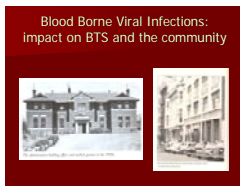


ARCBS Meeting, Sydney May 8, 2009



Ben's invitation has sparked a lot of memories – as I spent the first 25 years of my career at Fairfield Hospital for Communicable Diseases working largely on blood-borne viral infections – much of the time in close collaboration with Jack Morris, his team at the Melbourne Blood Bank and his colleagues interstate.

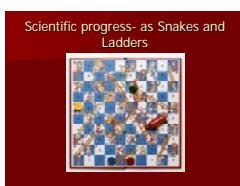
I thought it might be interesting to try revisit those times and some of the issues we faced, recognising that memory is not always reliable and inevitably selective.

It was a fascinating period, particularly for the Blood Transfusion Service and the nations plasma fractionator, CSL, as each moved from being a chronically underfunded, relatively low tech, protected, cottage industry accountable (through Boards of varying expertise and competence) only to God or the Minister – although not always in that order, and regarded fondly by the community – a bit like the Salvo's or the Country Women's Association, to the well funded, high profile, highly professional and highly regulated organisations of today.

It wasn't all straightforward, and reminds me that while, from the outside scientific and organizational progress may seem to be logical and carefully planned



A bit like a Grandmaster playing chess,



It is more like a game of snakes and ladders



and is often dependent on a fair slice of luck

There's no doubt that I've been very lucky.

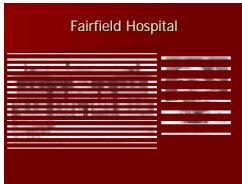


I graduated in December 1964 and started as Resident Medical Officer at the Alfred Hospital the following year.

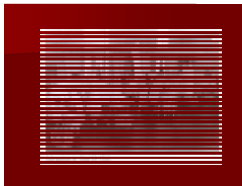
Life was tough. We worked 100 hour weeks, had 1 day off a fortnight and were paid the princely sum of £1000/yr of which the hospital took £250 for board and lodging. We lived in, with the male residents two to a room.

The prize job for an Alfred Hospital Resident was a 3 month rotation at Fairfield Hospital, where the work was not only fascinating, the pace more relaxed, the staff congenial, the food excellent but we had our own room.

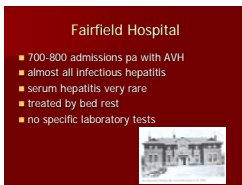
I lucked out and was rostered there over the summer of 1965/6, decided to become a medical virologist and apart from periods in the US and UK, remained there for the next 25 years.



Fairfield was a fascinating place – built by public subscription at the turn of the century it comprised a series of high roofed pavilions with wide verandas linked by covered walkways, set in a park like environment. It was led by John Forbes, a craggy faced, hard drinking, WWII veteran who lived on site and ran the hospital like a personal fiefdom. He was intensely loyal to his staff and encouraged everyone to be involved in research, because he was convinced that clinical practice could only progress through first class research.



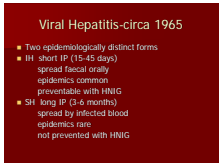
As well as multipurpose isolation wards, Fairfield had a ward full of patients in iron lungs – from the polio outbreaks of the 1950's and wards allocated to children with gastroenteritis, bronchitis or croup. Ward 20, the largest and most remote ward, was devoted to patients with viral hepatitis.



Viral hepatitis was a major public health problem in Australia in those days and Fairfield admitted some 7-800 patients with the disease each year, all of which were assumed to be, what we then called, infectious hepatitis (HA). Serum hepatitis (HB) was regarded as very rare and in the first 2 years I was there, I don't recall a single case coded as serum hepatitis.



The laboratory tests available for confirming a diagnosis and monitoring progress were crude and non specific – thymol turbidity, Cephalin Flocculation tests were widely used - the first specific tests for elevated levels of liver specific enzymes, SGOT and SGPT, were just being developed. Patients were treated by bed rest and allowed to go home in 3-4 weeks, when their bilirubin levels had declined to close to normal and they were assumed to be non-infectious.



Our knowledge of viral hepatitis at the time was gleaned from studies conducted in human volunteers, during WWII, by the British and American armies. These had demonstrated the existence of two aetiologically and epidemiologically distinct forms of the disease, which could be transmitted by bacteria free filtrates and were presumably caused by viruses. The diseases were then known as infectious and serum hepatitis rather than their current names HA and B.

Infectious Hepatitis – which was a particular problem for the military - had a shorter IP and was spread faecal orally; epidemics were common (especially associated with contaminated food or water) and the disease was preventable with γ globulin (HNIG).

By contrast serum hepatitis – which appeared to be rare, had a long incubation period - was transmitted by inoculation of infected blood or blood products – and was not prevented by HNIG.



Post Transfusion Hepatitis – presumably due to the SH virus, was a well known complication of blood transfusion. An Australian survey in the 1940's had shown that about 3% of recipients of blood who lived long enough, developed clinically apparent PTH (compared to 8% in the UK and over 20% in the US which relied on paid donors).

Post transfusion hepatitis was accepted as a risk of transfusion and tolerated because the risk/benefit equation was generally favourable.



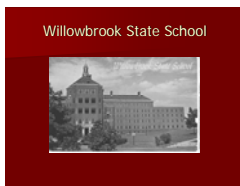
In the 1960's most Australian doctors who wanted to specialise, gained their post graduate qualifications in the UK. So in March 1967, I travelled to

London to study at the London School of Tropical Hygiene, working my way as the ships Doctor on the old English Star, which loaded apples in Port Huon, Albany, Bunbury and Fremantle before heading to the UK via the Suez Canal – a journey which took 8 weeks. By good fortune we were in the Red Sea when the 6 Day War broke out and were able to turn around and reach England via the Cape of Good Hope.

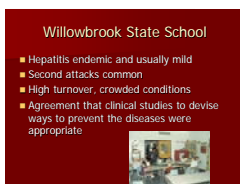
Our Sister Ship, the Scottish Star, which had been moored alongside in Fremantle and was a day ahead of us, wasn't so lucky. She was trapped in the Bitter Lakes – the crew not rescued for 6 months and the ship eventually sold for scrap metal.



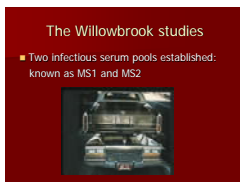
Just before I left, Fairfield had a visit from Saul Krugman a distinguished American paediatrician, who worked at the old Bellevue Hospital in New York and was a good friend of John Forbes. With his colleagues Bob Ward and Joan Giles, Krugman had just been recruited by the Willowbrook State School, on Staten Island to advise them on the control of infectious diseases.



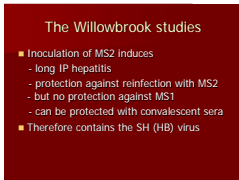
Willowbrook was a large, overcrowded institution for mentally retarded children, with a high turnover of patients and staff. Epidemics of respiratory and enteric disease, measles and rubella were common and hepatitis was endemic.



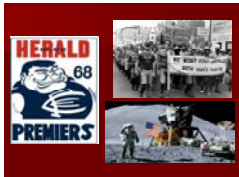
A high proportion of children acquired the disease in the first year after admission and second attacks were common, suggesting both strains of the virus were circulating.



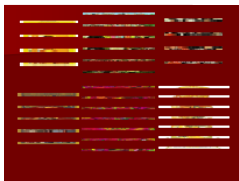
Because the disease was so mild, Krugman obtained consent from the parents to deliberately infect children to try and devise ways of protecting them against the disease. Starting with samples from one child who had two separate attacks of hepatitis, he established two infectious pools of serum ... named MS1 and MS2 which induced short and long incubation period hepatitis respectively.



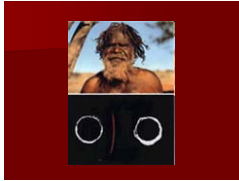
While children fed or inoculated with MS1 developed short incubation period hepatitis and were protected from re-infection, children inoculated with MS2 developed long incubation period hepatitis – were protected against re-infection from MS2 – but not from challenge with MS1. Krugman’s greatest contribution may have been that he collected and stockpiled pre and post infection sera from these studies, which made it possible to readily test candidate aetiological agents.



1967 and 1968 were extraordinary times - not only did Carlton win its 1st premiership in 20 years it was the time of the student revolts in Paris and Berlin – the Great Vietnam War demonstrations – and the first landing on the moon.



London was at the centre of the universe – it was the time of the Beatles and the Stones, Tariq Ali and Daniel Cohn Bendit. The London School of Hygiene and Tropical Medicine was an exciting place to work, especially as the Head of Microbiology, Ari Zuckerman was passionately interested in viral hepatitis and had a large team trying to identify the aetiological agents.



A year earlier Barry Blumberg had described the existence of a novel precipitating antigen in the serum of some Australian Aborigines (the Au/Ag) and noted that it seemed to be more common in people with leukaemia and institutionalised Down’s syndrome patients.



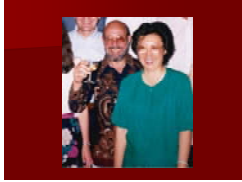
He then had a bit of luck.

In his studies, Blumberg used the serum of one of his lab technicians as a negative control. The young women then contracted hepatitis and was off work for several weeks. Frustrated because his negative control was exhausted, Blumberg went to

her home, bled her, separated the serum and included it in his next test, only to find that she had become Au Ag positive.

This was the first hint that the antigen might be a marker of one or both forms of viral hepatitis.

It took a blood banker to resolve the dilemma.

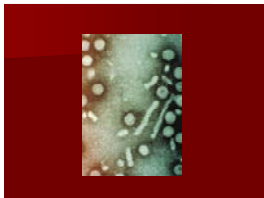


I remember going into the library of the London School of Hygiene and Tropical Medicine in 1968 and coming across a copy of *Vox Sanguinis* with Fred Prince's paper conclusively showing that Blumberg's antigen was a specific marker of the virus causing LIP hepatitis.

Prince who worked at the New York Blood Centre had anticipated that candidate agents would be discovered and had undertaken a long term follow up of patients receiving multiple blood transfusions some of whom had developed PTH.

When he tested those samples under code, he found that patients, who developed long incubation period hepatitis, became Au Ag positive during the IP and that Ag titres peaked shortly before the onset of symptoms. In each case, one of the donors was also Ag positive – just as would have been expected from an aetiological agent.

I rushed to Ari Zuckerman's office to show him the paper and was surprised by his dismay – he had clearly hoped to get there first.



Prince's findings were confirmed by blinded studies on Krugman's Willowbrook samples and the final piece of the puzzle solved, when Manfred Bayer and David Dane showed that the antigen was present on the surface of virus like particles – the hepatitis virions.



In late 1968 I moved to Glasgow to continue my studies with Norman Grist in the Regional Virus Laboratory at Ruchill Hospital and became part of a group which played squash on a Wednesday night, with another Australian expat, working at the Moredon Vet Lab down the road – Peter Doherty.



In those pre fax, pre internet, pre international meeting days, personal contacts were critical. I corresponded with Prince and Blumberg and arranged to visit both on my way back to Melbourne in late 1969, which I did, even marching with Blumberg's wife in a huge anti Vietnam War rally in Philadelphia.



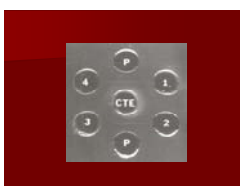
Both were incredibly generous and I left the US with an amazing gift, several sealed glass ampoules containing Au Ag and high titre chimpanzee anti sera raised against it – which I wrapped in cotton wool, packed in a cigarette tin which I put in my luggage and walked through customs with.

Imagine trying that today?



With the help of my Fairfield colleagues, biochemist Jacov Kaldor and clinician Ron Lucas I set up an AGD assay and from December 1969 began testing sera from every patient admitted to Fairfield Hospital with suspected viral hepatitis.

It was a Heath Robinson system.



Every day I would pour agar onto 3 1/4" square glass photographic plates, punch a rosette of holes, place the high titre chimpanzee antibody containing sera in the central well and test sera around the periphery and incubate the plates in a humidified chamber. Tupperware container and wet blotting paper.

I lived about 10 minutes walk from the hospital and would set up the plate in the late afternoon, go home for dinner then rush back to see if any lines had developed. The following morning, Ron Lucas and I would head to the ward to interview the patients and obtain relevant epidemiological information.

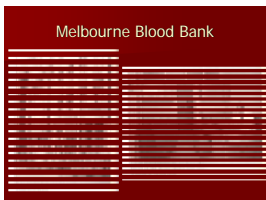


From these and similar studies around the world it quickly became apparent that infection with HB was much more common than previously imagined – that acute infection was often associated with onset of a chronic state – that the prevalence of chronic carriers varied enormously world wide and that long term carriage was associated with an increased risk of developing chronic active hepatitis or primary hepato cellular carcinoma.

It was clear that these findings have implications for Blood Transfusion and Plasma Fractionation services. Through Brian Farragher, who with Ron Sawer, ran the Haematology Department at the Alfred, and was the consultant haematologist at Fairfield in early 1970, I was able to access sera from a number of patients with haemophilia being treated at that hospital.

By chance one had high titre antibody to HB – and regular, generous donations from him, enabled us to replace the precious chimps anti sera provided by Fred Prince, with local material.

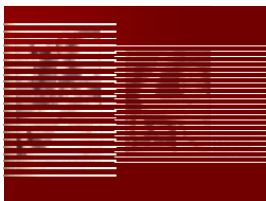
Although, in those days, the Blood Bank was somewhat isolated from clinical and academic medicine, initiating contact proved remarkably easy. I had met the Director, Jack Morris through our joint membership of the State Branch of the RCPA and my wife had just started working as a part time Medical Officer at the Blood Bank.



In those days the transfusion service was located in Flinders Street, near the old Herald Sun building and Lou Richards Pub The Phoenix in a rather down at heel area.

Jack, whose office was as spare as the surroundings, was a marvellous man, highly intelligent, curious, considerate and collegial, an ideal collaborator.

He was devoted to his donors, and took great pains to ensure that they were healthy including taking a history to exclude a PH of malaria or hepatitis conducting a physical examination and performing limited blood tests.



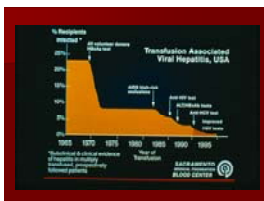
Jack recognised the need to introduce routine screening of all donations for HBs Ag and delegated implementation to his deputy, Murray Verso and serologist Rachel Jacobowitz.

By this time we had moved our testing at Fairfield Hospital from agar gel diffusion to counter immuno electrophoresis because of its higher throughput and greater sensitivity. Over a period of weeks we trained key Blood Bank staff on how to perform the assays, helped them select and commission appropriate equipment, and acted as trouble shooters for problems or doubtful results and performed the same service for regional collections centres. To ensure the quality of these programs, we established and circulated proficiency panels and became the conduit for a number of local and international reference reagents. The Fairfield lab was designed as a WHO CC for Viral Hepatitis. The annual meetings of Directors became an invaluable forum for receiving new information long before it was published.

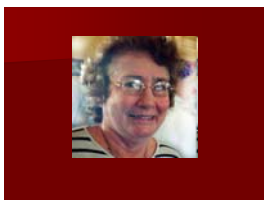
In 1974, Abbot Laboratories, under the scientific leadership of Lacy Overby, Isa Mushawar and Jean Pierre Allain developed a radio immunoassay for HBsAg which was automated about a year later. This was a quantum leap in technology for the Red Cross with a commensurate quantum leap in cost – the harbinger of a new era.

Today IA's are used to screen against a formidable array of BB viruses.

In 1978 I spent a sabbatical year with Bob Purcell's group at NIH. With Bob's drive and NIH's resources, including access to a large colony of marmosets and juvenile chimpanzees, his lab had become the centre of the hepatitis universe. Bob and John Gerin had developed and were testing a prototype hepatitis B vaccine, Dick Daemer, Steve Feinstone and I had isolated HAV in cell culture and were developing candidate HA vaccines, Mario Rizzetto was conducting his pioneering work on HBeAg while in the Clinical Centre, Harvey Alter was beginning his classic studies on NANB hepatitis. These were exciting times and an important set of contacts.



World wide, the introduction of screening tests for HB in the early 1970's led to a dramatic reduction, but not elimination of post transfusion hepatitis. As can be seen from this data from the Sacramento Blood Transfusion Service where the incidence of PTH fell from 23% to 7%.

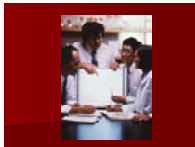


A study by Yvonne Cossart and her colleagues in Sydney in the early 80's showed that the rate in Australia had fallen to 1.7% or about 4 cases per 1000 units blood collected.



In the mid 1980's in the absence of specific markers for the remaining causes of post transfusion hepatitis, a debate took place whether surrogate markers, such as elevated ALT levels or the presence of anti HBc might detect donors at increased risk of transmitting the disease.

These tests were introduced into some overseas blood banks and closely monitored by Harvey Alter with variable, generally modest effect, but because of their lack of specificity, resulted in the loss of many donors.

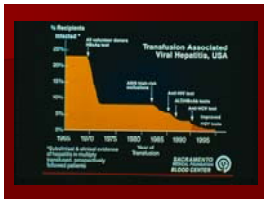


In July 1987 Australia decided to conduct a prospective study of the value of these markers – which was overtaken by subsequent events.

In 1989 following collaboration between Dan Bradley at CDC and Mike Houghton and his team at the Chiron Corporation and the use of modern molecular biological approaches, the Hepatitis C virus was identified and a relatively crude 1st generation diagnostic assay established.

Through my friendship with both Houghton and Alter our lab was involved in the evaluation of early versions of this assay and was again able to assist the Blood Bank in its introduction. The assay was rapidly introduced, making Australia the 2nd country in the world to have such a regime in place.

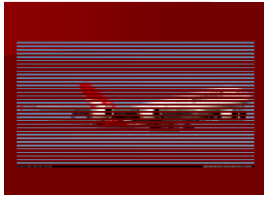
The pace of development was so swift that within 2 years these were supersede by assays of greater, sensitivity and specificity.



While this additional screening led to a loss of some 0.8% of donors the incidence of post transfusion hepatitis fell to very low levels.



When I recall the blood bank laboratories of the early 1970's with their crude labour intensive, Heath Robinson systems with the fully automated



computerised labs of today it's like comparing flying in a tiger moth to the new A380.



While hepatitis alerted the transfusion community to the potential of blood borne viral infections, the disease which shattered its innocence, was AIDS.

Sometimes events occur which are so dramatic, that everyone can tell you exactly where they were and what they were doing when they occurred.

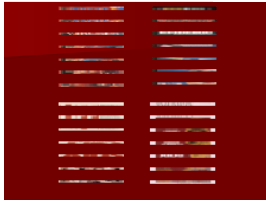
For my parent's generation it was the Great Depression and WW2, for me the Cuban missile crisis, the assassination of JFK and man landing on the moon;



for my kids it's probably 9/11 and Black Saturday.

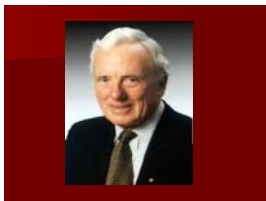


But sometimes equally momentous events sneak up on you by surprise their importance only being recognised in retrospect. Such a moment occurred for me in 1982 in Auckland. I was attending an early meeting of the ASID and over a morning tea discussion with a visiting US, infectious diseases physician, Morton Schwartz heard of the first time of the unusual cluster of cases of severe opportunistic infections in groups of promiscuous male homosexuals in California and New York, which was accompanied by a profound immunosurppression and selective depletion of CD4 cells. The disease, which became known as GRID (Gay Related Immunodeficiency) was thought to be a medical curiosity – possibly a toxic effect of Amyl Nitrate. Of course it was AIDS – a disease, which was to change all our lives. As the disease began to spread and its predilection for homosexual men and IV drug users became apparent, the press and the religious right had a field day.



It was impossible to open a newspaper or magazine or turn on the TV without encountering stories on AIDS – a bit like the saturation coverage of the H1N1 outbreak recently.

At first Australia regarded itself as somewhat of a bystander but publication of the first case report in April 1983 was evidence that we were unlikely to be spared.



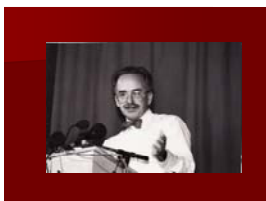
The Government moved quickly, establishing the NHMRC Working Party on AIDS in July and installing David Pennington – then Head of the National Blood Transfusion Committee – as its Chair. The WP included several members of the blood community including Bob Beal (SA RCBTS) Peter Schiff from CSL and myself.



Then we had several pieces of luck.

Firstly, my colleague Ron Lucas was invited to spend a sabbatical with Jim Curran's newly established AIDS group at CDC, where he was involved in several key meetings with blood bankers and plasma fractionators and I was invited to a meeting in Geneva to help WHO plan its response, to what seemed likely to become a major public health problem.

I've already mentioned how important personal contact was for access to the gaining latest information. In addition we had a secret weapon, unlimited use of one of Telstra's first IDD phones. Telstra was just expanding its international communications and wanting to test the reliability of the system. Graeme Laver at JCSMR who was collaborating with US and Russian scientists to grow influenza neuraminidase crystals in space and I, who was working with NIH on development of the HA vaccine, were given access to a phone each – held in locked cupboards in our offices. By dialling an amazing sequence of numbers we were able to communicate with international colleagues as though we had internet access. It proved a huge advantage.



The meeting in Geneva comprised about 20 people, mainly virologists and epidemiologists familiar with blood borne viral infections.

Fakery Assaad who had convened the meeting had invited two outsiders, a young CDC trained epidemiologist Jonathan Mann, who was working in Uganda and was familiar with the inroads the disease was making. Jonathan went on to become Director of the Global Program for AIDS (and perished in a tragic plane crash).



The second outsider was Luc Montagnier, a virologist from the Pasteur Institute in Paris, last years Nobel Laureate. By chance I was sitting beside Luc as he described in broken English his belief that he had identified the causative virus which he had named LAV, the lymphadenopathy associated virus.



These claims were met with almost universal scepticism. During an evacuation for a fire drill, we started talking and he agreed to send us, samples of LAV infected cells so that we could set up our own assays and attempt to confirm his work.



Over the summer of 1983 – 84 he made two attempts, both of which failed as the cells failed to survive. Later in the year I sent my colleague Rob Prinagle to CDC to acquire the technology which enabled us to establish local assays for HIV infection. It was not until Montagnier's work was confirmed by Bob Gallo at NIH, that the aetiology of the disease was universally accepted.

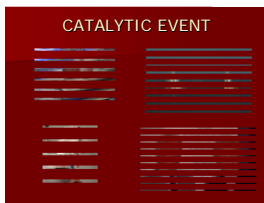
During the period that we were trying to establish the assay, AIDS had been detected in some American patients with haemophilia, raising concern that the US blood collection system was contaminated. Ron Lucas sent back a long letter describing a meeting between CDC epidemiologists and members of the US blood collection industry. When evidence was tabled that some patients with AIDS appeared to have acquired it by transfusion – many blood bankers argued that this was probably a chance association, leading Don Francis to demand “how many deaths would satisfy your need for statistical significance?”

Francis persuaded the blood bankers that they had a problem and CDC began studies to determine whether heat inactivation of clotting factors, would inactivate the virus. Information that Ron Lucas relayed to me on a regular basis and which I passed on to Peter Schiff. About this time the US Government issued samples of

the virus to 6 International manufacturers of diagnostic tests and invited them to devise assays which could be used to screen blood donors. The names of the manufacturers are a kind of fossil record of the diagnostics industry, Electronucleonics, Litton, Du Pont, Travenol, Organon and Abbott only one of which remains today.

When Robbie Pringle returned from CDC in August 1984, I contacted Ron Sawers, Brian Farragher and Henry Eckhart, who cared for many Victorian patients with haemophilia and was able to access fresh and stored samples of serum. We tested them and found, not only were 20% already infected with HIV, but that there was evidence that infection had been occurring since 1981.

The concept that supposedly life saving therapy was putting their patient's lives at risk, was devastating to some clinicians and led to recrimination and legal action, both here and abroad.



November 1984 rolled around and as Australia prepared for a Federal Election a catalytic event occurred which knocked politics off the front pages. John O'Duffy a Queensland paediatrician was asked to see a baby that was failing to thrive, had developed an opportunistic infection and had a low T cell count. The case was unusual and the following weekend he mentioned it to a pathologist friend, while they were out sailing.

The pathologist said that it reminded him very much of a child on whom he had recently performed an autopsy.

On Monday morning, they returned to the hospital, pulled out both children's records and found that not only had both been premature, but both had received top up transfusions from the same donor, a middle aged unmarried man.

Two other Aliquots of this donors blood had been transfused to two other babies, one of whom had died shortly afterwards, the other whom had developed similar symptoms.

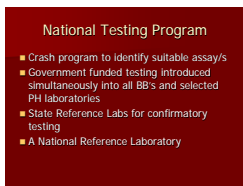
O'Duffy wondered if the 3 children could be suffering from paediatric AIDS; he rang me and arranged for samples from the donor and index case to be flown down to Melbourne. We tested them overnight and both were positive.

To say that this caused concern was an understatement.



With the election looming, Neil Blewett, the Federal Health Minister called a crisis meeting of Health Ministers in Melbourne for Sunday November 18.

By chance I was in Canberra on the previous Friday, when David Pennington and Spike Langford, asked me what I thought we should do to accelerate the introduction of screening tests to blood banks.



My advice was to work closely with the US based manufacturers and to evaluate the assays for their sensitivity, specificity and operational characteristics.

Assuming that these were satisfactory, the Government should control the importation of the assays, introducing them simultaneously with counselling, to blood banks, plasma fractionation centres and selected PH centres in each State or Territory – this to avoid the worried well donating blood as a way of checking their status.

I sketched out a possible 3 tier testing scheme on the back of an envelope – with screening tests in blood banks and selected public health laboratories comprising the first tier, backed by State Reference Laboratories offering confirmatory tests and at the apex of the pyramid a National Reference Laboratory to evaluate the assays, provide QA panels, train staff, developing consensus on interpretation of results and providing the final court of appeal for contentious samples.

We prepared an indicative budget and David presented the proposal to the meeting which endorsed it. Tom Roper, the Victorian Health Minister turned to me and asked how much additional support was needed – I thought of a number, doubled it and he said “OK – when can you start?” – “In the morning” I said.

I had expected the NRL to be a sunset organization, but it proved to be so valuable, it remains an important asset 25 years later.



So the National AIDS Reference Lab was established the next day.

I became its first Director.

It was a remarkable example of planning and decision making under fire.

Over Christmas we worked tirelessly, renovating an old animal house to provide a base for the work, assembling a panel of 1000 sera from healthy adults and HIV infected individuals which were divided into duplicate aliquots and coded to provide 5 panels each containing 2000 samples.

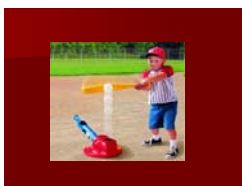
Five laboratories in South and East Australia (for logistic reasons) the Victorian, NSW, South Australian Blood Banks, St. Vincent's Sydney, Fairfield Hospital Melbourne agreed to participate as did all of the manufacturers, who provided their assays, equipment and staff to help train local technicians.

It was a huge and logistic Exercise, two thousand samples, tested by 6 different locations over a 2 month period. We decoded the data on Friday March 8, 1985 and I discussed the results, which showed that two assays had extremely high sensitivity, specificity and reproducibility, with the Directors of the BTS that day.

I presented the results to the executive of the Task Force on Tuesday March 12 and to the full Task Force shortly after.

As a result of our recommendation the Government opened tenders for supply of the kits to the two manufacturers (ENI and Abbott) on March 19 and closed them at the end of that week. In practice most transfusion services elected to purchase the ENI assay because its micro titre plates fitted their existing equipment, whereas the Abbott assay, which utilized antibody covered beads, was rather more cumbersome and required new equipment.

When the kits were introduced on April 29 Australia became the first country in the world to introduce routine screening to Blood Banks and Public Health Institutions. Of the first 250,000 donors tested 7 were found to be HIV positive.



It was a remarkable achievement – partly good management but also good luck, as compared to other countries we were unique in having all the balls aligned – no country had the combination

- an intelligent, involved Minister with the Cabinet clout and the ability to obtain resources
- an expert Advisory body led by David Pennington who was not only familiar with transfusion issues but politically astute
- a depth of scientific knowledge on management of blood borne infections
- the catalytic effect of the Queensland babies and
- the impetus of an imminent election

Where others procrastinated and paid the price, we were able to crash through.

It was an example of the importance of political and scientific leadership, the benefits of empowering individuals and the value of collegiality.



In confronting the challenge of AIDS, blood transfusion services in Australia and overseas have been both challenged and revitalised.

Not only are they now better funded and equipped, there has been a sea change in technology and management style. Lab tests are performed by sophisticated automated machines, reporting errors are minimized through computerization and vast amounts of data are being generated and available for analysis.

Rather than being a cottage industry the level of documentation and validation required by blood banks is similar to that required of the pharmaceutical industry. This has resulted not only in a greater level of professionalism but greater integration of the discipline into the medical and academic community.

It has been both fun and a privilege to be a participant in such momentous times.