Recollections of My Research in Developing the Heart-Lung Machine at Jefferson Medical College

Bernard J. Miller

Thomas Jefferson University

Follow this and additional works at: https://jdc.jefferson.edu/jeffbiographies

Part of the History of Science, Technology, and Medicine Commons, and the Surgery Commons

Let us know how access to this document benefits you

Recommended Citation
https://jdc.jefferson.edu/jeffbiographies/12

This Article is brought to you for free and open access by the Jefferson Digital Commons. The Jefferson Digital Commons is a service of Thomas Jefferson University's Center for Teaching and Learning (CTL). The Commons is a showcase for Jefferson books and journals, peer-reviewed scholarly publications, unique historical collections from the University archives, and teaching tools. The Jefferson Digital Commons allows researchers and interested readers anywhere in the world to learn about and keep up to date with Jefferson scholarship. This article has been accepted for inclusion in Jefferson Biographies by an authorized administrator of the Jefferson Digital Commons. For more information, please contact: JeffersonDigitalCommons@jefferson.edu.
Recollections of My Research in Developing the Heart-Lung Machine at Jefferson Medical College

[typescript, 24 pages]

Thomas Jefferson University Archives & Special Collections

Manuscript # 113

Dr. Bernard J. Miller, M.D.
My initial interest and fascination with research began while I was attending high school.

Following graduation, I succeeded in obtaining a volunteer position in the laboratory of the Cancer Research Institute of Lankenau Hospital, which is now the Fox Chase Cancer Research Institute of Philadelphia. During vacation periods while at college, I would work in the laboratory on a full time basis and, during the winter, I would manage to work 1-2 evenings a week and, in addition, the entire weekend. My initial assignments consisted in cleaning the urine specimen bottles and then I very quickly advanced to the pathology laboratory, learning to prepare specimen slides, and then to doing experimental embryology, under the direction of Dr. Stanley P. Reimann, the Director of the institute. I was involved in the recovery of human ova from operative specimens, mitotic manipulation of the human ova, and, eventually, the use of fertilized rabbit ova in circulating tissue culture as a method for studying the affects on growth of various amino acids. There were a number of publications and presentations that I made during that period. The friendship with Dr. Reimann continued on a father/son basis for many years thereafter until his death. At graduation from Villanova College, I was first in my class.

Following my graduation from Jefferson in 1943 and having achieved the Dean’s List, the Honor Society AOA, and recipient of the Medal in Surgery at graduation, I was offered the internship at Jefferson, which had been reduced to a nine month period at that time because of the need for medical officers in the armed services during World War II. I completed my internship and was then retained for an additional two nine month periods as a resident in general surgery, under Dr. George P. Muller. My work during my residency at Jefferson and prior to entering the armed services was exemplary, and I had hoped to return to Jefferson at the conclusion of my tour of duty in the armed services to complete my surgical training.
As my tour of duty was coming to an end, I requested an appointment with Dr. Gibbon, who had been appointed as the Professor of Surgery and Chief of the Surgical Services following Dr. Muller’s death, for the purpose of discussing the resumption of my residency training after separation from the armed services. I came to Philadelphia to meet with Dr. Gibbon and, as I recall, I was immediately told that there was no longer any place for me in the surgical residency training program at Jefferson nor would there be in the future. I was truly amazed, first since my performance in the early part of my residency at Jefferson had been exemplary, and it was my impression that returning veterans would be welcomed to the positions they had occupied prior to entering the service. I knew of no reason for Dr. Gibbon’s feelings, although I did have some personal thoughts about that.

Influential friends and admirers of my work at both The Lankenau Research Institute and Jefferson became aware of this. They immediately contacted the dean of Jefferson Medical College and Mr. Rosenwald, Chairman of the Board at Sears, Roebuck and also a member of the Board of Trustees at Jefferson Medical College. Within a few days following my interview with Mr. Rosenwald, Dr. Gibbon wrote to me at Fort Bragg informing me that I would be able to continue my surgical training at Jefferson following honorable discharge from the service.

In July of 1947, I was separated from the Army with honorable discharge and returned to Jefferson to meet with Dr. Gibbon. He informed me that there was no place available in the hospital for me at that time and that I would need to spend a year in the surgical research laboratory prior to clinical experience. His attitude was one of complete indifference. He did,
however, suggest that I interest myself in the problem of blood volume changes in surgical
patients undergoing major thoracic operations in which he was interested, and he directed me to
the laboratory where “I could find some test tubes and some blue dye T-1824.” This was
completely satisfactory to me since I was eager to return to Jefferson for continuation of my
surgical training in any manner.

I started the new research project very energetically and, within a short time, was making
measurements of blood volumes before and after major thoracic operations and correlating this
with changes in the extracellular fluid space and then relating this study to a parallel study where
actual intraoperative blood loss was determined by the weighing of sponges used during the
operations. By Thanksgiving of 1947, I had accumulated a significant amount of data and, for
the first time, requested an interview with Dr. Gibbon for the purpose of discussing the findings
of the blood volume study. I met with him and Frank Allbritton, and they reviewed my work
very critically. They were very impressed with my findings, remarking to each other “Gee
Frank, have we done all this damn work.” They then directed me to write a paper and submit it
to the American Thoracic Association for presentation at the spring meeting in Quebec, Canada.
This paper was well received. The photo that was taken of Allbritton, Templeton, and me at the
Frontenac was actually made during that meeting. Following the presentation of the paper, both
Gibbon and Allbritton’s attitude changed appreciably, and they actually became cordial. In fact,
by Christmas of that year, Dr. Gibbon invited Ethel, my wife, and me to a reception at his home.
He met us at the door and, with his arms around both of us, directed us to his study, at which
time he said, “BJ, let bygones be bygones.” Ethel and I were absolutely amazed.
From that time on, work in the laboratory became extremely enjoyable, pleasant, and also progressed very rapidly. I extended the work to blood volume measurements in patients with tuberculosis during stages of thoracoplasty and developed a method of objective measuring the circulation time by detecting the moment of arrival of intravenously injected blue dye at the lobe of the ear with an oximeter which I had designed and constructed. The study was further extended to correlating dye dilution patterns in the blood of the ear lobe as detected by the recording oximeter with various levels of cardiac shunts. I did not publish this work because there were only a few such cases available at that time. There was, however, a very definite dilution pattern specifically for each level of intracardiac shunt. In July of 1948, I was transferred into the hospital to complete the clinical aspects of my surgical residency and spent the next 2 ½ years in intense surgical training. I thoroughly enjoyed this time and developed a fondness and respect for Dr. Gibbon.

At the end of the surgical residency, Dr. Gibbon offered me the position of research assistant on a full time basis. I could not accept this on the basis of full time because we were having our first child and, during my surgical residency at Jefferson, I did not receive any financial support from the hospital so I needed to get on with making a few dollars. He did, however, change his offer, offering me the same salary, namely $3,600 a year, on a part time basis. Within a few months, after my position as research assistant began, he increased my salary to $4,800 a year on a part time basis and appointed me research associate. I had the opportunity of developing a small surgical practice of my own by that time which I managed in the afternoon and evening.
Following World War II, Mr. Thomas J. Watson, Chairman of the Board of the International Business Machine Corporation, became interested in helping Gibbon develop a heart/lung machine, and he assigned the experimental laboratory at Binghampton, New York to the construction of such a machine. Don Rex was the engineer appointed for the project. In 1947, the first of the IBM heart/lung machines arrived and was used extensively in animal experimentation by Stokes and Flick. In essence, the machine was a duplicate of the original machine used by Gibbon in his 1937 and 1938 experiments and actually similar to that originally patented in 1922 by Pierre DeLosolot in France. The only difference between the new IBM machine and the device used by Gibbon originally was that it had a much larger smooth film type oxygenator and the collecting bowl at the bottom of the oxygenator was gold plated with the thought that there would be less interaction between gold and stainless steel by the blood.

After an extensive animal experience, the machine appeared to be completely inadequate for human perfusion. There were a number of defects in the performance of the device. 1. The oxygenator was completely inadequate and could not support the respiratory requirement of even a large dog. The control of the arterial pump was photoelectric and malfunctioned frequently resulting in air embolization of the subject being perfused. The machine occupied two stainless steel and glass cabinets and was beautifully machined. To have enlarged a smooth film oxygenator such as this to the point where it would have had sufficient capacity to oxygenate a large dog or a very small human would require a geometric design of unrealistic proportion and also blood hold up. 2. There was, in addition, a large amount of hemolysis. 3. As the work progressed, it became evident very quickly that the matter of respiratory acidosis developing during the thoracotomy and perfusion needed to be corrected. Other problems also needed
solution, namely the continuous removal, without loss, of a large volume of cardiac venous blood returning to the right atrium during open atriotomy and removal of foam within the left ventricle at the time an interventricular or interatrial septal defect existed when atmospheric air entered the left side of the heart by way of the defect. At this point in time, it was clearly obvious that another machine needed to be built based hopefully on the solution of the problems which I have outlined. Mr. Watson assigned three engineers, Alf Malmros, senior engineer, John Engstrom, and Leo Farr, to the project concerned with the construction of a new machine. Animal experiments were suspended.

Richard and Drinker many years before introduced the element of turbulence in a smooth film oxygenator by forming the blood film on a silk screen and demonstrated that the degree of oxygenation could be vastly increased. Accordingly, Stokes and Flick lined the existing cylinder with a stainless steel screen and were able to show a significant improvement in oxygenation. At this point, all animal experiments were terminated. It was decided to use the existing machine as a supporting structure for further testing of new and advanced components.

The laboratory staff consisted of six technical assistants who maintained and prepared the machine, tended to the experimental animals, and procuring and typing of donor blood. In addition, two surgical residents or fellows were usually assigned for a nine month period to the research laboratory. This was a superb learning experience for the residents. Many of the residents of that period achieved high positions in surgery here and abroad; for example, Anthony Dobell, in Canada, and Dr. Hans Engle, in Denmark.
I directed the work in the laboratory, performed all of the perfusion experiments, and did the experimental design and development of the various components that would eventually be incorporated into the new machine. The engineers and I constantly communicated by phone with each other. They would come to the laboratory when I had designed a new component for demonstration. This usually occurred twice monthly. In 1952, engineer Engstrom, of IBM, gave a paper entitled “Electronic Control Circuits of the Mechanical Heart/Lung Apparatus” to the Industrial Electronic Group of the Society of Mechanical Engineers and acknowledged that I had conceived and tested the new control circuit. In the 1990’s, I received the Holley Medal from the American Society of Mechanical Engineers for the fluid control servo system that I had designed. The system that I developed made the machine completely automatic and provided a safeguard against embolization and, in addition, maintained constant blood volume hold up.

On one or two occasions, Dr. Gibbon and I went to Binghamton, New York to observe the progress on the construction of the new machine. Dr. Gibbon rarely came to the laboratory, only when prominent guests, such as Crafoord, Dennis, Byork, etc., came to the laboratory to visit and discuss the problems of the heart/lung machine. This fact was noted in the Gibbon lecture which Dr. Dobell had given at the American College of Surgeons in the 90’s. Subsequently, as the experiments proceeded, additional problems became evident, namely that of acidosis during the thoracotomy and perfusion, the recovery of cardiac venous blood without loss, and the recovery of blood and foam originating in the contracting left ventricle. At this place in time, the heart continued to pulsate during perfusion and the repair of experimentally produced septal defects.
The first priority was to develop an oxygenator capable of supporting the cardiorespiratory function of a large dog. A small test oxygenator was constructed using 2 x 12 inch strips of stainless steel screens of different wire sizes, mesh sizes, and punch metals suspended from a weir of 0.015 inches in width. This test device was then suspended in a cylinder containing oxygen. The oxygenation characteristic of each surface was determined as reduced cow blood flowed down each surface. Data derived from these experiments provided the basis of a new oxygenator using a turbulent film.

The first configuration consisted of a screen cylinder with a super-imposed cone upon which blood was deposited from a rotating jet. Filming of the blood was not uniform, and the blood tended to descend on the screen surface in rivulets producing a thick film of small surface area. Only after painting the screen with a protein solution, such as a dilute suspension of blood, could a complete film be established. The final design of the oxygenator consisted of six stainless steel screens, measuring 30.5 x 45 centimeters in size, suspended from individual weirs or a series of slots, each measuring 0.015 inches in width, and the entire assembly contained within a plastic case. This configuration was suggested by Alf Malmros and presented a 1.64 square meter surface area for oxygenation.

It was expected that blood pumped through the weir, which was located in the floor of the distribution chamber at the top of each screen, would be uniformly distributed along the entire width of the screen. Again, filming was incomplete and erratic. A uniform film of blood could be established only by flooding the oxygenator case with saline solution and then, as blood was being pumped into the distribution chamber, the mixture of saline solution and blood was rapidly
emptied. The falling layer of blood located at the top of the saline solution painted the screen, and the film was then maintained by the continuous recirculation of blood from the bottom of the oxygenator case to the distribution chamber. To reduce the volume of blood contained within the oxygenator assembly, the pool at the bottom was replaced, in part, by a block of plastic material that also served to maintain the position of the screen. The rate of flow through the recirculating pump was maintained at the maximum rate for which the oxygenator was designed. With six screens, this oxygenator could raise the saturation of blood with oxygen from 65% to 95% at a flow rate of 250 milliliters per minute.

The original photoelectric level control device used for controlling the rate of evacuation of the oxygenator by the arterial pump functioned erratically with resulting fatal air embolism. Proper control of this pump was extremely critical lest air be pumped into the arterial tree of the subject being perfused. A new device was conceived, the blood level in the bottom of the oxygenator was considered one element of a capacitor. The other element of the capacitor was a fixed electrode separated by a dielectric which, in this case, was the plastic case of the oxygenator. Accordingly, a small metal electrode was incorporated within the case and completely sealed at the control point at which the blood level was to be maintained. The capacity between the blood level and the fixed plate, in conjunction with an inductance, formed a tuned circuit operating at 10.7 megacycles. Changes in the blood level altered the frequency of the tuned circuit and provided the error signal for the control of the arterial pump. The arterial pump was then completely controlled by the blood level in the bottom of the oxygenator case. The variable capacitor circuit functioned well and appeared to be completely reliable providing proportional control of the arterial pump at a maximum flow within 0.1% and guarded against air
embolization of the systemic arteries. The accurate and instantaneous control of the capacitor system maintained the blood volume in both machine and subject at a constant level.

A filter of new design based on the reverse flow of blood was then included in the arterial line. This filter provided for the removal of air or fibrin from the chamber during the initial filling if required. Nichrome wires were embedded in the plastic case and heated electrically only sufficiently to prevent condensation on the interior of the oxygenator case.

With this modified machine, a number of dogs were successfully perfused during total bypass. At this time, the venous pumps were placed at a higher level than the heart of the subject being perfused. It was necessary to withdraw the blood from the vena cava with a moderate degree of suction. Since the venous DeBakey pumps were pulsatile, the moment the rotating roller contacted with the rubber tube, there was a marked increase in velocity of blood flow with a concurrent decrease in the lateral pressure and, therefore, the caval walls would be drawn into the orifice of the cannula at low flow rates and interrupting vena caval blood flow. When this occurred, blood could not be withdrawn through the occluded cannula despite the fact that the venous blood continued to flow into the cava. In order to control this, a small segment of thin wall collapsible rubber tubing was placed within the venous line and used as a pressure sensor. In the final design, a linear variable differential transformer sensed the intermittent collapse or change in diameter of the rubber tube in the venous circuit. A spring loaded lever rested against this segment of rubber tubing and was linked to the core of a different transformer. Minute changes in the diameter of the tube as a result of variations in pressure during the pumping cycle produced the necessary control signal to affect reliable automatic
control. At the moment of occlusion of the cava, the rubber tubing collapse was sensed by the device and the venous pump motors were stopped instantaneously. Intermittent erratic pressure changes preceding complete occlusion were indicated by fluctuation of a meter, known as a "flutter meter." By watching the oscillations of the flutter meter, the perfusionist was then alerted to reduce the speed of the venous pump and so prevent repeat occlusion of the vena cava and a cessation of blood flow into the system.

At this point, the new machine arrived in the laboratory, and an intense program of animal experimentation was undertaken. The motors of the venous pump in the first IBM machine were direct current motors operating directly from rectifiers. The low torque at reduced speed of direct current motors made them unsuited for this purpose. Accordingly, in the new machine, the alternating current motors replaced the direct current motors of the venous pumps with the exception of the arterial pump motor. Variable speed control was affected by Graham mechanical transmissions. Quick braking of alternating current motors was achieved by instantaneously applying a direct current to the motor.

The early phases of animal experimentation with the new machine took place early in 1951. Experiments with the new machine were only moderately successful. The morality was unduly high despite the apparent satisfactory condition of the animals during perfusion. Attention was now focused on this problem. Gasometric studies during anesthesia with a laboratory respirator revealed marked hypoxia and acidosis, probably the result of inadequate tidal exchange. Increasing respiratory rate or volume was ineffective. The notion of assisting expiration by increasing tidal volume of expired gas at the expense of the reserve gas volume without
increasing intratracheal pressure by the use of suction was investigated. Additionally, by rapidly reducing intratracheal pressure, alveolar capillary blood pressure and tissue perfusion have been shown to be markedly enhanced. Accordingly, a laboratory model of a respirator containing a timing circuit that alternately controlled electrically operated solenoid valves on both the inspiratory and the expiratory lines was constructed. Expiration was assisted by suction produced with a Venturi jet operating from the same air pressure source. The respirator operated at 5-pound line pressure of compressed room air. The frequency of respiration was controlled by a multivibrator circuit, which contained provisions for not only altering the rate of respiration but also changing the ratio of inspiration and expiration. Using room air during anesthesia with Pentothal and the new respirator, the saturation of blood with oxygen was 100%, and the carbon dioxide levels had fallen to extremely low values producing apnea after extubation of at least two minutes. Accordingly then, this device was used in all subsequent perfusions in the animals and also in the second total bypass in a child at Gibbon’s request. At this point, the survival rate of perfused animals rose precipitously.

In the latter part of 1950, I demonstrated to both Dr. Gibbon and to Dr. Allbritton the respirator which I had conceived and constructed. They were both very impressed by the performance of the respirator and the gasometric studies which I had performed. Very shortly after the initial demonstration, Dr. Gibbon asked me to demonstrate the device to his brother, Sam Gibbon, who was the owner of the Air Shields Corporation, in Warminster, Pennsylvania. His company manufactured incubators for premature infants and a number of other devices for the medical profession. Sam Gibbon came to the laboratory to see the demonstration and then invited me to visit his factory. Within a few days, I did visit him at his request at his factory one Sunday. He
showed me the facilities that were available there. He seemed to be extremely interested in continuing his interest in the respirator and suggested a future meeting. Following this, I did not have any further contact or communication with Sam Gibbon. There was likewise no further discussion in the laboratory concerning the respirator. At this point in time, I did not report the respirator in the literature. I did state, however, very clearly, in a paper published in 1952, entitled "The Repair of Intra-Atrial Septal Defects" that all animals were anesthetized with a positive negative pressure respirator.

The entrance to Allbritton’s office was directly opposite the entrance to the experimental laboratory where the respirator was in constant use with perfusion experiments. Within a few months, I became aware of the fact that George Haupt, a resident at that time assigned to Allbritton, were in the process of further investigating expiratory assistance with a mechanical version of the respirator using negative pressure. At this point, the door to Allbritton’s office, which was directly opposite, was always kept closed. As I look back, the reason being, of course, to secret from my view what they were doing. Following my departure from the Department of Surgery as research associate in 1954, I did publish an article describing the respirator using expiratory assistance purely as a matter of record, but this, of course, was many years after its original demonstration. I was no longer privy to what Allbritton and Haupt were doing.

I must refer now to the book entitled “John Gibbon and His Heart/Lung Machine”, by Ada Romain Davis, published in 1991, and the statement made by her at the very outset, namely, “Frank Allbritton, Jr., M.D. and George J. Haupt, M.D. were consultants extraordinaire who
helped make this book a reality.” Importantly, at this point in time (1992), George Haupt was very ill and was confined to a wheel chair at his home. Although he may have provided some information to the author, the bulk of the information concerning the book was provided by Frank Allbritton. On Page 112 of the book entitled “Gibbon and His Heart/Lung Machine,” under the title of the Early 1950’s, the information referable to the respirator which I had originally conceived and demonstrated is taken directly from my article. At this point, let me state the pertinent feature of that article. “The mortality rate of animals was unduly high despite the fact their condition during perfusion appeared satisfactory. After examining possible factors, the critical problem related to the method of anesthesia which neither provided sufficient oxygenation nor prevented acidosis. This was further substantiated by determination of blood gases in the subject during anesthesia, prior to perfusion, and also following perfusion. The problem was inadequate washing out of carbon dioxide and inadequate saturation of blood with oxygen. The concept of assisting expiration by introducing negative pressure was considered a possible solution. Therefore, a laboratory model of a ventilator device containing a timing circuit resulted which alternately operated solenoid valves both on the inspiratory and expiratory lines of the device was conceived by Haupt, then a resident – untrue. Expiration was assisted by using the Venturi jet to provide suction. Tests using this device showed that oxygenation was complete. It was possible to super saturate blood with oxygen using room air; a large amount of carbon dioxide could then be removed from the circulating blood to the point where the animal would remain apneic for a number of minutes. The ventilator was incorporated into the procedure and used in all subsequent animal experiments.”
In 1954, Allbritton, senior author, and Haupt, then a resident, read a paper at the American Surgical Association entitled "The Changes in Pulmonary Ventilation Achieved by Aiding the Deflation Phase of Respiration During Anesthesia for Surgical Operations." Incredibly, in this paper, there is no mention of the fact that they, including Gibbon, were aware of this development at least three years prior to their so-called original research. On Page 71, second column of Allbritton's paper, one is left with the thought that expiratory assistance during anesthesia originated completely at this time. Unbelievably, Gibbon was the editor of the Annals of Surgery and highly respected at that time, and I am amazed that he approved the publication of such a deception. In fact, in the discussion of Allbritton's paper, Gibbon repeats the same description of the anatomic findings as I had originally made. Since I had left the laboratory about this time, I was not aware that Sam Gibbon was in the process of manufacturing and distributing the device which was also patented by Haupt and cannot conceive of such scientific dishonesty.

In Ada Davis' book, Page 169, she indicates clearly that the two Gibbon brothers were frequently associated in various aspects of business as it related to the oxygenator and other kinds of surgical equipment and technology. At this point, Sam Gibbon's Air Shields Corporation became the sole manufacturer of the Haupt Jefferson ventilator development, and, obviously, they had many interests in common. Jefferson ventilators were distributed in large numbers both here and abroad. I believe that Gibbon and Allbritton benefitted financially immeasurably. George Haupt assigned the royalties to Jefferson and, following the completion of his residency, he was appointed Chief of Cardiac Surgery at the Lankenau Hospital, an affiliated hospital of the Jefferson network.
The importance of expiratory assistance as stated by Dr. Jay Jacoby, Chairman of the Department of Anesthesia of Thomas Jefferson University, is found in "Tradition and Heritage, Thomas Jefferson University." Let me quote at this point, on Page 631, "A second significant contribution of Jefferson surgeons through anesthesiology consisted in the development of the Jefferson ventilator, the patent obtained by George Haupt, was signed by Andrew Jefferson. He was aided in his work by Frank Allbritton and Jose Amadao, who co-investigated expiratory assistance as the means of improving ventilation and avoiding respiratory acidosis during anesthesia. Dr. Bernard J. Miller, Jefferson '43, had previously conducted the research on the important role of expiratory assistance during anesthesia. It was the same Dr. Miller who was also closely associated with Dr. Gibbon in the design of electronic and other components for the heart/lung machine." Further on, he states clearly that "The Jefferson ventilator was the prototype of the thousands of volume ventilators used throughout the world in operating rooms and intensive care units. Untold numbers of patients owe their lives to this development."

Following the appearance of the book and the availability of the contained information to me for the first time, I visited Dr. Haupt at his home who, at that point, was wheel chair bound and discussed the matter of scientific honesty and his role in the statement contained in the book that involved him as the one who first conceived of and developed expiratory assistance. Haupt informed me that the information which was provided to the author, Davis, was given completely by Frank Allbritton, and he was not sure what his motive was. I have enclosed a copy of Haupt's statement which says as follows: "The statement by Ada Romain Davis in her book entitled "John Gibbon and His Heart/Lung Machine" describes the use of negative pressure in assisting
expiration during anesthesia and the description of the laboratory ventilator attributes the
development to me. I was not associated in any way with this work. Dr. Bernard J. Miller
deserves all the credit for conceiving of the use of negative pressure to assist expiration during
anesthesia and the design and the construction of the laboratory ventilator. He made the first
laboratory tests in experimental animals with the method, and he established the usefulness of
negative pressure for this purpose." As an addendum in Haupt’s statement, he states the name
B.J. Miller should be substituted for Haupt, then a resident, because I was not associated with the
experiments using ventilation in conjunction with the heart/lung machine." I am including
Haupt’s signed statement. When the University of Pennsylvania Press became aware of this
deception by Frank Allbritton, John Gibbon, and Haupt, they immediately suspended further
publication of the book and actually verbally remarked that they regretted they had ever
published the book. Within a few hours after the University Editorial Board became aware of
the problem, I was contacted by Davis, who I am sure was fearful of some problem. I did not
have any interaction with Allbritton at this time. The University of Pennsylvania then published
an addendum, a copy of which I have included for your information, which was sent to a number
of surgical journals both in the United States and abroad.

Since the ultimate goal of this work was to achieve a bloodless cardiac chamber during total
bypass providing a means for the performance of precise surgical operations in human patients
under direct vision, the next logical step was to create defects within the septa and then to
attempt repair under direct vision while the cardiorespiratory function was maintained by the
heart/lung machine. As we had hoped, the new machine functioned flawlessly during total
bypass experiments with most animals surviving. The blood volumes of both the subject and the
machine were precisely maintained; the pH was maintained within normal range, and the servocontrol systems protected against air embolization completely and maintained constant hold up in the machine. Therefore, the next logical step was to create defects within the septa under direct vision during total bypass and then attempt to repair them while the cardiorespiratory function was maintained by the heart/lung machine. The magnitude of cardiac venous blood entering the right atrium was not fully anticipated before the first atriotomy during bypass. The first experiment was a failure because the large volume of blood returning to the right atrium by way of the sinus and the anterior cardiac veins could not be coped without loss by simple means.

An additional and unforeseen complication appeared when experimental atrial defects were produced during bypass. Air entered the left atrium as soon as the interatrial septal defect was produced. Blood and air trapped beneath the mitral leaflets were then pumped by the pulsating left ventricle into the systemic circulation, thus embolizing the coronary circulation and other systemic arteries. This was indeed a profound complication, but the solution was actually quite simple. A tube was introduced into the left ventricle of the beating heart by ventriculotomy at the apex in the out-flow tract and secured with a purse string suture provided a low impedance pathway for the escape of the air from the contracting left ventricle with the assistance of mild suction. At a discussion in the laboratory of the problem, Allbritton, during this, the only such meeting which he had ever attended, suggested decompression of the left side by way of a catheter in the left appendage. Both the returning cardiac venous blood from the open atrium and the blood aspirated from the left intraventricular catheter were directed into a collecting chamber. Because a certain amount of air was always mixed with the blood returning from the atrium, the bubbles were dissipated during their gradual descent on the inner surface of the tall
cylinder. The negative pressure within the collecting chamber also assisted in dispersing bubbles. As the blood accumulated in a pool at the bottom of the collecting chamber, the elevation of the blood level was then sensed by the same variable capacitor used in the control of the arterial pump. This circuit energized an additional pump that returned both the cardiac venous and the left ventricular blood and bubbles to the extracorporeal circuit.

A number of atrial septal defects were produced in dogs during total bypass and repaired by suturing a pericardial patch to the edge of the defect. In some situations, the patch was introduced into the right atrium through a stab wound onto the medial surface of the atrium and left connected to its base with the aim of providing circulatory support to the graft. This procedure was found to be unnecessary. In another group of animals, intraventricular septal defects were produced under direct vision by the use of a sharp cork borer and suction. These defects were also repaired by direct suture of a pericardial patch. Ninety percent of these animals survived.

Because the venous pumps were situated at a slightly higher level than the heart, a moderate degree of suction by the venous pumps was needed to ensure a maximal flow from the venae cavae. To further minimize the problem of pulsatile blood flow from the DeBakey pumps, further modification of this circuit was necessary. Using mild negative pressure, caval blood was directed into a separate collecting chamber placed at a level below the heart. As the blood accumulated in the bottom of the chamber, the level was sensed by the same variable capacity electronic circuit used to automatically control the arterial pump. This system worked very well, and the pumping pulsation were effectively minimized. Four recording potentiometers were
included in the front panel of the machine as a means of providing permanent records of physiologic data. The pH recorder indicated the pH of arterialized blood and, in addition, maintained a pH of 7.2 by automatically adding carbon dioxide to the oxygenator when the pH rose above the normal value. Another recorder indicated the degree of oxygen saturation of the arterialized blood from a cuvette located in the arterial line measuring light transmission at both 540 and 620 A. Another recorder indicated subject temperature and also controlled the temperature of the blood in the machine. There was excessive hemolysis of the blood in the machine because the heat required to elevate the temperature of the blood required a very high temperature gradient of the small sized blood heater elements. Consequently, the use of this feature was discontinued. The remaining recorder measured flow rate by means of a square wave flow meter but never functioned satisfactorily.

Because cyclopropane and ether were anesthetics frequently used at that time, the machine was pressurized with nitrogen because of the possibility that sparks originating in the D.C. motor of the artery pump made explosion a real possibility. There was also, in addition, a battery operated emergency electrical supply in the event of failure of the hospital's electrical supply.

Under these conditions, the automatic controls would no longer function because of the energy requirement and the pumps would need to be controlled manually. Alternating 110 volts was made available by a bank of batteries operating a motor generator converter.

Under normal operating conditions, all that was needed to initiate bypass and perfusion was the removal of the hemostats obstructing the venous and the arterial lines from the patient and to the
machine. As blood from the subject entered the extracorporeal circuit, the heart/lung machine then functioned completely automatically requiring only occasional adjustments of the negative pressure to the venous collecting chamber or small additions of blood as needed to maintain blood pressure. A large and successful experimental experience had been achieved by this time. The apparatus functioned splendidly. Oxygenation with a new oxygenator of greater capacity than the original one appeared to be sufficient for perfusion of a human patient of average size. Dr. Gibbon and this author, together with the laboratory group, had every confidence that the next phase, the use of the apparatus in operations upon humans, would also be successful. Accordingly, Dr. Gibbon initiated the use of this device in human applications.

In preparation for the first operation on a human patient, Gibbon practiced the procedure on one dog. The heart/lung machine was sterilized prior to use by first filling the machine with zephiran solution the evening before the contemplated operation, followed by repeated flushes with saline prior to filling of the machine with three units of blood. The first patients were two small children who failed to survive for reasons other than failure of perfusion. Interestingly, Dr. Gibbon suggested the use of the laboratory positive/negative pressure respirator in the second case because the anesthesia in the first child had been completely unsatisfactory. In May of 1953, Dr. Gibbon successfully performed an open cardiotomy during bypass under direct vision at Jefferson and repaired an interatrial septal defect in a teenage girl. Unfortunately, because of the prolonged duration of the bilateral anterior thoracotomy and a marginal dose of heparin to the patient, blood clotting on the oxygenator screens took place during the closure of the defect. Automatic controls in the heart/lung machine sensed the problem instantaneously stopping all pumps. It was then necessary to make an emergency reconnection of the blood circuit and, while
I operated the pumps under manual control, maintain the patient’s blood volume with fluids and what blood was available at the time. At this point, ventricular fibrillation occurred, but a normal rhythm was established immediately with an electric shock. This patient had an uncomplicated postoperative course, recovered completely, and has remained well with normal cardiac function for many years postoperatively.

Unfortunately, at this point in time, an incident occurred that altered the direction and course of my career. My work had come to the attention of the Junior Chamber of Commerce, a national organization, who elected to present me with an award as 1 of the 10 Outstanding Young Men under the age of 35 of the Year. I had informed Dr. Gibbon first to obtain his permission to accept the award, which he gave. I had requested from the Junior Chamber of Commerce that any press release be first submitted to me and be approved prior to publication. I was aware of the very tender nature of Dr. Gibbon’s feelings and of my own and was concerned only that the statement be honest. However, in the citation that was to accompany the award, and was not shown to me for approval, described me as the co-inventor of the heart/lung machine. Nothing could be more traumatic. This information released to the press did not reach Dr. Gibbon or me until the day or so before the presentation was to be made. I had, when I first became aware of the award, refused to accept the award. I made this known to Dr. Gibbon. After Dr. Gibbon reconsidered the entire matter, he then directed me in a three way phone call to receive the award, which I refused to do. I completely declined the award. Because of unpleasant feelings that evolved at this time, within a few months, I found associations with Dr. Gibbons to have deteriorated to the point at which I resigned.
Within a few days, the word of my resignation as Gibbons’ research associate reached Dr. George Bennett, the Dean and also the Chairman of the Department of Anatomy at Thomas Jefferson University. He had been aware of my performance as a student from my freshman days. He invited me to become a member of his staff in the Department of Anatomy at Jefferson, provided for a new laboratory, which he had constructed for me, complete with basic apparatus, with a stipend and also the use of his personal technician. Because of the many scientific questions that are always with me, there were a number of problems that I began to investigate. Funds were provided from the National Institute of Health, Tobacco Commission, and private sources for me for the development of an extracorporeal device to be use as a means of perfusing extremities containing tumors with chemotherapeutic agents, serum proteins, etc. in cancer patients, and a number of other topics for many many years. I continued research in many areas and published many papers from this laboratory.

Frank Allbritton’s motives were obvious and his conduct far from the standard of scientific honesty. As a resident, I always considered him a very demanding rigid individual whose vocabulary lacked completely the word “Thank you.” These feelings were shared by the surgical residents.

In conclusion, as I look back, I thoroughly enjoyed my early association with Gibbon when I was his resident and research associate. At one point, when all the problems with the heart/lung machine were solved and septal defects were produced and repaired during bypass with survival, Dr. Gibbon called me into his office one day and said, “BJ, you’ve done all of this work and you should be co-investigator on the grant.” I had never expected such a response and was
astounded. The following day, he called me back to his office and said, “BJ, I thought it over
and I’m going to wait a while.” At that point, I could see the influence of Allbritton and Maley
Gibbon lurking in the background. At Gibbon’s request, I included Maley as one of the authors
and Allbritton as the other in a number of papers despite the fact that neither had made any
contribution in the four years during which time I directed the work. Finally, as time passed,
Gibbon excluded me from the clinical aspects of the work. A number of years later, Gibbon sent
me a letter indicating that he was removing my hospital and academic privileges. I was told by a
number of the staff that the Executive Committee unanimously rejected his effort to do so. I
continued to admit patients to Jefferson and operate upon them and also continued teaching my
special courses in anatomy, which I had been doing for 45 years. In addition, the Alumni
Association at Jefferson recently presented me with the Alumni Achievement Award and also
the Samuel D. Gross Award from the Department of Surgery. Unfortunately, my introduction to
Gibbon was unpleasant and my departure from his service even more unpleasant.