Albert William Liley (1929–1983) [1]

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Albert William Liley advanced the science of fetal physiology and the techniques of life-saving *in utero* blood transfusions for fetuses with Rh incompatibility [5], also known as hemolytic disease. Due to his advances, fetuses too young to survive premature delivery, and likely to die *in utero* if their Rh incompatibilities were left untreated, were successfully transfused and carried to term. Liley was as passionate as a clinician and researcher as he was about his views on the rights of the fetuses.

Liley was born in Auckland, New Zealand on 12 March, 1929 to Zilla Jean Cook and Albert Harvey Liley. Liley's father was a painter. Liley studied at Auckland Grammar School in Auckland, New Zealanad, and in his senior year Liley won the New Zealand National Scholarship. Liley loved forestry as much as science, and though some accounts indicate that Liley studied forestry for a period of time, he ultimately studied pre-medicine at Auckland University [6] College, University of New Zealand in Auckland, New Zealand, starting in 1948. Liley earned his undergraduate degree in 1951. He then studied medicine at the University of Otago [7] Medical School in Dunedin, New Zealand. During his required research year at medical school, Liley worked with Australian Nobelist John Eccles [8], the Chair of Physiology at University of Otago [7] Medical School. Their research focused on the successful measurement of mammalian neuromuscular transmission, or the process that allows the central nervous system [9] to control muscle movement.

In 1953, Liley married Helen Margaret Irwin Hunt [10], a classmate at University of Otago [7] Medical School who later became an obstetrician and pediatrician. Liley and his wife shared strong views on the rights and care of the fetus [11] and those views blended between their personal and public lives. In their years together they had three daughters, two sons, and adopted a daughter with Down's syndrome. Liley and his wife shared their love of the outdoors with their family at their 88 hector property, which was 250 kilometers south of Auckland. On that property, Liley experimented on forestry improvements, influenced forestry development in New Zealand, and inspired his eldest son, Bill Liley Jr., into career in forestry.

Liley graduated from The University of Otago [7] Medical School in 1954 with his medical degree, but he did not enter an internship or residency program. Instead, Liley accepted a two year research scholarship at Australian National University [12], or ANU, in Canberra, Australia. Eccles had returned to Australia to become a Professor of Physiology at that same university. Liley earned a PhD from ANU with Eccles as his advisor in 1956. By then, Liley had published four papers on neuromuscular cell physiology. Liley and his wife returned to New Zealand, where Liley had a two year Sandoz research fellowship at the Auckland Postgraduate School of Obstetrics and Gynecology [13] in Auckland, New Zealand, with Harvey M. Carey [14]. Liley earned a Diploma in Obstetrics in 1962.

In 1965, Liley took sabbatical leave to research at the Department of Obstetrics and Gynecology at Columbia University [15] College of Physicians and Surgeons in New York City, New York. He worked with Vincent Freda [16] and Karliss Adamsons [17], both obstetricians and researchers in reproductive and fetal medicine. The three men, with their combined knowledge of physiology, neurology [18], obstetrics, fetal medicine, and surgical techniques, collaborated on interventions for medically compromised fetuses.

The collaboration continued when Liley returned to New Zealand in 1966 to the National Women's Hospital in Auckland, New Zealand, where he became the Senior Research Fellow at the New Zealand Medical Research Council [19] in Obstetrics until 1969. The hospital and its affiliated postgraduate school provided care to women with high risk pregnancies from all over New Zealand and the South Pacific. It was in Auckland that Liley and a team of fellow researchers focused on the prevention and eradication of Rh incompatibility [5]. Others involved in the research were Florence Fraser [20], an obstetrician, Ross Howie [21], a pediatrician, Graham Liggins [22], an obstetrician with expertise in fetal physiology, Sally Kinnock [23], a researcher, Herb Green [24], a clinical researcher and Neal Patterson [25], another obstetrician.

The Rhesus factor [26], often shortened to Rh factor, is named after the rhesus monkeys, which scientists used for the original Rh research. The Rh factor is a protein that provides a thin chemical coating surrounding the red blood cell. Red blood cells are cells that carry oxygen to all parts of the organism, which makes them crucial to an organism's survival. About eighty-five percent of people have that protein, and are called Rh-positive. People with partial or no Rh protein on their red blood cells are called Rh-negative. People with Rh-negative blood cannot tolerate exposure to Rh-positive blood.

If a pregnant woman is Rh-negative and her fetus [11] is Rh-positive, the woman's immune system creates antibodies against the fetus's blood. An antibody is protective protein that the immune system makes to overcome a foreign substance in the body, often called an antigen. The antibodies filter across the placenta [27] to the fetus [11] and destroy the fetus's red blood cells. The

fetal red blood cells then start producing at an accelerated rate to replace the blood cells that the maternal antibodies destroyed, but the red blood cells only partially form due to their rapid production. Those partially formed cells are called erythroblasts to differentiate them from normal or fully formed red blood cells, called erythrocytes. The production of erythroblasts leads to conditions such as anemia [28], jaundice [29], erythroblastosis, and often the death of thefetus [11].

Prior to World War II, if an Rh-negative woman produced antibodies in response to Rh-incompatibility in one pregnancy [30], she carried the antibodies for the rest of her life. For all of her later pregnancies, if the fetuses were Rh-positive, they were at risk for anemia [28] due to Rh incompatibility [5]. Anemia is when there is a deficiency of red blood cells or of hemoglobin, the protein in red blood cells that carries oxygen to all parts of the body. Prevention and treatment of Rh incompatibility [5] in fetuses required an understanding of the mechanisms of immunology.

Liley began his research on Rh incompatibility [5] in the early 1960s by becoming proficient in the technique of amniocentesis. He stated that testing the amniotic fluid was a way of testing the fetus [11] and its environment. Liley was sure that amniocentesis was the most accurate way to assess the course of the hemolytic disease in Rh-impaired fetuses. Repeated tests of the amniotic fluid, along with other measurements, indicated whether the condition was stationary or whether anemia [28] was worsening, and if so, if doctors had to induce labor.

A geneticist visiting the National Women's Hospital in the early 1960s told Liley and the research team that when she put normal blood cells into the abdomens, or peritoneums, of neonates and infants who had a hemolytic disease, or a disease of blood, the neonates rapidly absorbed the cells in large quantities. Normal cells were then in the peritoneums, inspiring Liley and team to do the same with blood transfusions to fetuses with Rh incompatibility [5].

In 1963 Liley aided the woman reported as Mrs. E. McLeod with herfetus [11]. McLeod's first pregnancy [30] was successful, but her second ended in intrauterine death and the third resulted in stillborn twins. She was on her fourth pregnancy [30]. The fetus [11] had hemolytic disease and would likely die within a week. Liley had attempted, and failed, three previous fetal blood transfusions [31] on fetuses from different mothers. With the knowledge he had gained from those attempts, he realized that McLeod's fetus [11] might need multiple blood transfusions before birth, as well as a transfusion after birth, to survive. For McLeod's fetus [11], Liley gave it a transfusion *in utero* and post birth. That time the fetal transfusion was a success.

At the time, fetal transfusions were novel. *The New Zealand Herald* ran headlines such as "Pre-birth Transfusion Saves Baby Boy", and "Baby Makes Headlines Over World", which brought people from all over the world, patients and physicians alike, to inquire and request Liley's expertise in Rh incompatibility [5] and the new treatment.

The range of success of the transfusion treatment in the first few years was only about thirty-nine percent. That range reflected the experimental status of the treatment. Needle penetration of the uterus [32] was a precarious procedure, even under x-rays and hands-on examinations. The fetus [11] shifted, as did the woman, for different reasons, and when the needle missed the fetal peritoneal cavity, the risk of puncturing a fetus' body part or rupturing the amniotic sac [33] was high, both of which could cause significant damage to the fetus [11]. In some of Liley's surgeries, he had to insert needles two, three, or four times, especially with young fetuses where it was more difficult to perform the procedure accurately. Gradually, more fetal surgeons became involved with transfusion surgeries and the success rates increased.

Liley received international recognition for his work in fetal research and application of his bench science to the patient. He was elected a fellow of the Royal Society of New Zealand in 1965, and he became a Chair in the Postgraduate School of Obstetrics and Gynecology at the Auckland University [6] Medical School in 1968. In 1971 he became a fellow of the Royal College of Obstetricians and Gynecologists and in 1978 Pope Paul VI [34] recognized Liley for his pro-life work. Though Liley was not a Catholic, the Pope made him a member of the Pontifical Academy of Sciences [35]. Liley was recognized as a Companion of the Order of St. Michael and St. George, received a British knighthood [36] in 1967, and in 1973 he received the title Knight Commander (of the Order) of St. Michael and St. George.

Liley's work focused on the fetus [11], improving the possibility of birth to fetuses with fetal diseases. According to Barbara Hawgood, Liley believed that the fetus [11] was a person, whose impact on theuterus [32] was one where the fetus [11] was dominant, that the fetus [11] had rights over the pregnant woman, and that thefetus [11] deserved the same respect and consideration as an adult.

Both Liley and his wife were active anti-abortion [37] activists. According to Hawgood, Liley gradually became distressed that amniocentesis, which sometimes offers information about possible handicaps, became a tool for some parents to decide to abort. According to Hawgood, he also disliked that some doctors used the needle procedure he had pioneered for life-saving transfusions to administer saline for abortions. Liley, never quiet about his beliefs, formed the Society for the Protection of the Unborn Child [38] in 1970 in response to the creation of the pro-choice Abortion Law Reform Association. Liley addressed the US Supreme Court in 1974 for a case to determine whether abortion [37] infringed upon the constitutional rights of the unborn children. He also testified to the Royal Commission on Contraception, Sterilization and Abortion in New Zealand in 1977.

The public did not always respond positively to Liley's activism. According to Monica Casper, the strain of his dual roles as a medical researcher and an activist may have partly caused Liley to commit suicide on 15 June 1983, at age 54. At his funeral,

officiated by both the Roman Catholic and the Anglican churches' highest leaders in New Zealand, at Auckland's Holy Trinity Cathedral, Liley was recognized for his great contribution to techniques that helped with fetal survival in complicated pregnancies.

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