VOLUME XXIX

UNIVERSITY OF MINNESOTA

Medical Bulletin

OFFICIAL PUBLICATION OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
THE MINNESOTA MEDICAL FOUNDATION
AND THE MINNESOTA MEDICAL ALUMNI
ASSOCIATION

IN THIS ISSUE:

University of Minnesota Medical Bulletin

UNIVERSITY OF MINNESOTA

Medical Bulletin

OFFICIAL PUBLICATION OF THE UNIVERSITY OF MINNESOTA HOSPITALS, MINNESOTA MEDICAL FOUNDATION, AND MINNESOTA MEDICAL ALUMNI ASSOCIATION

VOLUME XXIX March 15, 1958 NUMBER 10

CONTENTS

Published semi-monthly from October 15 to June 15 at Minneapolis, Minnesota

Staff Meeting Report

Epidemic of ECHO 9 Aseptic Meningitis in Minnesota, 1957*

Herman Kleinman, M.D.†, David Rogers, M.D.‡, Paul M. Ellwood, Jr., M.D.§, Heinz Bruhl, M.D.¶, Leonard M. Schuman, M.D.**, and Henry Bauer, Ph.D.††

The term "aseptic meningitis" was originally applied to reactions of the central nervous system thought to be of nonspecific or allergic etiology. Wallgren,[1] in 1925, formulated a set of criteria to differentiate this syndrome from specific infections also causing meningeal symptoms, such as syphilis, tuberculosis, helminthiasis, leptospirosis, poliomyelitis, mumps, and typhoid fever. Wallgren's criteria are these:[2]

1. Acute onset with obvious signs and symptoms of meningeal involvement.
2. Alteration of cerebrospinal fluid typical of meningitis; the cerebrospinal fluid may show a small or large number of cells.
3. Absence of bacteria in the cerebrospinal fluid as demonstrated by appropriate direct or culture techniques.
4. Relatively short and benign course of illness.
5. Absence of local parameningeal infection (otitis, sinusitis, trauma, etc.), or of a general disease which might present meningitis as a secondary manifestation.
6. Absence from the community of epidemic disease of which meningitis is a feature.

Improved laboratory diagnostic techniques developed recently have shown that the acute presenting picture described by Wallgren was, indeed, a non-specific syndrome attributable to a number of

* This report was given at the Staff Meeting of the University of Minnesota Hospitals on February 21, 1958.
† Chief, Section of Chronic Diseases, Minnesota Department of Health
‡ Epidemic Intelligence Service Officer, Communicable Disease Center, Atlanta, Georgia
§ Assistant Professor, Department of Pediatrics, University of Minnesota; and Director of Inpatient Service, Elizabeth Kenny Institute, Minneapolis
¶ Assistant Professor, Department of Pediatrics, University of Minnesota; Faribault State School and Hospital
**Associate Professor of Epidemiology, School of Public Health, University of Minnesota
††Director, Division of Medical Laboratories, Minnesota Department of Health

etiological agents, including the organisms responsible for poliomyelitis, mumps, measles, chickenpox, the arthropod-borne encephalitides, herpes simplex, lymphocytic choriomeningitis, leptospirosis, tuberculosis, syphilis, and (in this "antibiotic era") partially treated bacterial meningitis.

In a large group of such cases, however, etiology remained undetermined until very recently. But accumulated evidence now makes it possible to assign to these cases as etiological agents certain viruses that are presently known collectively as the enteroviruses. These enteroviruses and the human diseases with which they are associated are listed in Table 1.[3] Each group of these viruses includes some types that have been implicated in the aseptic meningitis syndrome.

TABLE 1

ASSOCIATION OF ENTEROVIRUSES WITH HUMAN DISEASE

Enteroviruses	*Associated Disease*
Poliovirus	Paralysis (complete to slight muscle weakness) Aseptic meningitis Undifferentiated febrile illness, particularly during the summer
Coxsackie viruses, Group A	Herpangina Undifferentiated febrile illness, particularly during the summer Aseptic meningitis (Types A7, A9)
Coxsackie viruses, Group B	Aseptic meningitis Pleurodynia Myocarditis or encephalomyocarditis during neonatal period and early childhood Mild paralysis (?)
ECHO viruses	Aseptic meningitis (Types 2, 3, 4, 5, 6, 9, 14, 16) Summer rash (Types 4, 9, 16*) Summer febrile illness Mild paralysis (?) (Type 6) Summer diarrhea of infants and children (Type 18 and others)

*Neva[8] has found the etiologic agent of Boston exanthem to be antigenically related to ECHO virus Type 16.

In Minnesota in 1955, the aseptic meningitis syndrome was due almost entirely to poliovirus. In 1956, while poliovirus was still operative, there were 75 cases in which Coxsackie B5 virus could confi-

dently be named the etiological agent.[4] And in 1957 in Minnesota, there occurred an epidemic of aseptic meningitis, due this time to another virus – ECHO 9. It is with this epidemic that the present paper is concerned.

Beginning on July 1, 1957, the Minnesota Department of Health began to list cumulatively the cases that fell into the general category of aseptic meningitis, some with rash and some without. These cases had been reported as suspected nonparalytic poliomyelitis, suspected encephalitis, or aseptic meningitis. Some cases were reported as "Boston" exanthem or as "unknown exanthematous disease." A certain number had been gleaned from requests by physicians for "virus disease" laboratory studies on specimens submitted. These

TABLE 2

INCIDENCE OF ASEPTIC MENINGITIS AND PARALYTIC POLIOMYELITIS, MINNESOTA, 1957, BY WEEK OF ONSET

	Aseptic Meningitis		*Paralytic Poliomyelitis*	
	Number of Cases	*Cumulative Total*	*Number of Cases*	*Cumulative Total*
Week ending:				
			3 (prior)	
July 6, 1957	3	3		3
" 13	9	12		3
" 20	23	35	1	4
" 27	57	92	2	6
August 3, 1957	99	191		6
" 10	125	316	2	8
" 17	81	397	3	11
" 24	46	443	2	13
" 31	30	473	2	15
September 7, 1957	36	509	1	16
" 14	37	546	4	20
" 21	18	564	2	22
" 28	14	578	3	25
October 5, 1957	19	597		25
" 12	11	608	1	26
" 19	9	617		26
" 26	8	625		26
November 2, 1957	5	630		26
" 9	3	633		
" 16	4	637	1	27
" 23	3	640		
" 30	3	643		27
December 7, 1957	5	648		
" 14	2	650	1	28
" 21	3	653		
" 28	5	658		
Total	658	658	28	28

CUMULATIVE INCIDENCE, ASEPTIC MENINGITIS AND PARALYTIC POLIO
Minnesota, 1957

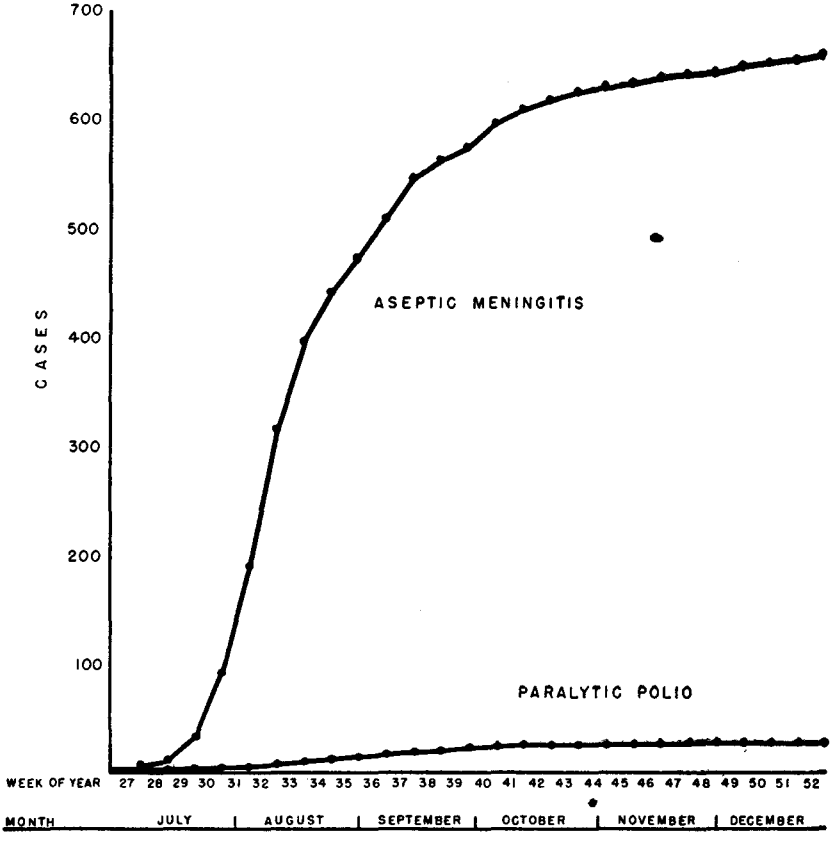

Fig. 1

cases, it was established by telephone query, exhibited the same general character as all the others.

Table 2 shows the incidence week by week since July 1, 1957, by dates of onset as well as cumulatively. Figure 1 depicts graphically this accumulating incidence of cases of aseptic meningitis and compares it with the cumulative incidence of paralytic poliomyelitis. The wide divergence of these two curves as they ascend is prima-facie evidence, at least, that the majority of nonparalytic cases did not represent poliomyelitis.

The epidemic curve shown in Figure 2 is typically prosodemic, with a peak during the week ending August 10.

EPIDEMIC CURVE, ASEPTIC MENINGITIS
Minnesota, 1957

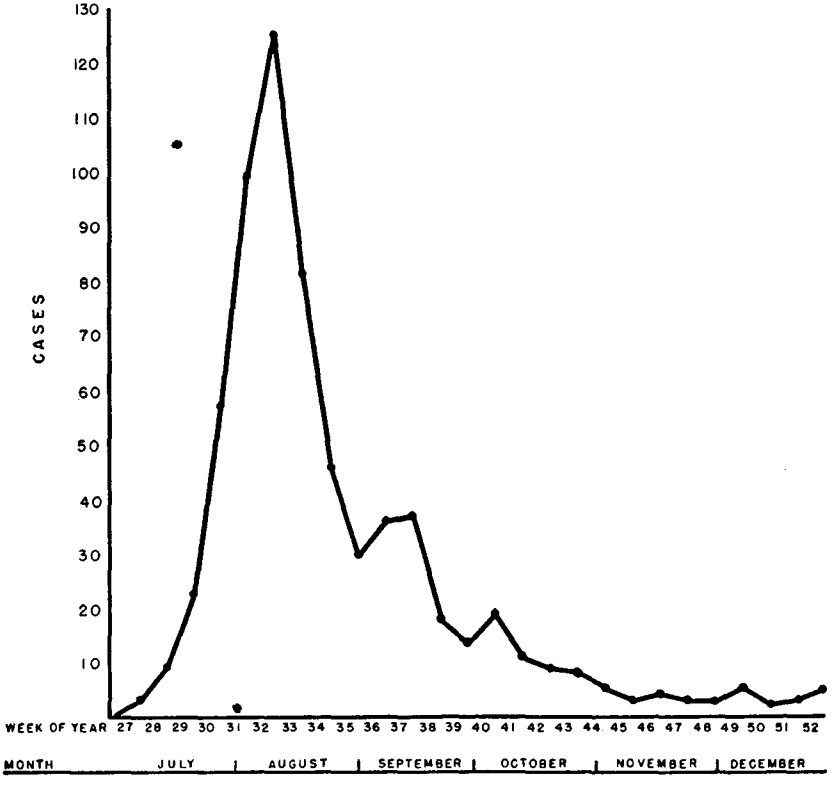

Fig. 2

Early in August, the influx of patients into various hospitals became so pressing, and the curiosity of physicians became so acute, that the health departments of Minnesota and of Minneapolis and St. Paul were impelled to issue an informational statement to physicians throughout the state.* The memorandum, which was issued

*They were aided in the preparation of this statement by Dr. Paul Ellwood of the Elizabeth Kenny Institute and by Dr. Richard Raile, Chief of Pediatric Service at Minneapolis General Hospital.

on August 13, is reproduced below as a concise summary of the essential clinical features of the disease:

Memorandum Re: ASEPTIC MENINGITIS
(VIRAL MENINGO-ENCEPHALITIS ASSOCIATED WITH RASH)

To: ALL PHYSICIANS

Throughout Minnesota, there has been occurring for the past few weeks an outbreak of a rather unusual syndrome which we would like to call to your attention.

The following impressions are based largely upon clinical impressions of the staffs at Minneapolis General Hospital and Elizabeth Kenny Institute where somewhat over 100 cases have been observed, and of reports from physicians throughout the State.

The disease apparently manifests itself in a wide variety of symptoms and varies greatly in its severity and duration.

The fever is generally low grade, rarely exceeding 102°, and lasts approximately 5 days. However, many cases with several separate spikes of fever have been noted.

The patients complain of generalized malaise, nausea, severe frontal headache, painful neck and back. Sore throat is occasionally mentioned. Very few patients report gastro-intestinal symptoms other than vomiting.

On examination, approximately one-third of the patients often have a maculopapular rash especially prominent over the abdomen and the flexor surfaces of the arms. This rash may be the most prominent finding. However, in families reporting several members ill, the rash may occur in only one or two persons.

In those patients with more severe headache there is often other evidence of CNS involvement, notably stiffness of the neck and back to such a degree that suspicion of nonparalytic polio or bacterial meningitis may occur. Bradycardia and respiratory irregularities occur in rare instances. Lumbar punctures reveal cell counts ranging from 20 to 1500 cells, predominantly mononuclears. Sugars have been normal, proteins occasionally slightly elevated. Leukopenia is observed in about one-third of the cases.

Although most of the cases seen to date are quite mild and without sequelae, we are of the opinion that aseptic meningitis of this type is most difficult to differentiate from nonparalytic polio. These patients should be carefully followed to rule out possible paralytic polio.

The presence of a rash would tend to exclude the diagnosis of polio. However, the rash should not be confused with that of the occasional meningococcemia.

It is our opinion that most of these cases are sufficiently mild to be managed in the home. Antibiotics have not been effective in altering the course of the illness. In giving symptomatic treatment caution should be exercised in the use of barbiturates, narcotics and other respiratory depressants since respiratory arrest has been observed in at least one case.

An etiological diagnosis has not yet been made. However, comprehensive virological studies are in progress. You will be kept informed as specific information is obtained regarding this entity.

* * * * *

By this time, those who were following the outbreak closely had noted its similarity to the outberaks primarily reported in England[5,6,12] and from the Low Countries and Belgium.[7] Some also saw a resemblance to the exanthematous disease reported in Boston[8] and Pittsburgh,[9] even though this type was not characterized by meningeal involvement. In fact, even at that time, there were grounds for ascribing the epidemic to infection with ECHO 9 virus, a possibility that was soon confirmed. Almost simultaneously with the issuance of the above bulletin, Prince, St. Geme, and Scherer[10] announced the isolation of ECHO 9 virus from a group of patients (case material from the Pediatric Department, University Hospitals) all of whom presented more or less, the clinical picture characteristic of the outbreak. Although Prince *et al.* were primarily interested in delineating the etiology of the exanthem, they did include patients with meningeal involvement. They were able to demonstrate ECHO 9 virus in about 20 instances and, along with this, serological evidence that the infection with this virus was current. (Further confirmation on etiology was to come from other case sources, discussed below.

TABLE 3

INCIDENCE OF ASEPTIC MENINGITIS, MINNESOTA, 1957, BY AGE AND SEX

Age	*Male*	*Rate per 100,000*	*Female*	*Rate per 100,000*	*Total*	*Rate per 100,000*
Under 5	30	15.9	16	8.5	46	12.2
5 to 9	59	40.2	35	24.0	94	32.2
10 to 14	68	52.1	45	34.8	113	43.5
15 to 19	49	42.9	35	30.9	84	36.9
20 to 29	72	31.3	75	32.9	147	32.1
30 to 39	61	27.3	48	21.7	109	24.5
40 and over	25	4.2	28	4.7	53	4.5
Unknown	8		4		12	
Total	372	22.8	286	17.7	658	20.3

The age and sex distribution of the reported cases (Table 3) indicates that males were more commonly attacked than females except in the 20 to 29 age group. This is reminiscent of the sex shift in incidence in poliomyelitis at about the same age level. The highest attack rate occurred in the 10 to 14 year age group; in general, the incidence was lowest in the very young and in older adults, and highest in older children and young adults. The plateau-like nature of the graph of age distribution (Fig. 3) has epidemiological significance and will be referred to later.

The geographical distribution of the cases is shown in Table 4. The area sub-divisions of the state conform to the pattern of the existing State Health Districts, with Hennepin and Ramsey counties and the cities of St. Paul and Minneapolis counted separately. Fifty-two per cent of the reported cases came from the metropolitan counties of Hennepin and Ramsey.

TABLE 4

GEOGRAPHICAL DISTRIBUTION OF ASEPTIC MENINGITIS
MINNESOTA, 1957

Area	*Cases*	*Per cent of Total*
Hennepin Co. exclusive of Minneapolis	114	17.3
Minneapolis City	149	22.6
Ramsey Co. exclusive of St. Paul	12	1.8
St. Paul City	66	10.1
District I (Northwest — 14 Counties)	31	4.7
District II (South central — 12 Counties)	30	4.6
District III (Southeast — 11 Counties)	59	9.0
District IV (Northeast — 6 Counties) (includes Duluth and Range Cities)	54	8.2
District V (Southwest — 13 Counties)	20	3.0
District VI (East central — 7 Counties) (excluding Hennepin and Ramsey)	37	5.6
District VII (West central — 11 Counties)	30	4.6
District VIII (Central — 11 Counties)	56	8.5
Total	658	100

AGE DISTRIBUTION, ASEPTIC MENINGITIS, MINNESOTA, 1957
Compared with age distribution, Paralytic Polio, U.S.A., 1956

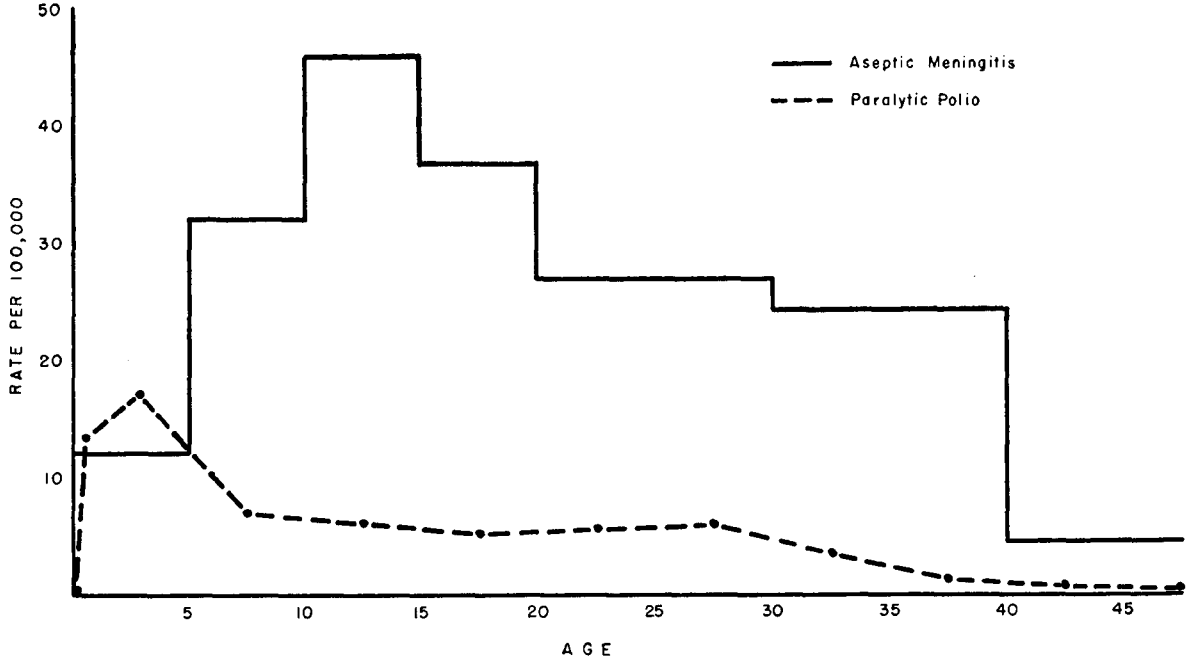

Fig. 3

This report would be incomplete without mention of an outbreak in late August and early September in some of the cottages at the State School and Hospital, Faribault, Minnesota. Most of the patients in this institution are mentally retarded. Hence, subjective symptoms would seldom be described spontaneously; moreover, the spasticity of some of the patients would impede detection of any recently acquired nuchal or back rigidity.

The essential features of this outbreak are presented in Tables 5 and 6. The overall attack rate was 31 per cent; a rash was present in 20 per cent of the cases; and, most interestingly, the fever curve was biphasic in 26 per cent of the cases. White blood cell counts were usually normal but in some cases leukopenic. Eosinophilia was present in 12 cases, with eosinophile percentages ranging from 5 to 18 per cent. The age and sex distribution (Table 6) parallels that found in the general population. Meningeal symptoms were absent; in only one case was nuchal rigidity recorded.

TABLE 5

CLINICAL FEATURES OF ECHO 9 DISEASE
FARIBAULT STATE SCHOOL AND HOSPITAL
MINNESOTA, 1957

Cottages	*Date of Outbreak*	*Population*	*Cases*	*Rash*	*Exanthema*	*Headache*	*Stiff Neck*	*Sore Throat*	*Nausea*	*Emesis*	*Diarrhea*	*Biphasic Fever*
Hillcrest	8/14–9/7	85	6	3		3		2	1	3		3
Center A	8/31–9/9	30	20	7		1				2		10
Center K	9/1–9/15	30	26	13	3	2				9		9
Center J	9/1–9/15	31	2	1		1				1		
Maple	8/23–9/2	105	51		2			1		4		10
Pine	8/28–9/10	106	44	1				5		6	2	9
Cedar	9/1–9/4	110	24				1	2		7	2	5
Holly B	8/29–9/18	115	17	14								3
		612	190	39	5	7	1	10	1	32	4	49

This particular outbreak at the State School offered an unusual opportunity to record detailed observation of the rash. In the fading stage the rash was noted on the face and upper chest as a measles-like eruption, pale but blotchy. One such instance was observed six days after the onset, but usually the rash was more transient. During the very first stages the rash was discrete with papules somewhat

smaller than those of measles. On the face, the rash appeared a little grosser and without coalescence. Small patches of petechiae were seen in one case. The rash was most abundant on the thorax, abdomen, face, and proximal portions of the extremities, in that order. The rash generally resembled the exanthem of rubella. But most of the children in the affected cottages had had typical German measles in April, 1957, five months earlier. These cases in April had displayed the characteristisic posterior cervical lymphadenopathy, which was not observed in the cases occurring in August and September.

TABLE 6

DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX
ECHO 9 DISEASE, FARIBAULT STATE SCHOOL AND HOSPITAL
MINNESOTA, 1957

Age	*Male*	*Female*	*Total*	*Cases in Percentage*
1- 4 years	1		1	0.52%
5- 9 years	34	3	37	19.47%
10-14 years	58	32	90	47.37%
15-19 years	27	14	41	21.57%
20 and over	1	20	21	11.05%
Total	121	69	190	100.00%
Sex distribution in Percentage	63.7%	36.3%		

In this series, ECHO 9 virus was isolated from the specimens of seven patients out of 16.

A more detailed study of the individual signs and symptoms associated with the outbreak appears in Table 7. The 172 cases represented are those patients who were hospitalized at the Elizabeth Kenny Institute, the Minneapolis General Hospital, or at Ancker Hospital in St. Paul, and on whom the clinical data were the most complete. This case total consists of both those in which an etiologic agent was isolated as well as those in which it was not -- whether for lack of specimens or any other reason. This group is tentatively referred to simply as aseptic meningitis. The roster of symptoms shows a preponderance of items associated with meningeal involvement. In addition, photophobia, nausea, vomiting, and abdominal pain were relatively common.

TABLE 7

SIGNS AND SYMPTOMS IN HOSPITAL CASES
ASEPTIC MENINGITIS, MINNESOTA, 1957
(172 CASES)

Signs and Symptoms	*No. of Cases*	*Percentage*
Fever	169	98%
Headache	168	98%
Mild Headache	96	56%
Severe Headache	72	42%
Stiff Neck	156	91%
Stiff Back	140	81%
Vomiting	100	58%
Nausea	94	55%
Rash	42	24%
Lethargy	34	20%
Photophobia	35	20%
Abdominal Pain	23	13%
Sore Throat	23	13%
Muscle Weakness	2	1%

Two of the clinical features, the headache and the rash, deserve some additional comment. The headache was often so severe that physicians reported it as such. One pediatrician reported the unusual fact that patients as young as three years of age had complained of headache. A woman patient averred that she would much rather endure an episode of migraine, to which she was subject, than the headache associated with this disease. Indeed physicians often stated that they had had to resort to prescribing Demerol® or morphine for relief of the pain; and physicians at the Elizabeth Kenny Institute reported that lumbar puncture relieved the headache in some instances.

The rash has been described previously, but, its distribution among the various age groups is shown in Table 8. Contrary to an earlier widespread impression, the rash was not confined principally to children but occurred with almost equal frequency in all age groups.

TABLE 8

AGE DISTRIBUTION OF RASH IN HOSPITAL CASES
ASEPTIC MENINGITIS, MINNESOTA, 1957

Age Group	*ECHO 9 Isolates*		*All Cases*	
Years	*No. Cases*	*Rate per 100 Cases*	*No. Cases*	*Rate per 100 Cases*
0- 4	1	100	1	14
5- 9	6	32	6	24
10-14	5	23	6	18
15-19	3	30	5	21
20-24	2	20	6	24
25-29	5	45	8	33
30-34	4	44	5	29
35-39	1	25	2	33
40-44	1	33	1	20
45-49	1	50	1	25
50-54	0		1	50
Total	29	30%	42	24%

The cellular responses in the cerebrospinal fluid are shown in Table 9, which includes for comparison similar date from poliomyelitis cases of 1955. The tendency for the cell counts in aseptic meningitis to exceed those in poliomyelitis is strongly suggested in the cell count range of 300 to 999 and even more strikingly in cell counts over 1,000. The cellular response was predominantly lymphocytic, although if the lumbar puncture was made early in the course of the disease, lymphocytes and neutrophiles often appeared about equal.

TABLE 9

DISTRIBUTION OF SPINAL FLUID CELL COUNTS
ASEPTIC MENINGITIS, MINNESOTA, 1957

	Number of Cells per mm^3			
	0-99	100-299	300-999	1000 and over
ECHO 9 Isolates 92 Cases	49%	29%	16%	5%
All Cases 167 Cases	40%	29%	23%	8%
Proved Polio, 1955 150 Cases	50%	39.3%	10%	.7%

An important aspect of the study of this epidemic consisted of visits to the families of the reported index cases in Hennepin and Ramsey counties. These families were queried as to the existence of concurrent and similar illness among the various household members. The results of these visits, although not yet completely analyzed, support the observation that multiple cases of illness did occur within single households. The data also indicate that the disease spectrum was wide: In many cases of concurrent illness the symptom constellations did not include stiff neck or stiff back. A rash was described as occurring with a mild set of symptoms as well as with a severe set which included the signs of meningeal irritation. Finally, in some cases the whole disease was represented only by a fever and rash.

In Minneapolis, for example, of 416 household exposures to an index case, there were 168 instances of illness occurring within 30 days before or after the onset of the illness in the index case. Of these 168 patients, 82 were males and 86 females; 104 had either headache, stiff neck, or stiff back; 83 were reported to have had fever; 60 had nausea, vomiting, diarrhea, and abdominal pain; 35 had sore throat; and 27 had a rash. The secondary attack rate thus comes out to at least 25 per cent and at most 40 per cent – a remarkably high secondary attack rate.

The data presented thus far have been crude, in the sense that, except for some hints in Tables 8 and 9, no reference was made to any group of cases in which laboratory findings permitted identification of the etiological agent. Through the reports of virus isolations effected at the Minnesota Department of Health's laboratories, some refinement of this crude picture is now possible. From the specimens of 1957 these laboratories have isolated six strains of poliovirus – one in a nonparalytic case; 18 strains of Coxsackie B5 virus; and 120 strains of ECHO 9 virus. The laboratory of Dr. J. T. Syverton (Professor and Head, Department of Bacteriology and Immunology, University of Minnesota), which is the virus reference laboratory for the Minnesota Department of Health, has so far confirmed the identity of sixty of the 120 ECHO 9 isolates. Etiologically, then, the ratio of poliovirus to Coxsackie B5 virus to ECHO 9 virus in the production of nonparalyzing aseptic meningitis is as one is to 18 is to 120. The specimen sources from which the ECHO 9 virus was isolated are summarized in Table 10. Noteworthy is the frequency with which the virus was found in the spinal fluid and throat washings, even though the number of stool specimens far exceeded the number of

throat washings and spinal fluids processed. As can be seen, isolation from multiple sources in the same patient was fairly common.

TABLE 10

SOURCES OF ECHO 9 VIRUS ISOLATIONS, 1957
MINNESOTA DEPARTMENT OF HEALTH

	Total
Stool Only	81
Throat Only	13
Spinal Fluid Only	2
Stool and Throat	13
Stool and Spinal Fluid	4
Throat and Spinal Fluid	3
Stool, Throat, Spinal Fluid	4
Total	120

The State Health Department also has the results of titrations for antibodies against prototype ECHO 9 virus in 196 pairs of blood specimens, representing in each case the acute phase and the convalescent phase. In 72 of these sets a significant (sixteen-fold) rise in antibody titer against ECHO 9 virus was demonstrated. Fifty-eight of these 72 represent the results in patients from whom ECHO 9 virus was isolated. A random sample of the character of specific antibody responses is given in Table 11.

The bank of 172 cases with the most complete data sheets which has been used in some of the analyses presented above, includes 97 cases designated as ECHO 9 isolates. This group can be considered as offering etiologically refined data. Figure 4 compares the signs and symptoms of the crude total with those of the etiologically refined component. The disease profiles of the two groups are very similar, almost identical, in fact. (The figures for the cerebrospinal fluid findings shown in Table 9 are an additional facet in the overall pattern of similarity.) Therefore when one has described this epidemic in terms of the crude data, one has, in effect, also described an epidemic of aseptic meningitis due to ECHO 9 virus.

The cases of aseptic meningitis associated with Coxsackie B5 virus deserve separate mention. Severe headache occurred only half as often in these patients as in those with ECHO 9 virus. With one

questionable exception, no patient with a Coxsackie virus infection exhibited a rash. None of the Coxsackie B5 cases of 1956 (when the incidence of this particular virus was more than four times as great) had showed any evidence of a rash either.[4]

TABLE 11

TYPICAL ANTIBODY RESPONSES IN PATIENTS FROM WHOM ECHO 9 VIRUS WAS ISOLATED, MINNESOTA, 1957

Patient	*Date of Onset*	*Interval of specimen from onset (days)*	*Titer*
	9-9-57	3	<4
		20	64
	8-11-57	3	4
		26	64
	8-19-57	7	64
		18	64
	7-26-57	4	16
		13	64
	9-14-57	0	<4
		16	16
	7-23-57	4	4
		13	64
	7-29-57	4	<4
		14	256
	7-27-57	4	16
		32	256
	8-10-57	4	4
		14	256
	9-19-57	0	4
		11	64
	7-26-57	4	4
		18	256
	7-26-57	1	16
		11	256

In 1957, Wisconsin too, was having outbreaks of nonparalyzing aseptic meningitis, not only in small communities but in its largest city, Milwaukee. Dr. E. R. Krumbiegel, Health Officer of Milwaukee, estimated that during the peak week of the season some 10,000 people had been affected, with as many as 100,000 cases during the entire season. The etiological agent associated with these cases was identified as ECHO 9 virus by Dr. Albert Sabin. By September 20, 1957,

the Poliomyelitis Surveillance Unit of the Communicable Disease Center in Atlanta was able to map the locations (see Figure 5) of outbreaks of aseptic meningitis from these newer causes.[11]

SIGNS AND SYMPTOMS IN HOSPITAL CASES, ASEPTIC MENINGITIS
Minnesota, 1957

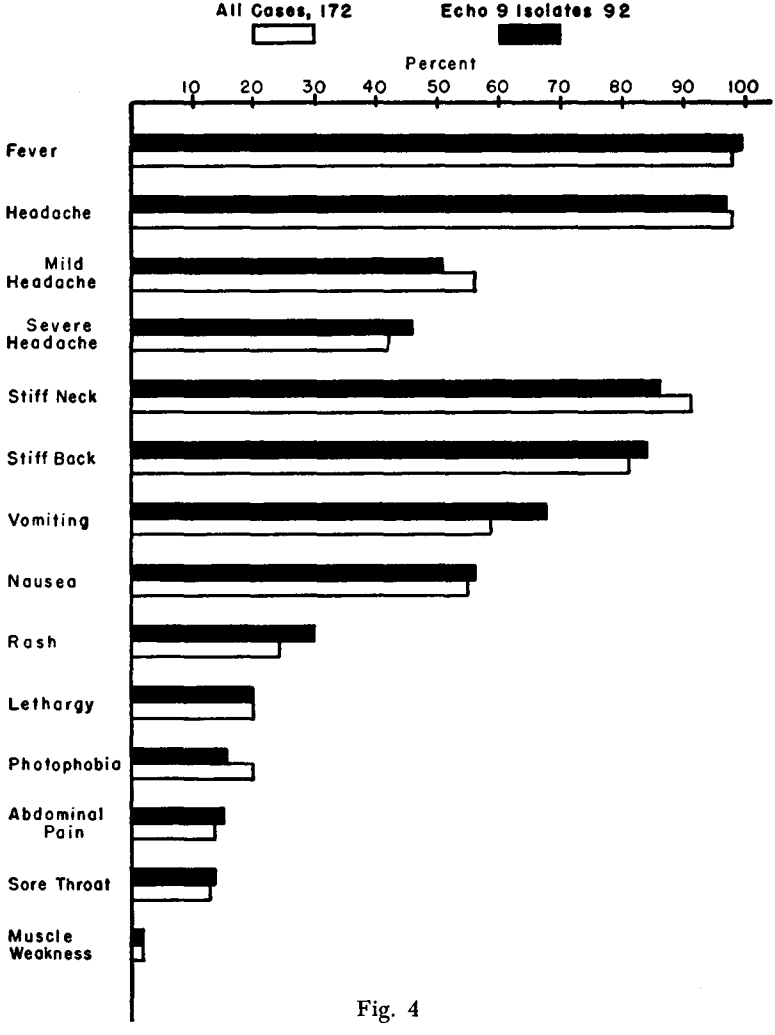

Fig. 4

ASEPTIC MENINGITIS,
United States, 1957

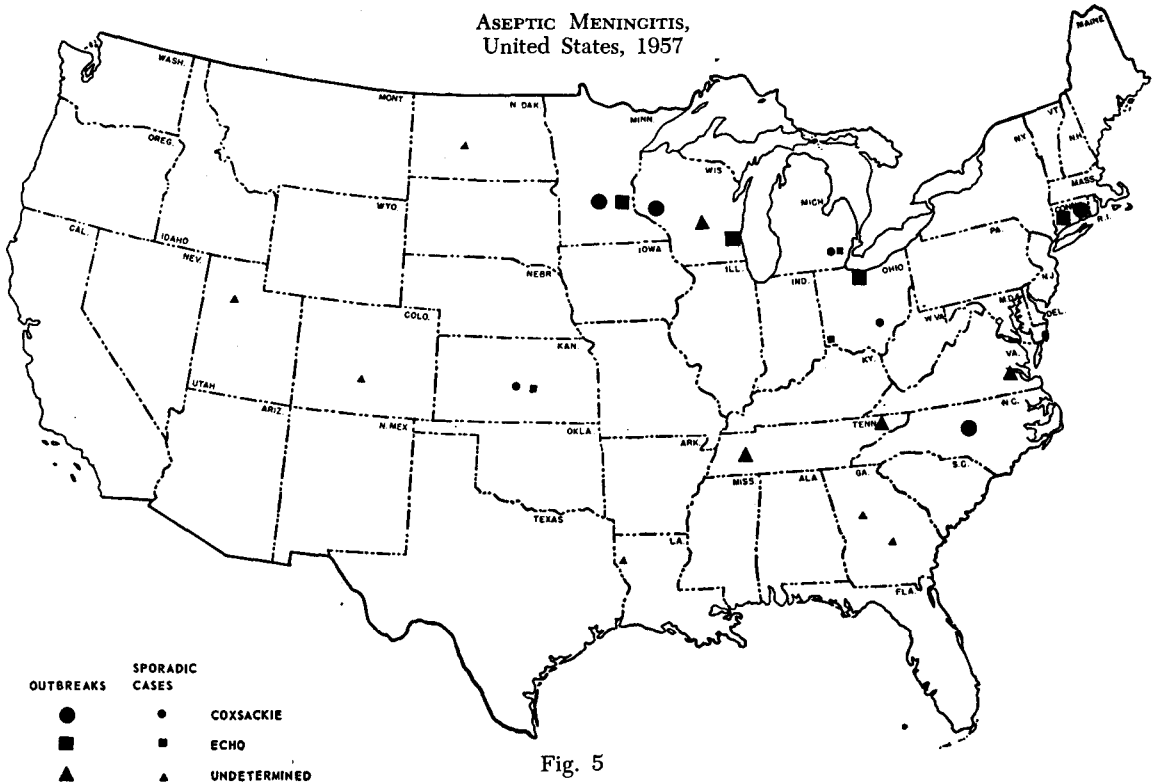

Fig. 5

It is not yet possible to prepare a final case list of aseptic meningitis in Minnesota for 1957 on the basis of etiology. When such a list is prepared, it will properly include the following categories:

1. Cases with virus isolated with or without positive serological evidence.
2. Cases with serological evidence but without virus isolation.
3. Cases acceptable on the basis of clinical evidence alone, i.e., cases with rash, severe headache, and high cell counts, or some reasonable combination of these.
4. Cases admissible on epidemiological grounds, i.e., cases in siblings or close contacts of a proved case, provided the clinical evidence is reasonable.

This final listing will of course not equal the 658 reported cases of Table 2, but it will approach this figure. The reported incidence was obviously but a fraction of the real incidence. The calculated secondary attack rates as well as the empirical observation of the widespread nature of this disease quite naturally inspired a number of questions: Just how extensive was this outbreak? How many people did it really involve? An attempt was made to answer these questions by designing a statistical survey to sample populations in Ramsey and Hennepin counties. Practical difficulties prevented its extension to other parts of the state. While the results obtained are not strictly applicable to the whole state, they do permit a reasonable overall approximation. The essential steps of the survey procedure were as follows:

1. A sample size was determined that would yield results at a 95 per cent confidence level. This, somewhat maximized, came to 400 households.
2. These 400 households were randomly located on the basis of population density by first randomly locating a city or village block and then randomly choosing a dwelling within that block.
3. Canvassers then interviewed the members of the selected household using a check list of symptoms, confining their queries to the period from July 1, 1957 to September 20, 1957.
4. The data accumulated by the canvassers were then reviewed, and on the basis of symptoms checked, the number of persons in any household who ostensibly had the illness was ascertained.
5. The proportion of the illnesses so turned up was then projected to the latest population estimates of the areas involved, to arrive at the estimated overall incidence.

Several considerations were involved in including or in rejecting any case in the survey total. First, it was evident that there was an epidemic. Second, the clinical features of the severer forms of the disease were known from hospital records. In addition, ample evidence indicated that there was a wide clinical spectrum for the disease and that it was common for a number in the same household to be stricken. Physicians, too, stated that they were seeing many more cases than they were reporting.

The decision to include a case, then, depended not only on the presence of an unequivocal constellation of symptoms but also on the evaluation of less characteristic symptom complexes. The latter had to be weighed against the time of occurrence in relation to the presence of more characteristic illness in the same household, as well as against the known width of the disease spectrum.

These factors are, of course, a source of bias and error. It is also admitted that some of the frankest cases may represent a Coxsackie infection or even nonparalytic poliomyelitis. Nevertheless, the results in Table 12 represent the best present estimate we have of the actual incidence of aseptic meningitis with or without rash due to ECHO 9 virus infection, as this disease occured in Hennepin and Ramsey counties from July 1, 1957 to September 20, 1957.

Table 12 also compares the proportions of estimated cases in various localities to the proportions of reported cases in these localities. Except for one area, the concordance is very good, which supports the view that the estimates are the reasonable result of a valid method. In fact, physicians to whom these results were reported preliminarily showed no surprise at the magnitude of the estimates; their experience had suggested that the figures were not at all disproportionate.

According to Table 12, the cases reported in Hennepin and Ramsey counties represented 52 per cent of the total reported. When this percentage is used in projecting the two-county estimate to the state as a whole, it appears that the total state incidence was 424,367 cases. This projected estimate, however, is not as certain as is the two-county estimate because the attack rates in other sections of the state, especially in rural regions, may have differed from those in the metropolitan area.

Epidemic theory strongly suggests that ECHO 9 infection is new to Minnesota. The most compelling reason for this view is found in the age distribution curve (Fig. 3), which suggests that the present adult population had had no previous immunizing experience with this

TABLE 12

ESTIMATED AND REPORTED INCIDENCE OF ASEPTIC MENINGITIS (ECHO 9) MINNESOTA, 1957, HENNEPIN AND RAMSEY COUNTIES

	Estimated Population	*Estimated Cases*	*Percent of Estimated Total*	*Reported Cases*	*Percent of Reported Total*
Hennepin County					
Minneapolis	552,000	91,061	41.3	127	43.0
Bloomington	35,000	17,888	8.1	22	7.5
Richfield	37,000	15,857	7.2	15	5.1
St. Louis Park	39,200	9,800	4.4	5	1.7
Other county areas	133,510	23,423	10.6	56	19.0
Total	796,710	158,029	71.6	225	76.3
Ramsey County					
St. Paul	336,000	45,073	20.4	60	20.3
Roseville	29,800	10,927	5.0	3	1.0
Other county areas	77,811	6,642	3.0	7	2.4
Total	443,611	62,642	28.4	70	23.7
Total	1,240,321	220,671	100	295	100

disease. The relatively lower attack rates at the extremes of age can be safely laid to differences in exposure potential. The age distribution curve for the 1956 Coxsackie B5 experience,[4] on the other hand, indicates that adults had had prior immunizing experience. When this occurred it is hard to say; it was almost certainly before 1955, because the laboratory procedures in use that year were the same as those used in 1956 and should have picked up the virus if it had been there.

It is tempting to look back in the records in a search for those years in which the reports of nonparalytic poliomyelitis seemed disproportionate to the reports of paralytic disease, and to conjecture from this that in those years some of the nonparalytic poliomyelitis might in truth have been aseptic meningitis due to some cause then unknown. Such divination, however, is hazardous. Fashions and vagaries in diagnosis and in reporting could very well push askew all such attempts at reconstruction. Nevertheless, these speculations in epidemiological archaeology might very well be right. Perhaps an intensive study in serological epidemiology, with all age groups well sampled, would show the relative antiquity of these newly recognized viruses. Such an investigation is worth trying.

Acknowledgment: Thanks are due the following people for their help in completing a project of considerable proportions in so short a space of time:

School of Public Health, University of Minnesota
Ernest Ager, M.D.
Jacob Bearman, Ph.D., and staff of graduate students

Minnesota Department of Health
Orpha LaCroix, R.N.
Kathrine Gram

Minneapolis Health Department
Karl R. Lundeberg, M.D.
F. G. Gunlaugson, M.D.
Beverly Schei, P.H.N.
Lorraine Baumgartner, P.H.N.
Tully Stopple, P.H.N.

St. Paul Health Department
R. B. J. Schoch, M.D.
Boris Levich
Public Health Nursing Staff

Hennepin County Nursing Service
Alma Hovde, P.H.N.

Ramsey County Nursing Service
Genevieve McDonald, P.H.N.

Medical Students
Gerald E. Nelson
Hovald K. Helseth
Sheldon G. Cable
Kenneth P. Manick
Rodney Langseth
Fred Featherstone

References

1. Wallgren, A.: Une Nouvelle Maladie Infectieuse du Systeme Nerveux Central?, Acta Paediat. 4:158, 1925.
2. Adair, C.; Gauld, R.; and Smadel, J.: Aseptic Meningitis, A Disease of Diverse Etiology: Clinical and Etiological Studies on 854 Cases, Ann. Int. Med. 39:675, 1953.
3. Committee on the Enteroviruses, National Foundation for Infantile Paralysis: The Enteroviruses, A.J.P.H. 47:1556, 1957.
4. Syverton, J. T.; McLean, D. M.; Martins da Silva, M.; Doany, H. B.; Cooney, M.; Kleinman, H.; and Bauer, H.: Outbreak of Aseptic Meningitis Caused by Coxsackie B5 Virus, J.A.M.A. 164:2015, 1957.
5. Boissard, G.P.B.; Stokes, Joan; Macrae, A. D.; and MacCallum, F. O.: Isolation of Viruses Related to ECHO Virus Type 9 from Outbreaks of Aseptic Meningitis, Lancet 1:500, 1957.
6. Tyrrell, D. A. J. and Snell, B.: Recovery of Viruses from Cases of Epidemic Exanthem Associated with Meningitis, Lancet 2:1028, 1956.
7. Quersin-Thiry, L.; Nihoul, E.; and Dekking, F.: ECHO Virus Type 9 (New Member of Coxsackie Group Type A?) as a Cause of Epidemic Meningitis, Science 125:744, 1957.
8. Neva, F. A.; Feemster, R. F.; and Gorbach, I. J.: Clinical and Epidemiological Features of Unusual Epidemic Exanthem, J.A.M.A. 155: 544, 1954.
9. Neva, F. A.: A Second Outbreak of Boston Exanthem Disease in Pittsburgh During 1954, N. Eng. J. Med. 254:838, 1956.

10. Prince, J.; St. Geme, J. W.; and Scherer, W.: ECHO 9 Virus Exanthem. In Preparation.
11. Poliomyelitis Surveillance Reports, 1957, Nos. 119, 120, 121, 122, 123, 127 and 128. Department of Health, Education and Welfare, Public Health Service, Bureau of State Services, Communicable Disease Center, Atlanta, Ga.
12. Garnett, D. G.; Burlingham, A.; and van Zwanenberg, D.: Outbreak of Aseptic Meningitis of Virus Origin in East Suffolk, Lancet 1:500-502, 1957.

Staff Meeting Report

The Patient's Presenting Complaint – Signpost or Goal?*

I. Philosophical Considerations

Richard M. Magraw, M.D.†

The purpose of this paper is to examine the patient's "presenting complaint," in regard to its traditional place in the medical history and in a larger philosophical sense to its place in medical practice. I have chosen this seemingly elementary and commonplace subject not only because it needs reexamination, but also because, in my hybrid role in internal medicine and psychiatry over almost ten years, I have been forced to recognize the crucial place of the patient's symptoms. This insight is far from new, but what needs emphasis is the frequency with which the patient's presenting complaint is not clarified during medical care. In my experience problems in diagnosis and treatment have been traceable repeatedly to the fact that about as often as not we physicians do not get this basic understanding of exactly how the patient is "hurting," and thus we do not exploit the potentialities for improved diagnosis and care that lie embedded in the presenting complaints. If the frequency of this failure is as high as indicated (see section II), we are faced with the paradox of trying to care for many patients without actually understanding what they need or want. I believe that this obvious fact is usually overlooked and that its seeming simplicity is deceptive.

We can find a common ground for this discussion by recalling some basic concepts of the diagnostic process and of medical history-taking. Of the three fundamental diagnostic tools in medicine, the history is not only the most fruitful, but is also the most difficult. It involves the same mastery of a large body of factual knowledge as do the physical examination and the interpretation of laboratory tests, and in addition, it requires the exacting skill of communication, of understanding another's thoughts or the interchange of thoughts and ideas.

*This report was given at the Staff Meeting of the University of Minnesota Hospitals on February 28, 1958.

†Associate Professor, Division of Psychiatry, and Associate Professor, Department of Medicine

The medical history is divided into three distinct and quite different parts: The *chief complaint* is a brief statement in the patient's own words, if possible, in answer to the question, "What bothers you the most?" or "What brings you to the hospital?" The *history of the present illness* is a chronological account of the events, symptoms, and treatments that have been involved in the patient's illness. The *past medical history* is an ordered inquiry into various organ systems and personal biography.

These parts differ in the level of skill in communication they require. The past medical history requires the least skill in communication; in fact, it readily lends itself to routine questionnaire and will undoubtedly be the first part of the diagnostic process to succumb to automation. The history of the present illness requires somewhat more effort in understanding the patient but is less concerned with the patient's meaning than with assigning a sequential rank to symptoms and happenings. It is in eliciting the patient's chief complaint that the physician's skills in communicating and understanding are tested; it is the most difficult part of the medical history. I do not think there is general agreement on the difficulty of this procedure. I doubt that most doctors would agree that getting the patient's answer to "What bothers you the most?" is usually the hardest part of history-taking. There might be more agreement were I to emphasize that the achievement sought is a real understanding of the patient's meaning rather than a perfunctory acceptance of the first phrase the patient uses. Much of the time we take this achievement for granted and accept the chief complaint at face value, assuming a mutual understanding that does not exist.

Now, what prevents or limits communication between patient and doctor? Let us begin with the patient, since the initiative for spontaneous and cooperative communication lies with him: Patients frequently may not be completely frank about what is bothering them. Every doctor has had exasperating confirmation of Oliver Goldsmith's comment that words are used as much to conceal thoughts and feelings as to express them. This is particularly true if the doctor wastes his best questions by pressing for a disclosure of symptoms before the patient has sized him up and feels ready to talk. In such an instance, the doctor may follow some trivial or trumped-up complaint whereas more deliberate questioning would have yielded a more relevant answer. Thus an elderly patient having complained vaguely to one doctor of "stomach pains," will tell a second who bided his time, "I'm no good to my wife anymore, Doc."

There are, of course, many other reasons for a patient's lack of frankness or inability to describe a symptom clearly. When a patient is ashamed or worried, the things he says are bothering him may be merely camouflaging his real trouble. Or the patient may speak allegorically, and only if the doctor listens alertly to the tune as well as the words, will he ask and find that the patient's gas pains and cramps go "way down . . . ah . . . down below my testicles" and that the patient fears that he may have strained himself sexually. Furthermore, the patient may not be frank about his symptom because he may have repressed what is troubling him; hence the doctor may have to help the patient redefine in his own mind what is actually bothering him. Then there is the rare patient who wants to use medical authority to gain his own ends and who may give a consciously misleading complaint.

The patient's chances of getting help are often compromised by his own habitual ambiguities of thought and speech. If, for example, he exhibits the "you know syndrome" ("Well, it's a sort of an ache. You know."), the doctor, in a reflex way, may find himself nodding in agreement when in fact he does not really know. This lazy ambiguity is closely related to the problem of psychological dependency which is often accentuated when a patient seeks a doctor's help, prompting him by vagueness and helplessness to have the doctor define the symptoms. He may say, "Well, it's a kind of pain," and his voice trails off. He pauses and looks helplessly and expectantly at the doctor. If the doctor is trapped into taking over with direct questions (such as, "Does the pain bore through to your back?" or "Is it a crampy pain?"), he soon finds he must take the initiative in extracting the rest of an unreliable history. When this happens he later becomes engaged in a sticky struggle with the patient, reminiscent of the difficulty Brer Rabbit had with the tar baby.

Dependency is only one of the issues about which patients struggle with their doctors. Ostensibly, the patient comes to the doctor for help against his symptom, but he often has attitudes that interfere, or he wants treatment on his own terms; once in a doctor's office, the doctor rather than the symptom may become the adversary. The ensuing covert struggles impede communication and prevent incisive medical care. Clarifying the presenting complaint is an extremely important device for minimizing these struggles between doctors and patients, for it focuses on what the real trouble is and precludes the doctor's entering into a false agreement[1] about the nature of the trouble and the treatment required. That is to say, the doctor avoids

the false implication that he can get the patient over his headaches by treating the high blood pressure he has found on physical examination, when the patient's symptom is, in fact, a tension headache resulting from problems with his boss and with other people in authority.

The point at issue is honesty: The doctor insists on frankness and honesty from the patient, and in turn he tries honestly to understand what bothers the patient and to help him, rather than merely to identify and treat disease entities. Legally and practically the doctor-patient relationship is a contract. For the patient this contract centers about his symptoms rather than about disease entities as the doctor sees them. If the things that are bothering him are concealed by the patient, ignored or misunderstood by the doctor, or in general not clarified by the diagnostic process, the agreement is likely to be unsatisfying, and help will be more or less fortuitous and nonspecific.

There are many other things a patient may do that obscure the doctor's understanding. He may use an impersonal form of speech in describing his symptoms, e. g., "when the vomiting comes" (rather than "when I vomit"), or "the arm aches" (rather than "my arm hurts me"). The patient seeks thus to objectify the symptom and remove it from analysis, and if the doctor is not careful he may find himself sanctioning this by asking about *the* vomiting and *the* arm. Or, the patient may adopt the particular kind of unspontaneous speech which has the reiterated rhythmic quality of iambic pentameter. When a patient slips into this rhythmic speech, he is usually entirely at ease because he is protecting his psychological defenses and avoiding facing up to what his symptoms are. Sometimes the doctor can figure out why the patient needs to protect himself and hence guess what his trouble is, but in general this is one of the few times when little is gained by letting the patient talk.*

It is clear, then, that if the doctor wants to understand the patient he must be ready to force the issue of frankness and insist that the patient show his hand. Nor should he concern himself excessively with unconscious motivation since the mental defense mechanisms of repression, projection, and the like are nothing more or less than techniques of self-deception. Standards of frankness set up at the outset

*A special comment should be made about the difficulties of understanding the patient's complaint once he is in the hospital.[2] Most physicians have learned the advisability of getting the history either before or just at the time of admission to the hospital, since once the patient has settled down into the hospital, he assumes that he will be taken care of and has less motivation to define his symptoms than he did prior to hospitalization.

will catalyze honesty throughout and minimize the problem of unconscious motivation in medical care.

If the doctor really understands the patient's complaint, he should be able to answer the following questions: 1) Exactly what does the patient mean by the words he uses? Is he, for example, speaking allegorically? "I have no strength in me," may mean something quite different from "I feel weak." 2) Exactly what about his symptom bothers the patient? How does it hurt or interfere with his life? 3) Why does he come at just this time? Whose idea was it? What was the last straw? 4) What does he think he has? In all these facets of the presenting complaint, the patient may deflect or counter the doctor's inquiring interest. The doctor will need to insist gently on clear answers from the patient as the price for help.

The doctor must be aware of the ways that patients may interfere with communication, but even more important, he must recognize his own habits of work and thought that may hinder understanding. Take, for example, the implications of the phrases we doctors use. We speak of "chief complaint," which implies that we want only one specific, brief complaint, as thought fearful that by opening this up we will let loose a Pandora's box of symptoms. By the phrase, "taking a history," we imply that we extract the information we want, rather than encouraging a spontaneous story from the patient. These phrases reflect our attitudes toward symptoms and toward communicating with patients. The doctor's orientation and training obligate him to identify the diseases present; consequently, he tends to treat first his own anxiety by deciding what disease entity the patient has,[3] rather than by focusing on what bothers the patient.

Moreover, after habitual failure in communicating with patients, the doctor learns to do without this information in diagnosis by using substitute techniques. One such substitute technique consists of what might be called "the IBM approach." This is

> a kind of matching operation[4] in which the patient's complaints are converted into a technical jargon and then matched against a list of likely disease possibilities. "I can't breathe deeply" becomes "dyspnea." "I have a burning taste" becomes "glossitis." "I don't feel able to do anything" becomes "easy fatiguability." These symptoms are then matched against appropriate disease entities to determine the diagnosis.[5]

Another diagnostic approach consists of a quest for a pathognomonic symptom or sign.

> History-taking becomes a kind of quiz session. "Does

> this burning in your stomach get worse when you lie down after eating? Are your stools tarry?", etc. If the patient says no despite the physician's hope for a positive answer, the doctor's face falls until he thinks of a new possibility. "Do you have alternate constipation and diarrhea?" he asks, etc.[5]

We have all seen that with a suggestible or pliable patient it is possible to construct almost any syndrome the doctor looks for by such directive questions. This "tailoring" of symptoms is opposed by a strongly entrenched and desirable medical tradition which holds that the patient's complaints ought to be expressed in his own words.

Moreover, it is perfectly possible to get the symptoms in the patient's own words and nevertheless to use them only partially, as in the following diagnostic technique:

> Here the patient's complaints are used to get a superficial idea of the locus of the trouble, and then the doctor puts out a kind of dragnet[6] of diagnostic procedures: laboratory, x-ray, psychological tests, etc. in the hope of finding among the data dredged up a pathological condition to use for a diagnosis. For example, if the patient has distress in the upper abdomen, x-rays of his gallbladder and upper and lower gastrointestinal tract are ordered without more ado.[5]

The frequency of this is suggested by recordings of the medical history which show that almost invariably the second question is not "What do you mean?" but "When did this begin?" or "How long have you had this?" This "when" question indicates that the doctor is satisfied with the statement of the chief complaint and has begun to elicit the history of the present illness. In learning to do without real communication by utilizing these pseudodiagnostic techniques, the doctor lessens his motivation and skill for understanding future patients.

Finally, making sense out of a patient's complaints or understanding his meaning requires thought. And this is hard work. When neurophysiologists are able to measure the energy used in the process of thinking, I am sure that they will find that it is immense, because we all will do almost anything to avoid it. I believe all of us are willing to do hours of routine work in order to avoid the necessity for one clear trail-blazing thought. In considering the problems of doctor-patient communication, this factor of medical laziness cannot be ignored.

Clarifying the patient's presenting complaint is likely to make

dealing with patients easier and more satisfying. It will help the doctor to avoid false agreements about the nature of the trouble, and this in turn will remove much of the unacknowledged subterfuge of medical practice. This relationship will be increasingly democratic, rather than authoritarian or paternalistic. Focusing on what bothers the patient not only helps him by making him feel that he is understood, but it implies accepting him as a partner in his own wellbeing. Moreover, it relieves the doctor of the burden of deciding unilaterally what is good for the patient.

Every symptom is likely to result from a series of processes and to represent a confluence of pathological, emotional, and social factors. Consequently, understanding the patient's complaint is a way of resolving operationally the mind-body dualism.

The physician often arrives at a stalemate, wondering where to start with a patient in whom he senses psychological problems. Usually this stalemate becomes obvious after the doctor has completed his diagnostic appraisal and has found nothing sufficiently pathological to require treatment. This impasse is often expressed in the question, "What should be done to the patient?" rather than the more appropriate question, "What is the trouble with or what troubles the patient?" Clarification of the patient's presenting complaint is the place to start in such a case, and it should prevent the impasse from ever arising.

So far I have regarded the patient's presenting complaint simply as a signpost in diagnosis. Now I should like to suggest that what is bothering the patient does more than *point* to what the patient has. In a very real sense it *is* what he has. It is the diagnosis. It is the illness. We doctors tend to think of ill health in terms of specific disease entities. We even speak of the patient as "a diabetic," "a coronary," or "a schizophrenic." The patient, on the other hand, thinks of his symptoms as the disease; indeed the very word "dis-ease" indicates that that was the original meaning. We regard the patient's symptoms as subjective and inexact and not really what is wrong, and we try to fit them into exact disease patterns. Because of our long familiarity with the concepts of diseases and because they are very helpful generally, we forget that they are essentially theoretical models, artefacts that we have introduced to give us some direction in working with complicated phenomena.

Whitehead[7] has said that any advance in any science will ultimately become a hindrance. Certainly something is hindering us in medicine from applying the concepts of comprehensive medical care

to which we all pay lip service. I think our theoretical models are inadequate. The narrow concept of diagnosis centered about disease entities is one thing that hinders us. I hope we will soon be able to supplement it with another approach to diagnosis whose goal is an understanding of the patient's complaints in terms of various mechanisms that produce them.

It has been said that in any language we start by speaking as we think but end by thinking as we speak. This is clearly true of the medical language. The "built-in" conceptual errors that cause us to think of the patient in terms of disease, instead of as a person with symptoms severely hamper us. If my suggested revision seems sweeping, remember that the dynamic concepts of modern physics leading to the Atomic Age grew out of a reevaluation of older models of space, time, and measurement. Those models were supplemented but retained as part of an expanded, more fruitful conception.

The presenting complaint, to conclude, is a signpost when we consider illness in terms of disease. But when we take a more comprehensive view of the whole of medicine and consider the symptom *as* the illness, then understanding the patient's complaints becomes the goal itself.

References

1. Balint, Michael: *The Doctor, His Patient and The Illness,* New York, International Universities Press, 1957.
2. Meares, Ainslie: *The Medical Interview,* Springfield, Charles C Thomas, 1957.
3. Glueck, Bernard C., Jr. Personal Communication.
4. Cohen, Sir Henry: In Foreword to *Bedside Diagnosis* by C. M. Seward, Edinburgh, Livingstone, 1949.
5. Magraw, R. M.: "Psychosomatic Medicine and the Diagnostic Process," To be published.
6. Engstrom, Denton. Personal Communication.
7. Whitehead, Alfred N.: *The Aims of Education,* New York, New American Library, 1957.

II. The Clinical Study

Everett P. Dulit, Ph.D.* and Richard M. Magraw, M.D.†

Last summer, we attempted to test clinically the ideas discussed in Section I, an understaking which required that these assertions be formulated in an experimentally verifiable manner. It has been said that one always has to do a good experiment three times; the first to find out how *not* to do it, the second, to find out *how* to do it, the third, to *do* it. By these standards this study is now in the second stage; nonetheless, it might be of interest to describe our aims, some of our difficulties, and our results.

The crucial questions in this study concerned (a) frequency: How often does the chief complaint go unclarified? And (b) consequence: How important is clarification, once achieved, in leading to the correct diagnosis and treatment?

As a means of measuring (a), each patient was interviewed twice—first in the routine manner, then in an interview aimed solely at clarifying the chief complaint. Assuming the second interviewer always achieved his goal, ideally, comparison with the immediately preceding routine interview would indicate whether or not the first interviewer had achieved clarification. Notwithstanding the fallibility of the second interviewer, it was reasoned, this method should at least reduce the frequency of nonclarification. Furthermore, comparison of the two interviews should enable us to detect any additional understanding provided by the second interview.

We chose as interviewers the most readily available group in the outpatient clinic – the senior medical students. Our patients were unselected. With the consent of patients and doctors all interviews were recorded on tape. For the first interview, we asked each student simply to take a routine history. When finished, we took two students aside and briefly explained to them the rationale of the study, as described in section I. The students were then asked to exchange patients and to conduct a second interview directed solely at clarifying the chief complaint. In that way the same group of interviewers was used for the routine and experimental interviews. The instructions were "to find out exactly what is troubling the patient, or in what way his symptoms or disease is bothering him, and why he comes in at this particular time." When each student had completed his second

*Senior Medical Student
†Associate Professor, Division of Psychiatry, and Associate Professor, Department of Medicine

interview, he was asked to write his impressions of what was bothering the patient.

Thus, for each patient we had the following data: 1) the first student's routine write-up of his interview; 2) the second student's written impressions of the presenting complaints; 3) our tape-recording of both interviews; 4) the chart record of the patient's subsequent progress.

Briefly, our results in a series of 24 patients were as follows: In 20–40 per cent of the cases, the second interview brought out something that was medically important. These results provide a measure of the frequency of nonclarification of the presenting complaint in an unselected group of medical outpatients. (That our results were as high as 20–40 per cent under the circumstances speaks, as well, for the ease of communicating the operational aspects of this approach and for the flexibility of our student interviewers.)

The upper and lower limits, 20 per cent and 40 per cent, reflect the extremes of skepticism and tolerance that were applied in judging whether the complaint was indeed clarified or whether it was indeed important. Unfortunately we could devise no method that did not require the two of us to decide those questions ourselves. The following is an example of what we judged to be clarification and what we judged to be important: Mr. A. came to the outpatient clinic with a chief complaint as recorded by the first interviewer of burning of the feet, pain in the thighs, and nervousness. The diagnosis in the outpatient clinic was psychoneurosis and chronic anxiety state. The patient having been seen three times in the outpatient psychiatry clinic, was referred to neurology, where the examination was negative. The clinical psychologist's report noted that the presenting bodily symptoms were rather peculiar for a patient in this diagnostic category, e. g. "feelings of water or blood running down his legs." The patient was discharged with a final diagnosis of anxiety reaction with transient phobic and conversion phenomena; and the following notation appeared in the discharge summary: "Frankly, what the basis for this reaction might have been is impossible to say." Now let's look back at the observations of our second interviewer, recorded when the patient was first seen and never again elicited: "The patient's complaint to a great extent reflects his concern about the possible spread to his legs of a tumor he had removed from his right shoulder several months ago." That is the sort of thing we considered to be an important clarification of the presenting complaint. And to allude even more briefly to another of our cases: Mrs. B's chief complaint

was recorded by the first student as: "Epileptic fits since 1952." The second student, inquiring more intensively into what was troubling the patient, came up with the rather different presenting complaints: "I have spells, and my husband is afraid that I'll kill my children." Subsequent developments have shown this to be important in the care of this patient.

Grouping the data in another way and utilizing only the first routine interviews revealed a high correlation between accuracy of diagnosis and clarification of the chief complaint. In each of the ten cases in which the first student's diagnosis proved accurate, the presenting complaint had been well clarified; in each of the 12 cases in which the first student's diagnosis was judged inaccurate or incomplete, the presenting complaint had been inadequately clarified. It should be noted that in only one of the 24 cases was the medical student's diagnosis changed by the staff checker. While the recordings we made were used primarily to supplement the written impressions submitted by each interviewer, they incidentally indicated two things: a) In the routine interviews, the students spent about five times as much time on the past medical history, including review of systems, as on the chief complaint and the history of present illness combined; b) The students missed many opportunities to elicit valuable information that probably would not have been overlooked by more experienced interviewers. Often a potentially fruitful revelation went unrecognized or was interrupted by a request for dates and duration. That had also been the case in a brief preliminary study using the resident and intern staff. These failures to follow through were apparent not only to the more experienced interviewers but often also to the students themselves, who were invited to listen to the playbacks.

Now I would like to conclude by describing our quest for a criterion that proved to be unexpectedly elusive, a criterion for successful diagnosis. We felt that we had a serviceable means of measuring frequency of clarification in the double interview method described. What was required further was an equally workable criterion for judging the importance of such clarification, once achieved, in establishing the correct final diagnosis. This, of course, raised the question: What is the correct final diagnosis? We thought of determining this by having a board of experts listen to the recorded interviews, or perhaps interview and examine the patients themselves; for this study, however, we had neither the funds nor the personnel to set up such a board. Happily, in many cases subsequent clinical and laboratory

evidence leads to an unassailably correct final diagnosis. But what criterion of successful diagnosis can be used in cases where no clear-cut diagnosis ever emerges? And what of those cases in which the clinical and laboratory evidence unmistakably reveals a disease that cannot be related by any physiological mechanism to the patient's complaints? Or those in which the complaint, related though it may be to the patient's disease, persists or becomes transformed when the disease is successfully treated? This problem, we suggest, is central to our thesis. The final test is: What happens to the patient's complaints or his symptoms? In short, following diagnosis and treatment, how does the patient feel? Such a criterion of successful diagnosis brings in the whole patient in the most natural way, circumventing the inclination to graft the concept of whole patient onto the more familiar concept of disease entities.

Thus, in seeking some reliable criterion for the correct final diagnosis, we have come full circle, back to the patient's presenting complaint. Understanding this complaint, which at first we regarded as a signpost, seems, on closer examination, to appear more and more as the goal.

Editorial

Medical Manpower in Minnesota

In the state of Minnesota, as in most other states, many of the isolated rural and semi-rural areas have not had resident physicians. This has caused great concern among our citizens, who have urged that our medical schools increase enrollment to graduate more physicians. But it is difficult to determine what would be an adequate number of physicians. Minnesota, with 140 physicians per 100,000 population, or one for every 714 people, ranked eighth among the states in the ratio of physicians to population in 1955. Since 1921 the physician-population ratio in Minnesota has increased 32 per cent, and Minnesota now has the highest such ratio of all the north central states. But the physician-population ratio does not indicate the realistic supply and demand picture for physicians as other factors must be considered, i.e., population distribution and availability of hospital and transportation facilities. It is, however, the most frequently quoted yardstick. Certainly the advances in medicine brought about by antibiotics, poliomyelitis vaccine, and early ambulation after surgery, have reduced the hours that a physician once had to spend with his patients during an illness. This might be expected to free him to serve more patients and thus would reduce the physician-population ratio quoted as standards for the past.

The proportion of graduates of the University of Minnesota Medical School entering general practice, as shown in Frank G. Dickinson's study,* is larger than that of graduates of other medical schools in the north central states. A January, 1957, survey of freshmen and sophomores of our school revealed that 137 of the 253 students (54.1%) were graduates of public rural high schools. With but 46.1 per cent of Minnesota's population considered rural, it would appear from this figure that the University is preparing many physicians who are acquainted with the needs of rural and semi-rural communities. A community with modern hospital facilities and a population sufficient to need the full time services of a doctor trained in the art and science of today's medicine and surgery, should be able to meet its needs from among those graduated by our state's University.

N. L. G.

*Dickinson, Frank G.: Distribution of Medical School Alumni in the United States as of April, 1950, J.A.M.A. 157:473, 1956.

Medical School Activities

Faculty News

DR. RICHARD A. DEWALL, Medical Fellow, Department of Surgery, was named one of America's Ten Outstanding Young Men of 1957 by The United States Junior Chamber of Commerce. Dr. DeWall was recognized for his development of the "bubble oxygenator," a simple inexpensive apparatus of plastic cylinders and tubing permitting complete cardio-pulmonary by-pass in the surgical correction of intracardiac lesions. He is a research fellow of the American Heart Association. For his contribution to cardiovascular problems, Dr. DeWall was previously awarded the Hektoen Gold Medal for scientific exhibit at the 1957 American Medical Association Convention and the Ida B. Gould Award (with Dr. C. Walton Lillehei) in 1956.

In Houston, Texas at the Fourth Pigment Cell Conference during November, DR. HERBERT M. HIRSCH, Assistant Professor, Division of Cancer Biology, presented a paper entitled, "Inhibition of Melanogenesis in Tissues and the Control of Intracellular Autoxidations."

In February, DR. WESLEY W. SPINK, Professor, Department of Medicine, was guest lecturer at the University of Oklahoma Medical School, speaking on "Staphylococcal Sepsis." He also spent four days as a guest at Johns Hopkins Medical School where he made ward rounds, participated in conferences, and presented an address entitled, "From Endotoxin to Snake Venom."

DR. DAVID GLICK, Professor, Department of Physiological Chemistry, spoke recently on "Quantitative Histochemical Studies on the Adrenal Gland" at the Iowa State College, Ames, Iowa. The lecture was one of a weekly series of interdepartmental seminars devoted to "Structural and Functional Aspects of Nutritional Deficiencies."

DR. DAVID LYKKEN, Assistant Professor, Division of Clinical Psychology, has been invited to participate for a year, 1958-59, at the Ford Foundation's Center for Advanced Study in the Behavioral Sciences at Palo Alto, Calif.

DR. C. J. WATSON, Professor and Head, Department of Medicine, was Director pro tem, Medical Education and Biological Sciences, Cook County Hospital and Hektoen Memorial Institute, Chicago, for two days in February; while there he delivered the Ludwig Hektoen Memorial Lecture.

In Memoriam

Members of the Minnesota Medical Foundation felt a deep sense of loss on learning of the death of Senator Archie Miller of Hopkins. As a state legislator he steadfastly supported the University of Minnesota in its program of education, research, and service to our citizens. His active interest in the Foundation will be missed. To surviving members of his family we extend our sincere sympathy.

Coming Events

March 20 . . . . . Continuation Course in Surgery for Surgeons

March 21 . . . . . E. STARR JUDD LECTURE: *A Concept of the Pathogenesis of Gastric and Duodenal Ulcers*; DR. LESTER R. DRAGSTEDT, Professor of Surgery, University of Illinois College of Medicine, Chicago, Illinois; Mayo Memorial Auditorium; 4:00 P.M.

March 25 . . . . . MINNESOTA PATHOLOGICAL SOCIETY LECTURE: Medical Student Program; *Experimental Cardiovascular-Renal and Arthritic Lesions;* J. M. ANDERSON, R. B. ARHELGER, P. L. ECKMAN, and W. H. SCHRADER, Department of Pathology, University of Minnesota; Amphitheater, Owre Hall; 8:00 P.M.

March 31–April 2 . . Continuation Course in Gynecology for Specialists

April 7–9 . . . . . Continuation Course in Radiology for General Physicians

April 10–12 . . . . . Continuation Course in Arthritis and Physical Medicine for General Physicians

April 12 . . . . . Continuation Course in Trauma for General Physicians

April 14–16 . . . . . Continuation Course in Gastroenterology for General Physicians

May 5–9 . . . . . Continuation Course in Electrocardiography for General Physicians and Specialists

May 12–16 . . . . . Continuation Course in Proctology for General Physicians

WEEKLY CONFERENCES OF GENERAL INTEREST

Physicians Welcome

Monday,	9:00 to 10:50 A.M.	OBSTETRICS AND GYNECOLOGY Old Nursery, Station 57 University Hospitals
	12:30 to 1:30 P.M.	PHYSIOLOGY- PHYSIOLOGICAL CHEMISTRY 214 Millard Hall
	4:00 to 6:00 P.M.	ANESTHESIOLOGY Classroom 100 Mayo Memorial
Tuesday,	12:30 to 1:20 P.M.	PATHOLOGY 104 Jackson Hall
Thursday,	11:30 A.M. to 12:30 P.M.	TUMOR Todd Amphitheater University Hospitals
Friday,	7:45 to 9:00 A.M.	PEDIATRICS McQuarrie Pediatric Library, 1450 Mayo Memorial
	8:00 to 10:00 A.M.	NEUROLOGY Station 50, University Hospitals
	9:00 to 10:00 A.M.	MEDICINE Todd Amphitheater, University Hospitals
	1:30 to 2:30 P.M.	DERMATOLOGY Eustis Amphitheater University Hospitals
Saturday,	7:45 to 9:00 A.M.	ORTHOPEDICS Powell Hall Amphitheater
	9:15 to 11:30 A.M.	SURGERY Todd Amphitheater, University Hospitals

For detailed information concerning all conferences, seminars, and ward rounds at University Hospitals, Ancker Hospital, Minneapolis General Hospitals, and the Minneapolis Veterans Administration Hospital, write to the Editor of the BULLETIN, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14, Minnesota.