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Poliomyelitis and Aseptic Meningitis

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I. POLIOMYELITIS AND ASEPTIC MENINGITIS

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The recent introduction of tissue culture methods[1] for the *in vitro* propagation of the poliomyelitis viruses has made available a relatively simple procedure for their isolation from clinical specimens and for the estimation of poliomyelitis antibodies in human and other serum samples.

Monkey kidney cells and a strain of human epithelial cells (HeLa) have been used extensively for these purposes. The latter cells have been in continuous culture on glass in our laboratory since 1952, shortly after this cell line was isolated from a cervical carcinoma by Gey. These cells are highly susceptible to the poliomyelitis viruses.[2,3] The poliomyelitis viruses grow rapidly in HeLa cells, rise to high titer and destroy tube cultures within 1 to 7 days depending on the amount of virus inoculated. The infected cells round up and fall off the glass, a phenomenon easily seen with the low power of the microscope. For isolation of virus,[3] feces, throat washings and other material suspected of containing virus are centrifuged to remove bacteria and inert material and the supernatant fluids after treatment with antibiotics, are inoculated into tubes containing HeLa cells. The tubes are inspected daily for the presence of virus, and if there, its identity proved by passing it to a series of 8 tubes, 3 pairs of which contain anti-serum prepared against the 3 types of poliomyelitis virus and one of which acts as the control for virus growth. The isolated virus will destroy the cells in all tubes except those containing its homologous antibody. This neutralization test serves to type the poliomyelitis viruses isolated and to separate them from other cytopathogenic agents which may occasionally be present in the material and which can also multiply in and destroy HeLa cells. Antibodies can be titrated in these cultures by adding different dilutions of serum to a small quantity of virus (25-100 $T.C.D._{50}$); the titer is the highest dilution which protects the cell from the cytopathic effect of this dose of virus. One of the problems which we are now studying by these techniques is the relation of non-paralytic poliomyelitis to "aseptic meningitis."

"Aseptic Meningitis"

Aseptic meningitis was the name given by Wallgren in 1925[4] to a syndrome he saw frequently in his Stockholm practice. He laid down 6 criteria for the diagnosis of this syndrome which he considered a nosological entity: 1) an illness with signs of meningitis as the main clinical finding; 2) a pleocytosis predominantly lymphocytic; 3) the absence of bacteria in the cerebrospinal fluid; 4) a short and benign course; 5) the absence of "neighborhood" infections in the middle ear, sinuses or nasopharynx, and 6) independence of epidemics or contact with known diseases producing the same symptoms. The diseases that were excluded in 1925 were poliomyelitis, mumps and leptospirosis. Since then many agents have been discovered which can cause this syndrome and in 1951 the same author[5] listed over 20 viruses and other microorganisms capable of producing aseptic meningitis. Of these agents, the viruses of poliomyelitis, lymphocytic choriomeningitis, arthropodal borne encephalitides, herpes simplex, mumps and the Coxsackie group and leptospirae are of particular concern to the present study since these are known to occur in Minnesota and its neighboring states.

Poliomyelitis in Minnesota

Poliomyelitis has been prevalent

for many years in the North Central states where it has recurred in epidemic proportions almost annually. The strains of poliomyelitis viruses recovered in our laboratory from patients since 1946 are arranged in Table I to show by years the distribution of types as compared with the number of notifications of the disease. Epidemics occurred in 1946, 1948, 1949, 1952, and 1953; the incidence of disease was much less in 1947, 1950, 1951 and 1954. Type 1 poliomyelitis virus was dominant in epidemic years; in some non-epidemic years, type 1 was no more frequent than one or both of the other types. It is of interest that the comparative numbers of paralytic and non-paralytic cases were reasonably constant with paralytic disease slightly more common, especially in 1946 when 2.2 paralytic cases occurred for each non-paralytic case. The incidence of disease as reported to the State Health Department would suggest that most non-paralytic cases were indeed poliomyelitis. This point is mentioned since it is accepted that the seasonal occurrence of poliomyelitis may result in a clinical diagnosis of non-paralytic poliomyelitis for any of a variety of aseptic meningoencephalitides which are not notifiable. That this mistake in diagnosis may occur is supported by our findings during the 1953 epidemic. These findings are summarized in part in this paper and will be reported in full elsewhere.[7]

Table I

Virus Isolations from Cases of Poliomyelitis

Minnesota 1946-54

Year	Total Number of Notified Cases	Relation of Paralytic to Non-paralytic Cases	Viruses Isolated		
			Type 1	Type 2	Type 3
1946	2881	2.2	7	0	0
1947	201	1.4	--	--	--
1948	1387	1.0	59	0	0
1949	1715	1.1	9	11	0
1950	502	1.4	21	1	32
1951	511	1.1	12	2	1
1952	3926	1.1	77	0	8
1953	2137	1.2	385	1	8
1954 Jan.-Oct.	624	0.9	35	34	32

Isolations of virus and antibody studies for 1953

Table I shows that 394 poliomyelitis viruses were isolated during 1953. Of these, 318 were recovered from 513 patients admitted with a clinical diagnosis of poliomyelitis to the

Elizabeth Kenny Institute from June through December. Since 314 strains were Type 1 and 4 were Type 3, the epidemic for 1953 is representative of Type 1 infection.[6] Of the 513 patients studied, 302 had paralytic and 211 non-paralytic disease. Virus was recovered from 78 per cent of all paralytic patients and from 42 per cent of all non-paralytic cases. This apparent discrepancy in the recoverability of virus from non-paralytic and paralytic cases is similar to the reduction in isolation rate reported for epidemics in New Haven and Lockhaven[8] where the percental recovery from non-paralytic disease and paralytic disease was 57 per cent and 86 per cent, and in Boston 42 per cent and 98 per cent respectively.[9] Our studies for 1954 have shown for this year that no immunologic type of poliomyelitis predominates and that isolations from non-paralytic cases are also much less frequent than from paralytic disease. The reason for this difference is not as yet known. Two possibilities under investigation are that a) patients with the non-paralytic form excrete less virus or a less virulent strain of virus, and b) the difference in isolation rates indicates that agents other than poliomyelitis virus share responsibility for much disease in the non-paralytic category. Evidence in support of the latter possibility has resulted from studies completed and under way.

The established predominance of Type 1 infection led to studies of the antibody levels to each of the three types of poliomyelitis virus in paralytic and non-paralytic patients from whom this virus was isolated, in a group of non-paralytic patients from whom no virus was recovered and for control purposes, in a group of healthy medical students. The results of this study are summarized in Table II and Table III. The distribution by groups of titers to the predominant Type 1 virus (Table II) demonstrated no difference in Type 1 antibody for Groups A and B from which virus was isolated; 84 per cent had a titer of 1:640 or more. In contrast to these findings, comparable titers were shown for only 43 per cent of Group C from whom no virus was recovered and for only 10 per cent in the normal control group. Since corollary studies have shown that recent poliomyelitis infection results in a higher titer than a remote infection, it was assumed from the comparative titer levels found for Groups A and D that the number of patients in Groups B and C combined with poliomyelitis Type 1 would be approximately 60 per cent. Thus, it appears that 40 per cent of the non-paralytic cases represent disease due to an agent other than this poliomyelitis virus. Evidence to rule out poliomyelitis infection in these cases was the findings that serum from 9 of the non-paralytic cases had no detectable antibody to Type 1 and that serum samples from 6 had no antibodies for any type. Moreover, all four groups of patients showed essentially the same distribution in antibody titer to Types 2 and 3 (Table III). This observation made it unlikely that poliomyelitis virus, Types 2 or 3, was responsible for more than an occasional case of poliomyelitis in 1953. This conclusion was probably valid since no Type 2 strains and only 4 strains of Type 3 were recovered from the 513 patients from The Elizabeth Kenny Institute, 99 per cent of the strains of virus isolated being Type 1.

Clinical observations on non-paralytic cases

A comparison of the clinical history and symptoms was made of 74 non-paralytic patients of whom 47 yielded virus and 27 did not. The cases studied serologically were included in this group. Since there are no pathognomonic signs of non-paralytic poliomyelitis the diagnosis can never be made clinically with absolute certainty. The patients that exhibit headache, fever, stiff neck and/or backache and moderate pleocytosis during the poliomyelitis season are generally diagnosed as non-paralytic poliomyelitis. The cases so diagnosed in this series had at least three of the four

Table II

Comparative Poliomyelitis Antibody Levels, Type 1, for Paralytic and Non-paralytic Cases and Normal Controls.

Minnesota 1953

	A Paralytic cases with Type 1 virus isolated	B Non-paralytic cases with Type 1 virus isolated	C Non-paralytic cases with no virus isolated	D Healthy medical students
Total	42	13	35	50
Number with a serum titer of 1) 1:640 or more	34	12	15	5
2) 1:160 or less	8	1	20	45
Number with no Type 1 antibody	0	0	9	24
Number with no antibody to all types.	--	--	6	9

Table III

Comparative Poliomyelitis Antibody Levels, Types 2 and 3, for Paralytic and Non-paralytic Cases and Normal Controls.

Minnesota 1953

	A Paralytic cases with Type 1 virus isolated	B Non-paralytic cases with Type 1 virus isolated	C Non-paralytic cases with no virus isolated	D Healthy medical students
Total	42	13	35	50
Number with a serum titer to Type 2 of				
1) 1:640 or more	10	2	7	11
2) 1:160 or less	32	11	28	39
Number with no Type 2 antibody	22	6	15	30
Number with a serum titer to Type 3 of				
1) 1:640 or more	8	5	8	6
2) 1:160 or less	35	8	27	44
Number with no Type 3 antibody	26	8	19	26

Table IV

Symptoms and Signs Observed for 74 Patients
Diagnosed Clinically as "Non-paralytic Poliomyelitis"

	Polio Virus Isolated			Polio Virus Not Isolated		
	Total	Positive	%	Total	Positive	%
Fever	47	47	100	27	27	100
Headache	42	35	83	27	24	89
Stiff neck	47	42	89	27	25	93
Pleocytosis	47	46	90	27	27	100
Duration of fever in days	40	4.75		27	4.82	
Sensory phenomena	47	30	64	27	10	37
A. Abdominal pain	47	16		27	8	
B. Chest pain	47	1		27	1	
C. Hyperesthesia	47	3		27	0	
D. Pain in ext.	47	10		27	1	
E. Hypesthesia	47	0		27	0	
Drowsiness or lethargy	47	28	60	27	9	33
Dizziness	47	8	17	27	9	33
Nausea and vomiting	47	20	43	27	0	
Transient weakness	45	6	13	27	4	15
Photophobia	47	1	2	27	2	7
Febrile prodrome	47	22	47	27	10	37
Exposure to polio	46	11	24	27	3	11

CLINICAL LAB. FINDINGS	Total	Range	Average	Total	Range	Average
# of days after onset of CNS symptoms tap done	46	1-9 days	2.9 days	26	1-5 days	2 days
Cell count - Spinal fluid	46	1-415	119	27	12-1382	236
Protein	29	8-86	36.59	20	11-146	48.7
White blood count	47	5,000-17,400	9100	27	5-14,000	8,800

parts of this syndrome. Fever commonly was less than 104° F. (R) and lasted an average of 4.75 days. Pleocytosis (10-1382 cells) was present in 98% of the patients, (average, 118 cells). Stiffness of the neck and back was usually less rigid than that observed with bacterial meningitis but persisted for a longer period of time, (average, 23 days).

Less common but nontheless frequent findings among confirmed cases of non-paralytic poliomyelitis were the following in the order of their frequency. Pains in extremities, chest or abdomen; drowsiness and lethargy; febrile prodrome; nausea and vomiting; history of exposure to polio; dizziness; transient weakness of less than 30 days duration.

Our attempts thus far to find clinical differences between patients with and without virus in their stools have been unsuccessful (Table IV). It is our feeling that attempts to be too rigid on clinical grounds will result in the exclusion of potentially severe pre-paralytic cases. Twenty-one per cent of the 1953 respirator patients were admitted with the non-paralytic syndrome. Shaw[10] contends that non-paralytic poliomyelitis is a relatively rare disease (3.9% of 798 cases of polio) if careful effort to find muscle weakness is made and if other similar diseases are excluded on clinical grounds. Since in 1953 virus was isolated from 88 patients in whom a physiatrist could find no signs of muscle weakness, at least 17% of these 513 cases must have been instances of non-paralytic poliomyelitis.

Other evidence bearing on the relation of poliomyelitis and aseptic meningitis

In the last two decades evidence has accumulated to indicate that the role of poliomyelitis in the incidence of the syndrome of aseptic meningitis varies considerably with season, annual prevalence, and geographic location. Since "aseptic meningitis" is not a notifiable disease, opinions of its incidence result from the few series of cases reported from hospital admissions and from receipts of specimens at laboratories. Most surveys under this title have included all illnesses diagnosed as aseptic meningoencephalitis, serous meningitis, lymphocytic meningitis and encephalitides with a lymphocytic pleocytosis. Meningitis is usually dominant, but evidence of an associated encephalitis results commonly in the latter label. It is suggested that "lymphocytic meningoencephalitis" designates better this widespread and common syndrome since all the viruses that produce 'aseptic meningitis' also give rise to a varying degree of encephalitis which occasionally produces discernible motor and sensory changes. However, "aseptic meningitis" is widely used and understood.

It is helpful to an understanding of the significance of a clinical diagnosis of nonparalytic poliomyelitis to review recent studies. Three reports from Sweden[11-13] indicate no relationship between the incidence of poliomyelitis and aseptic meningo-encephalitis, but the epidemiology of this latter condition differed from one community to another. In Gothenburg from 1932-1950 the number of hospital admissions for this syndrome in all age groups varied little by season or year.[11] Its incidence was not related to that of poliomyelitis, mumps, influenza, or encephalitis lethargica. The comparative incidence of this latter disease fell to an occasional case whereas aseptic meningo-encephalitis rose slowly from 50 cases per annum in the 1920's to 200 in the late 1940's. In Lund patients were admitted to hospitals throughout the year[12] in contrast to Stockholm[13] where from 1949-1952 a large increase in hospital admissions for meningo-encephalitis occurred during the summer and autumn of each year. In 1949 and 1950 when poliomyelitis epidemics occurred, the total

number of cases of aseptic meningitis increased only slightly as compared with 1951 and 1952 when paralytic poliomyelitis was infrequent. These figures also indicated that in Stockholm the 'aseptic meningitic' season preceded that of poliomyelitis by 6 weeks or so. In Manchester[14] during 1951 aseptic meningitis was widespread and more frequent than in 1952 although the number of cases of paralytic poliomyelitis was only two thirds that reported for 1950 and 1952 (Table V).

The age distribution of aseptic meningo-encephalitis is of interest. It was at least as frequent in adults as in children in Stockholm while in Gothenburg adult cases predominated in the ratio of 4 to 1. In the Manchester series there were 152 adult cases to 139 in children during 1951-2. In the Lund series, all the patients were children and there did seem to be a small increase in the number of cases in July through August which also occurred in the children in Gothenburg.

Table V

Relation of Paralytic Poliomyelitis (P) to Aseptic Meningo-encephalitis (A.M.)
Manchester area 1951-2

Month	1951		1952	
	P	A.M.	P	A.M.
J	1	2	1	11
F	1	6	0	8
M	0	4	0	7
A	1	8	1	10
M	1	11	6	16
J	3	23	3	9
J	4	17	3	14
A	3	23	8	11
S	4	29	6	11
O	2	28	4	8
N	1	10	3	8
D	2	5	1	12
Total	23	166	36	125

In 1951, in the Manchester epidemic of aseptic meningitis which occurred from July through October, more children were involved than adults. Of the cases of non-paralytic poliomyelitis admitted to the Elizabeth Kenny Institute in 1953 half were adults and a similar proportion has been found this year. The percental isolation of poliomyelitis virus from these cases in those over ten years of age was less than half that in those under ten, being only 23% as compared with 58%, suggesting that this infection played a more important role in aseptic meningitis in children than in adults. Thus, it appears that aseptic meningitis behaves independently of poliomyelitis and epidemiologically may differ somewhat in children and adults. Similarly, the proportion of other agents causing this syndrome varies in the two age groups.

Surveys of the comparative role of the causative agents of aseptic meningoencephalitis have been limited to the viruses of lymphocytic choriomeningitis, mumps and herpes simplex, and to leptospirosis. Adair, et al.,[16] in a survey of cases drawn from the Army and from the Veterans Administration, a group consisting mostly of young adults, found 8.1% due to lymphocytic choriomeningitis, 9.4% to mumps, 5.3% to herpes simplex and 7.6% to leptospirosis. In Manchester[14] the percentage of aseptic meningitis in adults due to the three viruses was 13.2, 1.4, 1.7, respectively, while in children under 15 it was 2.9, 3.1 and 3.2. In England, as a whole,[17] leptospirosis accounts for about 8% of all cases of this syndrome, while mumps and lymphocytic choriomeningitis cause about 3% and 10%. In Sweden, mumps and herpes simplex are responsible for about 10% and 7% of all cases.[11] Lymphocytic choriomeningitis or leptospirosis have not been implicated in aseptic meningitis in Sweden.

The seasonal incidence of these four agents varies. Although all occur throughout the year, in the United States[16] lymphocytic choriomeningitis is most prevalent towards the end and at the beginning of the year, mumps from February to May, leptospirosis around August,[18] but distribution may vary with locality and year. Lymphocytic choriomeningitis in Manchester occurs throughout the year with a low peak in the early summer. Mumps meningitis occurs whenever mumps is prevalent, 24% of all clinical cases of this disease having meningeal involvement while 8% do not suffer concurrent parotitis or orchitis.[11] The majority of these patients are not hospitalized while laboratory aids to diagnosis are sought only in those cases with no parotitis or in whom it appears after the onset of meningitis. Leptospirosis in Switzerland[19] and Great Britain[20] is prevalent throughout the year with its lowest incidence early in the year or in the Spring and with a low peak in the summer and fall.

In contrast to the occurrence of meningitis elsewhere, most of the Minnesota cases of aseptic meningitis coincide with the poliomyelitis season and the agents discussed above, except perhaps leptospirae, would not explain this distribution. The known agents found in this area and limited to this time of the year are the Coxsackie viruses and the two arthropodal borne encephalitides, Western equine encephalomyelitis and St. Louis encephalitis.

There is a reasonable amount of evidence to incriminate the B group of the Coxsackie viruses with the production of aseptic meningitis.[21,22] They are the causal agents of Bornholm disease or epidemic myalgia which is associated in a few instances with meningeal symptoms. Although these viruses have been isolated on only two occasions from the C.S.F. of patients,[22,23] investigators have reported in patients a rise in neutralizing antibody titer to these viruses and the isolation of virus from stools concurrently with the signs of meningitis. The investigations in Sweden[13] could indicate that these viruses were the chief cause of the aseptic meningitis

of summer and autumn there. However, in England during 1951 when Bornholm disease was widespread the causative Coxsackie strains were not held responsible for the large number of cases of aseptic meningitis which occurred that year.[14,15] In the Manchester district, the Bornholm epidemic and the isolation of "C" virus took place before the aseptic meningitis epidemic reached its peak. In Minnesota, only 3 strains, all group A, were isolated from the 78 cases tested in 1953, so they could have been responsible for only a few, if any, of the cases since the evidence for involvement of the group A Coxsackie viruses in this syndrome is not so convincing as for the B group. "C" viruses were isolated more frequently from children than adults with aseptic meningitis in Sweden.[13]

Western equine encephalomyelitis (WEE) and perhaps the St. Louis type occur in Minnesota during the late summer and fall as a seasonal disease. Serological evidence implicates the virus of WEE as responsible for 2% of all cases of aseptic meningitis in this area this year.[26]

The majority of the Minnesota cases whether definitely poliomyelitis or not were clinically indistinguishable one from the other. Clinically, a large proportion of cases of lymphocytic choriomeningitis and leptospirosis can be differentiated from others of the aseptic meningitides. From a third[16] to two thirds[24] of patients with lymphocytic choriomeningitis have a prodromal flu-like disease for from 1 to 3 weeks before the signs of meningitis intervene, a third have a low glucose (<45 mg%) and some show transient radiological changes in the lungs. This disease tends to incite a high lymphocytic pleocytosis, many patients having counts of over 1000 in the C.S.F. which is rare in the other forms of aseptic meningitis. There is also the association of infected house mice and of occurrence of cases in localized areas.[25] Leptospirosis when it presents primarily as aseptic meningitis is more often the result of infection with L. canicola, L. pomona or L. mitis than with L. icterohaemorrhagiae. In the first phase of the disease, pleocytosis is not observed usually; the rise in lymphocytes occurring in the second part of this often diphasic disease. Marked injection of the conjunctivae, albumen and red cells in the urine, and a pulse rate which does not parallel the temperature are common to leptospirosis but are not usually seen in non-paralytic poliomyelitis.[19]

In mumps, herpes simplex infection, and Bornholm disease the meningitis may be associated with parotitis, stomatitis, or myalgia but all, particularly herpes, may produce meningitic signs without the classical criteria of these infections. Herpes simplex tends to result in fever of longer duration than poliomyelitis and it is associated more frequently than the other agents with severe central nervous system symptoms.[11]

Three of our patients in 1953 had streptococcal throat infections with a rise in the neutrophils in the blood to 15,000 or more. Although this cell count may occur in WEE and leptospirosis, the lymphocytic meningitis in these cases must be considered as a 'neighborhood' reaction to local infection rather than due to an encephalitogenic agent.

None of our patients had a history of exposure to any of the exanthemata or whooping cough or had been vaccinated recently, so that post-infectious encephalitis was not important in the differential diagnosis of meningoencephalitis as it was in Lund. Other causes of this syndrome include the encephalomyocarditis group of viruses, arthropod-borne diseases, other than WEE and St. Louis, torulosis, toxoplasmosis and infectious mononucleosis. There was no evidence that this last disease played any part in our cases either in 1953 or 1954. In the differential diagnosis of aseptic

meningitis, tuberculosis and syphilis must also always be borne in mind.

Recently, cytopathogenic agents other than poliomyelitis (Orphan virus)[8] have been isolated in tissue culture by different workers from feces of non-paralytic poliomyelitis cases. Some of these were Coxsackie viruses, but others may be new viruses capable of causing aseptic meningitis. The role in this syndrome of these viruses and of other human and animal viruses may be considerable in certain areas.

This year we intend, with the help of Dr. H. Bauer's laboratory, to examine all patients with non-paralytic poliomyelitis admitted to the E.K.I. for laboratory evidence of infection with the poliomyelitis, Coxsackie, mumps, lymphocytic choriomeningitis, herpes simplex, WEE and St. Louis viruses and with leptospirae. This comprehensive study has not been done elsewhere and the 1954 material available to us seems admirably suited for this purpose. By these means, we hope to throw more light on the relationship of poliomyelitis and other agents to 'aseptic meningitis' in this area.

Conclusions and Summary

In our laboratory studies on 513 patients with poliomyelitis in Minnesota in 1953, isolation of virus was made in 78% of 302 paralytic patients and in 42% of 211 diagnosed as non-paralytic poliomyelitis. From serological investigations of these patients, it was concluded that of the non-paralytic cases about 60% were due to infection with poliomyelitis virus while 40% were caused by something else. The difference in the percental number of isolations of poliomyelitis virus in the two clinical categories could not be adequately explained as variations in amount or virulence of virus, but was rather an indication of a difference in its distribution in the two groups.

The role of lymphocytic choriomeningitis, herpes simplex, mumps, Coxsackie virus infection and leptospirosis in aseptic meningitis was discussed both from the epidemiological and clinical aspect, and related to the findings in our 1953 series of cases.

The relative importance of these viruses in this syndrome at different ages was noted, and it was pointed out that in our series, poliomyelitis was a much more important causal agent in children than in adults.

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II. MEDICAL SCHOOL NEWS

Coming Events

January 6 - 8	Continuation Course in Pediatrics for General Physicians
Jan. 31 - Feb. 2	Continuation Course in Ophthalmology for Specialists

* * *

Continuation Course

The University of Minnesota will present a continuation course in Pediatrics for General Physicians next January 6 to 8, 1955, at the Center for Continuation Study. This year's program will stress the recognition and management of the many and varied urinary tract problems seen in youngsters. Guest speaker will be Dr. Henry L. Barnett Associate Professor, Department of Pediatrics, The New York Hospital-Cornell Medical Center, New York City, an outstanding authority in this field. The program will be presented under the direction of Dr. Irvine McQuarrie, Professor and Head, Department of Pediatrics, University of Minnesota Medical School.

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Faculty News

On November 20 Dr. Donald W. Hastings, Professor and Head, Department of Psychiatry and Neurology, attended the Board meeting of the Corporate Foundation for Research and Training in Psychiatry which was held in New York City.

Dr. and Mrs. A. B. Baker have recently returned from a two-month visit to Norway where Dr. Baker gave a series of lectures under the auspices of the University of Oslo and the University of Bergen.

Congratulations are in order to Dr. Richard Teeter who has been promoted from Medical Fellow to Instructor in the Division of Psychiatry.

Dr. Wallace D. Armstrong, Professor and Head, Department of Physiological Chemistry, has been elected an honorary member of the Swedish Dental Association.

Dr. Cecil J. Watson, Professor and Head, Department of Medicine, was the guest speaker at the meeting of the Society of Graduate Internists of the Los Angeles County Hospital from November 12 to 14. He spoke on "The Clinical Significance of the Porphyrins."

Doctors James B. Carey, Jr., Mary Goepfert, and Ananda Prasad attended the November 5 meeting of the Society for Experimental Biology and Medicine in Rochester, Minnesota. Dr. Carey presented a paper entitled "Isolation and Identification of Desoxycholic Acid and other Bile Acids from Human Feces and Urine."

Dr. Ernst Simonson, Associate Professor, Laboratory of Physiological Hygiene, has been named a corresponding member of the German Physiological Society.

Doctors F. H. Van Bergen and D. Stuart Weatherhead of the Division of Anesthesiology attended the annual meeting of the American Society of Anesthesiologists held at Cincinnati on October 25 to 29. During the Fifth Annual Refresher Course which followed the annual meeting, Dr. Van Bergen presented a lecture entitled "The Anesthesiologist in the Care of Tetanus and Polio Cases." On November 10 Dr. Van Bergen presented his inaugural thesis entitled "Hexamethonium-Induced Hypotension" before a regular meeting of the Minnesota Academy of Medicine at the Town & Country Club. The Division of Anesthesiology was host on November 27 to Dr. George Thomas, Director of the Department of Anesthesiology at the St. Francis Hospital in Pittsburgh. Dr. Thomas gave a lecture and accompanying demonstration on "Fire and Explosion Hazards in the Hospital."

Publications of the Medical School Faculty

Bloom, David, Swigart, R. H., Scherer, W. F., and Glick, David: Studies in Histochemistry XXX. A Study by Phase Contrast Microscopy of Cytological Effects of Freezing-Drying Procedures on Cultured Fibrobrasts and Guinea Pig Tissues. J. Histochem. & Cytochem., 2: 178, 1954.

Brumfield, Helene P. and Ross, J. D.: A Sheep Erythrocyte Lysin Found in Guinea Pig Serum. J. Immunol., 72: 489, 1954.

Chandler, H. C., French, L. A., and Peyton, W. T.: Surgical Treatment of Metastatic Tumors of the Spine. Ann. of Surg., 140: 197, 1954.

French, L. A. and Watson, J. C.: Angiography Versus Pneumoencephalography. The X-Ray Technician, 25: 251, 1954.

French, L. A. and Scholtis, Genevieve: A Neurosurgical Instrument Table. J. Neurosurg., 11: 430, 1954.

French, L. A. and Peyton, W. T.: Vascular Malformations in the Region of the Great Vein of Galen. J. Neurosurg., 11: Sept., 1954.

Gifford, G. E., Robertson, H. E., and Syverton, J. T.: Application of Manometric Method to Testing Chemical Agents in vitro for Interference with Poliomyelitis Virus Synthesis. Proc. Soc. Exp. Biol. & Med., 86: 515, 1954.

Howard, R. B., Balfour, W. M., and Cullen, R.: Extreme Hyperferremia in Two Instances of Hemochromatosis with Notes on the Treatment of One Patient by Means of Repeated Venesection. J. Lab. & Clin. Med., 43: 848, 1954.

Kubicek, W. G. and Kottke, F. J.: Vasometer Nerve Stimulation and Mechanical Obstruction Factors in Renal Hypertension. Am. J. Physiol., 178: No. 2, Aug., 1954.

Lasser, E. C. and Rigler, L. G.: Observation on the Structure and Function of the Ileocecal Valve. Radiol., 63: 176, 1954.

Madden, J. F.: Tryptar in Enzymatic Debridement of Indolent Infected Cutaneous Ulcers. The Medical Clinics of North America, 38: 359, 1954.

Madden, John F.: Malignant Melanoma. A.M.A. Archives of Derm. & Syphilology, 69: 106, 1954.

Oneson, I. B. and Cohen, S. L.: Conjugated Steroids V. Hydrolysis of Total Ketosteroids in Urine. Proc. Soc. Exp. Biol. & Med., 86: 728, 1954.

Prasad, A. S. and Koza, D. W.: Agammaglobulinemia. Ann. Internal Med., 41: 629, 1954.

Quiggle, A. B., Kottke, F. J., and Magney, Jean: Metabolic Requirements of Occupational Therapy Procedures. Arch. Phys. Med. & Rehab., Sept., 1954.

Rigler, L. G. and Olfelt, P. A.: Abdominal Aortography for the Roentgen Demonstration of the Liver and Spleen. Am.J. Roentgenology, Radium Therapy, & Nuclear Med., 72: 586, 1954.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

December 13 - 18

Monday, December 13

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 Hitchcock, Zimmermann, and Stenstrom; Todd Amphitheater, U. H.

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

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

1:30 - 3:30 Dermatology Hospital Rounds; H. E. Michelson and Staff; Dermatology-Histopathology Room, C-394 Mayo Memorial.

4:00 - 6:00 Anesthesiology Conference; F. H. Van Bergen and Staff; Room 100, Mayo Memorial.

4:30 - Pediatric-Medicine Infectious Disease Rounds; Station 33, U. H.

5:00 - 6:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

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

Ancker Hospital

8:00 - 9:00 Pediatrics Contagion Rounds; L. R. Critchfield; Contagion 5.

8:30 - 10:30 Medical and Surgical Chest Conference; Dr. Gehlen and Staff; Auditorium.

10:00 - 12:00 Surgery Grand Ward Rounds; Begin Floor E4.

11:00 - 12:00 Medicine Resident Rounds.

11:00 - 12:00 Pediatric Rounds; Harry Orme; Contagion 1.

12:30 - 2:30 Surgery Out-Patient Clinic; Room 8.

2:00 - 3:00 Routine EKG Interpretation; Dr. Sommers and House Staff; Medical Record Library.

2:30 - 3:00 Discussion of Problem Case; Auditorium.

3:00 - 4:00 Surgery Journal Club; Classroom.

3:00 - 4:00 Lectures on Electrocardiography; Ben Sommers; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Richard Raile; Station K.

10:30 - 12:00 Medicine Rounds; Thomas Lowry; Station F.

Monday, December 13 (Cont.)

Minneapolis General Hospital (Cont.)

11:00 - Orthopedic and Fracture Rounds; Drs. John Moe and Arthur Zierold; Station B.

11:00 - Pediatric Seminar; Erling Platou; Classroom, Station M.

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

1:30 - 2:30 Tuberculosis Conference; J. A. Myers; Station M.

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

Veterans Administration Hospital

9:30 - Infectious Disease Rounds; Drs. Hall, Zinnemann, and Middlebrook.

1:30 - Cardiac Conference; Drs. Smith, Berman, Hoseth, Simonson, Swerdlow, Shapiro, and J. Brown; Conference Room, Bldg. I; Rounds immediately following conference.

Tuesday, December 14

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, Irvine McQuarrie and Staffs; Eustis Amphitheater, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 104 Jackson Hall.

12:30 - Physiology Seminar: Transport; Nathan Lifson; 129 Millard Hall.

3:30 - General Physiology Seminar; 323 Zoology Building.

3:30 - Pediatric Seminar; 1450 Mayo Memorial.

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

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases from Minneapolis General Hospital; Drs. Lipschultz and Paciotti; Eustis Amphitheater, U. H.

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Edward Strem; Contagion 1.

8:00 - 10:00 Visiting Staff Rounds.

9:00 - 12:00 Practical Diagnostic Clinic; Harry Orme; Out-Patient Department.

11:00 - 12:00 Medical X-ray Conference; Auditorium.

4:00 - 5:00 Medical-Pathological Conference; W. F. Mazzitello, Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Elizabeth Lowry; Station J.

10:00 - Psychiatry Grand Rounds; R. W. Anderson, Station H.

10:00 - Cardiac Rounds; Paul F. Dwan; Classroom, Station I.

11:00 - 12:00 Medicine-Surgery Conference; Classroom, Station M.

Tuesday, December 14 (Cont.)

Minneapolis General Hospital (Cont.)

12:30 - 2:30 Dermatology Rounds on Clinic; Carl W. Laymon and Staff.

12:30 - ECG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:00 - Tumor Clinic; Drs. Eder, Coe, and Lipschultz; Classroom.

3:30 - Pediatric-Psychiatry Rounds; Jack Wallinga; Station I.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Surgical Conference Room, Bldg. 43.

8:30 - Hematology Rounds; Drs. Hagen and Wexler.

8:30 - Surgery Journal Club; Conference Room, Bldg. I.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery-Tumor Conference; D. Ferguson and J. Jorgens.

1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

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

4:00 - Thoracic Surgery Problems; Conference Room, Bldg. I.

5:00 - Fluid Balance Conference; Conference Room, Bldg. I.

5:30 - Physiology Seminar; Surgical Conference Room, Bldg. 43.

Wednesday, December 15

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-Pediatrics Conference; Todd Amphitheater, U. H.

12:30 - 1:20 Radio-Isotope Seminar; Betatron Room in Cobalt Underground Section.

1:00 - 2:00 Dermatology Clinical Seminar; F. W. Lynch; 300 North Clinic.

1:30 - 3:00 Pediatric Allergy Clinic; Albert V. Stoesser and Lloyd Nelson; W-211, U. H.

3:30 - 4:30 Dermatology-Pharmacology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:30 - 5:50 Dermatology-Infectious Disease Seminar; 3rd Floor, Conference Room, Heart Hospital.

5:00 - 6:00 Residents Lectures; X-ray Circuits; John C. Watson; Todd Amphitheater, U. H.

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

5:30 - 7:30 Dermatology Journal Club and Discussion Group; Hospital Dining Room.

7:30 - 9:30 Dermatology Seminar; Review of Interesting Slides of the Week; Robert W. Goltz; Todd Amphitheater, U. H.

Wednesday, December 15 (Cont.)

Ancker Hospital

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

9:00 - 10:00 Contagion Rounds; L. R. Critchfield; Contagion 5.

11:00 - 12:00 Medicine Resident Rounds.

1:30 - 2:30 Pediatric Rounds; Ray Anderson; Contagion 1.

3:00 - 5:00 Infectious Disease Rounds; W. W. Spink; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Henry Staub; Station I.

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

12:00 - Surgery-Physiology Conference; Arthur Zierold and E. B. Brown; Classroom.

12:15 - Pediatrics Staff Meeting; Classroom, Station I.

1:30 - Pediatric House Staff Seminar; Erling Platou; Station I.

1:30 - Pediatric Rounds; Erling Platou; Classroom, Station I.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Surgical Conference Room, Bldg. 43.

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

9:00 - Gastro-Intestinal Rounds; Drs. Wilson, Zieve, Ferguson, Brakel, Konig, and Swenson.

10:30 - Psychosomatic Conference; C. K. Aldrich; 7th Floor, Bldg. 43.

12:30 - Medical Journal Club; Doctors' Dining Room.

12:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg. I.

1:30 - 3:00 Metabolic Disease Conference; Drs. Flink and Latts.

3:30 - Urology Pathology Slide Conference; Dr. Gleason;Conference Room, Bldg. I.

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

Thursday, December 16

Medical School and University Hospitals

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Room 3.148; Mayo Memorial.

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

12:30 - 1:30 Endocrine Seminar; Neurohumoral Mechanisms, Enteramine; F. Halberg; 271 Lyon Laboratories.

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

Thursday, December 16 (Cont.)

Medical School and University Hospitals (Cont.)

4:00 - 5:00 Anesthesiology Seminar; F. H. Van Bergen and Staff; Room 100, Mayo Memorial.

5:00 - 6:00 Radiology Seminar; Eosinophilic Granuloma; Leo Blank; Eustis Amphitheater, U. H.

7:30 - 9:30 Physiology 114A Seminar; Hemodynamic Problems; M. B. Visscher and Robert Evans; 271 Lyon Laboratories.

Ancker Hospital

8:30 - 9:30 Medical Grand Rounds; Auditorium; Visiting Staff Rounds immediately following Grand Rounds.

11:00 - 12:00 Medicine Resident Rounds.

2:00 - 3:00 Routine ECG Interpretation; Ben Sommers; Medical Record Library.

Minneapolis General Hospital

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

9:30 - Pediatric Contagion Rounds; R. B. Raile; Station K.

10:00 - Psychiatry Grand Rounds; R. W. Anderson and Staff; Station H.

11:30 - 12:30 Clinical Pathological Conference; John I. Coe; Classroom.

12:30 - 2:30 Dermatology Rounds and Clinic; Carl W. Laymon and Staff.

1:00 - Fracture X-ray Conference; Drs. Zierold and Moe; Classroom.

1:00 - House Staff Conference; Station I.

Veterans Administration Hospital

8:00 - Experimental Surgery Laboratory Meeting; Conference Room, Bldg. I.

8:30 - Hematology Rounds; Drs. Hagen and Williams.

9:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

9:00 - Surgery Ward Rounds; D. Ferguson and Staff; Ward 11.

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

1:00 - Infectious Disease Conference; Wesley W. Spink; Conference Room, Bldg. I. (Rounds immediately following conference.)

4:00 - 5:00 Medical-Surgical Conference; Medical Conference Room, Bldg. I.

Friday, December 17

Medical School and University Hospitals

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.

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

10:30 - 1:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

Friday, December 17 (Cont.)

Medical School and University Hospitals (Cont.)

11:00 - 12:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

11:45 - 12:50 University of Minnesota Hospitals Medical Staff Meeting; Football Highlights; Mayo Auditorium.

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

1:30 - 2:30 Dermatology Grand Rounds; Presentation of Cases from Grouped Hospitals (University, Ancker, General and Veterans) and Private Offices; H. E. Michelson and Staff; Eustis Amphitheater, U. H.

2:30 - 4:00 Dermatology Hospital Rounds; H. E. Michelson and Staff; Begin at Dermatological Histopathology Room, C-394 Mayo Memorial.

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

3:30 - 4:30 Dermatology-Physiology Seminar; 3rd Floor Conference Room, Heart Hospital.

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

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

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Edward Strem; Contagion 1.

11:00 - 12:00 Contagion Rounds; Harry Orme; Contagion 5.

3:00 - 4:00 Medical-Surgical-Pathological Conference; Auditorium.

4:00 - 5:00 Medical Journal Club; Conference Room, E5.

4:00 - 5:00 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Elizabeth Lowry; Station J.

10:30 - Pediatric Surgical Conference; Tague Chisholm and B. Spencer; Classroom, Station I.

12:00 - Surgery-Pathology Conference; Drs. Zierold and Coe; Classroom.

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

1:30 - Pediatric Contagion Rounds; L. Wannamaker; Station K.

Veterans Administration Hospital

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

12:30 - Urology X-ray Conference; X-ray Department.

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

2:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

Saturday, December 18

Medical School and University Hospitals

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

9:00 - 9:30 Pediatric Grand Rounds; Eustis Amphitheater, U. H.

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 Rounds; J. L. McKelvey and Staff; Station 44, U. H.

Ancker Hospital

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

9:30 - 11:00 Medicine Grand Ward Rounds.

Minneapolis General Hospital

8:00 - Urology Staff Conference; T. H. Sweetser; Main Classroom.

9:00 - Psychiatry Grand Rounds; R. W. Anderson; Station H.

9:30 - Pediatric Rounds on all Stations; R. B. Raile.

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 - Medical X-ray Conference; Conference Room, Bldg. I.