EXCERPTS FROM A PERSONAL PERSPECTIVE OF MEDICINE IN SINGAPORE IN THE LAST 50 YEARS

E.S. MONTEIRO

Vitamin B1 or Thiamine Chloride—first used in Singapore as a cure and as a proof that Cardiac Beri Beri is caused by an acute deficiency of Vitamin B1

Cardiac Beri Beri continued to exert its heavy toll on the Chinese labourers working in the docks in Singapore. No cure was available in a disease which was uniformly fatal with a mortality rate of 100 per cent.

Professor J.C. Tull, head of the Department of Pathology, invited Wenckebach to come to Singapore to assist. I was then assigned to assist Wenckebach and carry his syringes and tray in his quest to study the pathological dynamics of the circulation in these cases. He tried many procedures such as bandages of the limbs and intravenous injections of Adrenaline Hydrochloride. These procedures were of no avail and the Cardiac Beri Beri patients seemed to die faster after these procedures.

The period was 1937. I happened to be friendly with a biochemist Van Heen from Batavia Centrum who told me he was able to make an extract of Vitamin B1 from rice polishings and whether I was prepared to use it for my cases of Cardiac Beri Beri. At that period in time Vitamin B1 deficiency was suspected as the cause but no one could prove in fact that this was true. However, he sent me about 20 ampoules of his extract from rice polishings purported to be Vitamin B1. Professor R.B. Hawes gave me permission to try it. The first case of Cardiac Beri Beri admitted into the teaching unit was given intravenously an ampoule of the extract from Batavia Centrum purported to be the equivalent of 10-20 mg of the pure Vitamin B1. I gave the injection slowly by the intravenous route. The patient took it very well without any reaction. The nurse as usual covered the patient over with a blanket expecting that like the other cases the patient would soon die. But the patient sat up in bed within half an hour after the injection. He ate his lunch and walked

University of Singapore.
E.S. Monteiro, MD, FRCP (London), FRCP (Glasgow), DCH (London)
Emeritus Professor of Medicine
(lately Singapore's Ambassador to the United States of America)

towards me to thank for the injection. Unknown to me, the dramatic recovery of the first case caused quite an excitement in the whole hospital and calls to me were made everywhere except at the tennis court. I used the other ampoules for the next case and so on and I recorded a 100 per cent recovery rate with the 20 ampoules. At about this time, too, Williams in America had isolated Vitamin B1 and worked out the formula which he labelled chemically as Thiamine Chloride. Once the chemical formula was worked out, pharmaceutical chemists the world over began producing synthetic Vitamin B1 in extensive amounts at a cheat price. Cases of Cardiac Beri Beri were later given synthetic Vitamin B1 by me in doses of 30-50 mg intravenously and these cases receiving synthetic Vitamin B1 also showed the same quick response and recovery.

What struck me and my colleaques was the rapid recovery of the cases of Cardiac Beri Beri on Vitamin B1 therapy. The rapid recovery suggested to me a sort of catalyst action by the ferment which rectified a biochemical error. The pharmacological action of Vitamin B1 was soon ascribed to it being a co-carboxylase in the intermediate metabolism of carbohydrates. In an acute deficiency of Vitamin B1 in the system precipated by a rapid rise in the metabolic rate such as severe muscular exertion, fever or haemorrhage, the deficiency caused an incomplete metabolism of carbohydrates in the intermediate stage resulting in metabolites like the pyruvates, lactates and the bisulphide binding substances flooding the blood stream. Pyruvates are toxic and produce an incompetence at the arterio-venous capillary junction like an arterio-venous aneurysm. This incompetence causes the blood stream to race through to the right side of the heart while the speed of blood on the left side remains almost unchanged. This flooding of the right side of the heart results in overloading of the right ventricle where the conus is enlarged to accommodate the increased blood on the right side. The veins are full and the enlarged liver becomes exquisitely painful and tender so much so that some of the cases have been opened for "acute abdomen". The blood pressure records a normal systolic with a marked fall in the diastolic giving the characteristic pistol sounds on auscultation over the arteries. The patient is cyanosed, he is not unduly breathless, the lungs are dry and he lies down flat on bed. He is most likely to complain of pain and distress over the epigastrium from the tense enlarged liver.

I had the opportunity of seeing a case of alcoholic Cardiac Beri Beri catheterised in the Cardiac Laboratory. The cardiac output was very high and recorded 10 litres per minute. It is apparent that Vitamin B1 introduced intravenously in such cases in the early stages restores the altered cardiac dynamics described above to normal. The intermediary metabolics of carbohydrates—the pyruvates—are broken down to lactic acid, carbon dioxide and water and the patient's condition returns to normal. Pyruvates and the other intermediate products have a toxic action on the central and peripheral nervous system but the action is slower. The neuritic type of Beri Beri or the Dry Beri Beri as opposed to the wet or Cardiac Beri Beri takes at least 2-3 months to develop. The peripheral nerves at their most vulnerable points are affected. These points measured from the trophic centres of the anterio-horn cells in the cord are about a metre away. Thus the hands and feet are affected in the peripheral neuritis of Beri Beri and even in the alcoholic B1 conditioned peripheral neuritis. Some of these cases show gaps in their memory which they try to cover up by clever but detectable fabrication. This is known as the Korsakoff Syndrome which is apparently a toxic action on the frontal lobe memory area. Another effect described as Beri Beri of the brain or Polioencephalitis superior is seen in high protein eaters who suffer from deprivation of Vitamin B1. I saw numerous cases among European alcoholics at post mortem during the war who had severe haemorrhages in the corpus mamillaria of the hypothalamus. But this is a rare finding in a high rice or carbohydrate eater like the Chinese dock labourers in Tanjong Pagar. They are most likely to go down with Cardiac Beri Beri and attacks come on because their heavy muscular exertion like carrying 100 katis of rice bags up or down the gangway on a narrow wooden plank produces an acute B1 deficiency.

At about this time, my teacher and friend, Dr. Gopal Haridas, was doing research on Cardiac Beri Beri among breast-fed infants. Working and assisting him I learnt a great deal of Cardiac Beri Beri in breast-fed infants. The mothers all have evidence of neuritic Beri Beri (absent knee-jerk). When detected the infants are about 3 months old. They are all breast fed. The Japanese described the same condition as "mother's milk poisoning". The fact is that infants are fed on breast milk deficient in Vitamin B1 because their mothers have Beri Beri developed during their

period of gestation. The clinical types seen were:

1. frank cases of right heart failure with enlarged liver,
2. cases of aphonic Cardiac Beri Beri from pressure of the enlarged right heart on the recurrent laryngeal nerve,
3. cases of the meningitic type with convulsions and arching of the back, and
4. cases masquerading as diarrhoea and vomiting.

In all these cases prompt administration of Vitamin B1 intravenously or intramusculary saved most of them. As a preventive, all Chinese mothers of the poorer class were advised to eat Chow Bee or cargo rice (unpolished). With changes in their diet and a generous intake of Vitamin B1 during and after gestation, these cases of infantile Cardiac Beri Beri of breast-fed infants are no longer seen in the General Hospital.

The first trial on a big scale—the treatment of Typhoid Fever with Chloroamphemicol

Typhoid Fever is endemic in Singapore and periodically there are outbreaks of a sporadic nature. During the Japanese Occupation there was an epidemic in which I was a victim myself. There was no specific cure then. The course of the disease was a long and hectic one of 6-8 weeks fever and loss of flesh. Nursing of these cases imposes a severe strain on the nursing staff. The greatest danger is from Haemorrhage (30 per cent of cases) and perforation with fatal general peritonitis (30 per cent of cases) in the hospital record with an overall mortality rate of 40 per cent. Surgery of these peritonitis cases did not help. When Chloroamphemicol was discovered and used successfully in cases of Rickettsial infections of the Scrub Typhus Type, I suspected it would be valuable to use the antibiotic on Typhoid Fever. Such an outbreak occurred soon after my return from the United Kingdom and presented me with the opportunity of testing the effectiveness of Chloroamphemicol in proven cases of Typhoid Fever. The trial of a new drug requires patience and vigilance for any ill effects the patients may develop as a result of our therapy. Because we did not know the right dosage of Chloroamphemicol it was a question of erring on the side of safety rather than recklessness. Blood smears and blood counts were performed repeatedly on every case treated and the effect of the drug on the marrow carefully noted. Fortunately there were no side effects on the marrow. I found that this antibiotic which comes from streptomyces venezuelae is effective when given orally and effective blood levels appear within 30 minutes. After many trials I found that a dosage of 50 mg per kg of body weight in divided doses given 6 hourly were the most effective and simple. There were many schemes of dosage suggested but I stuck to the simple rule. When the temperature fell to normal usually in 3-4 days, the dosage was reduced to 30 mg per kg of body weight given 6 hourly for the next 2 weeks to prevent relapses. I found that premature stopping of the Chloroamphemicol after the temperature returned to normal could result in relapses and recrudescene. The introduction of chloroamphemicol in the treatment of Typhoid Fever is simplified what has always been a complex nursing problem in Typhoid nursing. It has reduced the overall mortality rate to less than 6 per cent in my series and haemorrhages and perforation have become uncommon. The carrier rate has not been drastically reduced in my view. I encountered no resistant cases to Chloroamphemicol. This may be due to the fact that I started with 50 mg per kg of body weight a day and after the temperature returned to normal in 3 or 4 days, the doses was cut down to 30 mg per kg of body weight for 2 weeks. I found that premature stopping less than 2 weeks during the afebrile period may induce a relapse. Having been a victim of this terrible disease, I naturally spent a lot of time in thinking about it. I came to some interesting conclusions. Typhoid Fever is an infection by the Typhoid bacilli of the Reticulo-Endothelial System. The portal of entry is the G.I. tract namely through the lymphoid follicles and into the blood stream where it remains as a bacillaemia for at least a week. This bacillaemia permits the bacilli to infect the whole of the Reticulo-Endothelial System of the body—the spleen, the bone marrow, the lymph glands and the liver. However, it is only in the liver that the bacilli could survive the longest because it has learnt to live in bile whereas the other competitors like the B coli cannot. From time to time, the bacilli now luxuriating in the liver bile channels, the bile duct and the gall bladder make their second entry into the small intestine and large intestine. The lymph follicles and the Peyers patches in the intestine set up an allergic inflammatory reaction to this second visitation. Depending on the extent and depth of the reaction in the follicles and Peyers patches, swelling and anaemic necrosis and ulceration result. It could be a haemorrhage or even a perforation or both combined.

This concept of the pathogenesis of Typhoid Fever explains why the carrier state is persistent because of the liver acting as a haven of refuge for these bacilli which are periodically squirted into the intestines even after the active phase of the disease. I am afraid this explains why there are so many Typhoid Marys in the world. In Singapore most of the outbreaks are due to carrier spread such as insanitary food handling and preparation by carriers and flies. The only remedy for this state of affairs is that everyone must be educated from young and in schools that they must wash their hands with soap and running water. Food handlers must be screened by tests and culture of their stools. Licence to food handlers must be strictly enforced and given only after they have been proved to be clean. The problem of the prevention of Typhoid Fever by carriers who are food handlers and flies is a major one. Ice cream vendors and manufacturers must be licenced. Quite early in my career at the General Hospital, Dr. Canton, the City Health Officer, and I traced an outbreak of Typhoid Fever among school children in 3 schools—St. Joseph's, St. Anthony's and St. Andrew's—to a unlicenced shop which prepared ice cream sold to these schools by vendors. The headman was an active carrier and he caused an outbreak of Typhoid Fever of about 750 school children in which 40 per cent of the children died from Typhoid Fever. Since then, ice cream manufacture and vending are under strict licence in Singapore.

Anti-diphtheria Toxoid inoculation made compulsory in Singapore

I was put in charge of the Middleton Hospital during the Japanese Occupation of Singapore having been evacuated from the General Hospital and the Tan Tock Seng Hospital because these hospitals had to make room for the Japanese wounded and sick. Apart from the epidemics raging in Singapore like diarrhoea, dysentery and typhoid, the hospital was inundated with sick people who were suffering from malnutrition and famine oedema and tropical ulcers of the legs from poor intake of proteins. The common infectious disease like Diphtheria took a heavy toll of lives of the children of occupied Singapore. The incidence of Diphtheria had risen. I fully expected that the scheme of inoculating the pre-school and the school child with anti-Diphtheria toxoid by the Singapore Municipality Health Department would work but I was disappointed as the scheme was a voluntary one. It did not work. Soon after the war I insisted on making anti-Diphtheria inoculation compulsory with the force of the law in a joint meeting with the Public Health officials of both Singapore and the Federation of Malaya. I succeeded in making it a legal requirement whereas the DMS of the Federation said he could not agree to compulsory anti-Diphtheria inoculation for Malaya.

Unlike the DMS Malaya I had seen enough of fatal cases of Diphtheria which died of Diphtheritic toxic myocarditis and of cases of laryngeal Diphtheria necessitating tracheostomy to relieve the fatal obstruction to the air passages. As an indication of the high incidence of Diphtheria in Singapore, I had to perform each year an average of 70 tracheostomies when I was at the Middleton. These Diphtheria cases usually came late. Furthermore, the Chinese medicine men to whom these cases were first sent would blow in a mercuric compound into the trachea and lungs to "dissolve" the membrane. These powders only added to the obstruction with a dirty mucoid gritty matter which was difficult to aspirate even after tracheostomy. The cases of pharyngeal diphtheria that usually came late, i.e. after the 7th day of onset usually died on the 16th day or as late as the 6th week. The fatal heart failure was the result of irreversible fixation of the diphtheria toxin on the myocardium which even massive doses of Diphtheria anti-serum could not counteract. Neither did the failing toxic myocardium respond to any of the known cardiac glycosides such as Digitalis or Strophanthin or Ouabain.

I was forced to the conclusion that the only way to save these children from Diphtheria is compulsory anti-Diphtheria inoculation for the pre-school and the school children. It may not be known that anti-Diphtheria serum in adequate dosage is only useful for cases seen early before the 7th day of illness. In no other illness is the saying truer that death lurks in the shadow of delay.

Antitoxic diphtheria serum prepared from goats

There was no end to the anxiety and frustration of working in a hospital during the Japanese occupation of Singapore. The hospital had run short of anti-diphtheria serum and the Japanese could not supply the hospital requirements. The children with diphtheria came in as usual only to die from lack of serum. It was a period of grave anxiety and frustration for me. But two facts gave me the solution to my problem. One was that I had a culture of diphtheria bacilli which were able to grow on the surface of a liquid media and the

toxins produced would sink to the fluid portion. In many ways it is like the famous Park 8 strain which was used to inoculate horses for anti-diphtheria serum. The second fact was that I had 8 goats presented to us for food in case of necessity. I injected firstly 2 goats with graduated doses of the diphtheria toxin which I obtained by filtration. I gradually increased the dosage. The goats showed no untoward reaction. After 8 weeks I bled the goats. The blood was allowed to clot and I separated the serum from the clot. I tried it on guinea pigs inoculated on the skin with diphtheria toxin and it worked. I used this crude serum from goats and cured many cases of diphtheria. The war ended very shortly after that and with fresh supply of diphtheria antitoxin from the Red Cross my anxiety and frustration came to an end. After the war I recounted my experience of using goat serum for diphtheria and suggested that it may be useful for cases of severe horse serum sensitivity. The chemist to whom I communicated this fact seemed not to be optimistic about its success. For me this adventure into preparing goat acti-diphtheria serum for diphtheria will always be a proud recollection of not giving up in the face of insurmountable odds. All the cases that received the goat serum developed serum sickness which passed off without any effects. The main thing is the serum worked and saved lives.

The El Tor Strain of cholera vibrio in Singapore and the story of the poisoned bread

Towards the end of the Japanese war in 1944, I was informed by the Japanese High Command that I was to go to Loyang to investigate an outbreak of cholera among the villagers of Loyang near Changi. A yellow flag general with a chauffeur-driven car came over to see me at the hospital and commanded me to investigate and prove whether the outbreak in fact was cholera. The attack of diarrhoea and vomiting had practically killed all the inhabitants. Some of them had fled from the village of Loyang as soon as the Japanese quarantined Loyang making it out of bounds for all civilians.

I asked Dr. T. Balasingam to accompany me and we took culture media for cholera and also autopsy tools in case we had to perform a post-mortem in the village. When I arrived late in the evening at the village, I found it was empty and deserted. I saw peculiar wicker work bamboo baskets nearly everywhere near the houses but I thought no more of them. When the yellow flag general was told there were no one in the village, he ordered us to examine the corpse of an elderly Malay whose remains were that morning. He obtained the services of 4 private Japanese soldiers from nearby Changi jail. They uncovered the grave. Balasingam and I descended into the grave and performed an examination of the corpse which was severely dehydrated. I had never before seen a Muslim grave. The body was lying on its side and touching the ground. We had to remove the cover of the coffin and took cultures from the gall bladder, and from the small and large intestines. After that, the body was neatly laid at rest and the grave was covered again. I proceeded back to the Middleton Hospital Bacteriological Laboratory and cultured the material we obtained. Three days later all the media grew cholera vibrios which were typical with their scintillating movements due to terminal flagella. The vibrio was typed and for the first time in Singapore the El Tor Strain was isolated with all the characteristics and biochemical reaction of the strain. However, I was somewhat dismayed that the pathogenicity of the El Tor Strain was in dispute. I was quite sure it was pathogenic as this strain had already decimated the whole village of Loyang. However, my doubts as to its pathogenity was soon laid to rest when a Japanese naval doctor visited my laboratory as he had heard we had cultured cholera vibrio. He told me he had also isolated the same El Tor Strain. I was surprised. He was emphatic it is a pathogenic strain. But he soon confided in me the following account. A Japanese vessel used by the navy was on the way from Korea to the Singapore naval base. Cholera had broken out among the crew members who ate vegetables which they were carrying to the naval base in Singapore. On nearing Pulau Ubin the Japanese fearing that their cargo was infectious jettisoned these vegetable baskets overboard. The villagers of Loyang seeing floating baskets of vegetables swam across the straits and took the baskets of vegetables to their homes. I later grew the isolated cholera vibrio in media containing the salinity as sea water and the growth lasted more than 80 days. On slabs of fish the vibrio was kept alive for well over 80 days. So the baskets containing infected vegetables which were thrown into the sea must have been responsible for the outbreak of cholera in Loyang and the salinity of sea water did not kill the cholera vibrio.

The mystery of the Poisoned Bread

I was again called to go to Loyang one Saturday

afternoon as cholera had broken out "again" at Loyang a few months after the Japanese had surrendered. I had become known as the cholera expert in the village and that was the reason why they wanted my expert opinion again. Peace had returned to the village. The war was just a bad memory. That Saturday morning, many of the villagers had become ill with vomiting and pains in the abdomen. The people who were ill had all eaten bread baked in the bakery. The master baker himself was ill, 2 of his children and about 20 village residents had died. I examined the bread and locked up the bakery. The loaves felt much heavier than similar loaves baked in the coffee shop. I suspected the bread had been contaminated with a heavy metallic poison and as it was heavy I suspected lead or barium. In the General Hospital, I X-rayed the Loyang bread and compared the X-rays with X-rays of bread of the same size in the General Hospital. There was a dense opaque shadow in the Loyang Bread. I sent the bread to the Department of Chemistry and suggested to them that I suspected barium. By evening, confirmation came that the Loyang bread was contaminated by barium. Post-mortem of the cases revealed most of them had died with the heart in systolic contraction. Those alive were treated symptomatically until the cause was confirmed where upon I gave magnesium sulphate to convert the barium carbonate into the insoluble barium sulphate. I was able to piece together a fascinating story. Soon after the Japanese occupation, the British Military Administration and the British Military distributed among other things sacks of wheat flour to the villagers in Loyang. The villagers in Loyang took their bags of flour to the village bakery. The master baker made bread from the supply of flour for the villagers for a fee. How one bag of barium carbonate got mixed up with the bags of flour is a mystery. It is known that barium carbonate is used as a rat poison by the British Military. Barium carbonate is strewn around army stores storing flour and other food. The rationale of using powder of barium carbonate is that the rat on eating the barium will become very thirsty and will rush out to the nearest drain to drink water. The ingested water then will dissolve the highly poisonous barium carbonate and kill the rat outside the warehouse immediately. Thus the story of Loyang from the isolation of the El Tor Strain of cholera vibrio to the episode of the poisoned bread mystery. Headlines in the press in Singapore at that period of the story of the poisoned bread made many people afraid of eating bread in Singapore for some time after the incident.

The use of oral live but attenuated Sabin vaccine on a massive scale in Singapore for prevention of Polio

This was one of the most important researches that was undertaken in Singapore which had beneficial and protective effects on millions of children in Singapore and the rest of the world. To appreciate the value of this pioneer work in preventive medicine, I must mention that acute anterior poliomyelitis, infantile paralysis or simply polio as it is known to-day had become a dangerous epidemic disease in Singapore after the Japanese occupation from 1945 onwards. Before this period, polio of the endemic type was seen, the ocasional child in the children ward with a flaccid paralysis of a lower limb. But after the war, Singapore was visited regularly with bigger and more virulent epidemics of the disease affecting children and young adults. A polio epidemic is a depressing period in the hospitals where the morale of everyone is at its lowest. My wards both in the General Hospital and in the Middleton Hospital were strewn with human wrecks of the disease. Some with paralysis from the waist downwards, some with paralysis of the upper and lower limbs. Those with paralysis of the chest muscles and of the diaphragm had to become permanent residents of the iron lung without which they are unable to breathe and would soon be suffocated to death. Those who had the bulbar variety would either get away with a mild facial paralysis or would die from involvement of the vital centres in the medulla. In fact these are the lucky ones as death is only a memory, while the paralysed polio children and adults pose great problems in medical and orthopaedic care and are a severe economic strain on the community which has to rehabilitate them. My colleague in the orthopaedic sections share with me the horros of the disease in their efforts to improve the paralysed muscle by orthopaedic means. Small heated swimming pools were donated by the public for the physiotherapist to exercise the paralysed muscles daily.

It was during a virulent outbreak of polio in Singapore caused by the Type I virus that Prof. J. Hale, myself and Prof. Sabin of the National Institute of Health, USA, held an important discussion. Hale and I decided to use the newly discovered live Polio Sabin vaccine in which the virus is alive but attenuated. At this point in time, Jonas Salk

had produced his formalised killed vaccine and used successfully as a preventive for polio. The vaccine had tobe given by injections and at least 4 such injections are required to boost the immune antibodies in the blood. Tragedy soon struck when injections of the Cutler brand of killed formalised vaccine produced fatal cases of polio among some of the children given the Cutler vaccine in the USA. This accident in some ways comparable to the Lubeck disaster when BCG was first introduced for protection of the children against tuberculosis. In the Lubeck disaster it was not the fault of the BCG. What happened was that by a serious bacteriological error, live virulent tubercle bacilli were used instead of the BCG strain of tubercle bacilli. But the disaster was enough to put back preventive BCG inoculation for tuberculosis for almost half a century. In my visit to the Commonwealth serum laboratory in Melbourne about this time, it was explained to me how probably the Cutler disaster took place. The polio virus is grown on tissue cultures of the kidney cells of Rhesus monkeys. The virus with the cells and the virus are killed by exposing them to formalin. In the process of formalising small pellets of kidney cells and virus are formed in the centre of which live virulent polio viruses are able to survive protected by the wall of the pellets. Constant shaking is necessary to prevent such pellets from forming and even after that the vaccine must be carefully examined to remove any pellets that may have been formed before it is given by injection. It was during this rather emotional period when we decided to use the oral Sabin vaccine to attempt.

1. to prove that oral Sabin vaccine could raise the immunity of the children so treated by studying the antibody levels in the blood and
2. to stop the epidemic caused by Type I virus that was raging in Singapore.

The school in Singapore were closed during the epidemic. We received assistance from the school teachers in our vaccination campaign. We gave the Sabin vaccine using Type II to the pre-school and the school children in Singapore. The particulars of each child given and the vaccine were carefully entered into the card for any further reference. The vaccine was flown by Pan Am from the USA and we gave the vaccine in teaspoonfuls of syrup. All the children did not show any untoward reaction. However I was being called to see many children with the slightest discomfort after the vaccine to reassure the parents. We gave the vaccine to 250,000 children in Singapore. The result:

1. The raging epidemic came to an abrupt end as my chart showed.
2. The antibody content of the blood of all these children showed a sharp rise of the level of antibodies.
3. Polio is contracted by the virus entering through the oro-paryngeal intestinal portal. We believe that the oral administration of the vaccine gives rise to an intestinal block from cellular immunity and is an additional advantage.
4. We found that the administration of the Sabin vaccine in syrup or candy is a pleasant, rapid and easy way to administer the vaccine and free from the psychological trauma which intramuscular or deep subcutaneous injections cause in young children and toddlers. As a child specialist and most paediatrician will agree, I always avoid injections for children and use the oral or rectal route whenever possible. Furthermore, during the epidemic of polio, it is my rule that injections to children and tonsillectomies and teeth extractions are stopped in the hospital. I have seen cases of muscular paralysis developing at the site of injections and bulbar polio developing after tonsillectomy.
5. We were amazed at the speed and extent of the spread of the vaccine virus. One child in the family receiving the vaccine would produce positive stool cultures from all other members of the household. The vaccine virus had spread very rapidly.

Objections to the use of the Sabin vaccine from members of the Singapore Medical Association

Quite rightly, concerned members of the Medical Association in Singapore took me and Hale to taks over the projected use of the Sabin vaccine because of insufficient information and data about the Sabin oral live vaccine. We were seriously cross-examined by members till well past midnight. Tempers were frayed. In my last answer when I was asked why I wanted to do this research in the face of insufficient information, I replied in all honesty and humility that I would go to the end of the world to try and find a preventive for this most dreadful of diseases that strikes at the flower of our youth and young children. It was my faith and conviction that the Sabin vaccine is the answer to my prayer. Reluctantly the Associa-

tion gave me and Hale the mandate to use the Sabin vaccine. Needless to say Hale and I were most worried and anxious about the outcome of the use of the vaccine. I spent many sleepless nights worrying over the matter. Calls to various parts of Singapore to reassure mothers of children given the vaccine occupied most of my time. I can very well understand the parent's anxiety but before the parents I had to put a bold front with all the pleasant and reassuring words and comfort I could command. I never experienced such happiness when at last Hale and I were able to announce that our efforts were successful as proved by our field and laboratory studies. Soon our results were confirmed by research workers in the United Kingdom and the USA and Europe. Today, polio is an excessively rare disease in Singapore and all our pre-school and school children receive their protective polio oral vaccination.