William Thornton Mustard (1914-1987) [1]

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William Thornton Mustard was a surgeon in Canada during the twentieth century who developed surgical techniques to treat children who had congenital heart defects. Mustard has two surgeries named after him, both of which he helped to develop. The first of these surgeries replaces damaged or paralyzed muscles in individuals who have polio, a virus that can cause paralysis. The other technique corrects a condition called the transposition of the great arteries (TGA) that is noticed at birth. Surgeons worldwide adopted that technique, leading to increased survival rates in infants afflicted with the condition. Mustard also published over 100 articles on congenital heart defects, surgical techniques, and the preparation of an artificial heart lung machine. Mustard helped perform the first blood transfusion of a newborn whose red blood cells (RBCs) had degraded, a condition called hemolytic anemia [2]. Throughout his career, Mustard developed surgical techniques that increased the survival rates of infants and children with congenital and developmental disorders.

Mustard was born in Clinton, Canada, on 8 August 1914. He was the fourth of five children born to Pearl Mustard and Thornton Mustard, both schoolteachers. During his childhood, Mustard collected butterflies and identified birds [3]. During high school, Mustard met Elise Howe, who he later married. In 1930, at the age of fifteen, Mustard graduated from high school at the University of Toronto in Toronto, Canada, which offered high school diplomas, though Mustard was too young to begin the application process for university, so he stayed another year before beginning university.

Mustard began his university education in 1931 at the University of Toronto, with a focus in forestry. However, his older brother Donald, already in medical school, persuaded Mustard to pursue medicine at the University of Toronto Medical School. In 1937, the youngest in his class at the age of twenty-two, Mustard graduated with his medical degree. Shortly after, he worked as an intern at the Toronto General Hospital in Toronto, and then he moved to the New York Orthopedic Hospital in New York City, New York.

At the beginning of World War II, in 1939, Mustard's parents boarded the British passenger ship Athenia to visit their eldest son in England. When they returned to Canada, Britain declared war on Germany. And Germany torpedoed the Athenia. Both of Mustard's parents survived the initial attack, but they were trapped on the sinking ship. Mustard's mother survived the ordeal, but his father drowned that night. The death of his father left Mustard's younger brother with no money to finish his last year of school, so Mustard began working at a general practice in Northern Ontario, Canada, to pay for his brother's tuition. In 1940, Mustard returned to Toronto to continue his own education, and he received six additional months of training in surgery before he joined the Royal Canadian Medical Corps in 1941. During that year, Mustard married Elise Howe and the couple would have three children.

During his time in the Royal Canadian Medical Corps, Mustard developed his first surgical
In 1944, Mustard preserved the leg of Captain Graham Dixon, whose main artery was injured. Doctors often amputated legs after such injuries. Instead, Mustard inserted a tube to function as a bridge between the two ends of the severed artery until a vein graft could be transplanted to replace the glass tubing a few days later. Mustard's intervention was an early prosthetic tube used to mend a damaged artery, and it helped save the limb from amputation. For performing that surgery, Mustard became a member of the Order of the British Empire, an honor society that recognizes an individual's contribution to the arts and sciences. Despite the initial success of the prosthetic glass technique, the success rate of similar attempts was low. Between 1950 and 1953, doctors in the US improved the technique during the Korean War.

In 1948, Mustard and John Fraser, another doctor at the Hospital for Sick Children in Toronto, Canada performed the first successful total blood transfusion on a newborn infant suffering from hemolytic disease, which leads to hemolytic anemia, a condition for which red blood cells are abnormally broken down and removed form the blood stream. Hemolytic anemia in newborns occurs when a pregnant woman's body recognizes the fetus's blood as foreign. This condition is called Rh incompatibility and occurs when the mother is Rh negative and the baby is Rh positive.

Rh designates the presence or absence of the D antigen, which is a type of marker on the surface of red blood cells. Rh positive red blood cells have the D antigen present on the surface of their cells while Rh negative red blood cells lack the D antigen. When a pregnant woman's blood comes in contact with a fetus's blood, the woman's immune system produces antibodies against the blood of the fetus, namely the D antigen on the red blood cell. Typically, the placenta prevents the crossing of blood between pregnant woman and the developing fetus. However, during childbirth or traumatic injuries during pregnancy, there is a higher chance that the blood of the fetus crosses the placenta. The crossing of the blood triggers the formation of antibodies in the woman's immune system. The antibodies attack the fetus's cells that are identified as foreign to the woman's body. The pregnant woman's antibodies bind to the D antigen present on the Rh positive fetus's red blood cells. This attack on the fetus's red blood cells causes them to lyse, or break open, a condition called hemolytic disease. Hemolytic disease causes anemia, which is fatal without a total blood transfusion, because the blood cannot transport oxygen to the tissues and organs throughout the body.

In 1948, Mustard and Fraser completed a total blood transfusion in a newborn through the umbilical vein soon after the baby was born. The umbilical vein is part of the umbilical cord that connects the developing fetus to the placenta during development and remains visible for several days after birth. Mustard and Fraser published their results in 1948. Together, Mustard and Fraser treated twenty-one infants who suffered from hemolytic disease caused by Rh incompatibility.

In 1949, Mustard began developing an orthopedic surgery to replace paralyzed and damaged muscles of the hip abductors in patients afflicted with the lasting consequences of the Polio virus. Some of the common consequences of this virus include paralysis of the hip abductor muscle and the gastrocnemius muscle in the calf of the leg. During that procedure, Mustard replaced the damaged hip abductor muscles with the iliopsoas muscles from the upper thigh and abdomen.

In 1952, Mustard published a preliminary report about his surgical technique in the Journal of Bone and Joint.
and later in May 1959, he published a follow up study in the same journal. In this second article, Mustard reviewed fifty cases of patients aged four to fifty five, who underwent the Mustard Procedure. He found that almost all patients could walk for longer distances, with less fatigue in their leg. Mustard received international recognition for the Mustard Procedure.

After Mustard's work on Polio, he focused his work in cardiac surgery, particularly with the cardiovascular congenital defect called the transposition of the great arteries (TGA), also called transposition of the great vessels. The cardiovascular system includes the heart and the blood vessels and is responsible for distributing oxygen and nutrients to cells and tissues within the body via the blood. The human cardiovascular system comprises the systemic circuit, which supplies oxygen-rich blood from the aorta to the body, and the pulmonary circuit, that takes deoxygenated blood from the body and transports it to the lungs for gas exchange. In a fetus with transposition of the great arteries, the positions of the aorta and of the pulmonary artery are switched. In this case, the aorta gets deoxygenated blood, while the pulmonary gets oxygenated blood. Before the mid-twentieth century, the mortality rate for such a condition was 89 percent. Many infants were either stillborn or died of systemic organ failure within the first few months of life. Mustard developed a procedure, which came to be called the Mustard Operation, to fix the switched arteries in children with TGA.

Mustard's first attempt to repair TGA involved cutting a hole in the septum, the heart tissue that separates the atria, which are the top chambers of the heart that enable oxygenated and deoxygenated blood to mix. That incision enabled blood from the systemic circuit to enter the blood from the pulmonary circuit and thus increased the amount of oxygenated blood available for the body. Mustard used this technique on twenty-eight children, fourteen of which survived. However, their life expectancies did not significantly increase with this intervention alone.

In 1963 Mustard, together with George Trusler, a physician at the Hospital for Sick Children, refined the TGA operation with a refined technique to redirect blood flow and to introduce oxygenated blood into the body. They performed it on an eighteen-month old girl named Maria Surnoski, on whom Mustard had earlier cut a hole in the atrial septum. Surnoski surgery on the infant proved successful and she recovered completely. Five years after Surnoski's surgery, Mustard operated on twenty-six children with transposition of the great arteries, nineteen of which survived.

Mustard remained at the Hospital for Sick Children until he retired in 1976. He published dozens of reports about surgeries to correct abnormalities in infants, and he further refined the TGA procedure that became named for him. He received many awards for his contribution to the medical field and community, and he was inducted into the Canadian Medical Hall of Fame in 1995. In 1979, Mustard's wife and his brother Donald died. In 1986, Mustard was diagnosed with poor blood outflow. Mustard opted not to have surgery to fix the issue, and he died from a heart attack in Florida on 11 December 1987.

Sources


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