Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Cardiac Cirrhosis

Volume XX | Friday, October 15, 1948 | Number 3

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
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INDEX

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UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

Visitors Welcome

October 18 - 23, 1948

No. 218

Monday, October 18

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Interns' Quarters, U. H.

8:00 - Fracture Rounds; A. A. Zierold and Staff; Ward A, Minneapolis General Hospital.

10:00 - 12:00 Neurology Ward Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Staff, Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

11:00 - 11:50 Physical Medicine Seminar; E-101, U. H.

12:00 - 1:00 Physiology Seminar; The Adrenalin Sensitivity of the Denervated Kidney and Some Adrenalytic Actions of Dihyrogenated Ergot Derivations; W. G. Kubicek; 214 M. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; M-435, U. H.

12:30 - 1:20 Pathology Seminar; Pyoderma Gangrenosum; John Coe; 104 I. A.

12:30 - 1:50 Surgery Grand Rounds; A. A. Zierold, Clarence Dennis and Staff; Minneapolis General Hospital.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jonsen, A. B. Baker and Staff; U. H.

2:00 - 3:00 Surgery Problem Case Conference; C. Dennis and Staff; Small Class Room, General Hospital.

3:00 - 5:00 Kellogg Lecture; Pancreatic Insufficiency; C. D. May; 229 Center for Continuation Study.

3:45 - Pediatric Seminar; Renal Function Tests; V. C. Kelley; 6th Floor, Child Psychiatry, U. H.

4:00 - 6:00 School of Public Health Seminar; Dr. Herman Hilleboe, State Commissioner of Health, N.Y. State Health Dept.; 113 MeS.

5:00 - 6:00 Urology-Roentgenology Conference; D. Creevy and H. M. Stauffer and Staffs; M-109, U. H.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

Tuesday, October 19

8:30 - 10:20 Surgery Seminar; Lyle Hay; Small Conference Room, Bldg. I, Veterans' Hospital.

9:00 - 9:50 Roentgenology Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Robert Hebbel; Veterans' Hospital.

? - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I. A.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans' Hospital.

2:00 - 4:00 Kellogg Lecture; Antispasmodic Preparations; R. N. Bieter; Eustis Amphitheater, U. H.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans' Hospital.

4:00 - 5:30 Surgery-Physiology Conference; O. H. Wangensteen and M. B. Visscher; Eustis Amphitheater, U. H.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

5:00 - 5:50 Urology Pathological Conference; C. D. Creevy and Staff; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Dr. Rigler and Staff; Powell Hall Amphitheater.

Wednesday, October 20

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U. H.

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans; Room 1AW, Veterams' Hospital.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker and Joe R. Brown; Veterans' Hospital.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

4:00 - 5:00 Infectious Disease Rounds; University Hospitals.

Thursday, October 21

8:15 - 9:00 Roentgenology-Surgical-Pathology Conference; Walter Walker and H. M. Stauffer; M-109, U. H.

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans' Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

11:30 - 12:30 Clinical Pathology Conference; Steven Barron, C. Dennis, George Fahr, A. V. Stoesser and Staffs; Large Class Room, Minneapolis General Hospital.

12:00 - 1:00 Physiological Chemistry Seminar; Determination of Testosterone and Related Steroids in Tissue Extracts; Marco Rabinovitz; 214 M. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

4:00 - 5:00 Bacteriology and Immunology Seminar; Brucellosis in Animals in Minnesota; W. L. Boyd, Head, Veterinary Medicine, University Farm; 214 M. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 X-ray Seminar; Vertebral Venous Plexus; Harry Mellins; Todd Amphitheater.

Friday, October 22

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Staff; Veterans' Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, A. V. Stoesser, and Staffs; Minneapolis General Hospital.

11:30 - 12:50 University of Minnesota Hospitals General Staff Meeting; Psychosomatic Medicine and Pediatrics; R. A. Jensen; Powell Hall Amphitheater.

12:00 - 1:00 Surgery Literature Conference; Clarence Dennis and Staff; Minneapolis General Hospital; Small Class Room.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

3:00 - Kellogg Lecture; Roentgen Diagnosis of the Stomach and Duodenum; Russell Morse; 229 Center for Continuation Study.

4:00 - Kellogg Lecture; Roentgen Diagnosis of Acute Abdominal Disorders; Leo Rigler; 229 Center for Continuation Study.

Saturday, October 23

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U. H.

8:00 - 9:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor West Wing, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. R. Rigler, H. M. Stauffer, and Staff; Todd Amphitheater, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-101, U. H.

9:00 - 12:00 Psychiatry Conference; Station 60, University Hospitals.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:00 - 11:50 Urology Seminar; Neurogenic Vesicle Disfunction; John Emmett, Mayo Clinic; E-101, U. H.

11:00 - 12:00 Anatomy Seminar; The Histology of Antigen-Antibody Reactions; Berry Campbell; 226 I. A.

II. CARDIAC CIRRHOSIS

J. S. McCartney

In 1840 Becquerel stated that one half of his cases of cirrhosis of the liver were due to heart failure. Ever since cardiac cirrhosis has been discussed pro and con with many persons of note ranged on each side of the question. Not only the question of chronic passive congestion as a cause of cirrhosis has been argued, but also what part of the liver lobule is affected. Some maintain that there is central fibrosis and reversed lobulation and others that the scarring process is portal in distribution. So authorities are divided into several camps on this subject of cardiac cirrhosis.

It appears that Becquerel's diagnoses were made by gross examination or with a hand lens, although he mentions using a microscope. Had modern methods of examination been available perhaps many of his cases would have been eliminated. It is probable that many of his 42 cases were merely instances of high degrees of chronic passive congestion.

Interference with venous outflow from the liver results in chronic passive congestion of this organ. This may be of all degrees, depending upon duration and severity. In mild degrees there is simply accumulation of blood in the central portion of the lobule with distention of the sinusoids. In more severe and long continued instances the various degenerative processes, atrophy, fatty metamorphosis, and necrosis, take place. When the process is of a certain degree the centers of the lobules stand out as red points and the peripheries are pale. With a more severe degree of congestion adjacent centers coalesce giving irregular red streaks and lines. In early congestion the liver is enlarged, but with more severe and prolonged congestion the liver shrinks and returns to normal or subnormal size and the surface may be nodular. The shrinkage is due to degeneration, atrophy and necrosis of the liver cells. Commonly in this latter stage the consistency is increased. By many this is called cyanotic induration and by others cardiac cirrhosis.

That this stage is to be classified as cirrhosis depends apparently on one's definition of cirrhosis or what criteria are necessary before the name cirrhosis is applied. If one believes that in cyanotic induration there is an actual increase in the reticulum and connective tissue, then perhaps the name is justified. If, on the other hand, one believes that there is only a relative increase of these elements due to disappearance of parenchyma and a collapse of the framework, then the name cirrhosis is hardly justified. If, however, one requires not only an actual increase in reticulum and fibrous tissue, but also disorganization and rearrangement of the architectural pattern as the result of regeneration and hyperplasia of liver parenchyma, then cardiac cirrhosis must be exceedingly rare, if it occurs at all.

Some authors, e.g., Rokitansky, Sabourin, Ghon, Kaufmann, Mallory and Villaret and Justin-Besancon, describe in advanced chronic passive congestion an increase in the fibrous elements in the central part of the lobule. Some believe there is a mono- or multilobular cirrhosis with a reversed lobulation. By this term is meant pseudolobulation with the portal space in a central situation and the joined lobular centers forming a ring-like fibrosis about it. Both Ghon and Kaufmann describe this but are rather guarded about calling it a true cirrhosis. Lamberton and Allison describe as the final change, in chronic passive congestion, a central collapse fibrosis. According to Adami and McCrae and Rolliston and McNee, with continued congestion there is some increase of connective tissue centrally--cyanotic induration, but a well marked cirrhosis is infrequent in chronic passive congestion. Mallory found in long continued passive congestion "a small, dense, indurated liver showing microscopically in each lobule a slight central fibrosis."

Other authors, e.g., Handfield, Jones, Green and Legg, claim an increase of connective tissue peripherally, but not a true multilobular cirrhosis.

Piery and others believe that chronic passive congestion alone cannot cause cirrhosis and that many cirrhoses allegedly due to passive congestion are to be explained on some other basis, e.g., alcohol, tuberculosis, rheumatic disease, etc. Villaret and Justin-Besancon think there is an annular cirrhosis connecting central veins, but that this type is rarely pure, and that there is an added periportal sclerosis, that the condition is really a cirrhosis in a cardiac.

MacCallum does not mention cardiac cirrhosis. Gerlach believes the designation stasis cirrhosis should be dropped. Rössle, on the other hand, believes that there are rare instances of true cardiac cirrhosis. White believes a real cirrhosis may develop after years of congestion of the liver. Boyd mentions cardiac cirrhosis as the condition which develops by condensation of connective tissue due to atrophy of the liver cells from passive congestion. According to Anderson the fibrosis may be portal and central.

Boland and Williams studied the livers of 75 patients who died after one or more bouts of cardiac decompensation and found that five showed true cirrhosis. However, the change was not that usually present in portal cirrhosis but was patchy. One of the five had recently had acute catarrhal jaundice and a second was a syphilitic who had had specific therapy. I think these two should be discarded. They included a sixth case where the cirrhosis was thought to have antedated the heart failure. Garvin studied 790 cases where heart disease was the chief cause of death and found 35 instances of cardiac cirrhosis. There was fibrosis of a degree to distort the lobules. The single case given in detail had distortion of lobules from extensive fibrosis. The connective tissue was young and mostly near the capsule. Elsewhere there was no constant distribution. There was chronic passive congestion with marked distention of the central veins. Boles and Clark said they found that of 243 instances of cirrhosis 33 were cardiac. They give no data on the microscopic findings, but mention that it is the so-called red atrophy.

The occurrence of cirrhosis in a person who has had one or more bouts of cardiac decompensation I have seen many times, but that the cirrhosis is to be attributed to the chronic passive congestion is doubtful. A degree of broadening of the portal spaces in the livers of older people is not a rare finding, but much of the time it is not sufficient to be appreciated on cutting this organ. In instances in our records where the gross diagnoses of cirrhosis and heart failure have been made, commonly one cannot see the evidences of heart failure in the liver. (In advanced cirrhosis it is very difficult to identify central veins.) One may find a cirrhosis of mild or severe grade, which can, by no means, be correlated with the duration of the clinical evidences of heart failure. In some of these persons the central veins are easily found and if there ever was chronic passive congestion the evidences of it have disappeared. Some livers from persons with cirrhosis and a history of recurring bouts of decompensation over a long period of years may show less disturbances of the normal lobulation than others with a history of only a few weeks of decompensation.

If one rejects from consideration those cases with cirrhosis and heart failure and a history of alcoholism, syphilis, acute catarrhal jaundice and other possible hepatotoxic agents, the number of instances in which the cirrhosis may possibly be attributed to heart failure becomes rather small. It is to be admitted that cirrhosis and heart disease are commonly associated but that the former is due to the latter is open to serious doubt.

During the last six years the diagnosis of cirrhosis has been made 376 times or in 2.5% of autopsies. I have examined sections, stained for connective tissue, from 291 of these cirrhotic livers and have rejected 20 cases because they showed no cirrhosis. In the

remaining 271 instances some form of organic heart disease was present in 139 or 50%. This does not mean that these 139 persons had a history of cardiac decompensation. From the 271 cases I have selected 64 in which historical or other evidence indicated heart failure, but not all gave a definite history. (It is of some interest that 9 gave a history of alcoholism, whereas in the whole group 110 used alcohol.) In 30 instances death was attributed to heart disease, in 16 to cirrhosis and in the remaining 18 to a variety of causes such as fracture or operation. In 36 instances there was some reason to believe that part of the patients' symptoms might have been due to cirrhosis. The persons with histories of one or more bouts of decompensation of the heart had had such symptoms for periods ranging from $1\frac{1}{2}$ months to 17 years. Two patients had congenital heart disease. One died at the age of 8 years and the other at the age of 28 years. The former had a patchy cirrhosis and chronic passive congestion. The latter had a marked portal cirrhosis, but the signs of passive congestion were not found. In 47 of the 64 I considered the cirrhosis as slight, early, patchy or subcapsular. Two livers showed hemochromatosis and two metastatic carcinoma and so are to be rejected. Of the remainder, four should be rejected because of chronic alcoholism and one perhaps because there was apparently a terminal acute yellow atrophy. This leaves at most 8 where chronic passive congestion might conceivably be accused of being the cause of the cirrhosis. One had syphilis and so should be excluded. Of the other 7 only one showed cirrhosis of more than moderate degree. Four with a moderate degree of cirrhosis had had periods of decompensation for 17, 2, 5, and 1 years and in two the duration of cardiac decompensation is unknown. In none of these livers is there a definite increase of connective tissue in the central portion of the lobule. The only one with severe cirrhosis was a patient with congenital heart disease who died at the age of 28 years (Cor triloculare, blue baby, cyanotic most of the time).

Conclusions

1. Cirrhosis of the liver and cardiac decompensation are not infrequently associated.

2. When associated there is little, if any, reason to believe that the cirrhosis is the result of the chronic passive congestion.

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III. MEDICAL SCHOOL NEWS

Medical Foundation Announces Litzenberg Lecture and Memorial

Alumni of the University of Minnesota Medical School through its Minnesota Medical Foundation will honor the name of Dr. Jennings C. Litzenberg on Friday, October 22, at 8:15 p.m. in the Medical Sciences Amphitheater. Dr. Everett D. Plass, Professor of Obstetrics and Gynecology at the University of Iowa will deliver the Litzenberg Memorial Lecture on the subject of "Advances in Maternal Welfare." Dr. Plass, Distinguished in the field of obstetrics and gynecology, was a devoted friend and admirer of Dr. Litzenberg; and both men have contributed enormously to the cause of maternal welfare in the United States.

Interest in a Litzenberg Memorial Fellowship initiated by alumni in the Foundation has already grown to the extent that some national organizations have expressed a desire to participate. Likewise, a number of prominent laymen have announced their intention to subscribe.

A committee from the Northwest Obstetrical and Gynecological Society, including Doctors Lawrence Randall, Rochester; Russell Moe, Duluth; and Claude Ehrenberg, Minneapolis, are now formulating plans to submit to the Board of Trustees of the Minnesota Medical Foundation.

It is reported that a fund for sending superior students in the field of obstetrics and gynecology to other centers is under consideration. First announcement of these plans will be carried in a subsequent issue of the Bulletin as well as in personal letters to those interested.

Erling S. Platou, President
Minnesota Medical Foundation

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Dr. K. W. Stenstrom was made Knight of the Order of the North Star by the King of Sweden.... Dr. Leo Rigler has been made Chairman of the Commission on Public Health of the American College of Radiology and a member of the Board of Chancellors.... Dr. Wesley Spink has been appointed to the Board of Governors of the American College of Physicians... Mr. James Hamilton, Professor and Director of the Course in Hospital Administration, has been named "The Man of the Year" in the hospital field by the American Hospital Association.

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Continuation Study Center

A course in Tuberculosis and Diseases of the Chest will be presented October 18 to 20. This course is sponsored by the American Trudeau Society and the Minnesota Public Health Association. A course in Gastroenterology will be presented October 21-23. Both of these courses are intended for general physicians.

Kellogg Foundation Lectures

Dr. B. R. Kirklin	Roentgen Diagnosis of Neoplasms of the Stomach and Roentgen Diagnosis of Gall Bladder Disease	Fri., Oct. 15, 3:00 p.m. Chapel, CCS
Dr. Chas. D. May	Pancreatic Insufficiency -	Mon., Oct. 18, 3:00 p.m. Rm. 229, CCS
Dr. R. N. Bieter	Antispasmodic Preparations -	Tues., Oct. 19, 2:00 p.m. Eustis Amph., U.H.
Dr. Russell Morse	Roentgen Diagnosis of the Stomach and Duodenum	Fri., Oct. 22, 3:00 p.m. Rm. 229, CCS
Dr. Leo Rigler	Roentgen Diagnosis of Acute Abdominal Disorders	Fri., Oct. 22, 4:00 p.m. Rm. 229, CCS