## Forty years of anaesthetic practice

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It occurred to me last year that I gave my first anaesthetic over 40 years ago in 1974 as a medical student. In the time I have available I will, through a series of stories give you a little insight into the changes in Anaesthesia, academically and professionally. I will talk about Wooley and Roe, Pearl Harbor, the first cardiac transplant in England, my first cardiac transplant, Digoxin babies, Pipeline swaps, vigilance, old drugs, new drugs, bullying, obsessionality, and Teflon I have given parts of this talk before and never finished and this will be no exception.

The first public demonstration of Anaesthesia in 1846 was not the first anaesthetic, with prior claims made for W Crawford Long in 1842. In 1823, Henry Hill Hickman anaesthetised animals with carbon dioxide and amputated limbs to assess the effects. He wrote about his findings to Sir Humphrey Davy, then President of the Royal Society. Apparently, the work was never read, and later an article in the Lancet rejected his ideas as "Surgical humbug". Despite support from Napoleon's field surgeon, Barron Larry, the French also did not pursue this idea. Hypoxic anaesthesia nevertheless was practiced for many years for Dental anaesthesia, the early McKesson apparatus able to deliver 100% Nitrous oxide which was used for induction. A variety of fail-safe devices, gas failure alarms and indexing of delivery systems have improved safety. Oxygen analysers in the breathing systems were suggested in the 1970s although not essential and most hospitals did not have these. My first anaesthetic was given in 1974 using a Boyles machine. Monitoring was simple, a finger on the pulse and a blood pressure cuff attached to a von Recklinghausen's oscillotonometer(1). At this stage almost all theatres relied on gas cylinders which were colour coded and attached to the machine using a pin index system (1952) and bodok seal. A mechanical oxygen failure device, the Ritchie whistle, was present on many but not all machines.

In 1981, I travelled to Rotterdam for a two-week locum. On my first day, the first patient was a Surinamese for hysterectomy and after 30mins the oxygen alarm signaled a falling oxygen level despite rotameters suggesting a 1:2 mixture of Oxygen:Nitrous oxide. At the same time the pipeline oxygen pressure alarm showed increased pressure. The oxygen level continued to fall to 18% before I opened the valve on the oxygen cylinder, and disconnected the pipeline. The oxygen immediately returned to 33%. Engineers had incorrectly connected the high pressure air line into the oxygen pipeline whilst doing renovations replacing the oxygen with air. Three years earlier, the UK had instituted a permit system for work on gas pipelines, including a physician sign-off prior to use. I wrote a letter to Anaesthesia suggesting the cost of defense of a hypoxia claim would be less than the cost of making analysers mandatory but was told that point had been made many times. Nothing changed really until pulse oximeters were available which actually monitored the patient. They were developed in Japan in 1972 by Aoyagi and Kishi and became available commercially from 1981. I would not see one until 1985. Capnometry and end tidal agent monitoring was available and in an effort to control escalating legal costs for medical malpractice, a minimum monitoring standard was adopted at Harvard and subsequently endorsed on 21st October 1986 by the ASA(2) (US). So in the space of ten years we had gone from total reliance on pulse, blood pressure and patient colour, to electronic devices capable of displaying physiological signals. A recent pipeline swap in Sydney reminds us of the requirement for anaesthetic involvement in checking pipelines after engineering work.

Epidural analgesia for labour in Sheffield was introduced at the Jessop Hospital by David Nicholas against a tide of midwife opposition. In 1978 I was personally instructed by David, and in order to become proficient we were resident on call 24/7 for one week during which we were expected to have the opportunity to insert 20-30 epidurals. Often the call came as the patient was nearing delivery, the patient was by this stage in severe pain and had worn the midwife down. Caesarian sections were in the

main performed under general anaesthesia. There was no anaesthetic technician, which required careful preparation of the machine and equipment so it was all within reach. Failure to intubate required a switch to spontaneous breathing via a mask using ether via an EMO draw-over inhaler. The Confidential enquiry into perioperative deaths in Scotland (1972-5) published in 1978 found anaesthesia had now become the leading cause of death in women delivered by Caesarian section (5 of 13 deaths). Maternal mortality from all causes had declined significantly whilst that for anaesthesia had not fallen. The two main causes of anaesthesia related death were aspiration and hypoxic cardiac arrest principally due to oesophageal intubation. These statistics resulted in a revaluation of general anaesthesia and a shift towards regional anaesthesia.

My introduction to Anaesthesia at the Jessop Hospital in Sheffield, as a medical student was at least with a trained anaesthetist, Dr David Edbrooke. At this time, Sheffield was one of the few medical schools where undergraduates were given instruction in anaesthesia. In the same year, a colleague of mine, Tim Shaw, also started anaesthesia but in a very different way. As a medical house officer, he was informed that his role was to give anaesthetics for patients in Casualty who required fracture manipulation or abscess drainage. His 'instruction' was given by the Casualty consultant and consisted of a brief introduction to the Boyles machine and how to operate it using nitrous oxide, oxygen and ether.

Specific training programs in anaesthesia were introduced after the second world war, and Pearl Harbor is in some way partly responsible for that. In December 1941 the Japanese bombed the 5<sup>th</sup> fleet at anchor in Pearl Harbor resulting in mass casualties. Anaesthetists were told that "iv anaesthesia was the cause of more fatal casualties among the servicemen at Pearl Harbor than were the enemy bombs".(3). Was it fair to blame thiopentone? The hospitals were overwhelmed by patients, had inadequate fluid and blood replacement, oxygen or ventilator support and pressed unskilled personnel to administer iv anaesthesia sometimes in over dosage. It became clear that better training was required and it was 'no longer appropriate for any junior doctors or nurses to administer sophisticated anaesthetic drugs'.(4). Prior to world war 2, there were few career anaesthetists, often the surgical dresser (intern) was pressed to give anaesthetics. The University of Sydney began a Diploma of Anaesthetics course in 1945 and in 1948 the Faculty of Anaesthetists at the Royal College of Surgeons of London was established. A similar Faculty of Anaesthetists at the Royal Australian College of Surgeons began in 1952. The UK Fellowship examination started in 1953.

When I began as an SHO in Anaesthetics, surgeons would limit surgical time as mortality increased with duration of anaesthesia, and often excluded patients on the basis of age, weight and smoking. Almost all patients over the age of 30 were edentulous and came to the operating theatre without teeth after premedication with Omnopon and Scopolamine. Vomiting on induction was fairly common and we were taught how to manage this very early in our careers. An oral airway was always used, and depth of anaesthesia was judged by the signs described by Guedel.

The Woolley and Roe case. As a junior anaesthetist training in Sheffield there was considerable reluctance to perform in spinal anaesthesia following the Wooley and Roe case in nearby Chesterfield. Two consecutive patients, namely Wooley and Roe became paraplegic following spinal injections of tetracaine for relatively minor surgery. The devastating complication was investigated and court proceedings ensued. Professor Sir Robert Macintosh was called upon for expert opinion and formulated the theory accepted at the time, that phenol had leaked into and contaminated the ampoules resulting in neurotoxicity. The anaesthetist, Dr Graham was acquitted based on the Bolam principle that he could not be expected to know this and had the standard of knowledge of competent anaesthetists in 1947. Dr Graham did not believe the 'invisible crack theory'. The case was revisited by Roger Maltby and more believable cause was found(5). It seems that the theatre sister responsible for sterilizing the needles had left acidic descaler in the sterilizing water boiler. No compensation was awarded to Wooley or Roe. A third patient severely ill from intestinal obstruction died a few days after operation, had probably also suffered neurological consequences. Interestingly, Lord Denning reviewed the case in 1955 and said there was no way compensation could be given. The case changed the legal judgements in medical

negligence to pro-doctor rather than pro-plaintiff. It is suspected that Lord Denning had in mind a no blame system akin to New Zealand's.

In the early 1974 there were six different different induction agents available to me and five muscle relaxants. Now at Auckland City Hospital we have refined this to 3 induction agents (Propofol, Etomidate and Methohexitone) and 4 relaxants (Atracurium, Rocuronium, Vecuronium, and Suxamethonium). The use of Suxamethonium has waned and Tetrahydraminacrime has disappeared. Methohexitone was widely held to be associated with seizure activity and promoted for ECT therapy due to its lowering of seizure threshold. Administration was associated with limbs movements and the literature, mainly anecdotal letters suggested this implied seizures. I was asked by Neurologists at GOS to give methohexitone to terminate a seizure which it did. Looking back I found that seizures attributed to the drug were merely the excitation it produced and this was based on a letter to a journal. This old pearl was handed down as being true from then on. Articles in journals usually used Student's t test for statistical analysis and 20 patients were the usual number needed to provide an answer. Nowadays, outcome research is based on thousands of patients. We tend to believe what we have learned in based on good foundation research but often that is not true.

I spent one year on fellowship at 'Sick kids' in Toronto. Only a few years earlier in 1980-81 Toronto was rocked by a murder enquiry as babies on Ward 41 Cardiology died unexpectedly during the night. When the story got out, immense pressure was put on the local police chief to solve this apparent crime(6). The pall of this event still hung over the hospital in my time there in 1985. So what did happen? In January 1981 the hospital mortality committee reviewed 22 cases aged between 9 days and 12 months who had died and found 15 cases with unaccountable causation. By March 1981, 32 more babies and 3 children would die on the unit - a 625% increase over similar periods, most dying from midnight to 6am. A post mortem examination of one baby showed higher than normal levels of digoxin and similar findings were discovered in other deceased babies. The Toronto police were called in to investigate. Under significant press coverage, the Mayor pressured the police department to rapidly control this and make early arrests. They began interviewing nurses on the night shift, who generally were surprised, tearful and exclaimed lack of knowledge, but when interviewing Susan Nelles, she merely asked to speak to an attorney. The police immediately took this to imply guilt and arrested her on March 25th 1981. After a four month preliminary hearing, Susan Nelles was released as she was not on duty for all cases. She successfully sued for false arrest and received \$190,000. The witch hunt then turned on Phyllis Traynor, who the US center for disease control in Atlanta identified as being present in all 29 suspicious deaths. Charles Smith was the pathologist involved, now disgraced. The cases remained unsolved. In April 2010, Nurse Lucia de Berk was cleared on seven charges of murder in a remarkably similar story to that of the Toronto. Gavin Hamilton believes no murders were committed. His book The nurses are innocent, believes the deaths were anaphylaxis to MBT, present in the rubber of pre-packaged syringes and which tests as Digoxin(7). The tests used to measure digoxin misread MBT as digoxin. At the same time as the Sick kid's deaths, Australian researchers warned of MBT's lethal effects especially in babies. Corporate governance is at the heart of a book by Sharon Stone, whose daughter died in another incident at Sick kid's hospital. The worrying feature is protection of institutional reputation perverting the availability of information and truth(8).

I was fortunate to be appointed as the only Registrar in the National Heart Hospital in 1981, working with Donald Ross and Magdi Yaccoub, famous cardiac surgeons. Donald was a contemporary of Christian Barnard at the University of Cape Town(9). A gifted surgeon he pioneered aortic homografts for right ventricular outflow tract reconstruction, developed methods of hypothermia and a pump oxygenator. In 1968 he performed the first cardiac transplant in the United Kingdom. The patient a 45 year old man survived 46 days before dying from sepsis. He performed 2 more heart transplants before halting further operations until rejection management was resolved. Two facts are of interest in this story. The first is the planning for the operation included practice on a pig. Unfortunately during preparations the pig escaped and was chased though the hospital. The second is that the anaesthetist involved, Alan Gilston, slept at the patient's bedside for the 46 days. His name is not mentioned in the

press reports. Alan was an inventor of the Gilston T piece and the Wallace cannula, and the founder of the UK Intensive Care Society in 197(10). During my time at the National Heart Hospital, Donald Ross decided a patient with critical multi-vessel coronary disease could be revascularised only by removing the heart to allow multiple grafts to be sutured. This was my only heart transplant case.

It was no doubt experimental in nature, and not repeated, but also probably marks the end of the era of surgical 'freedom'. I don't know the ethical situation surrounding the case, but I don't recollect any discussion on that particular issue.

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