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Coronary Embolization in Bacterial Endocarditis

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I. CORONARY EMBOLIZATION IN BACTERIAL ENDOCARDITIS

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Certain diseases affecting the coronary arteries, as intraluminal thrombosis, are well established clinical and pathological entities. Other abnormalities, as coronary embolism, have received comparatively little attention and, indeed, even the occurrence of such a phenomenon has been a matter of speculation by various writers.

Gallavardin and Dufourt[3], for example, in reporting a case in 1913, stated that embolism of the coronary arteries was one of the most exceptional of events. They attributed this to the angle at which the vessels left the aorta, their dimensions, and the speed and force of blood flow past the vessels. Coombs, however, in 1922, reported that the myocardial lesions in bacterial endocarditis are almost always due to emboli. This is in direct contrast to a statement by Hoseason[7], in 1935: "Coronary embolism is extremely uncommon; the probabilities are so strongly against an embolus of suitable size being swept into a coronary artery that it is remarkable that it should occur at all". Hoseason, in the same paper, in discussing the reasons for this seeming uncommon occurrence, wrote the following: "An important factor, no doubt, is that the rush of aortic blood sweeps any loose fragments well past the coronary openings which will be more or less masked by the aortic valve cusps. Also, the coronary circulation is unique in that it fills during diastole. A clot which has been dislodged probably sometime during systole will have passed well out of the way by the time the coronaries commence their diastolic filling from the residue of blood in the aorta".

The purpose of this paper is to review the literature concerning the frequency of coronary embolism in bacterial endocarditis and to report the study of eight successive cases of bacterial endocarditis seen in the Department of Pathology of the University of Minnesota between October, 1951 and July, 1952.

Literature

In 1933, Saphir[13] published an extensive historical review of the subject of coronary embolism at which time he compiled all reported cases beginning with one described by Virchow in 1856. He found a total of eleven acceptable cases and added three of his own. The author stressed the rarity of such an occurrence as judged from the number of reported cases. Certain obvious criteria, specifically the absence of intrinsic coronary disease and the presence of a source from which an embolus might arise, were emphasized by Saphir as being of value in separating thromboses from emboli.

Hamman and Rich[5], in 1933, reported two cases of bacterial endocarditis which presented interesting contrasts. The microscopic sections from one case, in which the vegetations were confined to the tricuspid valve, showed an essentially normal myocardium. In the other case, in which the vegetations were superimposed on a bicuspid aortic valve, microscopic sections of the myocardium revealed many small areas of scarring and necrosis, and the presence of masses of necrotic debris and cells within the small branches of the coronary arteries. These were similar, histologically, to the material present on the valve, and the authors stated, "This, clearly, represents embolism by a fragment of infected vegetation, and there is no doubt that the little scars in the myocardium are minute infarcts produced by such emboli". Perry, Fleming, and Edwards[12] have recently reported similar observations in a review of 52 cases from the Mayo Clinic, in which one case of endocarditis confined to the tricuspid valve failed to show myocardial lesions.

In 1935 Saphir[14], in describing the myocardial lesions in subacute bacterial endocarditis, reviewed 35 hearts from children and young adults. In all of these he found changes in the myocardium. These consisted of petechial hemorrhages, areas of necrosis and abscesses, minute

infarcts, perivascular cellular infiltrations, and Aschoff bodies. Of these changes, those described as minute infarcts were the most frequent, occurring in 28 of the 35 cases. Saphir attributed these to emboli arising from disrupted vegetations. Although an embolus was detected grossly in only one case, 18 of the cases showed microscopically the presence of embolic material in the lumina of the small branches of the coromary arteries. Saphir concluded by saying that small infarcts, produced in this manner, seem to be the most characteristic myocardial change in bacterial endocarditis. This, of course, represents a decided change of opinion from his previous (1933) paper on the subject.

In 1939, de Navasquez[10] reported the results of his studies of the hearts from 20 unselected cases of subacute bacterial endocarditis, in each of which the etiologic agent was Streptococcus viridans. The author states that "successive blocks" were cut from the left ventricle and interventricular septum. Microscopically, in 16 of these 20 cases (80%) he noted, "sharply defined masses lying loosely or impacted firmly within the lumen of the artery with or without accompanying changes in the wall and perivascular tissues". He noted the close resemblance of these masses to that of the valvular vegetations and in six of the 16 cases bacteria were demonstrated histologically within these emboli. Nine of the cases showed inflammatory changes of the arteriolar walls varying from slight arteritis to complete necrosis with fragmentation of the media. Serial sections of certain blocks were described as showing these lesions to be segmental and confined to that portion of the vessel containing the embolus. By measurement, it was found that the lesions were seldom longer than one millimeter.

Garvin and Work[14], however, in the same year, reviewed 12,300 consecutive autopsies at the Cleveland City Hospital (20 year period) and found only three cases of coronary embolism, each of which was associated with bacterial endocarditis. In each case the embolus was demonstrated grossly and was found in the lumen of a large branch of the coronary arteries.

Hamman[6], (1941) in discussing the incidence of coronary embolism, reported that the most frequent source was from bacterial vegetations on the heart valves and that, "this association is so frequently observed as to be almost a commonplace...". He estimated that of all cases of coronary occlusion, from 1% to 2% are due to embolism.

In this particular paper Hamman referred to 40 reported cases of coronary embolism, and listed six possible sources from which they might arise: a thrombus or atheromatous material within a coronary artery, a thrombus covering an arteriosclerotic plaque at the root of the aorta, bacterial vegetations upon the mitral or aortic valve leaflets, intracardiac mural thrombi, thrombi within the pulmonary veins, and thrombi within the peripheral veins (paradoxical embolism). Of these 40 cases the source of two was not specified, but of the remainder, 19, or 48%, had been reported as occurring in bacterial endocarditis.

Parks[11], in 1942, reported one case of coronary embolism in a 52 year old female with bacterial endocarditis. This embolus was detected grossly. It measured one centimeter in length, and lay within the lumen of the left coronary artery. The writer stressed the rarity of such an occurrence.

In the same year, Denman[2], reviewed the clinical characteristics and the autopsy findings in 50 cases of bacterial endocarditis, but made no mention of coronary emboli. It should be noted, however, that this paper was concerned with the frequency of valves involved and changes in other organs rather than with a detailed study of the myocardial lesions.

Latscha et al.[8], also in 1942, reported two cases of bacterial endocarditis in which the left coronary artery was occluded by emboli. In one, the embolus occluded the ostium of the vessel and in the other it was just below the ostium. The authors stated that generally the

left coronary artery is involved and only rarely are the small arteries or arterioles affected.

In 1946, Saphir[15] described the presence of "granulomas" in the myocardium of four patients who had received penicillin or a sulfa preparation in treatment for bacterial endocarditis. These were described as consisting of centers of dark bluish material which by the Von Kossa technique was found to be calcium. About the periphery of this calcific center were foreign body type giant cells and a few lymphocytes. No bacteria were demonstrated within the lesions. Sections from the involved heart valves showed the presence of necrotic material and bacteria at the periphery of the vegetations together with some calcium. Saphir thought that these "granulomatous" lesions were the result of the breaking off of particles of calcific material (emboli) from the valves; this deposition of calcium in the myocardium initiating the foreign body type of reaction. In one case he noted a similar type of lesion in the kidney. He again stressed the frequency with which coronary embolization occurs in bacterial endocarditis and emphasized the presence of minute infarcts as an indication of their occurrence.

Moragues, Bawell, and Shrader[9], in 1950, reported one case of coronary embolism in which the embolus consisted of calcific material similar to that seen on a leaflet of the aortic valve. The embolus measured three millimeters in length. The entire mass, as described from microscopic sections, was covered by endothelium and attached to the intima of the left coronary artery by endothelial covered connective tissue strands. There was no evidence of intimal atherosclerosis and no bacteria were noted either within the embolus or in sections taken from the aortic valve. Whether the patient had had previously treated bacterial endocarditis is not stated.

In another, more recent communication, (1950) Saphir, Katz, and Gore[16] described the histological changes in the myocardium in 76 cases of subacute bacterial endocarditis, two-thirds of the patients being under 40 years of age. Thirty-six of the cases are stated to have shown areas of ischemic necrosis in the myocardium. Of these, 17, or slightly less than 50%, showed the presence of emboli within small branches of the coronary arteries. The authors remarked that if the gross material had been available for further sectioning, the figures would almost certainly have been revised upwards. In 14 of the cases the emboli are described as being fibrinous with a few granulocytes and a surrounding perivascular leucocytic reaction. In three of the cases, the emboli were noted to contain calcium.

Perry, Flemming, and Edwards[12], in another recent publication (1952) reviewed 52 cases of subacute bacterial endocarditis in order to compare the changes found in the myocardium of treated and untreated patients. In their series, the most frequent finding was that of small myocardial infarcts, this being present in 47 (90%) of the cases. Microscopically, in 20 of the cases, emboli were noted within the small branches of the coronary arteries. These were described as being bland or septic, with the bland type predominating. In some of the cases they noted a "frank acute inflammation of the whole vessel wall", and this type of reaction was present in four cases in which they were unable to demonstrate emboli. This latter type of reaction they labeled "angiitis". In oen of the treated cases they noted a "granulomatous" type of lesion similar to that described by Saphir. Otherwise, there were no striking differences in the myocardial lesions between treated and untreated cases.

The pertinent findings concerning these reports are summarized in Table I. Certain overlapping of figures is almost inevitable because of sparcity of details in some reports, but it may be seen from the table that of all cases of coronary embolism reviewed, including both large and small branches of the coronary arteries (143 cases), 79% have been associated with bacterial endocarditis. It may also be noted that, in re-

TABLE I

SUMMARY OF REVIEWED CASES

AUTHOR	YEAR	CASES	INFARCTS		EMBOLI		ARTERITIS
			Large	Small	Large	Small	
Gallavardin	1913	1	1	--	1	--	--
Coombs	1922	20	--	1	--	3	3
Hoseason	1935	1	--	--	1	--	--
Saphir *	1933	14	3	--	14	--	--
Hamman	1933	2	--	1	--	1	--
Saphir	1935	35	--	28	1	18	11
de Navasquez	1939	20	1	--	1	16	9
Garvin	1939	3	--	--	3	--	--
Hamman **	1941	40	6	1	18	20	--
Parks	1942	1	1	--	1	--	--
Denman ***	1942	50	--	--	--	--	--
Latscha	1942	2	1	--	2	--	--
Saphir	1946	4	--	--	--	4	--
Moragues	1950	1	1	--	1	--	--
Saphir	1950	76	--	36	--	17	--
Perry	1952	52	--	47	--	21	4
TOTALS:							
A) All Above:		322	14	114	43	100	27
B) Endocarditis, omitting repetitions & statistical analyses.		227	3	113	33	80	27

References to Table I:

* Historical review of incidence of coronary embolism without specific references to gross or microscopic changes in myocardium except in author's own cases.

** General review of causes of coronary embolism without reference to myocardial changes. Source of two emboli not mentioned.

*** Statistical analysis rather than detailed study of myocardial changes.

lation to the total cases of bacterial endocarditis studied with respect to the occurrence of coronary emboli, these have been detected in slightly less than 50% of the cases.

If one limits the reported cases to those of bacterial endocarditis in which detailed studies of the myocardium were carried out (Saphir, de Navasquez, and Perry), 183 cases fall into this category. Of these, the most frequently reported alteration noted has been that described as the miliary infarct. It occurred in 111 cases (60%), while small emboli were detected in 72 of the cases (slightly less than 40%).

Material

The materials used in this study consisted of the hearts from eight successive cases of bacterial endocarditis which were seen at the University of Minnesota Department of Pathology between October, 1951, and July, 1952. The study was directed toward the gross and microscopic demonstration of emboli within the large and small branches of the coronary arteries, small in this paper referring to the direct, epicardial branches and not to the very small, intramural branches of the coronary arteries. No particular effort was made to demonstrate emboli within these latter branches.

Methods of study

Following the removal and opening of the heart at autopsy, the entire specimen was fixed in 10% formalin. The coronary arteries were then cut transversely at a distance of not more than three millimeters between sections with a sharp knife. This procedure frequently required from one and a half to two hours' time for a single specimen.

After gross sectioning and selection of those vessels which appeared to be occluded, the involved vessels were treated in the routine fashion and then embedded in paraffin. These blocks were then serially sectioned at seven micra and every fifth section was mounted. At the same time, routine blocks were cut from the myocardium and the involved valves; the former consisted of from three to five blocks from different areas depending on the gross appearance. All of these sections were then stained with hematoxylin and eosin in the usual manner and, in addition, certain sections of the valves and vessels were stained by the Gram-Weigert method.

Observations

The pertinent gross findings were summarized in Table II. Of particular interest is the fact that in all but one case (4) there were areas of scarring in the myocardium and, in one case (8) there were multiple areas of recent infarctions three to four centimeters in diameter. Two of the cases (1 & 2) also showed the presence of hard, calcific masses in the myocardium; these will be discussed with the histological findings. As may be noted in the table, emboli were detected in six of the cases, and in four of these cases (1,4,6 and 8) multiple occlusions were noted.

Microscopically, the sections taken from the myocardium showed definite alterations in every case. Areas of ischemic necrosis were noted in all but one case (4) and these showed varying degrees of resolution and replacement fibrosis (Fib. 1). These changes were particularly prominent and variable in those cases in which multiple emboli were detected grossly (1,6 and 8) and in one additional case (5) in which emboli were noted within the intramural branches of the coronary arteries (Fig. 2). In two cases there were also multiple abscesses in the myocardium. (cases 4 and 8). Areas of recent necrosis of the muscle fibers, associated with granulocytic and mononuclear cellular infiltration were noted in four of the cases. In case 8, in particular, these areas were quite numerous. No particular attempt was made to determine the presence of Aschoff bodies in the myocardium and consequently, small perivascular areas of scarring were not given special emphasis.

In two of the cases, as noted previously (cases 1 & 2) calcific densities, or granulomas as they have been called by other writers, were noted within the

TABLE II

GROSS PATHOLOGICAL FINDINGS

Case	Age & Sex	Heart Weight In Grams	Valvular Involvement	Myocardium	Emboli	Other
1	16, Male	900	Mitral Aortic	Large areas of scarring.	+	Rheumatic mitral and aortic valves.
2	37, Male	700	Pulmonary Mitral Aortic	Small scars	+	Rheumatic pulmonary, mitral, aortic valves. I.V. septal defect.
3	26, Female	350	Mitral Aortic	Petechiae. Large areas of scarring	-	Mild rheumatic mitral and aortic valves.
4	56, Male	460	Mitral	Perforated. No gross areas of fibrosis	+	Cardiac tamponade. Valves not grossly rheumatic.
5	32, Female	275	Aortic	Large scars	+	Mild rheumatic mitral and aortic valves. Gastric carcinoma.
6	54, Female	420	Aortic	Small scars	+	Valves not grossly rheumatic. Pancreatic carcinoma.
7	25, Female	440	Mitral, perforated leaflet	Petechiae Large scars	-	Mild rheumatic mitral valve.
8	50, Male	740	Aortic	Petechiae. Fresh areas of infarctions.	+	Valves not grossly rheumatic. Portal cirrhosis.

myocardium. In one of these cases (2) this lesion was quite large and was located near the endocardial surface. Its exact relationship to a vessel could not be determined and there was little or no cellular reaction about the calcium (Fig. 3). By the Gram-Weigert method, bacteria were noted in clumps about the periphery of this lesion. In the other case, however, it was possible by serial sections to demonstrate the relationship of the calcium to a vessel and particles of this material were found in the lumen of the vessel as well as in an extravascular location within the myocardium. In this lesion bacteria were present in large numbers and there was a marked cellular reaction which consisted of neutrophiles, lymphocytes, plasma cells, and a few multinucleated giant cells (Fig. 4).

It seems reasonable to conclude that this may represent an earlier stage of the previously described lesion, and that they both resulted from embolic occlusion of branches of the coronary arteries by particles of calcified vegetations from a valve leaflet, since in both instances sections from the valves revealed similar material. In both of these cases penicillin therapy has been employed.

Microscopically, none of the vessels showed any significant degree of atherosclerosis, and a study of the serial sections of the vessels showed them to contain material which was similar to that on the valve. These emboli were designated as being bland or septic, (Figs. 5 & 6) although it must be admitted that the distinction in some cases was more or less arbitrary and was dependent on which particular section one chose and the manner in which the

vessel was sectioned. In three of the cases bacteria were noted within the emboli. These were in large clumps in two instances (6,7,10,11).

In one case (4) which received ACTH and cortisone the emboli were septic and there was a pronounced arteritis with partial necrosis of the vessel wall (Fig. 7). In two other cases varying degrees of arterial inflammation were noted and in one (case 8) it was particularly striking, with almost complete necrosis of a portion of the media (Figs. 8 and 9).

In many instances, sections revealed what appeared to be a sudden distention of the lumen of the vessel when the largest diameter of the embolus was reached, and this was particularly striking when the embolus lodged near a bifurcation of a vessel. It was noted that this material stopped rather sharply at the point of devision; not extending down into the junctional vessel for any appreciable distance (Fig. 10).

In three of the cases (1,5 and 6) there was evidence of organization of the emboli and in case 1, particularly, there was a marked degree of normal appearing media beyond the limits of the embolus. Cross sections of a similar vessel showed that organization was progressing from all portions of the intima in contrast to the organization of a thrombus associated with atherosclerosis.

Table III summarizes the salient histological findings in these cases.

The clinical histories and diagnoses are summarized in Table IV. It is of interest that in 50% of the cases there

TABLE III

MICROSCOPIC FINDINGS

Case	Granulomas	INFARCTS				EMBOLI				Arteritis
		Recent	Old	Large	Small	Calcific	Bland	Septic	Bacteria	
1	+	+	+	+	+	+	+	-	+	+
2	+	-	+	-	+	-	-	+	-	-
3	-	+	+	+	+	-	-	-	-	-
4	-	-	-	-	-	-	-	+	+	+
5	-	-	+	+	+	-	+	-	-	-
6	-	+	+	+	+	-	+	-	-	-
7	-	+	+	+	+	-	-	-	-	-
8	-	+	+	+	+	+	-	+	+	+
TOTALS:	2+ 6-	5+ 3-	7+ 1-	6+ 2-	7+ 1-	2+ 4-	3+ 3-	3+ 3-	3+ 3-	3+ 5-

TABLE IV

CLINICAL SUMMARY

Case	Diagnosis	History	EKG	Blood Culture	Drugs Received	Death
1	Rheumatic heart disease Bacterial endocarditis	Rheumatic fever	Tachycardia. Auricular fibrillation. Right and left ventricular strain.	Sterile	Penicillin Digitoxin	Sudden
2	Congenital heart disease Mitral stenosis	Rheumatic fever	None	None	Penicillin Digitoxin	Sudden
3*	Rheumatic heart disease Bacterial endocarditis	Rheumatic fever	Prolonged PR. Depression $ST_{1\&2}$ $V_{4\&6}$	Strep. Fecalis, X 2	Penicillin Aureomycin Terramycin	Progressive
4	Pemphigus Bacterial endocarditis	------	None	Coagulase positive Staph.	(ACTH) Penicillin Streptomycin Dicumerol	Sudden
5	Undetermined	Thirteen months postpartum	None	None	No specific therapy	Sudden
6	Diabetes Portal Cirrhosis	Diabetes & hypertension for 2 years	Abnormal No specific T wave changes	None	No specific therapy	Progressive
7*	Rheumatic heart disease Bacterial endocarditis	Rheumatic fever	L. ventricular strain	Staph. Aureus	Penicillin Streptomycin Aureomycin Bacitracin Digitoxin	Sudden
8	Portal Cirrhosis Bacterial endocarditis	------	A-V Dissoc. Premature ventricular contr.	Species of Staph.	Penicillin Streptomycin Aureomycin Digitalis	Progressive

* Emboli not detected

was historical evidence of previous rheumatic heart disease. As may be noted, six of the patients received penicillin and four of these received one or more drugs in addition. Also, four of the cases showed clinical evidence of cardiac decomposition and were digitalized.

No correlation could be noted between the occurrence of bland and septic emboli as regards treatment, though it is of interest that in one case (4) which has been referred to previously, in which ACTH and cortisone were given during the course of the disease, the emboli were septic and were associated with a pronounced arteritis (Fig. 7). In this particular case the inflammatory reaction apparently progressed rapidly, with myocardial perforation and cardiac tamponade.

There were no constant changes in the electrocardiograms which would lead one to suspect that occlusion of a branch of a coronary artery had occurred.

In five of the cases death appeared to be of a sudden nature, and in one case (5) in particular, was the direct basis for postmortem examination.

Summary and Conclusions

Eight cases of bacterial endocarditis which have been studied from the standpoint of coronary embolization have been presented. In six of these, emboli were demonstrated grossly in the epicardial branches of the coronary arteries, and in four of these multiple occlusions were noted. Gross changes were noted in the myocardium in all but one case. These consisted of petechial hemorrhages, scarring, and areas of recent infarctions. These latter two findings stand in direct contrast to most reported observations. Two cases demonstrated the presence of calcific masses in the myocardium, which have been called 'granulomas' by other writers.

Microscopically, in four of the cases, emboli were noted in the intramural branches of the coronary arteries in addition to those described grossly, and small and large areas of ischemic necrosis were noted in all but one case. The latter finding is in accord with most reported cases, but contrasts sharply with the report of Coombs[1], who stated that he examined 274 sections from the left ventricle in a single case of bacterial endocarditis and found only 20 small areas of fibrosis; and with de Navasquez[10], who found only one infarction in a series of 20 cases in which he reported detailed histological alterations in the myocardium.

It is believed that coronary embolization in bacterial endocarditis is a common occurrence and, indeed, probably the most common complication of the disease. As regards the various theories concerning the improbability of the occurrence of emboli to these vessels, it seems more reasonable to conclude that the location of the arteries, the mechanics of the blood flow past them, and the manner of opening of the aortic valve leaflets, aptly predispose them to embolization in this condition or in any other in which small particles are free in the blood stream emitted from the left ventricle.

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II. MEDICAL SCHOOL NEWS

Coming Events

November 13-15 Continuation Course in Fractures and the Surgery of Trauma for General Physicians

November 21 J. B. Johnston Lectureship in Neurology; "Hypophysectomy in Man," Prof. Herbert Olivecrona, Professor of Neurosurgery, Stockholm, Sweden, Museum of Natural History Auditorium; 8:00 p.m.

November 21-22 Continuation Course: Conference on Pemphigus and the Bullous Dermatoses for Dermatologists

December 4-6 Continuation Course in Endocrinology for General Physicians

December 5 Journal-Lancet Lecture; "Some Studies on Experimental Diabetes," Dr. Dwight J. Ingle, Senior Research Scientist, Research Division, The Upjohn Company, Kalamazoo, Michigan; Owre Amphitheater; 8:00 p.m.

December 15-17 Continuation Course in Gynecology for Specialists

* * *

Homecoming Program

Last week's Homecoming Program for Physicians was attended by more than 44 medical alumni. Dr. Russell Spittler, Lodi, California, travelled the greatest distance. Visitors and staff alike enjoyed Dr. Sheppard's remarks on "The Medical School in Relation to Medical Practice in the State" and the Homecoming Clinics were well received. At the meeting of the Minnesota Medical Alumni Association a new slate of officers was chosen: President, Dr. Harold Benjamin, Minneapolis; Vice-Presidents, Doctors William C. Bernstein, St. Paul, and Cyrus O. Hansen, Minneapolis; Secretary, Dr. Sheldon Lagaard, Minneapolis; and Treasurer, Dr. Donald Lannin, St. Paul. Named to the Executive Committee were Doctors O. M. Heiberg, Worthington; Dr. Herman E. Drill, Hopkins; Dr. Byron Cochrane, St. Paul; and Dr. Robert J. Tenner, Minneapolis.

* * *

Minnesota Medical Foundation Meeting

On Friday, October 31, the Board of Trustees of the Minnesota Medical Foundation held a dinner meeting at the Curtis Hotel. Two of the new members of the Board, Doctors Moses Barron of Minneapolis and Herman E. Drill of Hopkins, attended their first meeting. The other new member, Dr. Raymond F. Hedin of Red Wing, was unable to be present. The Board expressed its thanks to those members retiring this year, Doctors William Hanson, Charles F. Code, and Russell J. Moe for their interest in and outstanding contributions to the objectives of the Foundation.

The Board selected officers for the coming year. All of the current year's officers were re-elected: President, Owen H. Wangensteen; Vice-President, Francis W. Lynch; and Secretary-Treasurer, Wesley W. Spink.

* * *

Minnesota Alumnus Honored

One of Minnesota's most distinguished alumni received an outstanding achievement award medal at the meeting of the University Board of Regents which was held on Saturday, November 1. Dr. Raymond B. Allen, Chancellor of the University of California and formerly President of the University of Washington was presented the award by President Morrill on behalf of the regents for his "far-reaching accomplishments as medical scientist, university administrator, and formulator of top level policy in the service of international enlightenment and democracy." His many friends in Minnesota know that this award was well deserved and join in extending congratulations to him.

* * *

III. MINNESOTA MEDICAL FOUNDATION

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IV. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

November 10 - 15, 1952

Monday, November 10

Medical School and University Hospitals

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; W-612, U. H.

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

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom; Todd Amphitheater, U. H.

11:30 - 12:30 Physical Medicine Seminar; Heart Hospital Auditorium.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:30 Physiology Seminar; The Theoretical Inadequacy of the Fick Principle for Practical Measurements of Cardiac Output; Maurice B. Visscher; 214 Millard Hall.

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

4:00 - 5:30 Seminar on Fluid and Electrolyte Balance; Gerald T. Evans; Todd Amphitheater, U. H.

4:00 - 5:00 Pediatric Seminar; Epidemiology of Streptococcal Infections; Lewis Wannemaker; Sixth Floor West, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

4:30 - Public Health Seminar; 12 Owre Hall.

4:30 - 6:00 Physiology 114A and Cancer Biology 140 -- Research Conference on Cancer, Nutrition, and Endocrinology; Drs. Visscher, Bittner, and King; "Rhythm Studies," F. Halberg; 129 Millard Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Eldon Berglund; Newborn Nursery, Station C.

10:30 - 12:00 Medical Rounds; Thomas Lowry and Staff; Station F.

Monday, November 10 (Cont.)

Minneapolis General Hospital (Cont.)

11:00 - Pediatric Rounds; Erling Platou; Station K.

11:45 - Grand Rounds; Fractures; Dr. Henrikson, et al; Station A.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. E.

2:00 - Pediatric Rounds; Robert A. Ulstrom; Stations I and J.

Ancker Hospital

8:30 - 10:00 Chest Disease Conference.

1:00 - 2:00 Medical Grand Rounds.

Veterans Administration Hospital

8:00 - 9:00 Neuroradiology Conference; J. Jorgens, R. C. Gray; 2nd Floor Annex.

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; J. Jorgens, Conference Room, Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

3:30 - Psychosomatic Rounds; C. K. Aldrich; Bldg. I.

Tuesday, November 11 (HOLIDAY)

Wednesday, November 12

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

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

12:30 - 1:20 Radioisotope seminar; Use of Radioisotopes in Control of Body Cavity Effusions; Robert Bronson; 110 Botany Building.

1:30 - 3:00 Physiology 114B -- Circulatory and Renal System Problems Seminar; Dr. M. B. Visscher, et al; 214 Millard Hall.

4:00 - 5:30 Physiology 114C -- Permeability and Metabolism Seminar; Nathan Lifson; 214 Millard Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

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

Wednesday, November 12 (Cont.)

Medical School and University Hospitals (Cont.)

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

2:00 - 4:00 Medical Ward Rounds;

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Max Seham; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

11:00 - Pediatric Rounds; Erling S. Platou; Station K.

12:30 - Pediatric Conference; Oral Manifestation of Systemic Disease in Children; Dale Cumming; Station I, Classroom.

1:30 - Visiting Staff Case Presentation; Station I, Classroom.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

2:00 - 4:00 Infectious Disease Rounds; Main Conference Room, Bldg. I.

4:00 - 5:00 Infectious Disease Conference; W. Spink; Conference Room, Bldg. I.

4:00 - Combined Medical-Surgical Conference; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, November 13

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

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

Thursday, November 13 (Cont.)

Medical School and University Hospitals (Cont.)

12:30 - Physiological Chemistry Seminar; Endocrine Regulation of Amino Acid Metabolism; Mickey Goldfine; 214 Millard Hall.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

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

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 X-ray Seminar; Report of American Cancer Society Clinical Meeting; Donn G. Mosser; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Ancker Hospital

4:00 - Medical Pathological Conference; Auditorium.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station I.

10:00 - Pediatric Rounds; Spencer F. Brown; Station K.

10:00 - Psychiatry Grand Rounds; J. C. Michael and Staff; Sta. H.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

1:00 - Fracture - X-ray Conference; Dr. Zierold; Classroom.

1:00 - Pediatric House Staff Conference; Station I.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

Friday, November 14

Medical School and University Hospitals (Cont.)

8:00 - 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.

Friday, November 14 (Cont.)

Medical School and University Hospitals (Cont.)

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

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

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Some Recent Studies of the Urobilin Problem; Paul T. Lowry and C. J. Watson; Powell Hall Amphitheater.

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

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Physiology 124 -- Seminar in Neurophysiology; Ernst Gelhorn; 113 Owre Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Wallace Lueck; Station J.

10:30 - Pediatric Surgery Conference; Oswald Wyatt; Tague Chisholm; Station I., Classroom.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:15 - X-ray Conference; Oscar Lipschultz; Classroom, Main Building.

2:00 - Pediatric Rounds; Robert Ulstrom; Stations I and J.

Veterans Administration Hospital

1:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

Saturday, November 15

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

Saturday, November 15 (Cont.)

Medical School and University Hospitals (Cont.)

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

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

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

11:30 - Anatomy Seminar; Development of the Human Diaphragm and Pleural Sacs; L. J. Wells; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - 12:00 Medical - X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - 11:15 Hematology Rounds; Drs. Hagen, Goldish, and Aufderheide
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide