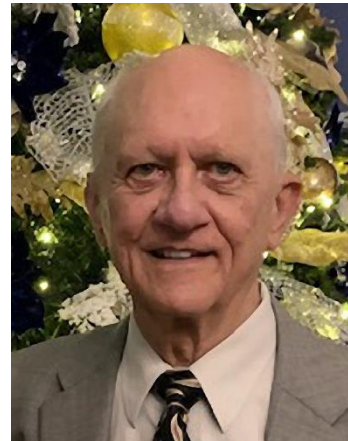


‘Fevers’ by Andre van As

A personal recollection of working at the Johannesburg Fever Hospital, 1963-1964

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History of ‘FEVERS’

The Johannesburg Fever Hospital was designed by the architect JS Cleland and constructed by the Public Works Department of Johannesburg. Opened in 1916, it served the white population of Johannesburg, suffering from infectious diseases. Cases were sent by the Public Health Department and admission was compulsory when isolation at home was not possible. It could accommodate 80 patients in five separate wards, two of which were isolation wards with cubicles. The buildings still stand behind the Civic Centre on Hoofd Street across the road from the old Woman’s Prison. Parts of this hospital were declared a National Monument 1995.

Below: Johannesburg Fever Hospital administration and nurses block in 1916.



Rietfontein (now Sizwe) Tropical Diseases Hospital

More than two decades earlier, there had been a significant need for the isolation and treatment of non-white patients with infectious diseases. In the early 1890s, three lazarettos (quarantine stations) made of tents and metal huts, had been hastily created in the Johannesburg area, to manage the smallpox epidemic of that time. When that epidemic subsided, one of these stations, the one furthest from the residential population of Johannesburg, on the Rietfontein mine farm, in the vicinity of what became Edenvale, became the Rietfontein (now called Sizwe) Tropical Diseases Hospital. It opened in 1895 and has been in use ever since. It provided treatment to non-European patients with smallpox, tuberculosis, malaria, leprosy, typhoid, Congo fever, Spanish flu, bubonic plague, anthrax and Ebola as well as venereal diseases. A report on the history of the Sizwe Hospital can be seen [here](#).

While some students in our class have no recollection of working at the Johannesburg Fever Hospital, we were certainly never rotated through the Sizwe hospital for our training in infectious diseases.

Medical Education at Wits

In 1911, Professor Wilkinson, who was the Professor of Chemistry of the SA School of Mines and Technology, proposed evening lectures in Pharmacology. These lectures commenced in 1912 in the School of Mines building on Plein Street in central Johannesburg, affectionately known as the 'Tin Temple'. Lectures were broadened to include anatomy, physiology and histology. This Tin Temple can thus be considered 'the first medical school' of the University of the Witwatersrand.

By 1919 it was decided that a Medical School needed to be built. Construction began in January 1920. It was well located on Hospital Street allowing students to gain clinical experience in the Hospital Hill area at the Johannesburg General Hospital, and later, the Fever Hospital, the Queen Victoria Maternity Hospital, the Transvaal Memorial Hospital for Children and the Non-European Hospital. Teaching was provided by private practitioners who became honorary appointees on a part-time basis to the new Faculty of Medicine. The first full time clinicians were appointed in the mid 1940s, after WW2. They had joint appointment with the Province and the University. Later in the 1960-70s many senior positions became full-time, with the exception of Fevers, that still had only part-time consultants.



Above: **The Wits Medical School** building on Hospital Street, where we studied in the 1950s, had opened in the early 1920s. When this picture was taken, nearly a hundred years later, in 2017, it had become the University of the Witwatersrand Department of Forensic Medicine while the medical school had moved to Parktown in the late 1970s.

FEVERS – my work there 1963/64

After spending eighteen months in post-graduation internships, as well as six months with the South African Blood Transfusion Service, I felt that it was time for me to get back into clinical medicine and resume my training. I saw an advertisement in the Johannesburg Star for a position at Fevers and had an interview with Dr Jackson, who was the head of the hospital. The next day I was notified that I had the position. I joined the staff as Medical Officer, Infectious Diseases, on the 28th of January 1963 and worked there until the 9th of March 1964.

Full time medical coverage was given by a Senior Houseman and the Medical Officer. Call, which included covering all the 80 beds, was split between the two doctors who were on call every second day and every second weekend. A small building near the entrance of Fevers had a living/dining area. Two bedrooms and a bathroom were our accommodation while on call and three meals a day were provided.

Our work schedule



We had one formal round a week, on Thursdays, which was run by Dr Jackson and always attended by Professor J S Gear (left) respected and renowned for his deep knowledge of virology and parasitology. It was a privilege to be taught about infectious and tropical diseases by him. He was a tremendous asset and had a comprehensive knowledge of his specialty as well as a vast clinical experience. The round lasted 90 minutes and selected cases were presented. After the weekly round we all gathered for morning tea and biscuits in the doctors' quarters. About six months into my tenure Dr Jackson retired and he was replaced by Dr Michael Malk, and another part-timer, Dr Ray Dando, joined us.

Left to our own devices

Fevers was a hospital where you were left to your own devices and decision making. In the time I was there the only additional contact I had with Dr Jackson was a phone call I made a few weeks after I started there, to get input on the management of a patient with polio. Initially the houseman position was filled by a General Practitioner, but later on, Nick Joyce-Clarke (Class of 1961) took the position.

The work schedule was individually driven by the two of us. As we alternated, we did not have the opportunity of working together and doing our daily ward rounds together. Effectively we were each working single-handedly. The Medical Officer had the additional responsibility of teaching 5th year medical students once a week, and also assisting Dr Bollie Sieff, a venereologist in private practice, in running the venereal disease clinic, one morning a week, at the Non-European Hospital (NEH).

No protocol for infection control

Despite being an infectious disease hospital I can recall no routine protocol on infection control such as use of masks, protective gowns or hand washing that is standard practice today. Fortunately, I had had all the childhood infectious diseases, had a positive Purified Protein Derivatives (PPD) for tuberculosis and was immunized against polio with the Sabin oral vaccine.

The case load

The case load was light in comparison with the 20 months spent at Baragwanath Hospital. It was rare to have a patient admitted after 10:00 PM. The majority of patients were young children and adolescents. Cases mostly consisted of the common or garden variety of childhood infections such as measles, German measles and whooping cough. Less frequently we would get patients with diphtheria, viral encephalitis, and tourists picking up a variety of rarer viral diseases from territories north of South Africa. Because there was no formal screening of admissions a number of cases were admitted that were not infectious diseases, but medical conditions that had been misdiagnosed.

Some of my more memorable patients are summarized below:

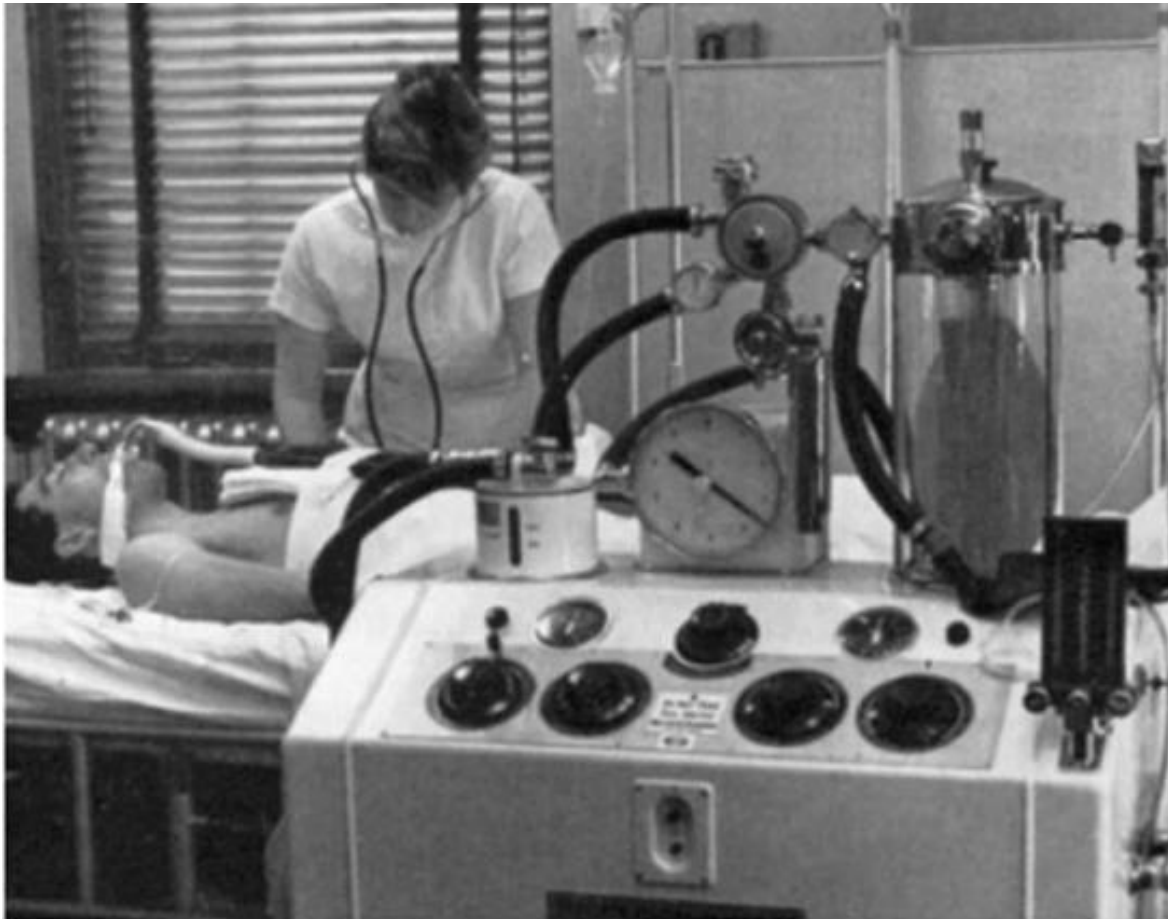
Case #1. In February 1963, a 36-year-old electrical engineer was admitted with bulbar polio. He deteriorated rapidly and needed assisted ventilation. He was placed in an iron lung and we initiated negative pressure assisted ventilation. He turned out to be the last patient to be treated in an iron lung in South Africa. (Picture above of a patient in an Iron Lung)



He developed widespread paralysis and posed a challenge to being adequately ventilated, nursed and attended to. We were able to get an Engström ventilator urgently and his gas exchange improved on IPPV (Intermittent Positive Pressure Ventilation).

He developed a staphylococcal pneumonia and despite an aggressive antibiotic regimen his pneumonia did not come under control and he died.

Case #2. A nine-year-old girl was admitted with viral encephalitis and signs of brain stem involvement. She was in coma and went into respiratory failure requiring IPPV with the Engström ventilator (see below). After several weeks she showed no signs of recovery, lost



blood pressure control, had fixed dilated pupils, did not respond to painful stimuli and was areflexic. At a weekly round we discussed what should be done, as it seemed that there was sentiment among the nurses that she could recover, an opinion we did not share. This was a first experience for many on the round, in that modern technology had presented us with an ethical dilemma, namely making a decision to discontinue life support. At that time we did not have access to modern technology for determining viability. After the round I stayed with her and did a thorough evaluation of her viability, and found that her status had deteriorated, as she had in addition become hypothermic. After careful consideration I turned off the ventilator and announced to the nursing staff that she had died, which they accepted.

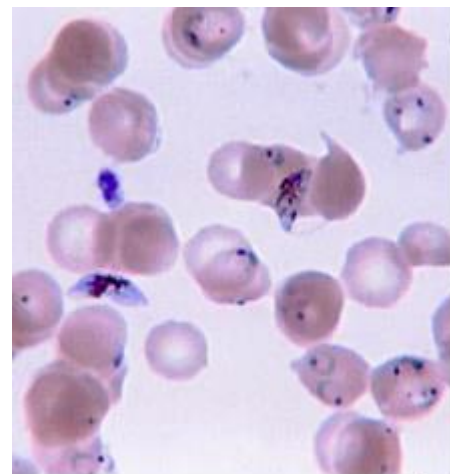
Case #3. A prominent northern suburbs General Practitioner (GP) admitted a woman in her 50s with a diagnosis of typhoid. She had no bowel symptoms or splenomegaly. Her blood sugar

was elevated confirming that her diabetes was out of control and she had a left sided 6th nerve palsy which is associated with diabetes. Her white blood count and pyrexia suggested an aggressive infection. Examination of the urine showed that it was loaded with PMNs (diagnostic of pyelonephritis). She responded to antibiotics and her diabetes was brought under control with insulin. The GP refused to accept that she did not have typhoid despite negative stool and blood cultures for Salmonella. By making the correct diagnosis we were able to prevent a typhoid scare.

Case #4. A 24-year-old man was admitted with a presumptive diagnosis of encephalitis. A lumbar puncture (LP) was normal except for an elevated pressure. Ophthalmoscopy showed a unilateral left sided papilledema. Specific questioning revealed that several days before he had a head injury playing rugby and was quite severely concussed. A diagnosis of a subdural hemorrhage was considered and a neurosurgeon was called in for an urgent consultation. A few hours later I got a call that a large left sided temporal subdural hematoma had been drained.

Case #5. A male in his late 20s was admitted with a headache and a stiff neck with a provisional diagnosis of meningitis. An LP showed a clear fluid with a normal pressure and a slightly elevated protein and an increase in lymphocytes. The VDRL (test for syphilis) and other treponemal tests were positive in the cerebrospinal fluid and blood. He had no history or evidence of a genital primary chancre. He responded well to penicillin for 14 days. Review of his history revealed that he had surgery to remove a rectal fissure several weeks before. I was not able to track the pathology specimen to ascertain if this was the primary site of infection.

Case #6. A ten-year-old boy living in Bryanston was admitted with a two-day history of cyclical fever and rigors. The only additional physical sign was splenomegaly. A workup for the usual causes of fever and splenomegaly was done. One hour after sending the blood specimens to the SAIMR, I received a call from the hematology technician that his blood smear was loaded with malarial parasites (*Plasmodium falciparum*). A chloroquine IV infusion was started, and he responded well. Today, because of widespread antimicrobial resistance, we would use a combination of newer drugs and selected antimalarials [known



Ring-forms and gametocytes of *Plasmodium falciparum* in human blood

as artemisinin-combination therapy, or ACT] which is about 90% effective when used to treat uncomplicated malaria. A careful history revealed no travel out of the high veld area for the past several months. At our weekly round Prof Gear related an instance of a sailor getting malaria while living on a Navy ship docked one mile out from Durban harbor. Apparently this is the maximum distance that a mosquito can fly. As the *Anopheles stephensi* mosquito does not occur naturally in the high veld, we assumed an infected mosquito had hitched a ride in a vehicle travelling from the low veld (Kruger Park) to Bryanston (high veld) and infected our patient.



An *Anopheles stephensi* mosquito

Case # 7. A five-year-old boy was admitted with diphtheria and started on penicillin. While on our round the next day at this patient's bedside he suddenly developed respiratory obstruction and became cyanosed. A patent airway had to be urgently secured. I saw a scalpel blade lying on the windowsill of the isolation cubicle, and grabbed it, to immediately do a tracheotomy. While inserting the tracheotomy tube I removed a large membrane that had sloughed off his tracheal epithelium and obstructed ventilation. The patient recovered fully. The Centre for Disease Control's estimates, that even with treatment, 1 in 10 people with respiratory diphtheria will die, airway obstruction being at the top of the list of cause of death.

Case # 8. At about 9 PM I was called because a three-year-old admitted the evening before with whooping cough, was having severe coughing and apneic spells, followed by convulsions. As I arrived in the ward, he was having his third spell which ended with apnea followed by convulsions. One of the nurses told me that his twin brother had died the evening before with the same severe coughing spells. I immediately had him transported, while bagging him with oxygen, to Ward 5, where we kept the Engström ventilator. I performed a tracheotomy and inserted a silver tracheostomy tube and commenced IPPV. His coughing spells stopped immediately and did not recur again. Four days later we extubated him and watched carefully for return of apneic coughing spells. None occurred. It was a bad season for whooping cough and over the next 6 months six more cases (all younger than 5 years) were admitted with severe apneic events, all of which needed intervention. All were treated with a tracheostomy and IPPV and responded as described above. All of the first six survived. The seventh patient

was admitted on the day before I left on vacation. The General Practitioner who regularly did our locums was fully briefed on the management and I took off to the Game Reserve. When I returned I learned that the patient had died. The nursing staff reported that the GP for some reason kept on turning up the minute volume, and the peak inspiratory pressure kept on increasing. I attended the autopsy, and on gross and microscopic examination, all of the alveoli were ruptured. Cause of death was iatrogenic (physician induced) barotrauma.

Papers on whooping cough

I found two papers in the French literature from the 1920s, in our Medical library, that reported treatment of severe apneic whooping cough with chloroform anesthesia, which resulted in coma and required subsequent resuscitation by plunging the infant in a mustard bath. They also reported a high mortality.

The isolated death that had occurred, had really worried me as it was clear that all of the other children had recovered. A mother sent me a note of thanks together with a photograph of her 4 year old daughter. I wanted to publish this series of patients but had no experience and could not get any guidance on writing a paper of this kind, so it never materialized. This account is the first public description of a treatment that I stumbled on, and that really worked. Clearly this is a drastic last resort treatment and needs to be carried out in a specialized pediatric intensive care unit (ICU) environment.

I have thought of a rational explanation why the intervention worked so well. It is possible that the positive pressure delivered by the ventilator interfered with the cough reflex. A more likely explanation is that the infection which causes a significant inflammation in the upper airways (larynx and epiglottis) stimulates the cough reflex resulting in deep inspiratory effort that produces the “whoop” that is so characteristic of the cough. While a deep inspiration will result in dilatation of the intrathoracic airways it could result in collapse of extrathoracic airways. The negative internal pressure in the upper airways results in the external atmospheric pressure exerting a compressing effect on the tissues of the neck (similar to the pathophysiology of adult obstructive sleep apnea). Under normal circumstances adult extrathoracic airways, which are large and well protected by mature cartilage, prevent collapse during inspiration. Infants younger than five years have a much smaller airway diameter as well as cartilage that has not matured sufficiently to withstand the external compressive forces. The apposition of the inflamed tissues then will act as a further stimulus of the cough reflex, leading to the repeated coughing spells, which eventually end with an apneic episode, often followed by vomiting. By doing a tracheotomy, the upper airway and larynx are disconnected from the trachea, and the

insertion of a rigid tracheotomy tube prevents the collapse of the trachea. The cycle of the cough reflex stimulated by the initial inspiratory whoop is then interrupted because breathing is provided by IPPV.

Immunisation

Pertussis (whooping cough) is mostly well controlled by immunization, developed in 1914 and widely introduced in the USA in 1941, but there are large areas where it is still prevalent, and thousands of infants still die from this eminently preventable disease. In 2010 ten infants died from whooping cough in California. A recent paper estimated that worldwide there were 24.1 million pertussis cases, and 160,700 deaths from pertussis in children younger than 5 years in 2014. Africa contributed the largest proportions (7.8 million [33%] cases and 92,500 [58%] deaths) - [Lancet Infect Dis. 2017 Sep;17\(9\):974-980](#). Current estimates suggest that, compared with the 1999 estimates, published in 2003, 30.6 million pertussis cases and 390,000 deaths from pertussis in children younger than 5 years will occur globally. This illustrates that children in underserved populations are the hardest hit by infectious diseases, and that immunization remains the most cost effective prevention. Most of the children admitted to Fevers were from a poor population to the South East of Johannesburg where there was practically no adherence to immunization.

Moving on from Fevers

Working at Fevers was very instructive, and my experience with IPPV, in many critically ill patients over the previous year, helped prepare me for the next phase of my life that I did not fully anticipate. In the first week of March 1964 I got a call from Derek Stables ([obituary](#)) asking me if I was prepared to take a position of registrar at the Johannesburg General in Professor Elliot's ward, as a locum. His co-registrar, Arthur Rubenstein, was going to London for a year. When I arrived on Tuesday March 10th, 1964 to start, I vividly recall Arthur showing me the new three-bed ICU that had been created very recently, and the new ventilators they had – an Engström volume cycled and a Bird pressure cycled ventilator. I felt right at home.

Andre van As, Florida, December 2020.

Read his biography here [Van As, Andre](#)

Edited by Geraldine Auerbach MBE, London, December 2020.