Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Arteriosclerosis

Volume XIX | Friday, June 4, 1948 | Number 29

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
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INDEX

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I.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

Visitors Welcome

June 7 - June 12, 1948

No. 206

Monday, June 7

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.

9:15 - 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.

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

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. Jensen, A. B. Baker and Staff; U. H.

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

4:00 - 5:00 Pediatric Seminar; Reports of Scientific Meetings; I. M. McQuarrie, J. Adams, C. D. May and E. N. Nelson; 6th Floor Seminar Room, U. H.

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

Tuesday, June 8

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; Eustis Amphitheater, U. H.

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

12:30 - 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.

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.

Wednesday, June 9

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

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

11:00 - 11:50 Pathology-Medicine-Surgery Conference; Aortic Stenosis; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

4:00 - 5:00 Infectious Disease Rounds; Todd Amphitheater, General Hospital, Veterans' Hospital.

Thursday, June 10

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; Todd Amphitheater, 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, General Hospital.

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

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

Friday, June 11

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; Radiology of Congenital Heart Disease; H. M. Stauffer; New 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.

Saturday, June 12

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.

8:00 - 9:30 Psychiatry and Neurology Grand Rounds; Staff; Veterans' Hospital.

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. G. Rigler and Staff; Todd Amphitheater, U. H.

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

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; The Metabolism of Calcium and Its Relation to Urinary Lithiasis; E. B. Flink; E-101, U. H.

II. ARTERIOSCLEROSIS: A REVIEW OF THE PROBLEM WITH SPECIAL REFERENCE TO LIPOTROPIC SUBSTANCES*

E. R. Hayes**

An attempt will be made to very briefly review some of the significant aspects of the problem of atherosclerosis and to discuss some of the results of our work here as related to the pathogenesis and the possible relationship which lipotropic substances may bear to the prevention of the disorder.

The refractile material found in atheromatous plaques has for a long time been recognized as cholesterol. Numerous theories have been advanced as to the mechanism of the deposition of this lipoid in the intima of blood vessels.

One of the earliest theories, which has been largely abandoned at the present, was that the cholesterol was left from disintegrating tissues.

Hueper[1] has been one of the outstanding proponents of the theory that the cholesterol is derived entirely from the blood plasma filtering through the vessel walls. Briefly his concept is that under certain conditions of stagnation there is a disturbance of the colloidal equilibrium in the plasma with a coating of the endothelium by the inert cholesterol, which in turn then interferes with the nutritive exchange of the endothelial cells. This, according to his concept, leads to damage of these cells with a subsequent diffusion of the cholesterol into the subendothelial tissues, where it is later taken up by the tissue macrophages to produce the familiar lipophages.

Experimentally Hueper[2] has administered polyvinyl alcohol, methyl cellulose, pectin, and acacia intravenously to animals and has produced an atherosis which is histologically similar to that seen in man. The localization of the lesions is similar to that found in cholesterol atherosis, but there is no species difference in the response. This is supposedly because none of the animals used had a metabolic system for handling these substances.

Moreton[3,4] has recently been studying the physical state of the lipids in the blood. He has found that certain individuals have large particles of lipid in suspension in the blood. These particles can be seen under the darkfield microscope and can be separated by the ultracentrifuge. He has correlated particle size, as estimated by the two above mentioned methods, with the degree of diffusion of light in a specially constructed nephelometer, and has found that there is no correlation between particle size and the absolute values of cholesterol in the plasma.

Further he has found similar large particles following the administration of methyl cellulose, pectin, acacia and polyvinyl alcohol, the substances used by Hueper in the production of atherosis. It was his observation also that a like amount of energy was necessary to separate these particles from the plasma as was necessary to separate the lipid particles. He then thinks of two conditions which might predispose to the formation of atherosis; first, the individual who has persistently large lipid particles in the plasma and secondly, the individual who following the ingestion of a meal has large particles for a short period of time in the plasma. It is his feeling that atherosclerosis is the result of changes in the particle size of the lipoid material with local stagnation in the blood vessels. It is hard to understand though why a certain group of people will have persistently larger particles of lipoid in the blood plasma unless it represents some general underlying defect in the metabolism of these lipoids.

Leary[5], on the other hand, proposes that the lipoid is brought to the blood vessels in the lipophages. His concept of pathogenesis is that the cholesterol is esterified in the liver by the hepatic parenchymal cells. During periods of overload, he supposes an accumulation of the material in the parenchyma of the liver with the production of damage to the hepatic cells. Following this damage of the liver cells, they are phagocytosed by the reticulo-endothelial cells in the liver. These reticulo-endothelial cells, now lipophages, become wandering cells

and enter the sinusoids of the liver where they pass into the systemic circulation. They then adhere to the endothelial lining of the blood vessels and enter into the subendothelial spaces producing the atheromatous lesion. In his work on animals Leary has demonstrated the plugging of lung capillaries with lipophages, and has demonstrated all stages of the entry into the subendothelial space. As an explanation for the localization of these atheromatous lesions to certain blood vessels Leary advances the rather attractive theory that the fibroblasts in the subendothelial tissue are capable of metabolizing the cholesterol and thus removing it from this location. He further then suggests that in certain blood vessels in adult life this capacity of fibroblasts to metabolize cholesterol is lost. This then allows the development of the atheromatous lesion.

Gordon[6] has recently suggested another concept to carry the theory of Leary to the final development of the atheromata. He points out that these lipophages are lighter than the other blood cells and that with the slowing of the current of blood they would be the first to leave the axial stream and thus be the first to come into contact with the endothelium. He suggests that because of their inertness they would be pushed into the intima by the arterial blood pressure. Once inside the intima the majority would be retained by the internal elastic membrane, which is impermeable to these fat cells. He further points out that atherosclerosis occurs mainly in the elastic vessels, which are filled only in systole and are partially emptied during the remainder of the cardiac cycle, the current having an intermittency which is less manifest in other vessels that are farther from the heart.

Faber[7] has recently made an interesting observation in a study of the cholesterol content of the aorta and the blood serum from a group of autopsies. His observations were on normals of ages 18 to 73, on patients with coronary occlusion between the ages of 32 and 78, and on patients with hypertension and early atheromatosis between the ages of 49 and 65 years. In the normals there was, with increasing age, a gradually increasing amount of cholesterol in the aorta which was not correlated with an increasing serum cholesterol value. In the patients with coronary occlusion a number of younger patients showed an increase in the cholesterol content of the aorta above the value found in normals in a much higher age group. This increase was accompanied by an increase in the serum cholesterol value. In the group with hypertension there was an increase in the cholesterol content of the aorta without a corresponding increase in the serum cholesterol values.

Numerous studies have been reported in the literature on cholesterol values in patients with vascular disease. These are extremely difficult to evaluate because of the variations in analytical methods and the inclusion of hypertensive and arteriosclerotic patients in the same group. However, more recently there has been an increasing number of observers who have reported what seem to be significantly elevated serum cholesterol levels in proven cases of atherosclerosis as compared to normal controls. Such results have been reported by Davis, Stern, and Lesnick[8], Herrmann[9], and Steiner and Domanski[10]. These last observers reported on a group of patients with coronary occlusion who were followed over a two year period and compared with normal subjects. They made two significant observations, first that the group of patients with atherosclerosis demonstrated higher serum cholesterol values than did the controls, and second that this group of patients demonstrated wide variations in serum cholesterol values as followed from day to day. This was in striking contrast to the normal controls who demonstrated remarkably constant values from day to day.

This is only a very brief review of some of the significant contributions to this subject, but it would seem to lead to certain conclusions relative to the pathogenesis of atherosclerosis. First of all, it seems reasonably well established that cholesterol is causally related to the development of the atheromatous lesion. Secondly, it seems that certain individuals demonstrate increased serum cholesterol values, variable values, or variations in the physical state of the cholesterol as it

appears in the blood serum. Any one or all of these factors might predispose to the development of atherosclerosis.

Struck by the fact that certain individuals might have some rather basic metabolic defect which led to their faulty handling of cholesterol, we have been led to investigate the effect of certain lipotropic substances upon the development of this disorder.

By definition lipotropic substances are those which will mobilize fat and lipoid from the liver. The most familiar of these substances are choline, methionine, inositol, and pancreatic substances. Our work has been with one of the pancreatic substances.

Early in the experimental work on diabetes it was recognized by Fischer[11] that depancreatized dogs maintained on insulin developed a typical syndrome characterized by loss of appetite, loss of weight, increasing sensitivity to insulin, hepatomegaly, decreasing serum cholesterol, and finally death. It was found that the addition of whole pancreas to the diets of these animals prevented the development of this syndrome and could reverse it after its beginning.

Dragstedt, Van Prohaska, and Harms[12] then extracted from pancreas a substance which they called lipocaic and which was effective in preventing this syndrome in depancreatized dogs. They felt that this substance was an internal secretion of the pancreas. It is not necessary at this time to go into the discussion that has arisen about this point; simply to say that there is not agreement on this point at the present time. One contention that the lipotropic activity of lipocaic arose from the choline which it contained has been pretty well disproven. Another view has been that the lipocaic is really the external secretion of the pancreas and that its use in the dogs has lead to a greater digestion of protein making a greater quantity of choline and methionine available to the animal. The primary fact has been accepted by all that pancreas contains a substance which will prevent the development of a fatty liver in the depancreatized dog.

Those who have been interested in the study of atherosclerosis realize that there are marked species variations in the development of the disease. The rat and dog rarely develop sclerosis of this type; the rabbit will almost uniformly develop atherosclerosis if fed cholesterol. It has been our thought that the development of a fatty liver in the depancreatized dog might be a species response which is represented in the human by the deposition of lipoid in the blood vessels. Consequently we have attempted to evaluate the effect of a pancreatic lipotropic material upon the development of atherosclerosis in the rabbit.

The material used has been derived from pancreas by the method of Bosshardt, Cierszko, and Barnes[13]. The preparation has been found to contain 0.10% choline (expressed as choline chloride), 0.94% methionine, and 0.64% inositol. The material has been tested by Bosshardt et al for proteolytic activity without any evidence of digestion of a standard protein solution. Schwert and Neurath[14] have observed no splitting of the synthetic substrate, benzoyl-l-argenineamide, which is further evidence that the material contains no trypsin-like activity. Entenman and Chaikoff[15] have stated that this material when fed in daily amounts of 60 mgs. to depancreatized dogs maintained on insulin prevented the development of fatty livers.

Experimentally rabbits have been rendered atherosclerotic by the feeding of 500 mgs. of cholesterol, one egg yolk, and a few drops of linseed oil six days out of seven for three months. This procedure has resulted in the production of severe atherosclerosis in 100% of 14 rabbits.

Other groups of rabbits have been fed the control diet plus varying amounts of the pancreatic lipotropic substance. These results are summarized in the following tables. The degree of sclerosis has been graded as follows. If the entire length of the aorta is involved, sclerosis has been graded as "severe"; if the ascending aorta and the arch are involved, the sclerosis has been graded as moderate.

Animals showing only slight changes have been graded as "moderate." Those with no changes have been graded as "none." We have regarded animals graded "moderate" or "none" as having been protected.

Table 1

PLS* mgm.	No.	SCLEROSIS Severe	Moderate	None
0	14	14	0	0
25	7	1	6	0
100	6	3	2	1
200-500	6	1	3	2

For details see Table 3.
All animals fed sclerosis producing diet.
*Pancreatic Lipotropic Substance

Table 2

Animal	PLS* mgm.	Control	Serum Cholesterol 1 wk.	4 wk.	8 wk.	12 wk.	Sclerosis
1	100	36	350	876	1780	1400	Severe
2	100	36	290	1300	1000	1630	Moderate
3	100	60	310	912	1700	1900	Moderate
4	100	20	224	585	280	330	None
5	100	30	304	1920	2380	2260	Severe
6	100	38	394	1980	2280	2060	Severe
7	None	28	288	830	770	940	Severe
8	None	50	306	1150	1680	2200	Severe

All animals fed sclerosis-producing diet.

*Pancreatic Lipotropic Substance

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It will be noted in Table 1 that there was some degree of protection afforded fourteen out of nineteen rabbits, whereas there was severe sclerosis produced in all of fourteen control animals.

Table 2 shows the serum cholesterol levels of some of the control animals as well as of some of the animals that received the pancreatic lipotropic material as well as the degree of sclerosis in the animals.

The next experiment was an attempt to determine whether or not it would be possible to reduce the serum cholesterol levels of animals fed the sclerosis producing diet by increasing the dose of the lipotropic material. In an attempt to do this the animals were followed at weekly intervals and the dose of the lipotropic material was increased. The details are shown in Table 3.

It will be noted that there was a significant degree of protection of the animals in spite of the markedly elevated serum cholesterol levels. The reason for this is not as yet apparent, but a possibility is suggested in that the lipoid might have been held in a stable form in the plasma until it could be destroyed in the body instead of being deposited in the vessel walls. All analyses for cholesterol were done by a modified Schoenheimer Sperry[16] method, determining free and total cholesterol. There was no departure from the normal

Table 3

Animal	Control	1 wk.	2 wk.	3 wk.	4 wk.	5 wk.	6 wk.	7 wk.	8 wk.	9 wk.	10 wk.	11 wk.	12 wk.	Sclerosis
1	34	578	1550	1820	1840	2340	2370	1990						None
2	44	244	760	1000	1050	1080	1260	1220	1480	1580	1400	1025	960	Moderate
3	25	86	1190	1650	1920	2470	2570	2660	2770	2500	2180	2640	2050	Moderate
4	47	206	760	950	960	1000	1120	1260	1550	1740	2010	1560	1220	Severe
5	82	160	970	780	580	930	1350	1470	1490	1470	1440	1500	810	Moderate
6	31	132	620	580	750	1100	1350	1490	1780	1810	1540	1740	1170	None
P.L.S* (mgms.)		0	200	300	400	500	500	500	500	500	500	500	500	

*Pancreatic Lipotropic Substance

partition of the cholesterol.

Summary and Conclusion

1. A brief review of the relationship of cholesterol metabolism to the pathogenesis of atherosclerosis has been presented.

2. It seems safe to conclude that cholesterol is causally related to the pathogenesis of the disease.

3. A concept of atherosclerosis being the result of an underlying defect in cholesterol metabolism is evidenced by variable serum levels, elevated serum levels, or changes in the physical state of the lipoid is proposed.

4. The results of preliminary animal experimentation using rabbits as the experimental animal are as follows:

   a. Fourteen control animals fed a sclerosis producing diet all developed severe atherosclerosis.

   b. Fourteen out of eighteen animals receiving varying amounts of a pancreatic lipotropic material received a significant degree of protection against the development of atherosclerosis. This protection was afforded without lowering of serum cholesterol levels.

5. It seems safe to conclude that the pancreatic substance used contains something which is effective in preventing the development of atherosclerosis in rabbits fed cholesterol.

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III. MEDICAL SCHOOL NEWS

Psychotherapy For General Physicians

In April of 1946 an experiment in Postgraduate Medical Education was conducted in the Center for Continuation Study, University of Minnesota. The Medical School and the Commonwealth Fund presented a course in Psychotherapeutic Medicine. Twenty-five representative physicians from this area spent two weeks studying the meaning and value of the doctor-patient relationship, the development of personality, the significance of psychoneurotic behavior and the role of the physician in the prevention and treatment of simple everyday psychiatric problems. Although most of the student physicians were general practitioners, there were some specialists including internists, a dermatologist and a pediatrician. Outstanding teachers of psychiatry from our own faculty and other universities gave lectures, conducted seminars and conferences, and supervised practical clinical work of the student physicians in the Out-Patient Department of the University Hospitals. Personal follow-up interviews with the student physicians by members of the Commonwealth Fund staff and our faculty were conducted six to twelve months after the course was finished. Uniformly the physicians reported that the course had had a profound effect on their attitude towards all their patients but particularly towards those who had emotional problems. They were finding greater satisfaction in treating functional problems and were pleased with the results which they had obtained.

A second course in psychotherapy for general physicians, internists, pediatricians and other specialists outside of the field of psychiatry will be presented at the Center for Continuation Study September 20 to October 2. Dr. Donald Hastings, Head of the Department of Psychiatry and Neurology, will head up a staff of psychiatrists from Minnesota and elsewhere. Enrollment will be limited to a small number in order that supervised clinical work can be a part of the course.

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Dr. Flink Leaves For Study

Dr. Edmund B. Flink, Assistant Professor of Medicine, Admitting Physician to the University Hospitals, and Director of the Medicine Out-Patient Department, will leave for his sabbatical year in September, 1948. Dr. Flink will go to Boston, Massachusetts, where he will study at the Peter Bent Brigham Hospital and Harvard University Medical School. He will work in the laboratories of Drs. George Thorn and A. Baird Hastings. He will study certain fluid and electrolyte problems which relate to the endocrine gland. Dr. Flink will be sorely missed here at the University because of his great contribution to undergraduate and graduate medical education. In addition he has carried a heavy load of clinical responsibility in the Out-Patient Department rendering services to a large number of patients and maintaining excellent relations with physicians over our entire state.

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Dr. Gaylord Anderson Visits South America

Dr. Gaylord Anderson, Head of the School of Public Health, is at present in South America on a mission for the Division of International Exchange of Persons of the State Department. The greater share of his time will be spent in Sao Paulo, Brazil, where he is delivering a series of lectures on Public Health Administration. He will also make brief visits to schools of Public Health in San Juan, Puerto Rico; Rio de Janiero, Brazil; Buenos Aires, Argentina; Santiago, Chile; Lima, Peru; and Balboa, Canal Zone.

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Minnesota Medical Association Meets

The 95th Annual Meeting of the Minnesota State Medical Association will be held in the Minneapolis Auditorium June 7, 8, and 9. This promises to be an extraordinarily worthwhile meeting. All interns and fellows are invited to attend the scientific meetings and to view the scientific and commercial exhibits. Tickets are available in the office of Postgraduate Medical Education, 332M, University Hospitals.