower core temperature of athletes between exercise bouts.

Occupational Settings

Unfortunately, less is known of the response to CII in occupational settings. A recent investigation to examine intermittent cooling of fire fighters in tropical field conditions found no core body temperature benefit for CII compared to ad libitum cool fluid consumption during rest periods[44]. The fire fighters were unable to ingest the 7.5g/kg body mass bolus, allowing much of the ice to melt prior to consumption and forfeiting its cooling potential. Since the fire fighters could not match the CII of athletes, alternative ingestion schedules ought to be examined. Another approach is to provide ad libitum access to crushed ice and cold fluids, allowing self regulation of consumption. Such an approach during simulated mining resulted in the miners consuming 34% less crushed ice when compared to cold fluids[45]. Despite the lower ice consumption, core temperature was not different between the groups due to the superior cooling power of ice. The impact of ad libitum CII remains poorly understood, and while many factors contribute to ad libitum fluid consumption, the cooler thermal sensation following CII may diminish the drive to drink. Such an outcome over an extended period may limit the ability of CII to influence core body temperature and also manifest in dehydration.

The threshold ingestion volume to improve thermoregulatory and performance responses also remains to be investigated, and is likely to vary based upon the task, uniform/protective attire and environmental conditions. In the absence of specific guidelines, consumption of 4-5 g/kg body mass of

crushed ice for soldiers during scheduled breaks of ~15 minutes seems a logical starting point. For an 80kg soldier consumption of 320-400 g of ice over a 15 minute period does not seem onerous, yet it would provide 156-196 kJ of cooling compared to 44-55 kJ of cooling for the equivalent volume of 4°C fluid. Whether soldiers could repeatedly consume such a volume of ice to prevent EEHS remains to be tested.

The logistics of providing crushed ice for soldiers are vastly different to those encountered when providing for small groups of athletes. Availability of adequate volumes of ice in the field will be a challenge. Within the Northern Territory, power and water utility crews have access to ice machines at each depot, allowing for ice transportation to work sites. McKenzie[20] details a similar system will exist on Australian military bases allowing training platoons to take ice into the field to be used with water and an individual sleeping shelter as a makeshift immersion bath. Whether adequate volumes of ice can be stored and transported for training platoons to use for treatment (immersion) and prevention (CII) of EEHS remains to be determined. Therefore, military bases are the ideal starting point for field testing. Should CII prove to be a worthwhile strategy during training on base, the logistics of CII in remote field settings will warrant addressing.

Conclusions and Recommendations

The limited research to test the ingestion of ice as a cooling modality reports lower core body temperatures before and during scheduled breaks in physical activity. Core body temperatures during the initial stages of exercise are generally lower following CII while also improving endurance performance for athletes. Based upon these findings, CII is worthy of consideration as a cooling modality in military and occupational settings as a preventative measure for EEHS. While its application is limited by the availability of ice, a slushie requires minimal preparation and can be administered without the removal of uniforms. Military bases appear the logical starting point to evaluate this strategy given the access to ice.

Research should test the ability of military personnel to consume adequate volumes of ice in a short time frame. The threshold ingestion volume to improve thermoregulatory and performance responses is yet to be determined, however 5g/kg body mass over a 10-15 minute period seems a logical starting point. Ad libitum fluid consumption following CII should also be examined. It is possible that internal cooling following CII may diminish the drive to drink, thereby lowering ad libitum fluid consumption and manifesting in dehydration.

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Trends in traumatic limb amputation in Allied Forces in Iraq and Afghanistan

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Abstract

Background: Limb amputation has been a common injury occurring in the conflicts in Iraq and Afghanistan. Compared to other injuries, less attention has been given to this serious, disabling wound.

Purpose: The article describes the Allied military experience of traumatic limb amputation in Iraq and Afghanistan. It intends to inform health care personnel involved in the care of serving military personnel and veterans about the scale of these casualties.

Methods: A literature search of both civilian and military academic databases was conducted.

Results: Both the US and UK have incurred very significant numbers of casualties involving traumatic limb amputation, many of whom have suffered multiple limb loss. The rate of blast injuries causing traumatic limb amputation among US forces has increased since the surge of troops in Afghanistan. Dismounted Complex Blast Injury (DCBI) consisting of multiple limb amputations with pelvic, abdominal or genito-urinary injuries has been reported as increasing in frequency among US troops in Afghanistan since 2010. Australian Defence Force casualties suffering traumatic limb amputation remain low.

Conclusions: Significant casualties involving traumatic limb amputation are likely to continue among Allied troops while current counter-insurgency tactics are continued. Planned troop withdrawals should eventually result in fewer casualties, including reduced numbers of traumatic limb amputation.

Introduction

Traumatic limb amputation is a highly visible wound that causes enormous personal distress and disability as well as incurring considerable national cost in physical and vocational rehabilitation. Recently, this injury appears to be increasing in frequency in the war in Afghanistan[1]. A pattern of multiple lower limb amputations, with associated severe abdominal, pelvic or genito-urinary injuries, has been dubbed the 'new signature wound of the war'[1], a term previously often used in relation to mild traumatic brain injury[2].

The purpose of this article is to describe the Allied military experience of traumatic limb amputation in the conflicts in Iraq and Afghanistan, and inform health care personnel involved in the care of serving military personnel and veterans about the scale of these particular casualties. Based on a review of the literature, it examines the numbers and causes of traumatic limb amputations in Allied military personnel in the current conflicts.

In doing so, it should be noted that military casualty statistics can be complex[3,)] and politically sensitive, with reports on traumatic limb amputations being especially so. Some countries (e.g. Canada and The Netherlands) have chosen not to disclose figures on these injuries. Furthermore, official figures on traumatic or surgical amputations sometimes include a range of injuries from the loss of part of a finger or toe, to the loss of entire limbs. Finally, the exact number of individuals who have undergone multiple limb amputations is not always obvious from some official reports.

Casualty trends in Iraq and Afghanistan

Compared to previous conflicts, the overall pattern of military casualties in Iraq and Afghanistan has been one of lower mortality rates[5] and improved wound survival[6,7]. However, this has coincided with an increase in injury severity and the number of wounds per casualty [8] - with many wounds occurring to the head, neck and upper extremities[9,10]. A review of all deaths of allied military personnel to 2009 found thoracic or abdominal wounds (40%) were the main cause of death, followed by traumatic brain injuries (35%)[11].

Explosive mechanisms, in particular Improvised Explosive Devices (IEDs), were the leading cause of all combat casualties in Iraq and Afghanistan, accounting for 70-75% of killed and wounded[11,12]. Rocket propelled grenades and mortar ordnance were the next most common cause of blast injury. Gunshot wounds, followed by motor vehicle collisions and aircraft crashes accounted for the remainder of

casualties [13]. Explosive devices were the mechanism of injury associated with most (87.9%) amputations[14].

A prospective longitudinal study of wound patterns on a large US Army unit during 'The Surge' in Iraq in 2007 found the distribution of wounds was approximately 50% to the extremities[13]. A British study of injuries requiring surgery found a similar distribution of injuries to extremities (50%)[15]. This burden of extremity injuries is similar to the US experience in previous conflicts from World War II to the First Gulf War 1990-91[16].

The parts of a soldier's body that may be injured in combat are strongly affected by the personal protective equipment (PPE) – with PPE also significantly increasing the chances of survival in the current conflicts in Iraq and Afghanistan[8,17]. Body armour has been improved with blast resistant ballistic goggles or glasses worn with improved Kevlar Advanced Combat Helmets. However, this equipment still leaves a soldier's face, hemicranium and extremities vulnerable[18,19], particularly when blast forces are directed upward through the floor of a vehicle[(20)]. For dismounted troops, the lower extremities are particularly vulnerable to blast injury, as most IEDs are detonated from ground level. Commercially available 'Ballistic Boxers' or Kevlar underpants, are being studied to assess their ability to provide some protection from genito-urinary and femoral artery injury [21].

Counterinsurgency doctrine implemented through the Surge of US Forces in Iraq from 2007[22] and in Afghanistan from 2009, calls for securing the local population by building a local presence within threatened communities[23]. Such local engagement requires troops to live in small outposts and to conduct frequent foot patrols[24], exposing them to greater risk from IEDs than if they were in armoured vehicles.

Allied military personnel are issued tourniquets capable of being applied with one hand, as individual first-aid for wounds sustained under fire. Notwithstanding some controversy over their benefit [25,26], several studies have found tourniquets to be effective in controlling severe haemorrhage, especially in upper limbs[27-29]. Numerous confronting accounts exist of US and UK military personnel routinely wearing their tourniquets on their legs, ready to be tightened in preparation for IED strikes[30-33].

United States

By theatre of operations to September 2010, 1,158 US military personnel suffered major or partial limb amputations as a result of the conflict in Iraq, 249 in Afghanistan, and 214 in 'unaffiliated conflicts'[34] in Yemen, Pakistan and Uzbekistan.

From mid-2008, the rate of blast injuries resulting in traumatic limb amputation in US Forces in Afghanistan began to consistently exceed those occurring in Iraq. By 2010, blast injuries to US Forces in Iraq declined to near zero (from a peak prevalence of 3.3 per 10,000); whereas by mid to late 2010, a significant increase in blast injuries to US personnel in Afghanistan emerged (with a prevalence of 5.2 per 10,000)[35]. For the whole of 2010, a total of 196 US military personnel suffered the loss of at least one limb, increasing to 240 in 2011[36]. Even though amputations increased, US combat deaths actually declined for the same period, from 437 to 368, further confirming improvements in wound survivability [37].

Most were attributed to ground-placed IEDs or land-mines, with 88% of survivors being on foot. An emerging pattern of high, multiple extremity amputations with pelvic, abdominal or genito-urinary injuries was described as Dismounted Complex Blast Injury[35]. Rates of genito-urinary injury among all casualty admissions from Afghanistan in 2010 were two to three times higher than historical averages. Furthermore, in 2010-11, nearly half a sample of US combat fatalities suffered bilateral lower limb extremity amputations, with almost a third losing three limbs[35].

United Kingdom

Prior to April 2006, the United Kingdom Ministry of Defence (MOD) resisted calls to publish data on British military personnel who had undergone traumatic or surgical limb amputation in Iraq (Operation Telic) and Afghanistan (Operation Herrick)[38]. Since then, quarterly amputation statistics have been released. However, the MOD 'suppresses' results when fewer than five persons experiencing amputations have been recorded in a particular reporting period for reasons of operational security and patient confidentiality[39]. This meant that a recent decision to publish historical figures from both conflicts between 2001-2006 failed to shed light on the exact number of these particular casualties[40].

Between April 2006 and December 2011, at least 20 British military personnel suffered traumatic limb amputations in Iraq, and 237 in Afghanistan. UK limb amputation casualties in Afghanistan have significantly increased since 2009 with 55 sustained in 2009, 79 in 2010, and 53 in 2011[39, 40]. Multiple amputee casualties were also the worst to date with 32 in FY2009/10 and 36 in 2010/11[39, 40].

Australia

At least three Australian soldiers have suffered traumatic limb amputations[41], among the 32 killed and 218 wounded in Afghanistan from 2002 to 14 January 2012[42]. The Australian Department of Veterans' Affairs reported one veteran of the Iraq War in 2003 and one veteran of the conflict in Afghanistan with accepted disabilities for limb amputation[43].

Conclusion

Both the US and UK have incurred very significant numbers of casualties involving traumatic limb amputation, many of whom have suffered multiple limb loss. Numbers of US limb amputation casualties peaked following the surges in troop numbers in Iraq in 2007 and Afghanistan in 2009. During counter-insurgency operations in Afghanistan since late 2010, the use of dismounted troops as foot patrols has been associated with the emergence of what has been termed Dismounted Complex Blast Injury, involving multiple limb amputations. Australian casualties involving limb amputations in Afghanistan to date have fortunately remained low.

Significant rates of traumatic limb amputation among allied military personnel in Afghanistan are likely to persist while current counter-insurgency tactics continue. The withdrawal of US forces, which commenced in July 2011 and which will increase in 2012, should be accompanied by a fall in overall casualties, including traumatic limb amputation, particularly as their role is anticipated to change from combat to more mentoring of Afghan security forces.

Even when all allied troops are eventually withdrawn from Iraq and Afghanistan, it must be recognized that a major, enduring burden has been imposed on personnel who have suffered traumatic limb amputations, and on their families. US and UK veterans' health care and rehabilitation services face an expensive commitment of years of work ahead to assist veterans in their adjustment to these disfiguring and life-changing wounds of war.

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The Canberra Class Landing Helicopter Docks (LHDs): A New Maritime Role 2 Enhanced (MR2E) Capability for the ADF

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The *Canberra* class Landing Helicopter Dock (LHD) ships will replace the LPAs *Manoora* and *Kanimbla*, and the LSH Tobruk. Planning for these new ships began in 2000, based on Australia's experience with INTERFET in East Timor. In 2004, invitations for tender were sought from a French company offering the *Mistral* class ships, and the Spanish company Navantia offering what became the *Juan Carlos* I design. The latter was selected in 2007, with Navantia responsible for building the ships from the keel to the flight deck, after which they will be transported to Australia for fitting of the island superstructure, by BAE Systems Australia.[1]
The roles of these ships will include:

- Embarking, transporting and deploying an embarked force (Army in the case of the ADF, but could also be an Allied army or marines), along with their equipment and aviation units, and
- Carrying out and supporting humanitarian aid missions.[2]

The provision of medical support for the LHD embarked force will be crucial to the latter's ability to meet its mission. The aim of this article is to describe some of the medical issues.

LHD Statistics

The LHDs are 230m long, 32m wide, 7.2m draught and displace nearly 30,000 tonnes. Although Navy has bought larger ships (in particular the tankers *Westralia* and *Sirius*), these are the largest ships ever built for the RAN.

They have a range of 6,000nm at 20 knots, or 9,000nm at 15 knots without refuelling. The flight deck has six spots for Blackhawk, Seahawk or MRH-90s, or four spots for Chinooks. They have two vehicle decks that can carry up to 110 vehicles depending on their size.

The LHDs also have a well dock that can carry up to four Landing Craft Mechanised, or LCM-1Es. They can be deployed up to Sea State Four, and operate over-the-horizon up to 20 nautical miles from their parent LHD.

The LCM-1E incorporates a stern gate, which allows the loading/unloading of vehicles up to 12 tons from LCM-1E to another. Propulsion is supplied by two diesel engines powering one waterjet each, allowing the LCM-1E to reach 22 knots empty, or 13.5 knots loaded. The maximum range at economical speeds is 190 nautical miles.[3]

Figure 1. A Spanish Navy LCM-1E[4]

The LHDs have bunks for 1403 personnel. Of these, about 240 bunks will be for the ship's company, plus about 160 more for the LCM-1E and flight deck crews. The remaining 1000 bunks are for the embarked landing force, aviation assets, HQ staff, and health personnel. The finite bunk space means that additional health staff means fewer non-health embarked personnel, and vice versa.

The size of these new ships can be illustrated by comparing them with the LPAs that many ADF health personnel are familiar with.

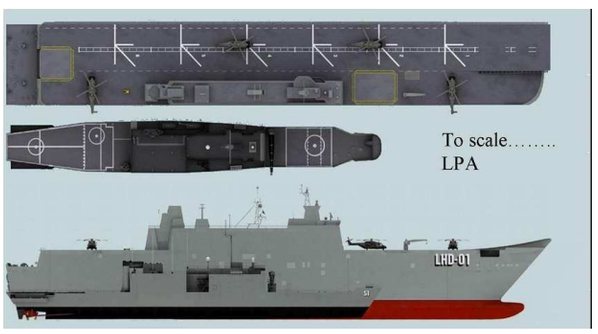

Figure 2. Comparison of LHD with LPA: side elevation[5]

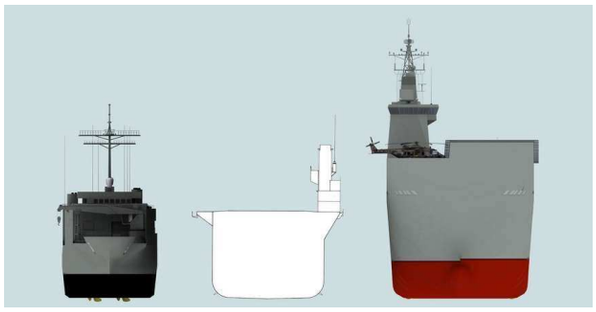

Figure 3. Comparison of LHD with LPA: bow view.[6]

The silhouette between is a cross section of the aircraft carrier HMAS *Melbourne* (II), which decommissioned in 1982. The LHDs are about 50% larger than the old carrier.[7]

Maritime Role 2 Enhanced (MR2E)

The LHD MR2E is located amidships, immediately below the hangar upper vehicle deck and above the lower vehicle/well deck. Patients enter the MR2E via a dedicated lift, either from the hangar or flight decks above, or the vehicle deck below.

From there they move to a triage/resuscitation area, then to one of two operating theatres, which are supported by a sterilisation area between them and two scrub rooms.

After surgery, casualties are moved to the High Dependency Unit (HDU), then to either the Medium Dependency Unit (MDU) or Low Dependency Unit (LDU). The latter uses the adjacent embarked forces cabins once they have moved ashore, on comparable terms as the troop messes aboard the LPAs.

These facilities are supported by x-ray, laboratory, and pharmacy. Primary health care is provided from a patient administration area, outpatient consulting room, medical office, and dental surgery. There are three medical stores, including a medical gas store.

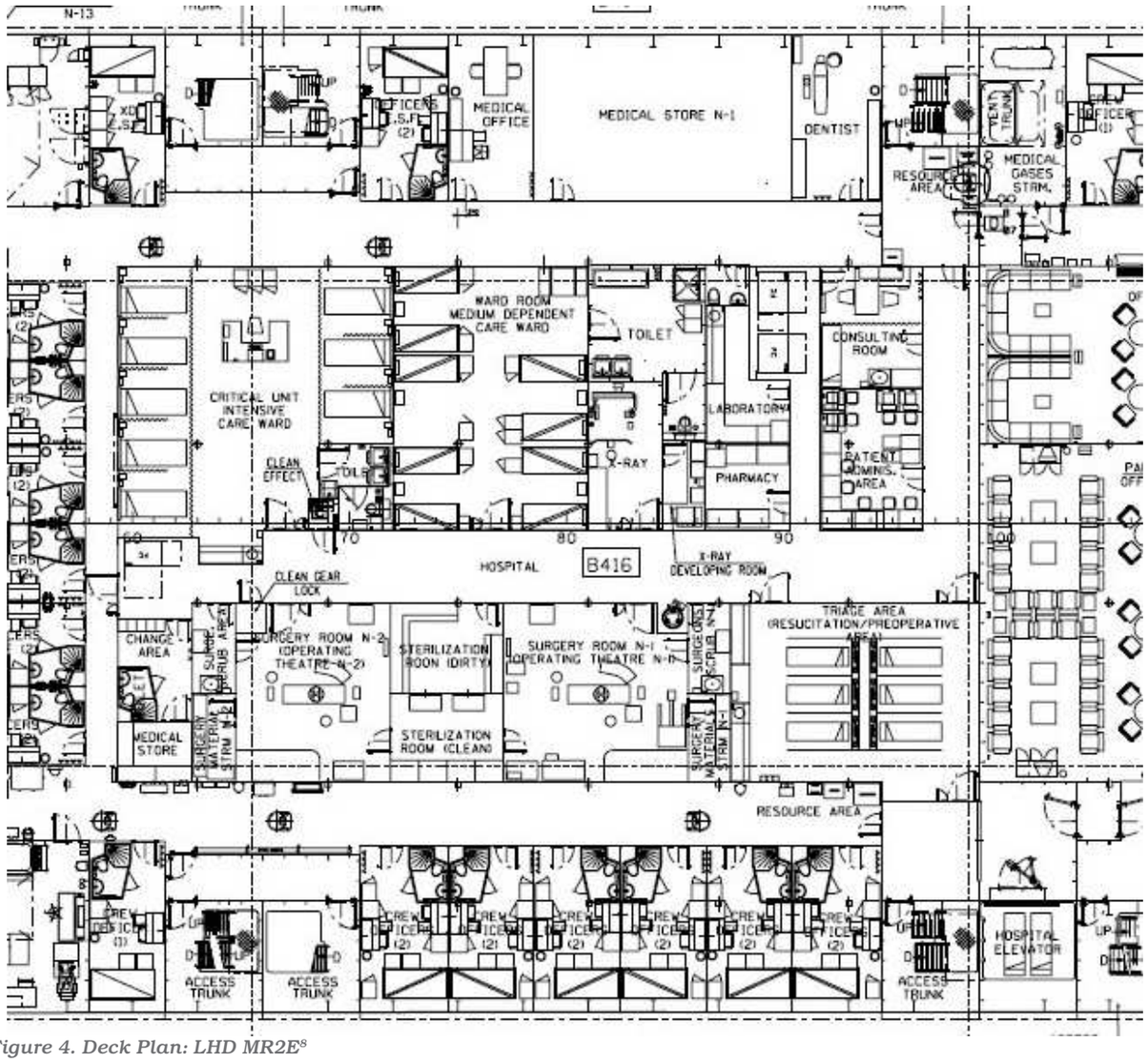

Figure 4. Deck Plan: LHD MR2E[8]

MR2E Staffing

Navy is required to sustain either one MR2E for an extended period or possibly two MR2Es for shorter periods. The MR2E response will be tailored to the mission, and delivered through capability bricks or elements organised by notice to move at 48 hours, 7 days, 28 days, and 6 months as follows:

- The elements at 48 hours notice will provide a limited MR2E capability. It will be staffed primarily by Permanent Navy personnel, although they may require additional Navy Reserve specialist support.
- The elements at 7 days notice will provide a medium level capability. It will be staffed by Permanent Navy personnel, some of whom will be augmenting garrison health support services when not undertaking MR2E duties as well as Navy Reserve specialist support.
- The elements at 28 days notice will provide a large level capability. It will be staffed by Permanent Navy personnel, some of whom will be posted to Joint Health Command positions with a MR2E obligation when required and Navy Reserve specialists.
- The elements at six month notice will provide the sustainment capability. It will be staffed by a combination of Permanent and Navy Reserve personnel.

The at times very short notice to move means it is necessary to maintain a high level of both clinical and military skills. This will be achieved by a combination of individual and group training at sea when the MR2E is activated for training, civilian hospital clinical placements, and the MR2E Training and Support Facility (TSF), which was established at HMAS PENGUIN in January 2011. The TSF has a medium fidelity simulator for individual and team clinical training.

LHD Timeframes

First steel was cut for *Canberra* in September 2008 and she was launched on 17 February 2011. She will be delivered to Williamstown in mid-2012, and is due to commence First Of Class Trials (FOCTs) in January 2014.

First steel was cut for *Adelaide* in February 2010. She is due for launch at the end of 2012, delivery to Australia in 2013 and to start FOCTs sometime in 2016.

HMAS Choules

Losing *Manoora* led to the decision to buy Largs Bay (now HMAS *Choules*) from the UK. She first commissioned as a Royal Fleet Auxiliary in 2006, and arrived in Australia in December 2011. She is about half the size of the LHDs, but roughly double that of the LPAs and three times that of *Tobruk*.

Choules has a crew of 158 plus 360 troops. She has two flight deck spots for Chinooks, and a docking well for a Landing Craft Utility (LCU) and two Landing Craft Vehicle and Personnel (LCVPs). She can carry 30 tanks or 150 light trucks (a capacity equivalent to that of the LPAs and Tobruk combined).[9]

Unlike *Tobruk*, *Choules* has stretcher access between the flight deck and the sick bay. The sick bay has a treatment room, one 6-bed and one 2-bed ward, bathroom, toilet and a medical store/dispensary. In addition, there is a room with one operating table that can be used for surgical cases.

Choules' sickbay is therefore similar in size and capability to an enlarged Tobruk sickbay, rather than a small LPA MR2E. This will pose some challenges in Navy's ability to provide a MR2E capability until *Canberra* enters service.

Conclusion

The LHDs offer the ADF a vastly expanded amphibious capability, compared to that provided by the LPAs and *Tobruk*. This particularly refers to the MR2E capability: although the LHD MR2E is generally comparable to the LPA MR2E in terms of theatre capacity (both have/had two operating tables), the on-board LPA medical supporting infrastructure (in particular bed space) is significantly greater.

This in turn poses particular challenges with respect to ensuring that these assets are supported by adequate numbers of appropriately trained and credentialed health staff, so that the MR2E can reach its full potential. The timeframe for decommissioning the LPAs has added to those challenges, as has the limited ability of *Choules* to be used for MR2E training to date, as well as the time now available for these staffing issues to be addressed before *Canberra* enters service.

Nevertheless, the introduction of the MR2E capability for the LHDs is clearly an exciting time for both Permanent and Reserve Navy Health personnel, which will stand them, the Navy and the ADF in good stead for the expected 30-year life of these ships.

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Pioneer Aviation and a Medical Legacy: The T.W. White Society Prize for Thoracic Research. A Tribute to Group Captain Sir Thomas Walter White (1888 - 1957) - Australian's pioneer military aviator

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Abstract

The twentieth century has seen many great inventions but few of greater significance than that of aviation. Within the single window of one century the world has seen not only the invention of flight, but also its ascendance to interplanetary probes. The discipline of military aviation likewise has developed from the first tentative flights of "those magnificent men" of the Royal Flying Corps to the development of aviation medicine as a crucial specialty not only within civilian aviation and space medicine but as a part of Defence Health more broadly.

It is appropriate that Australia's pioneer military aviator, Major T.W. White, should be commemorated particularly in an outreach prize for thoracic medicine. This Prize, the T.W. White Society Prize for Thoracic Research, commemorates the life and works of one whose service contributed significantly to both Australia as a Nation and to the genesis of military aviation.

Group Captain Sir Thomas Walter White (1888-1957)

Thomas Walter White was born in Melbourne; and in 1914 enlisted as a soldier in the First World War. He was one of the first four volunteers to be trained as a pilot in the A.I.F.; and was the first to be awarded his wings. As an Australian, he was a member of an exclusive group of several officers of the first Australian Half-Flight, which was raised in 1915. He was posted to the Indian Expeditionary Force D ("Force D") in May 1915. This pioneer unit of military aviation was posted to the Middle East; and engaged in its initial operations in Mesopotamia from May 19151. Captain Thomas White was then commissioned in the Royal Flying Corps from June 1915, but as he said "We retained our own [Australian] uniforms and always wore our "Australia" shoulder patches"1. Flying the early military biplanes of those pioneering days, the unit operated in the skies in the Ctesiphon region of Mesopotamia, flying over the Tigris and Euphrates Rivers and the northern littoral of the Persian Gulf. Whilst flying a low-level sortie, Captain White crashed his plane into a telephone pole some 12 kilometres south of Baghdad in November 1915 and was captured. He was physically abused by his Arab captors; and his life was undoubtedly saved by his being handed over to Turkish soldiers2. He kept a secret account (at mortal risk) of his captivity, including its abuses and privations, and recorded evidence of atrocities against the Armenians2. His notes became the basis for his book "Guests of the Unspeakable". In the traditions expected of captured officers of the day he refused to sign a "No Escape" document forced upon him by his captors. Together with a fellow British officer, he disguised himself as a Turk and escaped from captivity on board a train taking him north to further incarceration. He hid for 33 days aboard a Ukrainian steamer in port, before it finally sailed from Constantinople. Ultimately he made his way to a British Consulate in Eastern Europe prior to the end of the First World War.

White's experiences in captivity and his basic altruistic and humanitarian outlook lead him to

champion welfare programmes for those whose families who had been touched and disadvantaged by a breadwinner's service in the First World War. He supported Major General John Jellibrand's movement to establish the Legacy Club; this latter formed in Melbourne in September 1923. White promoted these ideals and entered Federal Parliament in 1929. He was to serve therein for 22 years, in a Cabinet Post as Minister for Customs and later as Minister for Aviation. After leaving Parliament he served as one of Australia's senior ambassadors. He was posted to London as Australia's High Commissioner and served in that post from 1951 until 1956.

At the outbreak of the Second World War, Major White transferred to the Royal Australian Air Force, and as Group Captain White served as Commanding Officer in Air Training Schools in Australia and at the RAAF Base in Bournemouth in southern England. It is recorded that:

> "Breaking all the rules concerning age, he flew as a co-pilot in several bombing raids over Germany"[2]

Knighted for his services to aviation, to humanitarian welfare and to the people of Australia, he died in 1957.

The T.W. White Society

The T.W. White Society was founded by a group of young officers of the Queensland University Regiment in 1982. Two of its founding members were Robert Johnston and David Monaghan - the former who became a senior administrator at the University of Queensland, and the latter a senior solicitor. The founder group had been boarders together at The Southport School on Queensland's Gold Coast, a School known for its traditions of public service and outreach. Whilst at University and whilst serving in their "second careers" in the Royal Australian Infantry, they saw a need to highlight the outreach and services of Australia's pioneer aviator; and to promote medical research, particularly in the area of thoracic medicine - a discipline which is so important in aviation health. Thus was established the T.W. White Society Prize for Thoracic Research.

The medical custodian of the Prize is The Thoracic Society of Queensland, which awards the Prize annually to the top researcher for his or her contributions to the advance of knowledge of thoracic medicine.

The Fusion of Science, Service and Military Art

Best-practice medicine today depends on the unity of science and of art; on the promotion of research and its communication to patients and doctors alike, and on the concepts of service. The T.W. White prize for medical research symbolises the need for balance between focussed specialisation on the one hand, and the retention of a broad outlook the continued mix of art and science in professional life — on the other. In the best of all worlds the specialist retains some of the outlook of the artist. Indeed, scientists are artists insofar as they create delicate instruments and refine methods for the purpose of discovery. It has been said also that "artists are scientists insofar as they seek knowledge of the world to help them with the creation of their sculptures, music or painting"[3]. Marble is handled in different ways by the sculptor when compared with that of the geologist[3].

It is possible to have the best of both worlds - the specialist as exemplified by the focussed medical researcher, and the broad perspective of those who render wide service to the community - particularly in applied sciences such as agricultural science, engineering and medicine. But how to be both a good specialist and one with the wisdom which perspective brings is a great professional challenge to all who follow the profession of medicine and of arms.

One approach is to attempt a superficial study of many interests. This does not necessarily give broad insight, and seldom brings wisdom. It has been said that a doctor or other health professional "cannot understand other peoples problems by interrupting his or her own work to climb a few feet up another's tree"[3].

An alternative approach is to adopt the historical paradigm. As each climbs his or her own tree, it is essential to look back and learn the history of one's own specialty in great detail. Such an approach soon leads to a study of origins; and the origins of our own specialty tree are entwined in the roots of others. In every case, if one does this, one sees the common ground of many disciplines and sees them in perspective. One sees, as in the early days of the development of aviation and of medical research both, the fusion of art and science.

Thomas Walter White's legacy, the T.W. White Prize for Thoracic Research, highlights the importance of maintaining a broad outlook, irrespective of whether or not one is working at the most minute point of the medical research coalface. The need to maintain a balanced approach is no better exemplified by examples in the history of respiratory medicine — those of the discoveries of Auenbrugger and Laennec.

Auenbrugger

Leopold Auenbrugger (1722-1809) was the pioneer of chest percussion, and one of the significant pioneers of thoracic medicine. He was appointed Physician-in-Chief of the Hospital of The Holy Trinity in Vienna in 1751. He was first to write concerning the use of percussion of the chest, in diagnosis. He was a consummate clinical artist[4] - but a great scientist also. Auenbrugger appreciated that sounds could be generated by tapping over an air-filled or fluid-filled surface; and that such could give much information about the nature of any underlying substance. He ascertained the different sounds obtained by tapping the chest wall; and conducted experiments to determine the changes in pitch and timbre over fluids injected into the chest of a cadaver, having injected them post-mortem. He extrapolated his findings into bedside practice.

It transpires that Auenbrugger was not a narrow clinician but a man of refined taste and broad interests in the arts and sciences. He was what would be called today a music "buff". He loved opera and wrote music. He composed the libretto for "The Chimney Sweep" (Der Rauchfangkehrer), written by Salieri[5]. Auenbrugger was indeed a specialist, but one whose roots were broad.

Laennec

Laennec (1781-1826) was the inventor of the stethoscope. Known to his mother (but perhaps not to the local street children) as Rene-Theophile-Hyacinthe, he was a great lover of the outdoors. He received a commission in the Army, and served as a Regimental Surgeon during the French Revolution. He was appointed Physician to the Hopital Neckar in 1816; and in 1819 invented the stethoscope — at first, only a cylinder of paper. He appreciated that sound could be channelled along hollow tubes; and that its air transmission was not as good as that emanating from a diaphragm or from a solid body. His invention of that first stethoscope and the development of its successors enabled the diagnostic sounds of cardiac and lung diseases to be placed on a reliable basis. Laennec was the first to describe the conditions of bronchiectasis, pneumothorax and haemorrhagic pleurisy. He was the first to use the terms of aegophony, pectoriloquoy and rales — these latter either sonorous or sibilant[5].

Like Auenbrugger, Laennec did not research or practice medicine in a social vacuum. His love of the outdoors was extended by his extensive skill as a horseman. It is said that it was whilst watching children at play - a boy tapping the end of a see-saw while another listened with his ear to the wood -that dawned the idea of the instrument which today we call the stethoscope. Laennec's book "Traite de L'Auscultation Mediate"[6], published in 1819, has been described as "the most important treatise on diseases of the thoracic organs ever written"[8].

The T.W. White Prize has its primary terms of reference, the encouragement of new discoveries in medical research. What new discoveries in thoracic medicine are waiting to be made? Percussion was invented only in 1751, and required no new tools - only an enquiring mind and the hands of the clinician. Auscultation with the stethoscope was invented only in 1819 and then needed only a tube of paper. Charcot, the French Neurologist, summed up the philosophy of the enquiring mind as the most important ingredient of research, by asking the question:

> "How is it that, one fine morning, Duchenne discovered a disease [muscular dystrophy] which had probably existed since the time of Hippocrates?"

In Grey's Elegy it was said that the good deeds of individuals might be "interred with their bones". Such is not the case with the example of service of Group Captain Sir Thomas White. His legacy of service today is the encouragement of research in thoracic medicine, a discipline which has become crucial to the further development of both underwater and aviation medicine; and to their civilian and military applications.

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Epidemiology

*Petra Buttner and Reinhold Muller**

**1st edn, (xxiv) + 600 pp, paperback with extensive illustrations, Oxford, Oxford University Press, RRP: $83.95, 2011. ISBN: 9780195573893.*

The field of epidemiology is a science and it remains "one of the many contributors to guiding action" in public health.[1] The field is crowded with published books with over 9500 books of varying publication dates on a recent Amazon.com search.[2] However, few of these books are relevant to the Australasian context and none appear to be recent. The 1st edition of *Epidemiology* fulfils the need for a reference textbook in this field relevant to the region and one which will no doubt establish itself as one of the leading reference textbooks in Australasia in the field of epidemiology.

Epidemiology is presented as a 600-page A5 publication that would fit easily into the briefcase or carry bag. It contains a table of Contents, a List of Figures, a List of Tables, a Preface, Acknowledgments, a Table (Find your level: From introduction to beyond the basics", 14 chapters, seven Appendices, and a comprehensive Index. There is no foreword, bibliography, glossary, or list of abbreviations. There are however explanations of key terms throughout the textbook. The primary target audience of *Epidemiology* would be students, particularly postgraduate students, and health professionals throughout Australia. It would also be a useful resource for any school or library in the health sciences area and a core reference for any school of public health.

Chapters include "1. What is Epidemiology?"; "2. Disease Concepts in Epidemiology"; "3. Identification of Disease - Diagnostic Tests and Screening"; "4. Measures of Disease Frequency"; "5. From Research Topic to Research Hypothesis"; "6. Quantitative Descriptive Study Designs"; "7. Experimental Designs"; "8. Observational Designs"; "9. Sources of Bias"; "10. Sampling Strategy and Sample Size Calculation"; 11. Quantitative Methods of Data Collection"; "12. Statistics with Confidence"; "13. Ethical Considerations"; and "14. How to Read and Write Scientific Publications". Of particular interest to students and teachers of epidemiology would be the locally drawn case studies and critical thinking exercises (answers given on a separate page). Needless to say, well-known historical examples, such as that described by John Snow, are also given. The chapter on "Ethical Considerations" (Ch. 13) is refreshingly well developed and helps to round off the attractiveness of the textbook to clinical researchers. Details of the authors are given on the back cover. Both are Associate Professors in the School of Public Health, Tropical Medicine and Rehabilitation Sciences, James Cook University.

The consistent and concise style ensures that *Epidemiology* is easy to read. Given that this is only the first edition of the *Epidemiology*, it is a remarkably mature reference textbook, which is a credit to the authors, reviewers and publishers. *Epidemiology* has little competition within the Australian context, although there are numerous textbooks in this field internationally. Some recent examples of other international textbooks of epidemiology targeting different groups have been reviewed elsewhere.[3,4] *Epidemiology* will certainly appeal to those undertaking research in the health sciences in Australasia, particularly those who do not already use a similar textbook or those who wish to include a local work in their reference portfolio. The cost is not prohibitive for clinicians and other health staff, and the *Epidemiology* is sure to become an important addition to the exclusive international portfolio of standard textbooks in the area of epidemiology.

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Declaration of Interests

The authors of Epidemiology are staff members in the same School as the reviewer. The reviewer did not participate in the production of this textbook.

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Rear Admiral Graeme Spencer Shirtley AM RFD RANR MB BS (NSW), DDR (Syd), FRANZCR Born 17 August 1950 Died 27 March 2012

Neil Westphalen

Rear Admiral Graeme Spencer Shirtley AM RFD RANR served as Surgeon General Australian Defence Force (SGADF) from 09 May 2005 to 03 July 2008. During that time he was AMMA Patron, where he provided strong support for its development, aims and objectives.

Graeme was born at Epping NSW, to Spen and Gwen Shirtley, on 17 August 1950. He was educated at Beecroft Public School and Epping Boys High School, where he completed his HSC in 1968, and won a scholarship for entry to the medical school at the University of NSW.

After graduating in 1974 and subsequent residency, Graeme commenced his radiology training. During his time at the Prince of Wales and Prince Henry Hospitals, Graeme met his future wife Debbie, with whom he had three children (Laura, Mark and Ian). Notwithstanding his many professional achievements, Graeme's greatest pride always remained his family.

Graeme was awarded a Diploma of Diagnostic Radiology from Sydney University in 1979 and his Fellowship from the Royal Australasian College of Radiologists (now the Royal Australian and New Zealand College of Radiologists) in 1980. He was in private practice in Sydney, developing his special interests in CT imaging, musculoskeletal imaging (particularly with ultrasound) and mammography. He was Chairman of his radiology group in 1995-9.

In addition, Graeme was a senior visiting medical officer with the Central and Eastern Sydney Breast Screening Program at the Royal Prince Alfred Hospital from 1989 to 2003. He was also chairman of the CT group of the RANZCR Accreditation and Quality Control Subcommittee, and the radiologist on the Professional Services Review Committee of the Health Insurance Commission.

In 1992 Graeme became a Visiting Fellow in MRI at the Barrows Neurological Institute in Phoenix Arizona, and at the MRI Institute Presbyterian Hospital in Pittsburgh Pennsylvania. He was also a member of the Radiological Society of North America.

On the Service side, Graeme joined the Navy as a reserve junior sailor in 1969. After topping his recruit course, he joined the Medical Branch as a Reserve Ordinary Sick Berth Attendant (ORD SBA, now SMNMED). Over the next six years he was promoted through the ranks to Leading Seaman in

the Reserve, while continuing his undergraduate medical studies.

On completing his medical degree, Graeme spent a brief period as a somewhat overqualified Reserve leading seaman medic. Although this rather appealed to his sense of humour, he nonetheless had to be commissioned as a Lieutenant RANR.

Graeme progressed to Lieutenant-Commander in 1981, and to Commander in 1987. From 1985 to 1987 he was Deputy Senior Medical Officer (SMO) of the Sydney Port Division, and in 1986 he completed and topped his Reserve Staff Acquaint Course. That year he was also awarded his Reserve Forces Decoration (RFD).

Following his promotion to Commander, Graeme became SMO of the Sydney Port Division. He was the senior reserve Medical Officer for Exercise Kangaroo 89, and was acting Executive Officer of the Sydney Port Division from 1991. In 1992 he was awarded a Flag Officer's Commendation for developing a program for training of Reservists in military medicine.

Graeme's seagoing service included the aircraft carriers Melbourne and Sydney, as well as Vendetta, Torrens, Stuart, Brisbane, Stalwart, Supply and Darwin. He also served ashore at Cerberus, Penguin, Kuttabul, Albatross, and Stirling, and in Canberra.

Graeme was appointed consultant radiologist to the Director General Naval Health Service in 1985, and to SGADF in 1990. In 1986 he was appointed ADF representative to the radiology committee of The Standards Association of Australia. He was a member of the SGADF Radiology Steering Group from 1985, and was appointed the inaugural chairman when it became the Medical Imaging Consultative Group in 2000.

Overseas, Graeme was a visiting lecturer to the Department of Radiology National Naval Medical Centre (NNMC) in Bethesda Maryland USA in 1994, 1998 and 1999, for which he was awarded the US Navy and Marine Commendation Medal, and the US Navy Achievement Medal. He was also a guest lecturer at the Uniformed Services University of the Health Sciences (USUHS) in Washington DC in 1998, and was appointed USUHS Adjunct Assistant Professor of Radiology and Nuclear Medicine in 2002.

Graeme researched the US military experience with computed and digital radiography systems, with a view to their implementation into the ADF as part of Joint Project 2060. He was a member of the Association of Military Surgeons of the United States and was appointed to its International Committee in 2006.

Graeme was promoted to Captain RANR on 31 December 1998 and appointed Director Health Reserves-Navy (DHR-N) the following day. In July 2000 he was appointed the inaugural chairman of the National Reserve Health Triumvirate. Whilst DHR-N, Graeme worked to achieve rank equity for Navy health officers compared to their Army and Air Force counterparts. Other issues included training, age of retirement and a new scheme of complement for naval health specialist reservists.

During this time, Graeme was the Reserve representative on the Naval Health Board Advisory Council. He also published articles in ADF Health and Australian Military Medicine on the military aspects of ultrasonography, telemedicine, virtual endoscopy and restructuring of the Navy Reserve Health Branch.

On 27 September 2002 Graeme was promoted to Commodore and appointed Assistant SGADF – Navy. In this role he liaised with State Departments of Health to establish strategic alliances with the teaching hospitals to increase the experience of Permanent Forces doctors, nurses and medics in trauma management. Graeme also established 'mini-fellowships' in trauma management in South Africa for reserve surgeons, anaesthetists and intensivists, established an affordable pilot e-health system for the ADF, and investigated personal digital assistants as an aid in the delivery of ADF health care.

In October 2004, Graeme became the first Reserve officer to undertake the Capstone program, a one-week live-in staff acquaint course for one-star officers.

On 09 May 2005 Graeme was promoted to Rear Admiral and appointed SGADF. As such he was the first Navy medical officer to become SGADF since that position was established in the early 1980's, the first to achieve the rank of Rear Admiral RANR, and the first to achieve the rank of Rear Admiral since Geoff Bayliss (DGNHS 1987-1990).

Besides chairing the Australian Defence Human Research Ethics Committee, Graeme was Adjunct Associate Professor at the University of Queensland in the Centre for Military and Veterans Health (CMVH), and Chairman of the CMVH e-Health Committee. He also continued to conduct courses in ultrasound for trauma surgeons, as part of the Royal Australian College of Surgeons teaching program.

On 04 July 2008, with the restructure of the senior ADF health leadership, Graeme was appointed Surgeon General Defence Health Reserves until 31 December 2008. He was appointed a Member of the Order of Australia in the Military Division in the Australia Day 2011 Honours List, for his exceptional performance of duties as a RANR medical officer.

Graeme always loved sport, either as a participant or a spectator. He represented Beecroft and later Strathfield in the Sydney Grass Court Tennis competition, and was a member of the Pennant Hills Golf Club Junior Pennant Team. Debbie later introduced Graeme to skiing, initially at Thredbo and later the Silver Star resort in Canada, which became his favourite.

After his term as Surgeon General, Graeme decided not to return to full time radiology practice but to relax a little, pursue his sporting interests and spend more time with his family. Tragically, these plans were curtailed by the onset of a brain tumour in March 2010. He underwent surgery and made a good recovery, however he died on 27 March 2012 after its expected recurrence.

Graeme was proud to be Patron of the Association, and Patron of the Navy Reserve Association, an honour normally reserved for seaman officers. His gregarious nature, enthusiasm and sense of humour made it very easy for anyone to forge a bond with him. He was a consummate gentleman who could manage the senior level politics while always giving a balanced opinion. These attributes were matched by his integrity, selflessness, common sense, and loyalty to his family, his civilian private practice staff, his Service and his Nation.

Graeme's family can be exceptionally proud of his long, distinguished military and clinical career. He will always be sadly missed by his military and civilian friends and colleagues.

Thanks for everything Graeme. God has many rooms and places for everyone, and we know that your sense of humour and humanity will brighten His day forevermore… even if you've started far sooner than any of us would have preferred.

Fair winds and following seas.

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