Dr. Albert Lyons, President.

Lyons: I bring you all greetings. Directly after this session this morning, we will all adjourn immediately and directly to the cafeteria, where we will be guests of the Hospital. I also would like to announce that at the AMA convention in June, June 23rd, there will be a reception in the Crystal Room of the Palmer House, and we certainly hope that you and all your friends will come to this reception, if you happen to be in Chicago.

We have an exceptional opportunity this morning to be present to receive the opinions and illuminations of pioneer contributors to medicine. In no other forum is this information available, not in the past, not now and not in the future, and so in a sense, this little body here is participating in a historic event. Please realize that numbers of others could have been invited here for the same reasons, but the pragmatic demands of time will have to limit the length of any session.

Some of the speakers may have, or may have not, received the rewards and recognition in these halls that they have deserved. But I think that I can safely predict that posterity will recognize and remember them, and some of us will do our best to see that that is so.

We are indebted to Dr. Alvin Gordon who will chair this meeting for his efforts in bringing these men before us. I wish you a fine day and evening, and I bid you welcome.

Gordon: Thank you, Al.

Medicine has made such fantastic advances and has become so complex in the past few decades that we sometimes lose sight of the fact that one, two or three individuals, working on a shoestring, and between office visits or house calls, have been able in the past to make basic contributions to medical science. This is the fiber of which Mount Sinai is made, and here are some of the men who accomplished this. They are part of the legacy left by such great people as Janeway, Jacobi, Brill, Lilienthal, Libman, Schick, Eli Moschcowitz, Klemperer and Otani.

The committee has chosen only a few of the many physicians who could have qualified for this program. One additional outstanding alumnus was invited but was unable to attend, Jonas Salk. Most of our speakers interned at Mount Sinai in the early Twenties and went out on their own just before the Great Depression. They supported themselves and their research by practice, perhaps aided by contributions from private sources. The government and the great medical foundations had not yet entered the lists in support of training and research. The golden era of medicine was just ahead, and there was much to be done.
The idea of today's program is to present personal reminiscences, not scientific observations; its mission is entertainment, not education. Our first speaker will be Dr. Moses Swick. He was an intern and house surgeon at Mount Sinai, vintage 1925-28. Mo then went to Germany on a Libman Fellowship, returning to New York in 1929. He rose through the ranks on the urology service. Then he joined the Army and was in charge of urology for the 3rd General Hospital, which was the Mount Sinai unit during the second World War. Later he was Attending and then Consulting Urologist to the Hospital and Emeritus Clinical Professor. His pioneer work in the development of intravenous urography has won increasing recognition, particularly in recent years. In 1933, he received the Billings Gold Medal for his AMA exhibit on this subject. He was awarded the Jacobi Medal by the Mount Sinai Hospital Alumni, the Valentine Lectureship and Award, and a commemorative medal as a distinguished alumnus on the 200th anniversary of the founding of Columbia's College of Physicians and Surgeons. Dr. Swick has lectured at many of the foremost hospitals and medical societies in the country. Like the rest of his brothers on this panel, Mo continues to serve the community in the private practice of urology. As handy with the scalpel as with the cystoscope, Mo is sometimes known in the cystoscopy room as "The Tiger in Your Tank." [laughter]

Mo will speak on the development of intravenous urography. Dr. Swick. [applause]

I trust I can do justice to the subject in the very limited time allotted me. I'll do my best, however, and I'll have to eliminate some of what I consider important aspects.

Now, we speak of the urographic media as they exist today. In actuality, one should apply them not only to urology but the entire field of medicine. There isn't a facet of medicine that isn't tapped for diagnostic acumen with the presently existing organically bound iodide compounds, which were first developed, as you will see, on the basis of urologic research.

Urography in its clinical application is intimately associated with the development and investigation of various radiopaque media. For historical purposes, one may consider two separate periods in the development of these media, namely, that of the inorganic iodide compounds administered cystoscopically, and the more recent one, that of the organically bound iodide compounds administered intravenously, the latter being responsible for the development of excretion urography and other diagnostic fields in medicine.

On this occasion, I'll confine myself to the present era. The organically-bound iodides as urographic media are responsible for the successful development of excretion urography and historical background is therefore not amiss. In 1923, Rowntree and his co-workers of the Mayo Clinic, treating a luetic with sodium iodide intravenously, noted on taking a film that the bladder was visualized. The pelvis and the ureters were just faintly indicated. And thus he published a paper on excretion urography by means of sodium iodide.

Others followed, for example, Roseno with a compound called palegos, which is a double compound, urea sodium iodide, but all these attempts were not satisfactory,
either as a result of large volume of fluids required for injection, toxicity, and poor visualization.

In 1929, I presented a paper on my researches in successful development of Uroselectan, in this country known as Iopax, for intravenous urography. It may be of interest to recount the thoughts, the steps and the trials that led to the final satisfactory result.

In 1928, under a Libman Fellowship -- I should mention that Dr. Libman was one of the outstanding clinicians in this country, was chief of medicine in this hospital, was rather a stimulating force for younger men, and directed them into various fields of research in the practice of medicine -- I went to work at the Altona Krankenhaus in Hamburg, Germany, where Professor Lichtwitz was using a compound called Selectan-Neutral purely for the treatment of coccus infection in man. This compound, iodomethanate sodium which I will later illustrate, one of the group synthesized by the chemist Professor Binz, had previously been used for coccal infections in cows. Since this compound contained iodine, 54 percent, an element known for its great roentgenological properties, it occurred to me that it might be of value in visualizing the urinary tract. I conducted simultaneous chemical efficiency determinations, toxicity studies, and roentgenologic studies. These studies revealed the compound was promptly excreted in the urine, and on roentgenologic examination in the rabbit and human, revealed encouraging results, that the kidney and the urinary bladder could be visualized not infrequently after the intravenous administration of the drug. The renal pelvis and ureter, on the other hand, were for the most part poorly seen. In one case, that of high occluding ureteral calculus, satisfactory visualization of the urinary tract was also demonstrated.

I won't go into that, because of limited time I won't go into my experimental work as far as excretion studies and toxicity studies except to say that based on toxicity studies in the rabbit, one could theoretically administer 12 grams of this stuff intravenously in the human, considerably less than the amount used. There were marked individual differences in tolerance. The disturbing factors were diplopia, vomiting, nausea and headache. All administrations produced similar results. In no case, however, were the symptoms of such a nature as to deter me from continuing with this work.

Simultaneous excretion studies, limited to quantitative determination of the iodine component, revealed that 80 percent of the injected iodine was recovered in the urine. The obtained results, though not sufficient for practical purposes, pointed to the potential of the methodology and warranted continued investigation.

The problem then resolved itself into modifying Selectan-Neutral in order to accomplish the following results: 1) to diminish the toxicity through the substitution of a methyl radical, which I will point out to you, in order to decrease toxicity, and thus
permit the administration of a larger dose, thereby attaining a higher concentration of excreted iodine component, a determining factor for roentgenologic success. The observation of double vision led me to the suspicion, at least empirically, that the methyl radical of Selectan-Neutral might be responsible for toxic manifestations. The latter thought was the stimulus for continued investigation that led to the ultimate successful compound. 2) To increase the solubility by means of a substituted radical so that a larger dose could be administered. 3) To increase the iodine content.

After carrying out these investigations for about seven months in Hamburg, I presented the ideas and results on Selectan to Professor Binz and sought his aid. He subsequently furnished me with two separate di-iodo-pyridon compounds with a methyl radical substituted. I returned to Hamburg. Both of these compounds, because of their poor solubility, were administered by mouth, not intravenously, to dogs. Neither one was absorbed and both produced X-ray shadows of the intestinal tract.

My investigation then transferred to the large urological service of Professor von Lichtenberg in the St. Hedwig’s Krankenhaus in Berlin. This afforded me close contact with Professor Binz. Meanwhile I continued to work with the Selectan-Neutral at the St. Hedwig’s until the development of a more suitable compound came into being.

In order to obtain greater solubility and eliminate toxicity, Professor Binz prepared the modification of Selectan-Neutral in which the methyl radical was replaced by the sodium acetate radical. Solubility was also increased by lowering the iodine content from a di-iodo-pyridon to the original mono-iodo-pyridon. This new compound, named Uroselectan, fulfilled the required specifications.

Now, can I have the slides, please? This is the original publication of a lecture that I presented before the 9th Congress of the German Urological Society in Munich in 1929. That is the title of the paper. I won’t go into the German. Next, please.

Now, here’s the original compound used against coccus infections. It’s a 5-carbon-nitrogen ring, with the oxon making the pyridon 5-ioda, 2-pyridon and an end methyl.

When I discussed the problem with Professor Binz, he supplied me with a double ioda compound in order to increase the iodine content, but instead of the methyl radical, the substitution by acetic acid radical. This was insoluble. I went back. Then he furnished me with a sodium salt, in the attempt to increase solubility. That, too, just raised solubility about 8 percent, still with a double ioda. And the final step was decreasing the iodine molecule to one of the original Selectan-Neutral, but with the methyl radical substituted by sodium acetate, and so we have a 5-ioda, 2-pyridon, and acetic acid acility, which was the first successful intravenous urographic medium, Uroselectan. Next, please.
Swick: And this [slide] is the compound, of course, with its molecular weight. I won't want to get too involved, time is of the essence. Next, please.

Excretion studies revealed the following, that in the first two hours -- now, this is a curve for Uroselectan, revealed that the precipitated substance, it could be precipitated in the urine, about 75 percent was excreted in the first two hours. This period I called the thrust-excretion ability of the normal function of the kidney. It is this period, upon the thrust-excretion ability of the normal concentrating kidney, that successful intravenous urography depends, and you will note, there you have the thrust-excretion period, then the drop to about 33 percent in the next two hours, and finally in about eight hours most of the substance is eliminated by the urine.

In distinction to that, if you study a kidney that is diseased and has lost its concentrating power, you'll note it never reaches thrust-excretion ability, but you have a curve indicating a level type of excretion over a prolonged period of time, and never an iodine content in the urinary tract to permit good visualization.

Next, please. This is a curve, it shows a similarity, done with Hippuran, to which I will later refer, a new compound that I had developed. Next, please.

These are the original reproductions of the original article presented in Germany before the German Urological Society, an instructive uropathy at the UP junction, and you notice the intense visualization with the Uroselectan. Next, please.

This is a case of perinephritis bilateral, also one of that group. Next, please.

These are all of Uroselectan. And here, too, there's a stone in this pelvis, a stone in the lower calix, double sided neo?, that is, bilateral renal calculi. Next, please.

Partial kidney with a calculus here, and a calculus in the lower calix here. Next, please.

These are all with Uroselectan. A nonfunctioning kidney with a staghorn stone, a stone in the lower calix, the pelvis and calix visualized, not here. Next, please.

Now, when I returned to the States in December, 1929, two sets of compounds came into existence. Based on this pyridon nucleus, you notice. This is a 5- carbon-nitrogen-pyridon ring, and here you have the Uroselectan B, or Neoiopax in this country, a decarboxylate disodium salt with a methyl radical, incidentally. This was purely an empirical observation of mine, which was a stimulus for further work and perseverance in the field.

And here you have also diagrams, also a double iodo-pyridon and acetic acid, though, di ethyl ? compound. Lights, please.
Swick: I'm not going into the characteristics of Uroselectan, its solubility, toxicity studies except to say it was very well tolerated, three grams per kilogram of bodyweight; in other words, 180 grams for a 60 kilogram individual. We were far below that level, 40 grams we administered in a 100 ccs.

I'm not going into the study of function or the study of excretion as it pertains to the field. If I have the time I will do that later. Or various principles that I developed in accordance with observations in this particular field.

Now, to continue on -- do I have enough time? I think I'll be able to do it. In 1939, in 1931, excuse me, further investigations of mine led to the formulation of another compound, Hippuran, which I presented and published in 1933. It was based on a metabolic principle differing from the above pyridon substances in that the iodine is organically bound. This is important, organically bound, to the 6-carbon-benzoic acid nucleus instead of the 5-carbon-nitrogen ring. Hippuran, a mono-iodo-benzoic acid derivative, interestingly and importantly bears similarity to our present urographic media, which I will point out subsequently, namely, what we use today, originally used Urocon, Hypaque, Miokon, Renografin and Conray, in that the latter are tri-iodo-benzoic acid compounds, Hippuran being, as mentioned, a mono-iodo-benzoic acid compound derivative. A detailed and illuminating treatment of the subject was carried out with James Hopper who was pharmacologist for Winthrop.

I won't go into the characteristics of Hippuran. It's a 38.8 percent iodine compound, administered in 50 percent solution. Very well tolerated. And the derivative is hippuric acid. Levic originally recovered this substance from a horse, hippos, and he called it hippuric acid. It's a detoxification product, the human puts out almost at 1 percent a day. It's the main metabolic product in a horse. In other words, for example, preservatives with benzoic acid are detoxified in the body with the simplest amino acid, amino acetic acid, glycine, and is thus excreted as hippuric acid. And this has been a well known observation for many years. There was a lot of experimental work done in order to determine where this process of detoxification takes place. Does it take place in the liver? Does it take place in the kidney?

Now, the sodium (?). It has long been known that the administration of benzoic acid or iodo-benzoic acid -- in experiments with iodo-benzoic acid -- the detoxification with the amino acid glycine results in the excretion of a sodium salt of hippuric acid, or iodo-hippuric acid, Hippuran. Hippuran satisfies the necessary requirements for excretion urography by both intravenous and oral routes.

Now, very importantly, as a result of this Hippuran, many fields have lent themselves to investigation in medicine. Many facets have been opened up for
concrete definite urologic and diagnostic resolution of problems, whether it be in the brain, the heart, the aorta, the liver, arteries -- any artery today is capable of being tapped for roentgenological visualization, localization and proper surgical approach.

Swick: Hed-iodine hippuran for example has also been found of value as a test of renal function, and a screening diagnostic method by means of the renogram.

In closing, I wish to point out that further important developments from the introduction of a tri-iodo-benzoic acid compounds, Hypaque, Renografin, Conray, have had wide applications throughout the field of medicine. For example, they were used for cerebral, cardiovascular, selective renal angiography, and other local arterial angiographic examination, have made possible proper localization of lesions for exact surgical approach in their respective fields.

These compounds, you will note, are derivative of the original Hippuran, and related to it, in that the nucleus of all of them is the benzoic acid ring. These radiographic organic iodides have made excretion urography the cornerstone of urologic diagnostic investigation, lending themselves to the studies of the dynamics of the urinary tract by the means of cine-roentgenography, and permitting important information to be obtained from the voiding cystourethrogram and from nephrotomography. May I have the slides, please?

Here you have the 6-carbon, 2-iodo-benzoic acid compounds. Administered in the body, it is detoxified with glycine which is an amino acetic acid, the simplest amino acid in the body, and you have as a result 2-iodo hippuric acid. It's a benzo-amino acetic acid. Next, please.

Now, here you have Hippuran, which is a 2-iodo, mono-iodo, hippuric acid sodium salt. You will observe now the subsequent compounds that have been developed and are presently used in excretion urography and in other fields for medical diagnosis. This compound Hypaque, for example, is a tri-iodo, here you have the benzoic acid radical, sodium salt, of diazamedo. It's a tri-iodo-benzoic acid compound. Renografin, for example, also is a tri-iodo-benzoic acid derivative of a metaglucamine salt. Next please. Miokon, and I originally turned over Hippuran to the Mallinckrodt people and subsequent developments, before I left for the Army, were di-iodo hippuric acids, which was a benzoic acid derivative, and here you have a tri-iodo-benzoic acid derivative of a di-propiamedal salt. And this is also used for angiography. Is that all? Next please.

And similarly with Conray, also tri-iodo-benzoic acid derivative. Next, please. Well, I won't go into the question of temporary functional inhibition and other aspects of excretion urography.

I think my time is up. But in closing, I hope that this, the end is not yet in sight and further developments will occur.
Gordon: Arthur M. Master, a famous name in cardiology, also interned at Mount Sinai, 1921-23, some years earlier than Dr. Swick. His postgraduate work with Sir Thomas Lewis was performed on a Cornell Medical College travelling fellowship. He later worked at Cornell, and for many years he headed the electrocardiographic department of Mount Sinai at a time when that was the only scientific discipline in clinical cardiology. Dr. Masters served in the Navy in World Wars I and II. In the latter, his tour included a period as chief of medicine of mobile hospital No. 10 based in the Solomon Islands. Later he was cardiologist in Honolulu and at the National Naval Medical Center at Bethesda.

In the midst of a busy consulting practice, which is active today, he found time to be president of the New York County Medical Society, vice president of the New York Academy of Medicine, and is currently president-elect of the American College of Chest Physicians.

The author or co-author of over 400 medical articles and five medical books, Arthur Master will be known as the P.T. Barnum of Medicine, a man who sold two steps to hundreds of cardiologists with offices on the ground floor. [applause & laughter] Dr. Arthur Master’s topic is "Early Experiences in Exercise Electrocardiology." Dr. Masters. [applause]

Masters: Dr. Lyons, Dr. Gordon. When I was a fourth year medical student at Cornell, I became interested in fatty degeneration of the heart. At that time, that was a very common diagnosis, more common than coronary disease, and I found that we were wrong to base the diagnosis on the presence of globules of fat, as evidenced by oxalic (?) acid or (?) stains. Of course, we took the hearts of perfectly healthy people killed in accidents and we found these globules. Fatty degeneration of the heart was given up.

B.S. Oppenheimer, who was one of the chiefs of medicine [at Mount Sinai], had heard of this and heard of my interest in electrocardiography and cardiology and in my fourth year, 1920, invited me up here to volunteer to do two afternoons in the Electrocardiographic Department. And I would come up Tuesday and Thursday afternoons in my fourth year.

At that time, the ECG department was in back of Ward C, and to take an electrocardiogram on the ward, you trundled a huge heavy cart, hundreds of feet of wire so that you could connect it into a socket in the wall. You carried jars of saline, bandages, synch electrodes, because you bandaged the arms and legs. It was quite a contraption. And the machine itself in the electrocardiographic room -- the optics were 20 feet long. Just think of those little portables we take now. We had an arc lamp to give us a source of light. It was always sputtering out. And we used plates, photographic plates to photograph.

We took the electrocardiogram in an Adirondack chair, and I’ll show you the picture later. An Adirondack chair is very comfortable and relaxes the patient and you don’t get muscular tremor, but the American Heart [Association] passed
a rule that everyone should take an ECG in a recumbent position so that they would match up.

Of course, we only used three leads.

At that time, Hubert Mann was part of the department, an M.D. and electrical engineer, and he worked out mathematically the monocardiogram, which is the vectorcardiogram. So that that really stems from this hospital, the vectorcardiogram used all over the world.

Masters: From Ward C we moved to the basement, and opposite was the dining room. And here I really come to something that’s extremely important, and it’s about Leon Ginzburg. The hell with this ileitis, this is something far more important, and I wonder if Lester Tuchman is here? Of course, in the dining room, Leon would take a -- it wasn’t deviltry -- he would take a knife and stab a pat of butter, and catapult it up on the ceiling. And that was because he knew cholesterol was very bad for coronary disease. So I think we should give him as much credit as you give Jonas Salk.

I was responsible for two innovations. At that time, when you took an electrocardiogram, you had to put the lights out, and I had Phillips, our engineer, get the tinsmith to make a tin cover just before the shadow of the electrocardiogram entered the ocular system. And also, instead of the girl who took the electrocardiograms on the wall running to the telephone and telephoning down, “You can start the electrocardiogram,” and then the girl in the basement telephoning back when she was finished, I had them put on a chest piece such as telephone operators use, so that one could talk to the other directly without calling up on the telephone.

Now, to come to exercise, I came on the two-step exercise for this reason. Everybody exercised the patient: dumbbells, hopping on the foot 20 times, 40 times, hands up and down, knee bending, dumbbells, and a ten pound dumbbell would be given to a kid of eight years and a boy of 18 or 28. Of course, that wasn’t standardized, and we looked around for standardization, and that’s how we evolved the two steps. We used all kinds of heights of steps and all times of performance to the test, and finally came up with the present two-step test.

We used, of course, at first the blood pressure and pulse response. We took a faculty of different colleges in New York, in age and weight and so on. We found out how many trips they could make and yet their blood pressure and pulse would return to normal. Incidentally, we found that if a Volunteer, in those days we had good looking Volunteers around the Hospital, took a blood pressure and pulse on a man, it would never return to normal. And so we’d have to have the doctor take a man and a nurse or volunteer take a woman patient.

And because the blood pressure wasn’t stable, I think is, the reason that people, investigators came to the electrocardiogram instead of the blood pressure. In this country [Francis] Wood and [Mary] Livesey in Philadelphia really first suggested
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taking an electrocardiogram after exercise, to distinguish chest pain, cardiac versus non-cardiac. But they said it was dangerous, watch out. And I'm afraid they haven't gotten full credence for their work because they frightened people.

Scherf in 1932 really did a lot of work on exercise and electrocardiography, but he didn't believe in standardization. He was always taking a whack at us because we feel very strongly about standardization. He would have the patient do whatever brought on pain. If sexual intercourse was the only thing that brought on pain, I wonder how he would do that in his office?

Masters: Well, [Louis] Katz, the father of our Arnold Katz, also came up with electrocardiography and exercise. Missal in 1938 suggested the two-step test, but he didn't standardize. He didn't have the patient walk according to age and weight and sex. We really were very early in the game. In 1930 or '31 we had a Sieman-Hausky(?) machine downstairs, and it was an ink-writing electrocardiogram, but the point was, it was NG, because if anyone turned the turbine on or some electricity, it would shoot off, and I've tried to find out from Sieman-Hausky (?) if they have any record of that, because we, I think, really tried out electrocardiography before anybody else.

I might say that we were a good deal responsible for the division of coronary disease into anginal syndromes, and subendocardial ischemia, so-called coronary insufficiency, and coronary occlusion. The basis is coronary disease, but we showed that the three really are separate entities, different causes, different electrocardiograms, different treatment.

At first we called any depression on the exercise electrocardiogram abnormal, and we would write: "You have to interpret it with the clinical findings." But along came Wood in 1951, Englishman, famous cardiologist, and he wrote on the so-called Eschemic Depression. Now we all use the Eschemic Depression as an abnormality in the two-step test.

We at that time, we used three leads and four leads, and then, of course, during the war twelve leads came on. I think the next advance in electrocardiography was monitoring the electrocardiogram, that is, actually recording it while the patient walked the steps. And we found, this is very valuable, to find the patient giddy or unsteady or faint-like, actual syncope or staggers, and you can't find the cause by examination. Very often in monitoring the electrocardiogram and the regular post-exercise electrocardiogram, you'll find that he has runs of premature beats that nobody suspected, or he has tachycardia or ventricular fibrillation.

We also, I think, are not innovators really, but we've advocated the two-step test to discover silent coronary disease. I think we found that it's very prevalent, that millions of people really have silent coronary disease.
Now, of course, everyone or many are advocating the strenuous test, and we don't think it's necessary. We think the two-step test increases the rate sufficiently.

I want to emphasize that we found a negative two-step test as valuable as a positive. In other words, it excludes coronary disease. The two-step test can be done in congenital heart disease and in rheumatic heart disease as I'll show you in a moment. Can I have the slides?

This is an early electrocardiogram taken by Einthoven. Next.

This is the first electrocardiogram taken after exercise. It's lead two, you see, upright "zwei rank" at rest, and then below is after walking up, running up a flight of stairs. That was published by Einthoven in 1908. Next.

Masters: Here's the galvanometer that we had downstairs. It's a huge thing and the ocular system you see here, and here, and the fourth string is somewhere in there, and the movement of the fourth string, the movements, the electromagnetism engendered by the current from the heart muscle interplaying with the electric, the magnetism between the north and south poles. This magnet produced the electrocardiogram. Next --

Here's the, I won't go into details, of course, of this, but here's your arc lamp, there's your galvanometer which isn't shown, this is all ocular apparatus. That's a time marker that was always stopping. And 30, 20 feet long, from this end of the room to that end of the room. Here we'd have a plate that would catch the electrocardiogram. Next --

Here you see the jars we had, clay jars full of saline, zinc electrodes, and leads 1, 2 and 3. Next --

Here's the Adirondack chair we used, and as I say it's extremely comfortable, and here's the two steps, each nine inches high, that we evolved. Next --

And here is the two-step, and this is Simon Dack, when we were still taking blood pressure and pulse. Next --

This shows why we standardized it. We found that the efficiency of the heart, blood pressure and pulse response was definitely related to age. For example, a man did the maximum, 3800 foot pounds of work around the age of 24, 22. After those ages, efficiency declined. Next --

And here you see, with weight. At a weight of 155 - 175, a man performed maximum work, and a woman around 135. When you got heavier, you were not as efficient. Next --

So you had to do, you had to standardize. The mathematicians after we gave them the material came up with this table, the one for the dark in parenthesis, for
women, and you see, as you get older you’re asked to do less and less trips, 48, 42, 40, and as you get heavier you’re asked to do less and less trips, 46 to 32. Next --

And here now is the monitoring. Yes, this was one of our technicians, and one of the reasons, of course, I went into electrocardiography, the technicians were good looking. And she became a stewardess. Here is the basis for our monitoring. Dean Mason took these two positions -- instead of the arms, the outer portions of the clavicle. You’d be amazed, you can prance and dance and you won’t interfere with the electrocardiogram, particularly when you use disposable electrodes. That was another worthwhile discovery. Next --

And this, now, for standardization, this is a man with heart disease. His resting electrocardiogram, in this left hand column, is normal. We exercised him, and you see these dramatic depressions. And yet this man, we’d given him Marsiled, which was a sympatholytic agent, and he had no complaints at all. He lost his angina completely. But still the two-step was positive. And so, by standardization, you could work on drugs. Paul Wood and I interchanged communications, he in London, because we did two-step tests exactly alike, and we found similar experience with Iproniazid. I thought it was a wonderful drug. It was too bad it was taken off the market. It was supposed to cause hepatic disease, and our own Hans Popper felt that it caused hepatic necrosis, but I still doubt it. Next --

Here’s another reason for standardization. This was given me by Bill Foley of New York Hospital. This table calls for 38 trips but people always innovate, and this doctor had his patient walk until he developed this bradycardia and collapsed and went into convulsions. Luckily he recovered. So that’s another reason for standardization. It makes for safety. Next --

Here, again standardization, here this man is monitored, and we show you tracings of the two or three trips, and you see only in the 42nd tracing -- it called for 42 trips, the table did -- did he first show eschemic depressions, another bit of evidence that you should standardize and ask the patient to do work according to his age, sex and weight. Next --

And here is a patient who had -- has a miraculous tachycardia, shown by the electrocardiogram -- next -- that you would not have suspected otherwise.

Now, a negative two-step test, as I said, is as valuable as a positive one. This is a man of 46 who’d been a "cardiac cripple" for 20 years. He’d sustained two coronary occlusions, and when we saw him in the OPD of Mount Sinai Hospital his control was perfectly normal. But, of course, with that history, we hesitated to do the regular three minute step and we did the single control, immediately to find a state of no change. The next day he came and we did the regular. No change in the immediate or five minute tracing. We now knew that he had never had two attacks of coronary occlusion, yet he’d been put to bed and received
morphine. This is the diaphragm down here, this is the, above the diaphragm, he had a hiatus hernia. That's it, a hiatus hernia. Next --

Now, this is a patient with idiopathic pulmonary dilation, and a loud harsh murmur. But believe it or not, he wanted to go into the Korean War. That's the only young man I ever examined who wanted to go to war. Next -- but they wouldn't take him on account of that murmur, but when we did the two-step test, this is the 12 lead control, perfectly normal, and here's lead two, control, two minutes, six minutes, four, no change. The two-step was negative. The Army accepted him and he did a three year hitch in the Army, and he's perfectly well now. Next --

This is, they advocate walking through angina. This is a patient with angina pectoris who walked to my office. He developed pain but he thought he'd walk it off and he walked faster, and sure enough, when he came to the office he was in collapse, and we took an electrocardiogram, and there you see the typical changes of so-called coronary insufficiency, RS-T depression. It took two months before that electrocardiogram came back to normal. We did the work, a lot of it I think original, on coronary insufficiency. Next --

Masters: Here's a man with silent coronary disease. His resting electrocardiogram is normal, the FRO, (?) but is seen when you ask him to do the two-step test, see how abnormal. And here, too, I think the two-step will help stop silent coronary disease. Next --

This is a man that we found, very often we tell the patient who does the two-step test to stop the moment he has pain. That's why we've never had an accident in more than 100,000 tests. And the patient would -- he did this, and yet after he stopped, there's no change, immediately, in all these leads, and we asked him how come. He said when he stopped he knew that if he went another two or three steps, it would be abnormal, next that he would develop pain. Next --

And sure enough, the next day we had him come back and do more steps, and you see it was very abnormal, R-S-T depression, and that has a very potent clinical application, because we teach our patients to become introspective, old ladies, to watch for that little sense of discomfort before real pain comes on, and then they stop, and then go along. Next --

In conclusion then, I want to say a word about the two-step test. You know heart transplantation hasn't panned out. Even the coronary care unit is not as good as we thought. 40, 50 or 60 percent of patients with acute myocardial infarction sustain it before they ever go into a hospital. That's new. And even when you get to the coronary care unit, they still die of shock. They die of power failure. That is, if they've had two or three attacks before, nothing you can do, and I think perhaps Lester [?] and the others working on assisted circulation will solve the problem. We all advocate going after the coronary profile of patients who have high cholesterol, reduce it, and hypertension, reduce it, but we've concluded that every person over 35 should have a two-step test or an equivalent, to spot the fellow that's going to have coronary disease. Thank you. [applause]
Gordon: Is this microphone adequate? Can all you in the back hear? No? We'll try to use this one. We'll try to use both.

Sam Rosen, our next speaker, interned at Mount Sinai from 1921 to 23, together with Arthur Master, and was Resident on the ENT service from 1923 to 26. He served on that service at Mount Sinai and was Associate Attending for many years. When penicillin proved so successful in the treatment of ear infections and practically eliminated mastoidectomy, Sam started a revolution in ear surgery which continues to this day. As a result he has been honored as few mortals have during their lifetimes. He received the AMA Hektoin Medal, Gold Medal, for a scientific exhibit on stapes mobilization in 1956, also the first International Quadrennial Pietro Calachedi Gold Medal for original contribution to otology, in Bologna, Italy, 1957; gold medals from the Association of Otolaryngologists of India and Egypt; the Jacobi Medallion from Mount Sinai and the George Arent [?] Pioneer Medical Citation from his alma mater, Syracuse University, in 1964. He was made Knight Officer in the Order of Merit of the Italian Republic; Honorary Member of Finland's Otological Society; Foreign Member of the Academy of Medical Sciences, USSR; and was awarded the Pirgov Medal by the Academy of Medical Sciences of the Soviet Union. Truthfully, it can be said that Sam has been widely honored on both sides of the iron tympanic membrane. Sam -- [applause]

Gordon: Today Dr. Rosen will receive one additional award. Some time ago when I requested his curricula vitae from our speakers I promised a special prize for the one who sent his in first. So, Sam, have a ten cent cigar. [applause]

Rosen: Thanks, Alvin.

I might start off by saying that this entire approach to the stapes was really made possible only by the great contribution of [Julius] Lempert. I studied with Lempert. I learned his fenestration operation. And I did it, liked it, enjoyed it, and no amount of praise can go to such a pioneer who really was the first one to revolutionize the surgery of deafness.

However, with the Lempert operation, and everyone who did it, the patient was usually quite dizzy for sometimes two or three weeks or sometimes a good deal longer, and at the same time it was necessary to hospitalize the patient for two to three weeks.

I remember one day in '51, I was doing a Lempert operation, a fenestration, and I was trying a new modification, which ultimately did not work, but I had great faith in it, and so I invited the late Franz Altman from Columbia-Presbyterian to come and see this modification and see what criticism he had of it.

And when he came, I was about two-thirds finished with the operation, and when you're that far in the operation you've already disposed of the incus and you bypass the stapes and make a fenestra in the semicircular canal, but usually or almost always before we did that, we then were able to see and touch the head of
the stapes, to see if it was truly rigid, as it would have to be if it were a true otosclerosis, for which the patient was operated.

And in this particular instance, I found that when I touched the head of the stapes, that instead of it being cement-like and rigid and immovable, it was movable. And so I turned to Franz and I said, "Have you ever seen this?"

He said, "Yes, it's happened to me."

And at that moment I realized that if it happened to him and happened to me, it must be happening on and off everywhere, and this could account for a misdiagnosis and a fruitless operation. I decided then and there that I would try to make it possible, before operating on a patient for otosclerosis, however clear the diagnosis was, to make certain that that stapes was truly rigid. And I decided then to do this maybe in 100 consecutive cases to see how often the stapes was not rigid, was actually mobile.

Again I used Lempert's technique for elevating the skin and the drum and exposing the middle ear, and I did this in five consecutive cases, and found that when I touched the head of the stapes, it was cement-like in rigidity. These patients, of course, in a few weeks were ultimately operated on according to the Lempert technique.

Rosen: But along the sixth patient was one that was referred to me by one of the ear surgeons in Chicago. This man was going to be in New York for a while and I was asked to operate on him. I told him the story of this woman that I operated on, that at operation had a mobile instead of a rigid stapes. And I said, "We can do one of two things. I can either go right ahead and do the fenestration of Lempert, or I can take you to the hospital, lift up the ear drum, test the stapes for rigidity, and if it is not rigid, I can save you a fruitless operation. If it is rigid then in two months we'll do the fenestration. But you can have your choice."

This man was a young engineer and he thought about it for a minute and said, "I think I'd rather have you try it first."

It was this patient that I operated on, that is, lifted up the ear drum to test this, and it evidently was not as rigid as many others have been, and while I was trying to test it for rigidity, which I didn't really see it move, all of a sudden he said, "I can hear everything."

He said, "Somebody just dropped something in a pail, in a galvanized pail," which was outside the operating room, and they had.

I thought to myself, "My God, is this man really hearing?"

So I whispered to him in a very, very soft whisper, and he said, "You're whispering too loud, I can hear everything you say." I then whispered to him, "Can you hear?" He said, "Yes, I can hear."
Of course, many of them would respond that way, I thought. Then I asked him in a whisper, very soft whisper, "Do you like scrambled eggs?"

Now, I knew that he did not expect to hear. And he said, "Yes, I love scrambled eggs."

Boy, I spent some restless night that night, and I realized what I had done. I realized what happened, but I didn't realize exactly, because I didn't try to do it. It was accidental really.

Then since I knew that that must have happened, I then turned around and started to work on cadavers, to do deliberately what I had done accidentally. That was in April of '52. Now, how was I going to do that? I had to develop a technique; I had to develop instruments. It was a kind of a, an operation that you had to be very delicate about. Cadavers were awfully hard to come by. And I discovered that there was a black market in heads. I can't tell you, that's a military secret. I never told anybody. But I used to buy these heads, and bring them home and work on them after the kids went to bed. I did quite a number of those, until I was sure that I could operate on the living without doing any harm.

Rosen: Speaking of cadavers, I remember about that time there was a very, very famous ear surgeon from South Africa who had come to America, and he wanted to see this fenestration operation that I was doing before I came on the stapes operation. He was in town for just a few days, and he called me in the country. We have a little house in the country, and I used to keep my heads in a root cellar that we dug out of the side of a hill, like the old farmers do in the countryside, they keep their potatoes and apples and stuff all winter. So I had a real fine place for my heads, in a big crock. I must have had 12 or 15 heads there. And it seems that--

So I invited him to come up to the country, and I had a setup in a garage there so that I was working weekends on the cadavers just the same. So he came and we worked for maybe three or four hours. He showed me what he was doing. I showed him what I was doing. And then I was on my way back to the root cellar, which was about 100 yards from the house, with a bushel basket, with these heads in it. I had four heads in there. And I was halfway down when my wife called me and said, "There's a phone call for you, you'd better come up right away." So I dropped the heads right then and there. Come back, forgot all about them -- all about them.

And the next thing I knew, we had to have somebody come and plow up the land there for about an acre, and the farmer found these heads. He never said anything to us, but he called the homicide squad. And one Sunday morning -- we were very peaceful at the house in the country -- up drives this car with four men in it, and he picked out one head, and he said, "Where's the body?"
Well, I had one hell of a time convincing him that it wasn't anything of that order. I took him down and showed him the heads in the root cellar. I took him into the garage and showed him all kinds of temple bones and what not, and so he called his office, and they said, "I guess it's all right." But that's the closest I ever got to being accused of murder.

It was awfully difficult to get a hearing on this stapes work, because so many men all over the country, in fact all over the world, who had trained with Lempert and had been doing very fine work, couldn't quite get the connection between what they were doing and doing something to the stapes. And so after I had done six cases, I reported them as a preliminary report in the New York State medical journal, and then I started to try to get some of the fellows to come up to watch me do it. But there were no takers. And I said, "Look, you test the patient before the operation. Let me do the operation. You come up and watch it. I'll teach you how to do it. Then you do the hearing tests after the operation." And I still had no takers.

Finally, I gave a paper at a national meeting in Hollywood, Florida in '55, and after that everybody seemed to be very interested in it. And so doctors were coming from all over the country and from many other countries to watch this surgery.

The first operation that I did was to put an instrument on the neck of the stapes and actually try to pry it loose. But in many of those the fixation at the footplate was so severe that these crura arms broke before the footplate would be mobilized. And so I began to work down around the footplate to loosen it up before I did this. Then later on I tried making a little hole in the footplate in those cases in which the other techniques did not work. Of course, since then there have been many modifications, many improvements, and whereas in the beginning we got about 25 percent of patients with a good result after surgery, some of whom regressed, at the present time I think we can get about 85 percent.

For the most part today men are taking this out altogether as stapedectomy and putting in a wire from the incus down to the vestibule. I rather believe it's better whenever it is possible to take out as much of the stapes as you can, leaving the articulation of the incus and the stapes intact, and then pushing the posterior portion of the footplate forward so that the posterior crus can act in the same way as the wire. I believe that the long postoperative results give less involvement of nerve loss after this operation than after using the artificial strut, though if I cannot do this, then, of course, I use the artificial strut.

Well, in a few more minutes, I can just talk a little bit about an experience. I traveled around to many countries to demonstrate this, and it's been a lot of fun and very heartwarming. It isn't really terribly often in any person's life, I think, that he gets a feeling of ecstasy. But to see a patient who is deaf, hard of hearing, wheeled into the operating room, who needs his hearing so badly, and then you do something to the stapes, and on the operating table his hearing comes back --
that is really something that I would call so gratifying, and has always given me a great feeling of ecstasy.

Now, I remember many years ago on the way to India where we were -- I say "we" because my wife always travels with me and helps me in the operating room -- we were on our way to India. We'd been invited to India, and so I thought on the way to India we might stop in Moscow. We'd never been there. This was in '56. And so I went to the Ministry of Health and told them that I'd like to see what they were doing about deafness, and could I visit a hospital or institute. The next day, they said "Yes" and they told us where to go.

It was about the time that we read in the press here that Russia was the first country to invent baseball, that it was the first country to invent damn near everything that we'd been doing, you know. And I said to Helen on the way to this institute, "If they say they've done this already, the first to do it, I'm getting the hell out on the next plane."

Well, we got there and we met the old professor and his staff, and we talked back and forth through an interpreter for a while, and finally he said, "Professor Rosen, we are familiar with your work," and with that he turned in his swivel chair and pulled out a volume that had a translation with diagrams, pictures of the instruments, of every paper that I had published up to that time. And he told me how it was Dr. Samuel Rosen, which I couldn't read in Russian, and under it, Mount Sinai Hospital.

So he said, "Now that you're here, will you show us how to do it?"

Rosen: So I said, "Sure." Well, I couldn't get out of Moscow for about twelve days, I operated every day, and I must say that while there, of course, most of the doctors there and especially some of the surgeons, many of the surgeons are women, and most of the women that I met there, especially the doctors, are pretty hefty. I must say, every time I did something, and I took my head away so that they could lean in and look in, it was as if I were being pushed around and back by a lot of Jello. [laughter]

But everything worked out all right. By the time we had gotten through India, and then we went to Ceylon and two or three other countries, and then on the way back we were stopped in London, and when I went to my instrument maker -- I go to every instrument maker in every country that I visit -- and when I went there, he said that they had already had an order for some forty sets of these instruments from the Soviet Union. And they're doing it like hot cakes over there, as they are everywhere in the world.

So I was really very, very lucky to have come upon this accidentally. But I knew what had happened, and I knew I shouldn't give up, and I didn't give up and I'm awfully glad I didn't. Thank you. [applause]
Dr. Crohn wasn't able to be here because of a fractured ankle, unfortunately, and I'd like to read some excerpts from a letter which he sent me.

I saw Leon Ginzburg yesterday and he has volunteered to give the whole historical background on granulomatous diseases of the bowel, and correctly so, since he was studying the pathology and clinical aspects with Gordon Oppenheimer before or during the time that I, on purely clinical experience, was describing the first clinical findings in regional or terminal ileitis. My first paper on granulomatous, or what I called segmental colitis, was in 1938 with A.A. Berg. I considered it a form of ulcerative colitis and so it was reported by Klemperer and Otani for years. But the British pathologists, Morson particularly, proved that at least 50 percent of the cases were granulomatous and so it now remains. At Prague in 1968 they, that is the British contingent, named it Crohn's disease of the colon. I was the only one who objected. Cordial regards to my fellow alumni. Burrell Crohn.

The names of Crohn, Ginzburg and Oppenheimer are associated with this definitive paper on regional ileitis, now often called granulomatous disease of the bowel. Before we hear from Leon, a word about Gordon D. Oppenheimer, who today will be the silent partner.

Oppie was an intern at the Hospital from 1922 to 24 on the services of Edwin Beer and A.V. Moschcowitz. He spent five years in the pathology laboratory and was appointed an Adjunct Surgeon in the urology service of Dr. Beer in 1929. You all know that he was Director of Urology here from 1947 to 1963, and a popular and well loved chief he was. He is in the active practice of urology at the present time. Gordon, will you stand for a minute? [applause]

Leon Ginzburg, with a "z" not an "s", a "u" not an "e". His medical story begins in the familiar way: P and S, 1920; intern at Mount Sinai, 1920-24. Leon was one of the few surgeons who also spent a period as house physician. I remind you that there were no medical or surgical residents in those days, and that the house physicians and surgeons were in complete charge of the services.

At the Hospital, Ginzy rose to Associate Attending Surgeon and then in 1947 became Director of Surgery at Beth Israel Hospital in New York. In between, and this was news to me, he directed the surgical service at Harlem Hospital for a short time and was also Attending Surgeon at Sydenham.

At present he is Consulting Surgeon at Mount Sinai, Beth Israel, Harlem, Bayonne, Monmouth and Beekman-Downtown, and is Clinical Professor of Surgery at Mount Sinai School of Medicine. Ginzy was on active duty with the Army during World War II. Like any proper surgeon, Dr. Ginzburg saves his greatest efforts, both technical and vocal, for the operating room, where his skill
is legendary. I give you a man who is near and dear to our abdomens, Leon Ginzburg. [applause]

Ginzburg: Al, fellow alumni. I just remarked something. All the men speaking here this morning were all here, contemporaries from a period of about 1922 to 1925. Just what caused that outburst of genius, I don’t know. [laughter] I wish I did. We’d get a few more outbursts.

If anything is Mount Sinai’s cosa nostra, it’s regional granulomota of the intestine. The first article written on the subject in American literature, as far as I know the English literature or any other literature, were by two Mount Sinai men. But the article was not from Mount Sinai. The two Sinai men were Eli Moschcowitz and A.L. [Abraham] Wilensky. Dr. Moschcowitz everyone will surely remember; he’s only been dead for a few years. And he was sort of Mr. Chips around here in his later years, one of the really thoughtful, kind and intelligent people around here.

Dr. Wilensky was a surgeon. He also had an extraordinary intelligence which was rather underrated. I think his surgery was a bit overrated. For my money, he was a rip snorting, tearing surgeon, but the medical men thought he was great. As they said, “He didn’t diddle.” Well, neither did a fair number of his patients afterwards. [laughter]

But the man really had a tremendously powerful intellect. One of the things he contributed was the first clear understanding of the septic osteomyelitis, osteomyelitis accompanied by bacteremia, usually staphylococcus, and in spite of all his aggressiveness as a surgeon, he learned enough to counsel restraint in the approach of this particular thing, saved a good deal of unnecessary surgery and a great deal of damage.

Here were two highly intelligent men who never got things straightened out, and the reason was very obvious. They didn’t have the material. They had managed to gather four cases from four different institutions over a period of five years. The first case that they studied was in 1918. It’s interesting to me that Dr. Moschcowitz at that time was the Pathologist at Beth Israel Hospital.

Ginzburg: But if these men didn’t have the material, there was somebody who did: Dr. A.A. Berg. I look around here; I think everybody here is old enough to remember him. To have seen him was to have never forgotten him. He was in many ways the most extraordinary combination of qualities that I’ve ever encountered. Those people who knew him, who saw him, believe him. Anyone coming here after 1950 wouldn’t believe it. They wouldn’t believe a description. It would be impossible. I don’t think, as a matter of fact, in the present day of rebellion of youth, that he would have lasted a week. [laughter] He was a great surgeon. He was at least thirty years ahead of his time. And he had a practice which was just incredible. He operated all day long, except for two hours in his office, every day of the week including Sunday, when he used to relax by going down to Long Branch to do a series of cases that had been set up for him.
I was the house surgeon on his service, and from 1926 to, through about 1931, I was his private assistant. As I used to say, I never saw any patients in Mount Sinai, and I believe it was his policy never to have any of his assistants take care of people who could talk English. They could take care of them in the Bronx and Sydenham, where the English-speaking facility was a little lower. But he had a tremendous – he’s the foundation of all the surgery in this hospital, and whatever of a surgeon I’ve become, I owe to him. I have a peculiar ambivalent reaction, which, if you know me, you’d understand. [laughter]

Anyway, working with him, the material I saw was enormous. And, I have to use "I" a little bit -- first I ran into a series of cases of tumors of the colon, the colon and abdominal wall, operated under the diagnosis of carcinoma, which proved to be tumors resulting from the penetration of fish bones through the colon, and the formation of pericolonic masses. And the old boy was so good, I remember him on two occasions saying, "This isn't a carcinoma," and he just fished around there, in two cases, until he came up with a fish bone. Well, I published an article on that subject in 1927.

The next article I published on the subject of granuloma was the type of disease which we encountered following strangulation of the bowel. You know, in those days, the story was, 'if in doubt, put the bowel back,' because a resection was not a minor event in those times. Now, of course, 'if in doubt, take it out,' which is much better. And some of these were put back. The patient did not die of peritonitis or intestinal obstruction, but later went on to form a fibrosing sort of lesion in the affected segment, which led to further operation because of intestinal obstruction. And in a very short time, Dr. Berg had about four or five of these, and those I reported in 1927.

I began to notice other things coming up. This is almost 45 years ago. A lot of this was quite new, and at that time Oppie was working in the laboratory. He was working in surgical pathology, and these things were beginning to really come in, as operation became more frequent.

I came down to see Oppie one day and said, "Look, Op, there’s a lot of these things coming and I can’t follow them all. You’re down here. Catch them as they come through and study them." And we discussed the whole thing. We decided various things had to be done. It seemed sort of superfluous today, but not then.

Ginzburg: First of all, we had to distinguish all these things from tuberculosis. And from the various luetic gummata. And from lymphomas, which were not as clearly understood then as now, and from amebomas and from actinomycosis. And we discussed the whole thing and Oppie took that over, with the help of Dr. [Paul] Klemperer, and we often got together and talked these things over.

We found, incidentally, it’s a pretty good article, it’s been lost -- I was telling one of these men that hadn’t Dr. Crohn gotten into this thing, this would have been lost in some surgical journal and the best we ever would have gotten was an asterisk with a reference, or maybe two or four after our name, in the discussion of a similar subject. We never would have gotten number one.
Anyway, as I re-read this article the other day, I really was amazed. I really was amazed at the perspicacity we showed. [laughter] No kidding. The granulomatous disease, we had it there, of the colon, it's there, it's described. There's a picture of it, showing it was not ulcerative colitis. But that had been described before. The vascular changes, incidentally, the full force of which we are beginning to appreciate only now, when people are getting old enough, live to be old enough to have vascular changes in the supply to their bowel. We're beginning to see these vocal necroses and strictures and so forth that we saw at that time only in strangulated bowels. Then, of course, we had the diverticulosis and diverticulitis, and tumors you got from appendicitis, and pseudo-tumors, real tumors but pseudo-carcinomas that you got from these things.

Then there began slowly to come out a series of cases which had never been described before. They were these cases that we saw in the terminal ilium largely, where you found a segment of bowel, from a few inches to a couple of feet, which were thick, demitous, garden hose like, we called it, big, huge, glands in the mesentery, demitous mesentery, abscesses in the mesentery, communications, internal fistulæ between other hollow fiscera and this diseased segment of bowel, at that time mostly colonic, and it was obvious that we were dealing with something which hadn't been described, at least described as such. I'm sure they were described: it was called tuberculosis. But we showed pretty surely that it wasn't tuberculosis. And those cases were being operated upon for tumor, carcinoma, sarcoma, tuberculosis; they were being operated under wrong diagnoses. I remember one of the early cases Dr. Berg operated upon for a sigmoid carcinoma - well, that's what the X-ray showed - but actually what it was, was a fistula between the terminal ilium and the sigmoid, which produced a fistula in the sigmoid.

Of course, the Hospital was different then. It was a small hospital. Everybody knew all about what was going on all over. And I remembered one specimen I saw in the pathological section [museum] that Dr. Libman had put up, where things were kept in glass cases, and I remember helping him shift them around, in his innumerable shifts. We went and got that specimen. It was typical ileitis. It appears in our article which appeared in 1933, in a glass case. We hunted the fellow up and added another case to our list.

Ginzburg: Then Gordon and I both remembered a cause celebre on the service of Dr. Beer, where a man was supposed to have a retroperitoneal infection and so forth in the ureter. And we smelled something funny about it and went and looked it up, and we discovered that eventually this man was proved to have regional enteritis. Of course, lately there's been a rediscovery of what shall we call it, ureteral changes secondary to regional enteritis. But we had all these things.

There was one thing though that still, we couldn't understand. Most of these patients were being operated on for carcinoma and other tumors and at that time there were a great many fistulæ following supposed appendectomies or drainage
of appendicular abscesses. We had begun to suspect that this had some connection with these specimens that we were finding. In the first place, Ralph Colp had made a very thorough study of cases of appendicitis, acute appendicitis in this hospital, where focal fistulae developed, where they could be sure it was an appendix. And he found that the overwhelming number of them were healed spontaneously, and that those that weren't, there was usually something like a lip fistula of the cecum or something else local in the cecum that could be easily repaired. But from all over the country there were reports coming of these fistulae that they treated by sewing up and doing ileostomies, doing one thing or another but never resecting it or bypassing it, and we were beginning to think that there was some connection between them.

I remember very well the case that finally shed, gave us the answer to these external fistulae, intractable regional enteritis. I see Sidney Grossman is sitting here. He was the house surgeon. It must have been about 1928 or '29. When I came up one Saturday night, there was an appendix abscess to be done, and I came up to shamuse it, and I shamused Sidney through this operation, and he couldn't find the appendix.

"Oh, let me have a look at this --" I couldn't find the appendix. So after continuing for a while, we just stuck some drains in and got out. Man closed up.

Came back about six months later with another abscess. It was drained. None of these smelt very focal. Came back a third time. And by that time, Dr. Berg had been clobbering me pretty badly for having given this fellow a focal fistula. I practically got down on my knees to him and asked him to operate on this man, because I had a feeling that it had something to do with regional enteritis.

Well, he went in and he operated, and resected the terminal ileum, and the ascending colon, and there was a perfectly normal appendix, and a number of fistulae coming out of the mesentery of the affected segment of ileum. And then we had it, the relation of all these external fistulae, focal fistulae following appendectomy, to regional enteritis.

We were very optimistic about the effect of surgery in those days. As a matter of fact, I didn't write it, but the original -- I never had that ebullient optimism -- but it was decided that it cured everything. All the patients operated on were cured. Well, it didn't take us long to find out that that wasn't so. By the time Gordon and I got our paper published in the surgical journal, we were able to report at least two recurrences at the site of anastomosis. And it's interesting, we

Ginzburg: almost never got that paper published. You know why? It was too good. We sent in a paper with case reports and everything else, and they sent it back to me. They said, "This should be a monograph." This was in 1932. We were going to finance a monograph? Oppie and I were barely keeping our heads above water. [laughter] Like most everybody else at that time. So I got in touch with the Annals of Surgery and said, "All right, I'll cut this down," and they finally printed it.
Now I come to the $64 question, which has been anticipated by the note that Dr. Crohn left with Dr. Gordon. I wish to hell he'd said that a long time ago. It's the first time I ever heard anything like that.

Any idea that Oppie and I were a couple of boys who came along for the ride on the tail coats of the mighty is so much phonus-balonus. The pathology was studied by Oppie and myself. The surgical parts were studied by Oppie and myself. I wrote the part on pathology. I wrote the different types of disease. Burrill added a few cases and considerable embellishment. I mean, he had the touch, we didn't -- as I always say about him, he's the St. Paul of ileitis. We'd have been left in some little Sinai desert where you get an occasional asterisk, but like St. Paul, he took it all over the world. It got to be known. He got to be known. And the Hospital got to be known.

Incidentally, of all the things reported this morning, the least important is ileitis. We have a completely cockeyed idea of its frequency: They congregate here. They come here from all over. If you go from here to Beth Israel, you're amazed at the difference in the incidence of ileitis and colitis cases that you see. This place gets 'em. And while it's important and it's cleared up, of all the things presented here this morning, this is by far the least important, and like so many other things that are unimportant, the most talked about, battled about, and fought about.

I've developed a sense of humor about this some time ago, many years ago. I used to get a hold -- when Oppie and I would go somewhere, meet some doctors, I'd say, "Meet the Ileitis boys, I'm Et and he's Al." [laughter]

So as a footnote, probably a very important footnote to think about, shows you the relation between history and actuality. I think when you don't understand what's going on in the big world, take a look at what happens closer to home where things can be seen in a much lesser perspective, and you get an idea what it's all about.

With malice toward none, charity for all -- [applause]
Gordon: Thank you. Mo would like to make an announcement. He's had some requests for the year of appearance of the article he showed and that was the *Klinische Wochenschrift* in 1929.

The last of our giants, Coleman Rabin, started his internship at the Hospital here in 1922. He followed the then current trend by working in the pathology laboratory from 1927 to 1938, where his keen eye and nimble brain quickly established him as an outstanding morbid anatomist. He never lost his abiding interest in pathology in the succeeding years when he ran the chest service at Mount Sinai and attracted several generations of house staff who waited expectantly for the pearls of wisdom to drop. And drop they did, for Kelly above all was and is a passionate and inspiring teacher.

Kelly has just finished his time as president of the American College of Chest Physicians. He practices from an office in the Klingenstein Pavilion and is widely sought as a consultant. He has always had appointments here in both medicine and radiology, and is even now working with Dr. Murray Baron on the revision of his book *X-Ray Diagnosis of Chest Diseases*, which promises to be a classic. Dr. Rabin will speak on the development of the concept of putrid lung abscess and its treatment. Dr. Rabin.

[applause]

I'd just like to announce, in case any of you have to leave early, that there'll be a luncheon in the cafeteria, and we'd like the speakers to be sitting at the main table.

Rabin: Friends and fellow alumni. I almost forgot that I'd been working in pathology this length of time, and with Leon here, it reminds me of the days, the time when he was working on ileitis, when we lived in the offices together. We couldn't live anywhere else. We sponged meals off the Hospital and paid no rent. It might be in the middle of the night, might be 12 o'clock at night, 1 o'clock in the morning, I'd get a telephone call. Ginz would answer the phone. I'd say "What is it?" He says, "An autopsy. Rush, the patient may die." It was an emergency in those days. It was an emergency.

It happened about that time there began to develop a new type of emergency, and that was putrid lung abscess. And the way we looked at it was as follows. That a putrid lung abscess, like appendicitis, as soon as you made the diagnosis, off you went, no matter what time it was, a good many of these cases being done at night. And a ruptured abscess, even though it was localized to a small area and just showed a little fluid level on the film, this was like a ruptured ulcer. This was [a] super-emergency. So we had three kinds of emergencies here, aside from the ones that he had, and these were, the autopsy, number 1, and those lung abscesses.
Rabin: Now, speaking about lung abscess reminds me of a talk I gave in Leyden last year. There was a symposium on diseases of the bronchi and I spoke on broncholithiasis. And I thought I gave a good, interesting talk on broncholithiasis. When I got finished, the chairman got up and smiled and he said, "Thank you very much for your interesting lesson in paleopathology. We don’t see this anymore." [laughter]

Well, you have pretty much the same thing here, in the case of lung abscess. We don’t see many putrid lung abscess, we don’t see many anymore. The patient who feels ill, has a pain in the chest, has some fever, immediately gets some penicillin and in most cases the disease is gone without anybody ever making an X-ray film or even knowing that he had any important disease. Few are the cases now that present a problem.

And yet, there are those that do. And because of this, it’s important for the physician to know and understand the disease. Unfortunately, we have a great many of the young chest surgeons who either have never seen a case in the acute stage, and haven’t seen the early stages of the disease at all and have very little understanding of the entire problem. So, the same way with broncholithiasis. The disease is getting rarer and rarer, as tuberculosis is rarer and rarer, and there are cases in which this is the problem and where you have to understand the disease and know its manifestations in order to handle it right, and the same holds for the lung abscess.

Now, the tradition of chest diseases in Mount Sinai goes back far. It goes back here to the time of Howard Lilienthal, who is rightly considered the dean of thoracic surgery in the United States. He operated on patients who were said to have pulmonary suppuration. These were patients who’d been ill for quite some time, coughing and spitting and miserable, and had reached the point of no return, and they finally got to Lilienthal, and in about 1920, he did lobectomies on these cases -- and was the first one to do lobectomies in suppurative disease of the lung.

He got a certain number of cures; a high mortality, many people weren't cured. Many people left with fistulae and empyemas and so forth and so on. It wasn't entirely satisfactory but it was a great advance.

And at about that time, soon thereafter, Harry Wessler became interested, around that time, in lung diseases, and eventually he pointed out an article that he’d written in 1912, and this was the first description of post-tonsillectomy lung abscess. He had great perspicacity. He’d seen some cases of putrid lung abscess, and he visualized the pathogenesis of the disease as we know it today. That is, the result of inhalation of a particle of material, in this case crypts of tonsillar crypt or blood clot, contaminated with a mixture of organisms, impacting in the small bronchus producing a gangrenous infection of the lung distal to the small bronchus, the lung supplied by this small bronchus, which quickly would soften up and form an abscess. This pathology that he visualized, without handling organs but simply from his clinical and X-ray observations, holds to the present time.
Rabin: I became interested when I began to work with Dr. Wessler on the chest service. At that time, [Sidney] Yankauer, whom most of you here, I see the age group is right, will remember, if not having known him personally, at least have heard his name, and [Rudolph] Kramer who worked with him, were treating acute putrid lung abscess through the bronchoscope. And they were putting a tube into the segmental bronchus, we know it now as the segmental bronchus, at that time it was the bronchus that leads to the abscess. They hoped to get into the abscess and they thought they did. They really didn't, but at least they got into this bronchus, and they not only sucked out but they irrigated. They used a weak solution, a lugol solution, and they had a tube which was double-barreled and through one barrel would go the solution, and the other was sucking, and so they were able to have a continuous form of irrigation, because they were afraid to simply inject solutions into the bronchi without this fear that the patient might drown or spread infection or what not. We know today that you can do these things. You can pour in all manner of solutions into the bronchi in large amounts and not cause any trouble. Of course, this wasn't known, but they had this ingenious apparatus, and they found that if they did this in a certain number of cases of acute lung abscess, they could be cured.

Now, this whole idea is based on the assumption that there is a bronchus entering the abscess of the lung. And if one goes back to Wessler's hypothesis, or postulate if you will, that the disease was caused by the inhalation of particulate material containing these destructive organisms, a mixture of them, one can understand that disease then began in that bronchus, and that destruction would begin in the bronchus and everything in the lung beyond it was really secondary to that infection. Therefore, a bronchus had to empty into the cavity, and it was very important to realize this from the standpoint of what became the standard operating procedure.

Well, we began to study these cases of lung abscess from several standpoints in the laboratory, the Path lab. [Harold] Neuhof who had begun to operate on some of these cases thought he was doing an operation for bronchotomy because Aschner, who had studied the material of Lilienthal, had come to the conclusion that the abscess was a blowout bronchiectasis, that is a blown-out bronchus and bronchiectasis, because he was operating on the late stages and this is what you saw. Because you saw the bronchus entering it, the bronchus entering it would be rather wide in a chronic case, and the epithelium would begin to grow into the, to line the cavity, in some chronic cases almost line it completely. It would look like a bronchiectasis.

And we went over this thing and hammered it out and discussed and discussed and discussed. Dr. Neuhof was a rather stubborn man when it came to ideas. He reached conclusions through logical reasoning. If he started off with the wrong premise, his logic would lead him out to left field, of course. But there was logical reasoning there, and once he reached a conclusion he would not change that unless he found a good reason for changing it. But he had an open mind. He would listen to anybody. Here I was, a young fellow, and I told him, "No, you're not doing a bronchotomy, you are draining a putrid abscess of the lung
which is formed by a communication with the bronchus, the bronchial opening is always there. Now, you may for practical purposes be doing the same thing."
Rabin: He said, "Yes, but I like to know exactly what I have here. How do you know that this is so?"

I said, "I know this is so for many reasons. I know it's so from studying X-ray films serially, and I see that the thing starts as a pneumonia and then breaks down, you get your fluid level, you have your cavity form. I know it from studying pathological material. I see several things. I see first of all that this is, it's true connected with the bronchus, but it's a bronchus leading to the periphery. These lesions are all on the periphery of the lobe. And interestingly enough, there are always adhesions over the lesion, so you don't always have to do what you've been doing, that is, a two stage operation. Because if you go where the adhesions are you can get into the lesion."

And he said, "Well, how do you differentiate it from bronchiectasis? In any event, how do you differentiate this? Why do you say this is all aspiration disease? How do you know it isn't metastatic abscess? What is the difference?"

I began to talk to him about suppurative bronchial pneumonia being a different disease.

He said, "What is a suppurative bronchial pneumonia?"

Had to explain it to him.

He said, "Well, it all sounds very good. But you gotta prove it to me. How can you prove it to me?"

I said, "You'll have to look at the specimens."

He said, "All right, let's come down now and look at your specimens."

I said, "That you can't do."

He said, "Why not?"

"Because we added them up a little while ago. There are 68 specimens, they form a series that give you the pathogenesis of these various conditions, and you have to understand them all and you have to look at 68 specimens."

He said, "All right, we'll spend an evening."

I said, "You'll never get through in an evening."

He said, "I'll stay up all night."

I said, "Well, I don't think you'll be able to cover it."

He said, "Well, we'll see."
So he got Wessler and [Paul] Klemperer and I and he and, that's all I think it was, at first. And I began to dig out these specimens from the jars where I'd put them, and we went and took them in order, to show him what is a metastatic abscess and how it differs from the other; what is suppurative pneumonia; how bronchiectasis comes about from suppurative bronchial pneumonia, and how putrid lung abscess develops and how it begins and how it develops into a chronic stage and looks like bronchiectasis.

Well, we did this, and the first night we stayed up till 4 o'clock. We started at 8 o'clock in the evening, we worked till 4 o'clock. Then we adjourned for another night and did the same thing. By this time, 12 o'clock at night, Wessler was finished; 2 o'clock in the morning, Klemperer was finished, but we kept up till 4 o'clock. Then we finished up the last night at 2 AM and just Neuhof and I were the only ones there. But he lasted through, and he said, "By golly, you're right."

This was always the way with Neuhof. If you could show him, prove something to him, no matter what his concepts were before, he'd change. But you had to have logical proof. He's a great task master that way. And we all learned a tremendous amount from him because of this. I did. I learned these diseases, so much about these diseases, because he always would insist on proof for everything, and I had to find the proof. In order to find the proof I had to work. This is what went on year after year. This was a great man.

And then comes the question of treatment. So we had the pathogenesis work done. And Wessler said, he was impressed by one thing, that if a patient had a localized and, in most cases a perforated lung abscess has a localized abscess of the pleura, localized pyopneumo-thorax, a localized empyema over them, if you went in and drained that properly, most often the abscess underneath would heal. He said, "Why not skip that step? Don't wait for the patient to perforate through, drain it beforehand in the acute stage?"

Well, the story was then, this had been done. It had been done many, many years before in the early part of the century, and the mortality of draining a lung abscess in the acute stage, before six weeks, was 75 percent. If you waited till after six weeks, the mortality came down to 20 to 40 percent. So the idea was always wait six weeks.

Well, we found this -- that if we, starting with the postulate that there were adhesions over the abscess, which we knew, and we knew this in two ways. Number 1, the patients always would start with pain over the site of the lesion, so we knew they always started with a pleurisy. And number 2, when you fluoroscope the patient, have them breathe and have them cough, we'd never see movement of the lesion in relation to the ribs. Now, if you have a carcinoma in the lungs, and not adherent, and you have the patient breathe and look on the fluoroscope, have them cough, you'll see movement between the ribs and the lesion. Of course, if it's adherent it won't, and this was always the case. I say,
always, there are some exceptions -- there are about 2%, 4% exceptions. In 2% the lesion was adherent to the diaphragm, and the other

Rabin: 2% the lesion was on the periphery of the lobe where it had to be, but facing a fissure, not facing the chest wall. But 96%, there were those adhesions at all times.

And so the idea was to go in where the lesion was most superficial, and always the abscess would be situated within two or three millimeters of the pleura, and that two or three millimeters of lung would be collapsed, inflamed lung, and over it adhesions. Now, the area of adhesions might be quite small. It might be no bigger than this.

What did this mean? If we were not going to open the pleural cavity and go directly into the abscess, it would mean you’d have to have extreme accuracy, localization. So we had to develop methods of localization. And what we did first was find that we needed oblique views, examination of oblique views. Nobody made examinations of the chest from the oblique views at all, for any reason. We found we had to do that and learn how to interpret them.

We had to then learn how to tell just if the abscess was on the front or the back, it was easy to tell where to go. If it was on the side or at the bends of the ribs, it was very difficult to tell over which rib one would, exactly where the operation should be performed. This we did on the fluoroscope by having the patient turn around so that the rib over the abscess was tangential to the abscess and parallel to the screen, and by using parallax, we were able to determine this.

Well, what we’d have to do then was make a mark on the skin and tell the surgeon that’s where it is. But that was no good, because when the patient went to the operating room, the arm would get pulled up, the skin would move, and you wouldn’t be over the lesion. And so we had to localize the relation to a rib. I would know exactly where, in relation to what rib and just where, front or back. Well, I couldn’t transmit this information. So I had to be in the operating room. And many of these cases, as I say, were done at night, and every patient who had a lung abscess operated on, I had to be in the operating room.

Well, after a while this became quite a chore, because you’d hang around there for quite a while and begin to lose a little interest. And so we developed a system we called spot localization. We’d take about a fifth of a CC of lipoidal and a fifth of a CC of methylene blue and inject it into the intracostal muscle, important to inject it into the intracostal muscle because the intracostal muscle is connected by fascia to the rib above and rib below, so the material couldn’t shift too much. And then the fibers run up across this way and they would hold the thing back so it wouldn’t spread too far, so it wouldn’t go up and down because of the attachment of the fascia to the ribs and wouldn’t go too far forward or backward because of the direction of the fibers in the intracostal muscle.

We put that in, and then X-rayed the patient in the proper position where there wouldn’t be distortion of the curve of the ribs, might be this oblique, that oblique,
lateral or whatever, and then count the ribs, and then tell, then see the relationship on the film between this spot which we injected, which was visible
Rabin: on the film because it had lipoidal in it, and would be visible to the surgeon because it had methylene blue in it, and we’d make films and see how much off we were. Sometimes we’d be right over the abscess, sometimes we’d be a rib too low or a rib too high or an inch or an inch and a half too far forward, and inch and half too far back, and all I would have to do is telephone Dr. Neuhof and say, “Look, I’ve got a spot there, unfortunately I’m a little bit off, you should take the rib above the spot and just one inch anterior to it and you’ll be right over it.”

And so he’d operate, find the spot, the methylene blue in the intracostal muscle would stain everything very intensely, be no problem, and know exactly where he was. This way we lessened the number of cases in which inadvertently pleura would be opened, because that would add to the danger of the procedure.

And he, Neuhof -- yes, about two minutes -- Neuhof was very much bound by surgical principles. When it came to drainage, for instance, infection, every focus had to be drained. He didn’t just make an opening in the abscess, he would unroof the abscess, he’d look for every little loculation, drain those, pack those, and the patients did remarkably well. The mortality was cut down from 75 percent to 2½ percent, almost all these patients cured. If we waited over six weeks, mortality went up, and also the number of cures were fewer and far between.

Now, just one more thing I want to tell you about, and this is an offshoot from lung abscess, more important than lung abscess itself. When we were studying these cases and dissecting bronchi and injecting the abscesses with lipoidal, we found that the bronchi were pretty consistent. They had no names. So we began to study with stereoscopic X-rays these post-mortem injected specimens, and we began to give them names, and Neuhof said, “We have a young fellow around here by the name of [Ameil] Glass, let him work with you.”

I said, “Fine.” Well, all I had to do was tell Glass what the problem was and then he disappeared, and before I knew it, he had 200 lungs that he’d worked upon. He said, “You know, the bronchi and the blood vessels all supply the same area. They don’t cross each other. Each part of the lung is supplied by a bronchus and a blood vessel.”

I said, “This you have to show me.”

He said, “You can cut in between, it won’t bleed. It’s like in the kidney where there’s a bloodless area between segments of the kidney, I suppose.”

He blew up a lung for me and proved it for me. He had gone to the X-ray department. He’d injected specimens. He had dissected specimens from the lung root to catch these bronchi. And we decided we got to call this something. We finally came up with the name "broncho-pulmonary segment." He said at that time, “The way to operate on bronchiectasis or diseases that have part of a lung involved is to go through the lung root, get the bronchus and the blood vessel
leading to the area, and you can then get out that segment with the disease in it without cutting through bronchi and without cutting through blood vessels."

Rabin: Well, he wanted Neuhof to let him do it but he wouldn't let him do it, he wasn't on his staff. And this was taken up later, and this is the origin of segmental resection. And the whole idea of the broncho-pulmonary segment then came out of this study, from the Mount Sinai Hospital, in the study of lung abscess and the credit, of course, goes to Dr. Glass. [applause]

Gordon: If any of you haven't signed up for the dinner dance tonight, there's still time to do so. You're all invited to luncheon in the cafeteria, courtesy of the Hospital, and the way to get there is through the exit in the rear. Thank you for coming.