
Mr. President, Members of the Association, Mrs. Gibbon, Ladies and Gentlemen.

A little less than a month ago I received a letter from your president telling me that I had been selected by your association to receive The John Gibbon Award for 1977. The honor that you have bestowed on me has touched me more deeply, both intellectually and emotionally, than any other one that I may have received. I was asked to give you a brief talk on this occasion. You kindly gave me freedom to choose any subject that I preferred. At my high age, I have not actively participated in the technicalities of extracorporeal circulation since my retirement over 10 years ago. I believe that you have already heard enough technical details in this field during this meeting, so I felt I should instead concentrate on the more historical aspects and the development of the professional life of the man in whose memory this award is given, to the similarity with my own career and how we came to work in the same direction since the early thirties without knowing anything of each other before 1939.

As perhaps some of you may know, I met Jack and Maly Gibbon and and their family way back in 1939 and, as time went by, they became close friends of my family and myself. Already from the first hours that Dr. Gibbon and I met in Los Angeles at the yearly meeting of The American Association For Thoracic Surgery, we found that our main professional interests were mutual. It was on postoperative thrombembolic disease that we had focused our efforts. Right from the beginning, Dr. Gibbon attacked the problem from the active surgical point of view and his principal task was to prolong the period that the patient could live after an attack of...
massive pulmonary embolism. If the embolus actually blocked almost 100% of the pulmonary circulation, the time a surgeon had at his disposal to remove it was a matter of minutes. Dr. Gibbon demonstrated this in experimental animals by acute clamping of the pulmonary artery. If the pulmonary artery was blocked for more than a few minutes, all the animals died with more or less widespread necrosis of the brain. The central nervous system is the organ that can stand lack of oxygen less well than any other tissue in the body. Both Dr. Gibbon and I, during our time as surgical residents, had experienced this in cases where pulmonary embolectomy was tried. It was not until 1924 that the Trendelenburg operation first performed in 1908, after a rather large number of failures, was successfully performed by Professor Kirschner in Germany, when a patient’s life was saved by pulmonary embolectomy. Both Dr. Gibbon and I had realized that only under very fortunate circumstances could individual patients be saved by this operation in the presence of almost 100% occlusion. Dr. Gibbon tackled the problem further by trying to find a method by which the venous blood could be safely and by technically simple means evacuated from the venous side, oxygenated outside the body and then returned to the arterial side of the heart. This procedure should be of such long duration that in cases of massive pulmonary embolism the pulmonary artery could be opened and the embolic masses removed without undue haste and with ample oxygenation of both the brain and other organs.

In view of the severity of the symptoms displayed by many patients, who survived spontaneously after a massive attack of postoperative thromboembolic complications and the survival of only exceptional cases after attempted pulmonary embolectomy, in my own experience only 2 of 10, I started the laborious task of trying to find some way of preventing this complication. Therefore I investigated numerous changes in the blood, which were produced by operative treatment, and could have some connection with thromboembolic complications. I read at that time McLean’s article of 1916 about heparin. He was a pupil of Howell and they tried to define the chemical nature of this substance which had a very marked influence on the coagulation time of blood. Back in the late twenties I had tried to persuade my friend Dr. Eric Jorpes to continue from where McLean and Howell had stopped in order to provide a purified substance without any toxic side-effects which could be used to prevent thromboembolic complications. He said then that this was too complicated. But a few years later he told me that he had come across some work performed in Canada which had caused him to intensify his work on heparin. In 1935 he had succeeded in the laboratory of the Vitrum Company, where he was employed as expert consultant, in producing a heparin preparation which had proved both in experimental animals and in man to have no toxic effects, even in doses high enough to render the blood incoagulable for short periods. In August 1935 I got the purified heparin from Vitrum through Dr. Jorpes and started my first series of preventive postoperative treatment to see if by this method the incidence of postoperative thromboembolic complications could be diminished or perhaps even eliminated.

At about this time, Dr. Gibbon was given a second period of employment as research assistant in Dr. Churchill’s laboratory in Boston with his wife Maly and his assistant also on that occasion. She had previously worked in Churchill’s experimental laboratory and helped Jack Gibbon during his first period with Churchill, when they worked on the thromboembolic problem in 1930, and it was
after Dr. Gibbon’s first period with Churchill that they married. Meanwhile, a heparin compound had also been produced by the Connaught Laboratories in Toronto and it was this preparation that Dr. Gibbon used during his experimental work on heart-lung machine construction. Without the discovery of heparin all extracorporeal technology would have been impossible.

During the years 1935-37, when I gave heparin postoperatively in order to prevent thromboembolic complications in certain groups of patients in whom experience had shown that the incidence of such complications was high, I also worked in the experimental laboratory. The purpose of that experimental work was to establish the role that insufficient brain circulation played, when the pulmonary circulation was almost completely blocked by massive pulmonary embolism. In all cases, in both rabbits and dogs, where the pulmonary artery was clamped for more than 6-7 minutes, the animals died and showed symptoms of serious brain damage. If the pulmonary occlusion time was diminished by only a couple of minutes they could survive, but all of them had symptoms indicating more or less severe damage to the brain. I therefore did a series of animal experiments in which the central parts of the carotid arteries and deep jugular veins in one animal were anastomosed to the peripheral parts of the same vessels in another animal of the same species. Immediately afterwards, the pulmonary artery was clamped in the animal with brain circulation preserved from the other animal. By this method I could show that the pulmonary artery could be clamped for a little more than 17 minutes in the animal with cross-circulation of the brain from the other one and, that after opening up of the pulmonary circulation by removing the clamp on the pulmonary artery, the normal circulation to the brain could be reestablished and the animals survive without any signs of brain damage or of changes in any other organ of the body.

During this same period, Emil Anderson, an experimental engineer at the big international AGA Company in Stockholm, cooperated with me on the problem of constructing an effective and physiologically functioning machine for artificial respiration and for anesthesia. Our combined efforts resulted in the construction of the spiropulsator. We considered that this machine was the best existing one at that time for keeping the ventilation as normal as possible during long-term intrathoracic operations. This was described in my doctoral thesis in 1938 entitled: Total Pneumonectomy In Man. It was thanks to the interest that this publication aroused in the U.S.A. that I was invited by the American Association for Thoracic Surgery as guest speaker in Los Angeles in 1939. There I met Dr. Gibbon without having had any previous knowledge of his work. On our way back to Sweden from Los Angeles, Engineer Anderson, who accompanied me in order to demonstrate all the technical details of our ventilating anesthesia machine, and I visited different centers where we were invited to use it in patients undergoing long-lasting intrathoracic operations and we also visited Dr. Gibbon’s experimental laboratory in Philadelphia. There we were extremely impressed by the function of a heart-lung machine under construction by him. This machine could take over the total circulatory blood leaving the right side of the heart, oxygenate it nearly as effectively as in the lungs of the animal (a cat) and then pump it back to the arterial circulation. During that time he could completely clamp the pulmonary artery for more than half an hour with long-term survival of the experimental animal and without signs of any untowards effect on either the brain or any other organ system.
In connection with the cross circulation experiments that I have just described, Anderson and I had discussed the possibility of constructing a mechanical device for oxygenating the blood outside an animal's circulatory system in sufficient quantities to keep the brain surviving, and here we saw such a device constructed and already in use which could carry a small animal's total circulation.

After our return from the U.S.A. 1939, Anderson and I started work on a device for oxygenating venous blood outside an experimental animal's body in the early 40s. The first experimental machine was a disc oxygenator. At that time, Anderson and I never thought that we could construct a machine for extracorporeal circulation of such magnitude that it could take over all the blood from a big animal or a human being, oxygenate it and then return it. My aim was to try to develop a machine large enough to take out sufficient blood from a big animal, or even from a human being, so as to provide the brain with oxygenated blood during the short time required to remove big pulmonary emboli or perhaps repair an intracardiac congenital malformation or diseased valves.

It was not until after the end of World War II that we had come so far, and it was at about the same time that Dr. Gibbon came back home from the Pacific War and could continue his work on the heart-lungmachine in Philadelphia. It was rather a strange coincidence that two letters, one from Dr. Gibbon to me and one from me to him with the idea of resuming discussions on the progress of extracorporeal circulation, crossed each other on their way over the Atlantic.

One of my associates Dr. Viking Olof Björk, showed in his doctoral thesis, that with the heart-lungmachine, which he and Anderson had improved upon very much from the stage it had reached in the slide I showed you, that blood from a big dog could be removed in such quantities and oxygenated in the heart-lungmachine so as to preserve the brain circulation for about half an hour's clamping of the pulmonary artery. The experimental animal could be kept alive and survive without any signs of brain or other organ damage.

In the immediately following years, Dr. Gibbon and I and often members of our families met at least once a year. He had come into contact with a big firm the International Business Machines in the U.S.A. where he was helped with technical problems just as I was by the AGA Company in Stockholm. The development of his heart-lungmachine proceeded much quicker than ours. My friend, Engineer Anderson, died from a severe mitral stenosis, and tragically enough we had not made such progress in our research that we could save him by operative treatment. Dr. Björk was temporarily on leave for studies abroad. During that time another of my associates Dr. Åke Senning, who is now Professor of Surgery in Zurich, and Engineer P. A. Åstradsson, who was an associate with Anderson, took over where Björk and Anderson had left off and we tried to catch up with Dr. Gibbon's work. I didn't know at that time that Clarence Dennis, working with Wangensteen in Minneapolis, was also engaged in experimental work on a heart-lungmachine. Then, at last, in the beginning of May 1953, Dr. Gibbon considered that his heart-lung machine was then so far advanced that it could be used for open cardiac operation in man. On May 6, 1953, with the aid of his machine he operated on a case of atrial septal defect where the patient was under total bypass for a little less than half an hour and with complete success. The patient survived, and is alive and well today. No further attempts were made to Dr. Gibbon's machine, as far as I know,
to operate clinically under extracorporeal circulation until July 16, 1954, when we operated on our first patient in Stockholm. Since that time, the machine was improved upon continuously by Dr. Åke Senning and Engineer Åstradsson in my clinic until 1961.

Before I finish my talk, I will tell you in just a few words about the first successful case operated on with heart-lung machine in my clinic in 1954. A female patient was observed on several occasions from the fall of 1952 for heart trouble and sometimes for short periods in the hospital. A mitral lesion was suspected and in December 1953 a right-sided heart catheterization was performed which showed elevated pressures in both the pulmonary artery and the left auricle. After catheterization, she had a sudden syncope attack, and in association with it, a slight right sided hemiplegia. The symptoms disappeared rapidly and when she left hospital the only sequel was slightly decreased strength in the right hand. She was under observation until May 1954 when it was decided to perform a new catheterization and also undertake angiocardiography. On that occasion, there was also increased pressures both in the pulmonary artery and in the left auricle. After injection of contrast we got pictures of the left auricle which showed the presence of a rounded solid structure in the auricle. Its origin was discussed and the presence of an intra-auricular myxoma was considered to be the explanation. The patient and her relatives were fully informed about the risk of operation and her prognosis later in life with or without an operation with removal of the growth that was found in the left auricle. She was also told that she was the first patient in our experience to be operated upon under extracorporeal circulation of the blood and that we considered it was impossible without extracorporeal circulation. The operation was carried out in combination with hypothermia around 30°C and extracorporeal circulation for 27 minutes during the intracardiac procedure. The operation went smoothly and was without complications. The intracardiac part was done under ventricular fibrillation, produced by electric shock of 50 mill/amp for half a second. In spite of a maximally wide incision in the left atrial wall, it was impossible to remove the intra-auricular tumor in one piece. It had to be divided and removed in several parts. The tumor, a myxoma, originated from the intra-auricular septum and the attachment area was small. Throughout the intracardiac manipulations, the mitral valvular opening was completely blocked by the finger of my first assistant, Dr. Senning. There were no signs of any embolization postoperatively. Cardiac massage and one more electric shock were given before the heart started to work with a sinus frequency of 120/min. The postoperative course was free from any dramatic complication. This patient is still alive today and just before I left Stockholm I had telephone conversation with her and she is still in good condition in spite of her 66 years of age, more than 20 years after the cardiotomy.

Our optimism after this first successful open heart operation in our clinic was dampened by the next unsuccessful attempts to correct congenital heart lesions mainly caused by our lack of knowledge of the hemodynamic pathophysiology that today is the basis of success and progress in the field of cardiovascular surgery. On the other hand, I had the pleasure to take part in the development of new methods in cardiac surgery in my department: e.g. the correction of total anomalous pulmonary venous return, of the transposition of the great vessels, of the treatment of total atrio-ventricular block with permanent implantable pacemakers, etc., etc.
From what I have now told you, I wanted to give you my own opinion about what I myself consider concerning the value of my contributions towards the field of extracorporeal circulation. I said in the paper I presented at the 2nd Henry Ford Hospital International Symposium on October 6, 1975, that it was more the fact (quote) "That I was born just at the time when new developments in biology and engineering could be combined and used in the revolution of surgery" (unquote) and that (quote) "I was also fortunate in being able to collect a group of very able, highly intelligent, hard-working men round me," (unquote) men such as Emil Anderson, P.A. Åstradsson, and surgical assistants such as Viking Olov Björk, Åke Senning, Lennart Johansson and Olof Dählback, now all of them heads of university departments of thoracic and cardiovascular surgery. In this way I was able to contribute to intracardiac surgery. I myself am really somewhat in doubt as to whether I am worthy of the high distinction that you have given to me tonight, but I do wish you to know that I value this, the John Gibbon Award, more than any other that I have received in my rather long life.