THE

BULLETIN

OF

The Vancouver Medical Association

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VOL. XXVI — AUGUST, 1950 — NUMBER 11

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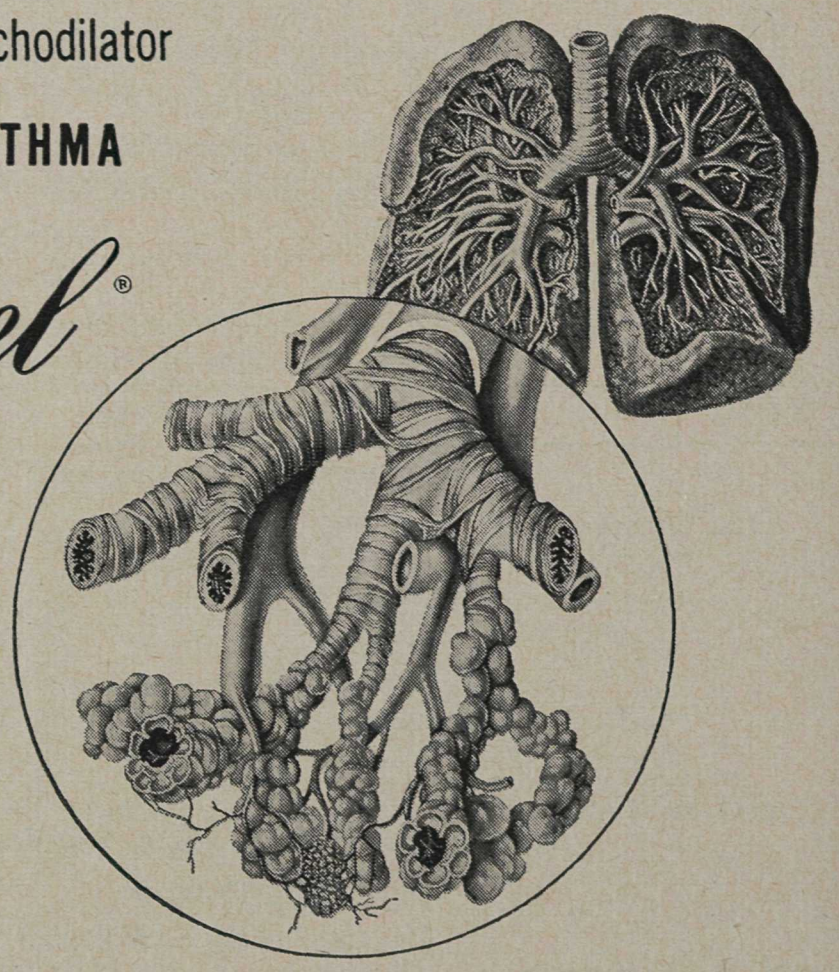

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VANCOUVER MEDICAL ASSOCIATION
PROGRAMME FOR THE FIFTY-THIRD ANNUAL SESSION
(Fall Season)

Founded 1898; Incorporated 1906.

OCTOBER 3rd—GENERAL MEETING—Speaker: Professor G. W. Pickering, Professor of Medicine, University of London. Subject: "Pain in Peptic Ulcer."

OCTOBER 13th—SPECIAL MEETING—Speaker: Dr. Donald Hunter, London Hospital, London, England.

NOVEMBER 7th—GENERAL MEETING—Speaker: Mr. L. Detwiller, Commissioner British Columbia Hospital Insurance. Also time devoted to Medical Economics.

NOVEMBER—ANNUAL DINNER (date and place to be announced).

DECEMBER 5th—GENERAL MEETING—Speaker: Dr. Sydney M. Friedman, Professor and Head of the Department of Anatomy, University of British Columbia.

All General Meetings will be held in the Auditorium,
British Columbia Tuberculosis Institute.

CLINICAL MEETINGS, which members of the Vancouver Medical Association are invited to attend, will be held each month as follows:

SECOND TUESDAY—SHAUGHNESSY HOSPITAL.

THIRD TUESDAY—ST. PAUL'S HOSPITAL.

FOURTH TUESDAY—VANCOUVER GENERAL HOSPITAL.

FIFTH TUESDAY (when one occurs in the month)—CHILDREN'S HOSPITAL.

Notice and programme of all meetings will be circularized as usual by the Executive Office of the Association.

THE BULLETIN

Publishing and Business Office — 17 - 675 Davie Street, Vancouver, B.C.
Editorial Office — 203 Medical-Dental Building, Vancouver, B.C.

The Bulletin of the Vancouver Medical Association is published on the first of each month.

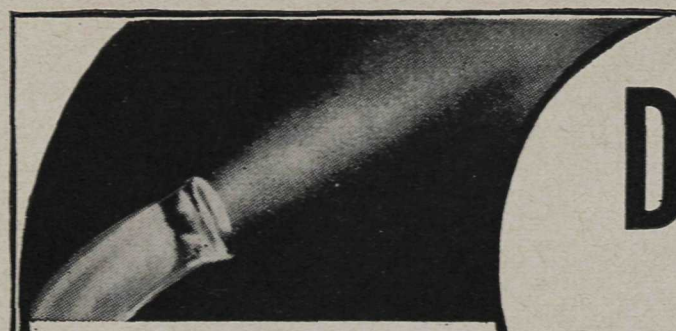
Closing Date for articles is the 10th of the month preceding date of issue.

Manuscripts must be typewritten, double spaced and the original copy.

Reprints must be ordered within 15 days after the appearance of the article in question, direct from the Publisher. Quotations on request.

Advertisements

Closing Date for advertisements is the 10th of the month preceding date of issue.
Advertising Rates on Request.



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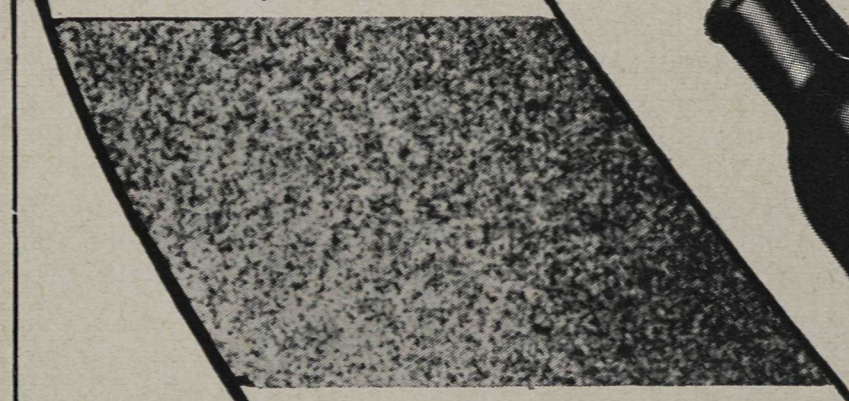

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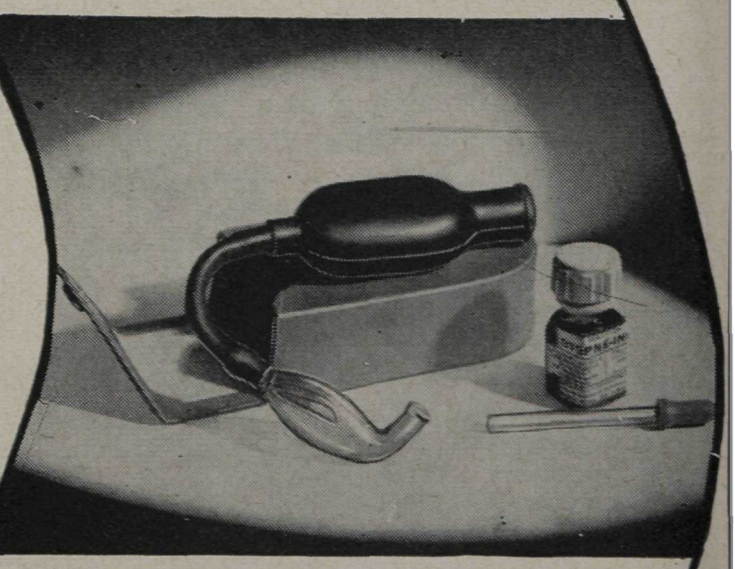

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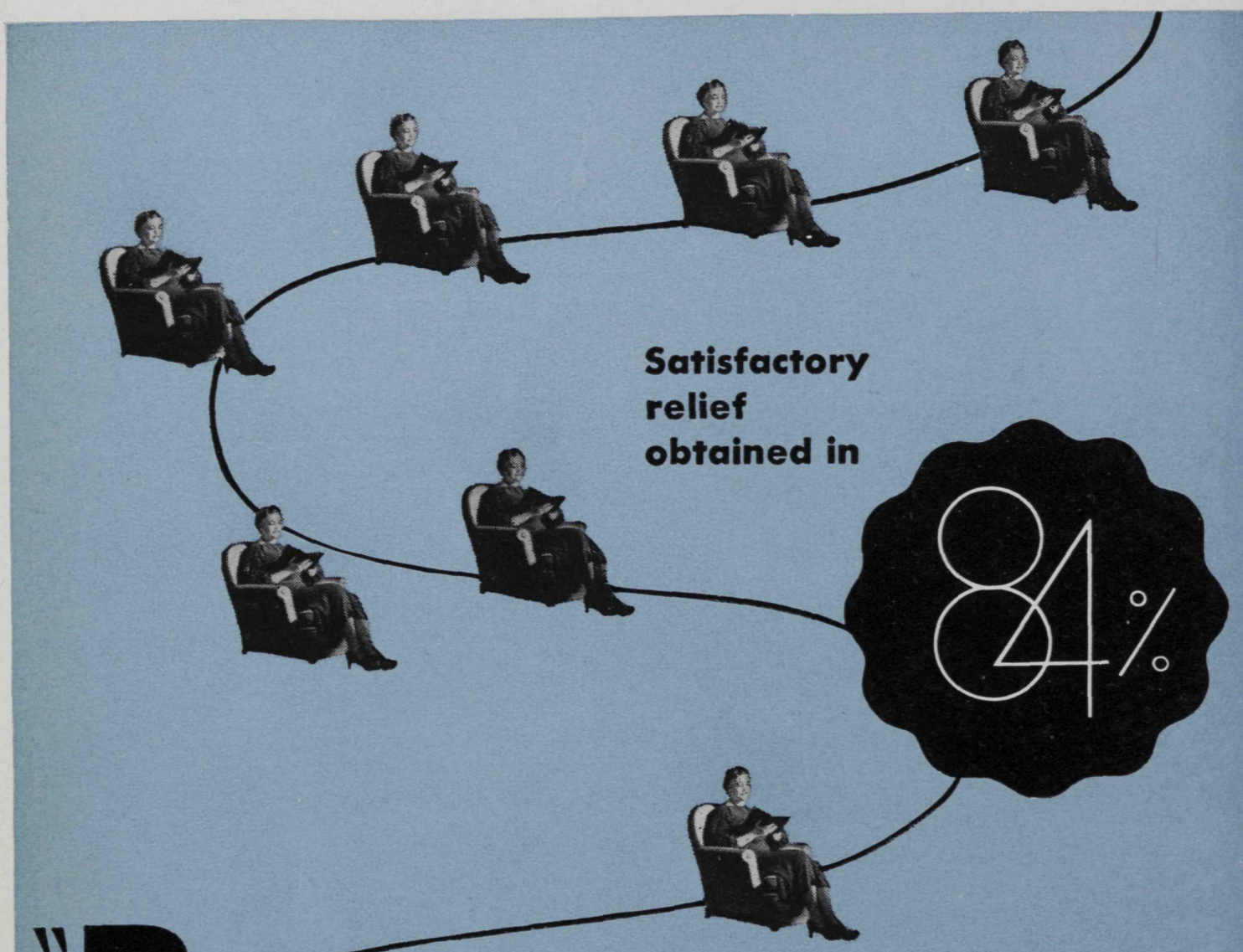

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Freed, S.C., Eisin, W.M. and Greenhill, J.P.: J. Clin. Endocrinol. **3**:89 (Feb.) 1943.
Fried, P.H. and Hair, Q.: J. Clin. Endocrinol. **3**:512 (Sept.) 1943.
Glass, S.J. and Rosenblum, G.: J. Clin. Endocrinol. **3**:95 (Feb.) 1943.
Gray, L.A.: J. Clin. Endocrinol. **3**:92 (Feb.) 1943.
Harding, F.E.: West. J. Surg. Obst. & Gynec. **52**:31 (Jan.) 1944.
Perloff, W.H.: Am. J. Obst. & Gynec. **58**:684 (Oct.) 1949.
Sevringhaus, E.L. and St. John, R.: J. Clin. Endocrinol. **3**:98 (Feb.) 1943.

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VANCOUVER HEALTH DEPARTMENT

CASES OF COMMUNICABLE DISEASES REPORTED IN THE CITY

STATISTICS, MAY, 1950.

Total population—estimated	385,500
Chinese population—estimated	6,877
Hindu population—estimated	133

	April, 1950	
	Number	Rate per 1000 Pop.
Total deaths (by occurrence)	333	10.4
Chinese deaths	9	15.7
Deaths, residents only	319	9.9

BIRTH REGISTRATIONS——RESIDENTS AND NON-RESIDENTS:
(includes late registrations)

	April, 1950
Male	407
Female	403

INFANT MORTALITY——RESIDENTS ONLY:

	April, 1950
Deaths under 1 year of age	18
Death rate per 1000 live births	30.4
Stillbirths (not included in above items)	7

CASES OF COMMUNICABLE DISEASES REPORTED IN THE CITY

	April, 1950		April, 1949	
	Cases	Deaths	Cases	Deaths
Scarlet Fever	10	—	2	—
Diphtheria	—	—	—	—
Diphtheria Carriers	—	—	—	—
Chicken Pox	157	—	319	—
Measles	312	1	245	—
Rubella	556	—	45	—
Mumps	616	—	58	—
Whooping Cough	16	—	—	—
Typhoid Fever	1	—	1	—
Typhoid Fever Carriers	—	—	—	—
Undulant Fever	—	—	—	—
Poliomyelitis	—	—	—	—
Tuberculosis	58	10	47	11
Erysipelas	—	—	—	—
Meningitis	1	—	1	—
Infectious Jaundice	—	—	—	—
Salmonellosis	4	—	2	—
Salmonellosis Carriers	—	—	—	—
Dysentery	1	—	—	—
Dysentery Carriers	—	—	—	—
Tetanus	—	—	—	—
Syphilis	17	4	29	1
Gonorrhoea	120	—	148	—
Cancer (Reportable)—Resident	103	59	96	44

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Recent References:

Stats, D., and Neuhof, H.: Am. J. Med. Sci., 1947, 214: 159.
Walker, J.: Surgery, 1945, 17: 54.
Cosgriff, S. W., Cross, R. J., and Habif, D. V.: Surgical Clinics of North America, 1948, 324.
De Takats, G.: J.A.M.A., 1950, 142: 527.

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The Editor's Page

This is an eventful year in medicine in British Columbia. In September, the British Columbia Medical Association will celebrate its fiftieth anniversary. A special booklet is in preparation, in commemoration of this event. It had been hoped that Dr. Hill, the General Secretary of the British Medical Association, would be one of our chief guests —But this has proved to be impossible, as events in England connected with his work have made it necessary for him to remain there. The programme, however, which will appear in this number of the Bulletin, is a very attractive one.

Then the opening of the new Medical School also in September, is an event of a significance which we all appreciate. This medical school has been long in coming, and its path will not be an easy one at first. But there is no question that British Columbia should have a medical school of its own, and we have ample clinical facilities to make of it a school equal to any. We are fortunate in the appointment of Dr. M. M. Weaver as Dean of the Medical Faculty. Those of us who have heard him speak on various occasions, can recognize in him a man of vision, with definite views of his own as to the type of medical school he wants to see established. He has laid emphasis, on various occasions, on two most important points, as we think. One is the need for good general practitioners, who will fill the crying need for family doctors, a need which has become more and more obvious of late. Another is the importance of research in connection with the medical school. No good medical school today, we gather from his words, can continue to grow and improve its standards, unless there is constant opportunity for research. It is also ncessary to integrate this research with the clinical practice of medicine, and this is not as easy as it sounds. In a very thoughtful address, Dr. Walshe brings out this point very clearly, shewing the urgent need for integration of the clinical side of medicine with the research that is now so widely carried on. To the new medical school, we extend a heartfelt welcome. We believe that it is starting under the happiest auspices.

A third happening of considerable importance to our medical life is the building of the new Academy of Medicine in Vancouver, which will act as a central focus for all our medical activities. This has been made possible by the wise vision of the College of Physicians and Surgeons, through its Council. It will collect into one area all our various departments—Library, Administrative Offices, the Bulletin, and so on. It will be an institution of which we can all be proud. In due time, we are told, an Auditorium adequate to our needs will be built. In this connection, we acknowledge with gratitude, the generosity of the T.B. Institute in offering us the use of their splendid auditorium. This has been a most kindly and gracious act. But it seems to us that, as time goes on, and the work of the Medical School develops, this auditorium will be more and more needed for the School's use—and in any case, we feel that we should have an auditorium of our own.

It is a pity that this year also has other aspects less happy, more dismaying, in fact, than these. But in the meantime, we must carry on with the work that must be done—and hope and pray that we shall have the peace and the opportunity to develop it all.

LIBRARY NOTES

Hours During the Summer Months:

Mondays to Fridays ..... 9:00 a.m. to 5:00 p.m.
Saturdays ..... 9:00 a.m. to 1:00 p.m.

The Library Committee wishes to draw the attention of members of the Association to the following special services offered by the library:

Messenger Service

There is a messenger service for the delivery and pick-up of books from the library to doctors' offices in the city area of Vancouver. For those outside this area and for associate members in the Province, there is a mailing service, postage being paid both ways by the library.

Microfilm and Photostat Service

For members wishing to see articles in medical and surgical journals not available in the library, there is an excellent microfilm and photostat service from the Army Medical Library in Washington, D. C. Microfilms can be obtained free, but, must be returned within three months to the Army Medical Library, otherwise there is a small charge. There are facilities for reading these microfilms in the library. Photostat copies can be retained, but, there is a charge of 50c for each five pages of an article. It takes about three weeks to obtain these articles, so the Library Committee urges members to make any requests to the Librarian in good time if they require such references for a paper, etc.

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BOOK REVIEW

MEDICINE AND HEALTH IN THE SOVIET UNION, by Henry Sigerist
New York: The Citadel Press, 1947 pp. 364 illus.

This book is written by a physician who is one of the few who has been permitted to make an extended survey of medicine in a vast country where access has been difficult and is now almost prohibitive. He has equipped himself for the task by spending three years in learning Russian and studying the literature before visiting the U.S.S.R. In 1935 he spent some months in this country and in 1936 returned again to the U.S.S.R. for further study of this medical problem. He states that he returned with boxes full of literature and statistics furnished by the government of the U.S.S.R. He therefore, in writing as an historian, speaks with a great deal of knowledge and his former advocacy of State Medicine has been accelerated. In reading the book, one has the impression that the historian in his zeal has become, to some extent, a propagandist for the type of social medicine existing in the U.S.S.R.

His short review of the background of social medicine in this immense area covering one sixth of the inhabited world, is interesting and instructive. Among others, he brings to mind some of the great medical names of the past, such as Pirogov (1810-1881), Metchnikoff (1845-1916), Botkin (1832-1898), Zakharin (1829-1894) and the incomparable Ivan Petrovich Pavlov (1849-1936). These medical giants, among many others, were educated and developed under the old Imperial regime.

The author's intricate account of Soviet Medicine is interesting and accompanied by a great deal of government statistics and charts. It is difficult for anyone not fully acquainted with conditions to challenge the enthusiastic acclaim of this system and no one wishes to minimize the tremendous achievements of this type of medicine in a vast country hampered by internal revolution and devastated from without by external forces, and through famines.

However, in retrospect, most of us are aware of the struggle of medicine in other countries down through the ages and much that is fine and courageous and scientific has been made possible through individual effort, free thought, international friendship, intermingling of medical ideas of all free nations, and a sense of responsibility by the various governments. One might gather from books of this kind that if such a drastic

social state as the one described, medically, could have full sway, suffering would be greatly decreased, hatred and distrust would be displaced by love and trust, preventable diseases would be conquered and poverty would cease. In other words we could ask for little more. Such conclusions are disconcerting and in the minds of most of us cannot be substantiated.

It should be remembered that in all the civilised world there are tremendous changes taking place. A few of the most obvious are the improvement and control of infections, the improvement in housing and social conditions, a more enlightening and sympathetic interest on the parts of governments, greater knowledge of physiology and bio-chemistry and the improved care and treatment of mentally ill people. All these and many other expanding problems remain to be dealt with by physicians, the government, and the people. A great harvest has been garnered in the past and much remains to be grown and reaped. None of us should be ignorant about past mistakes and failures, but most of us still believe that medical science should be allowed to grow and improve under the blessings of free international exchange of thought, medical colleges not too drastically controlled by changing governments, and by free access to all the discoveries in medicine and allied sciences.

Until Soviet Medicine can extend to the world opportunities for all medical men to see and study for themselves what is taking place in the medical U.S.S.R., and until they send their men of science to discuss freely with the rest of the world all medical and social problems, we cannot be content with the reports of a few, even though they may be as capable as the author.

Professor Sigerist has written a careful, challenging book on one system of medicine. Medical men will be better informed about this side of medical problems if they take time to study his views.

A. H. S.

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ACTH AND CORTISONE SHORT COURSE REPLACING THE SAME IN THE RHEUMATIC DISEASES WHICH HAS BEEN POSTPONED

Recent announcements have made supplies of Cortisone and ACTH available for use in approved hospitals. The British Columbia Medical Association was requested to undertake the supervision of this more extensive use clinically. An August 10th, 1950, your association set up the British Columbia Medical Association Committee on Cortisone and ACTH. The purpose of this Committee is to:

1. Guide the B.C.H.I.S. in a proficient attitude towards the distribution of these hormones to the hospitals of the province.
2. Assist the hospitals to establish minimum standards suitable for safe control of administration.
3. Instruct physicians in the province as far as possible in the use and dangers of these new hormones.

Through the courtesy of the Vancouver General Hospital Committee on Cortisone and ACTH, a short course is to be given on September 24th and 25th, replacing the course previously announced for these dates. Assistance will be given by both St. Paul's and Shaughnessy Hospitals. This course is open to all members of the profession and it is requested that each hospital send at least two men from its community to this course.

TIME: Beginning 9.30 a.m., September 24th and 25th.

PLACE: Auditorium, B. C. Institute of Tuberculosis.

SUBJECT: The Clinical Uses and Dangers of Cortisone and ACTH.

This course is being held prior to the Annual Meeting and there is no charge to members of the Association. Those intending to attend this course please notify the Secretary, B. C. Medical Association, Room 203, 925 West Georgia Street, Vancouver. Telephone: MArine 9634.

THE BRITISH COLUMBIA MEDICAL ASSOCIATION 1950 ANNUAL MEETING

— *Programme* —

WEDNESDAY September 27th	THURSDAY September 28th	FRIDAY September 29th
MORNING SESSIONS		
Chairman: Dr. James Wilson	**Chairman:** Dr. Howard Black.	**Chairman:** Dr. E. Christopherson.
8:30 a.m. Registration.	**8:30 a.m. Registration.**	**8:30 a.m. Registration.**
9:00 a.m. Dr. Hoyle Campbell: "Traumatic Surgery of the Extremities."	**9:00 a.m.—Dr. F. G. Ebaugh.** To be announced.	**9:00 a.m.** Dr. Robt. Williams "Adrenal Physiology and Therapy."
9:45 a.m. Dr. H. B. Atlee: "Chronic Right-sided Pain in Women."	**9:45 a.m.** Dr. Hoyle Campbell: "Rehabilitation—a Medical Problem."	**9:45 a.m.** Dr. J. W. Abbiss: "Banti's Syndrome."
10:30 a.m. Dr. Norman Gosse: "Carcinoma of the Sigmoid and Rectum."	**10:30 a.m.** Dr. Robt. Williams: "Hypertension."	**10:30 a.m.** Dr. Robt. Williams: "Thyroid Physiology and Therapy."
Ten-minute Interval.		

11:30 a.m. Dr. J. W. Abiss: "Cytology in Diagnosis of Cancer."

12:15 p.m. B.C.M.A. Luncheon.
(Banquet Room)
Guest Speaker: Dr. Norman H. Gosse.

11:30 a.m. Dr. H. B. Atlee: "Natural Childbirth and Such Other Considerations in Pregnancy and Labor."

11:30 a.m. Dr. V. Johnston.
"Relationship of the G. P. to Specialists and Hospital."

12:15 p.m. New Board of Directors of B.C.M.A. Luncheon Meeting. (Salon C)

12:15 p.m.—Special Luncheon, Banquet Room, Canadian Club.
Dr. Gorosh, speaker.

AFTERNOON SESSIONS

2:00 p.m. Tour of Medical School, U.B.C.

3:30 p.m. Special Congregation, U.B.C.

5:00 p.m. Sectional Meetings:
Paediatrics—Salon D.

1:30 p.m. Golf Tournament. Shaughnessy Golf Club. Please register for golf early.

2:15 p.m. Round Table Conference.
"New Drugs."
Moderator: Dr. Robt. Williams.
Collaborators: Dr. R. Farquarson, Dr. R. Kerr.

3:30 p.m. Round Table Conference.
"Biopsy Techniques and Interpretations."
Moderator: Dr. J. W. Abbiss.

4:30 p.m. Sectional Meetings.
General Practitioners—Salon A.
Anaesthetists—Hotel Board Room.
E.E.N.T.—Salon D.
Pathologists—Salon E.

EVENING SESSIONS

7:30 p.m.—General Practitioners
Business Meeting.

8:15 p.m.—Public Meeting.
U.B.C. Auditorium.
Subject: "Mental Hygiene."
Speaker: Dr. Ebaugh.

Informal Buffet Dinner Dance—
Stanley Park Pavilion.

7:00 p.m. Assembly

8:00-10:00 p.m. Buffet Dinner. Dancing.

7:15 Annual Dinner (Formal).
The British Columbia Medical Association. (Ladies and Gentlemen.)
Guest Speaker: Dr. N. A. M. MacKenzie, President, U.B.C.

6:30 p.m. Cocktails.

7:15 p.m. Dinner. Banquet Room, Hotel Vancouver.

GROUP PSYCHOTHERAPY IN THE TREATMENT OF ALCOHOLIC ADDICTION

By A. J. SHULMAN, M.D.
Provincial Mental Hospital, Essondale, B.C.

In the past year and a half the staff of the Provincial Hospital, Essondale has been faced with the problem of treating an increasingly large number of patients whose presenting symptom was the excessive use of alcohol. This influx was related to the efforts of Alcoholics Anonymous, who are intensely interested in facilities for the treatment of alcoholic patients. As part of the response to this demand for therapy I initiated a programme of group psychotherapy in June, 1949. Although the programme has been conducted somewhat haphazardly from a research point of view, it has nevertheless been possible to discover a number of facts and principles relating to this type of treatment. It is the observations made and the conclusions derived therefrom that I wish to describe here.

From a theoretical point of view it will be necessary to make certain hypotheses concerning the etiology of alcoholism, the nature of the problem, and the sort of therapeutic approach most likely to be profitable. With these hypotheses in mind, one can then consider the relevance of certain techniques of group therapy to the treatment process. Finally, I should like to say something of my experience in the past year.

I. The Nature of the Problem.

First, let us settle upon an operational definition of the problem. We shall define alcoholic addiction as the continued use of alcohol to a degree that leads to the partial or complete destruction—physically, psychologically, and socially—of the individual as a functioning organism. Myerson (1) makes an additional point that along with the "alcohol addict" we should place the "alcohol dependent", who, although not alcoholic according to the above definition, nevertheless needs alcohol in larger or smaller doses to assist him in any of his functions. This use of alcohol as a means of adjusting to reality is central to the problem. To sum up many views (ignoring as being both pernicious and irrelevant those views of alcoholism as a moral feature) I should say that the alcoholic is a person who has discevered that alcohol is of superlative value as a narcotic in the defence against anxiety, and that it has, by reason of the circumstances associated with its use, by reason of its symbolic meanings, both private and socially shared—it has a unique virtue for him in his particular problems in maintaining self-esteem and in relating to others. There are two corollaries to this statement.

(1) Alcoholism is almost invariably the symptom of a personality disorder—to quote Knight (2), "no excessive drinker is normal and well-adjusted when sober." To this I would add that neither was he well-adjusted even before he entered upon his alcoholic career.

(2) There are certain specific interpersonal variables associated in a cause-effect relationship with the choice of alcoholism as a symptom. By this I do not mean there is *one* type of pre-alcoholic personality—there are a number of routes to the final common path—but I do suggest that it would be possible to predict, in early adult life, an alcoholic future for many apparently social drinkers. As we go on, I shall mention a few of the significant interpersonal variables, particularly those that have relevance to the treatment process.

II. The Therapeutic Approach.

If we are justified in saying that alcoholism is a symptom of disturbed interpersonal relationships, we may then conclude that the ideal approach is psychotherapeutic, for it is only by psychological means that we can alter those characteristic ways of responding that constitute personality. In using the term "psychotherapy", I refer not only to the planned scientific techniques of the physician, but also to the constructive activities of friends and religious advisors, and to the remarkable work of A.A. This

view is not intended to deprecate the use of physical agents; antabuse and conditioning with emetine may be regarded as "crutches" (to use the language of A.A.); yet the crippled must navigate, and if legs are not available and crutches are, the handicapped must use them. Nevertheless, control of drinking is not the heart of the problem; in the first place, because no alcoholic will stop drinking—antabuse or no antabuse—unless he has effected at least some resolution of his personality problems, and secondly, should it be possible to whip him into a non-alcoholic submission, his personality difficulties will then manifest themselves in new symptoms, possibly as destructive, or even more detructive than his previous one.

III. Group as Contrasted With Individual Psychotherapy.

Ideally, in a programme of planned psychotherapy, one uses both individual and group methods. However, in this paper, it is the use of the group as a therapeutic agent that is to be discussed, and I should like to make a number of statements concerning its value in the treatment of this sort of patient.

First, all those therapeutic processes, described in the literature on group-work with other patients, operate with equal force for alcoholics (a) (4). We find the relief of anxiety and guilt through acceptance by other patients, the development of a sense of unity, and the awareness of common problems. Ventilation, abreaction, and the constant inter-action between members themselves and between members and the therapist allow for the recognition of certain relevant problems and the growth of insight. In addition, we cannot ignore that most appealing characteristic of group therapy—that of allowing for the treatment of a large number of patients with limited personnel.

However, certain points are particularly significant. Probably the most important one is the role of group membership and group participation in the life of the alcoholic. Bacon has written several very interesting papers (5) (6) in which he elaborates the concept of the alcoholic as a social isolate, with a life-long history of limited participation in primary face-to-face groups, such as the family, play-group, social club, etc. More insightful patients can verbalize this; they will tell you of their loneliness, their boredom, their lack of group-determined values, their feeling of not-belonging. Possibly at this point it would be profitable to present one hypothesis which would explain the careers of a large number of alcoholics, called "essential alcoholics" by Knight (1) and constituting the majority of the patients seen here. Without going into its genesis, we can say that basically the problem is one of a conflict between passive, masochistic dependency and aggressive, sadistic rebellion (7). It is these latter trends that carry the patient away from his first group—the family. In adolescence, the alcoholic symptom begins, probably in this way. Our future alcoholic makes contact with a group of other adolescent boys, all concerned, as they are at that age, with problems revolving around their assertion of independence from their parents, and the adoption of adult masculine attributes. "Independence" and "masculine' 'are key words; to prove their independence they perpetrate pranks and indulge in minor delinquencies and irresponsible adventures; to prove their masculinity, they exaggerate those easily grasped attributes found found in our culture as part of the adolescent stereotype of masculinity—to name a few, the use of profanity, of alcohol, and the adoption of a Don Juan exploitative attitude to women. Most of these lads, as they acquire more skill in their adult, masculine roles, pass on to productive good living. The alcoholic, faced with the necessity of denying his stronger passive dependent needs, becomes acutely anxious on entering the adolescent group. With great vehemence he seizes those "he-man" attributes—alcohol, profanity, and promiscuity—for they are very reassuring, and from then on uses them compulsively as proof of his manliness. Of the three, alcohol is of special value; in the first place, because even among the so-called adult members of our society it retains its adolescent meaning, "the two-fisted hard-drinking he-man"; secondly, because of its narcotic effect on anxiety, and thirdly because of the possibility, in an intoxicated state, of gratification of repressed dependent or other needs.

In any event, in adolescence, the alcoholic does enjoy group membership, although even then he cannot with security participate fully. Unfortunately, while he continues to be compulsively preoccupied with the problems of establishing his masculinity, his companions move on, leaving him alone. He cannot marry successfully and achieve group membership by establishing a family, because in his attitudes to women he alternates between a masochistic dependence on an over-idealized mother-image and the sadistic exploitation of women utilized as sexual objects.

Ignoring all the variations, we shall return to the main theme. The alcoholic is under-socialized, and group therapy provides him with an opportunity to work out at least some of the problems associated with group living. Just as it was in the adolescent rebel group that the problem of alcohol arose, now in a similar group of fellow-alcoholics, the patient can, either directly or indirectly work on those persistent problems of masculinity and independence. Stating it a little differently—for a large portion of his life his major interpersonal satisfactions have been obtained while sitting around a table, glass in hand, with a group of other drinkers. Thus now he is in a reassuringly familiar situation—the table is the same, the drinkers are there—only the glass is absent. In this way, without a too radical change in the sort of situation to which he is accustomed, he receives training in relating to a group without the assistance of alcohol.

The familiarity of this situation, found in A. A. as well as in the tavern, is important, not only because it functions as an effective bridge from the old to the new, but also because of its value in preventing anxiety, to which the alcoholic has a notoriously low tolerance, and which in individual therapy frequently results in a flight from treatment into an alcoholic bout. Another characteristic of group therapy is here significant—with mutliple interactions transference feelings are weaker, so that the interpersonal relationships are not sufficiently significant to those participating to over-stimulate old anxieties.

To summarize these values:

(1) Working in a group is a necessary experience for the poorly socialized alcoholic.

(2) The group protects his low self-esteem through the sharing of guilt and the recognition of common problems.

(3) As it was the problems associated with the acquisition of masculine role-taking skills in an adolescent group that created the initial alcoholic symptom, so these problems can profitably be re-examined in a similar group, without the use of alcohol.

(4) The familiarity of this type of group experience prevents excessive anxiety and a flight from therapy.

(5) The attenuated transference feelings operating in groups also serve to prevent an excessive affective disturbance.

IV. Group Psychotherapy in This Hospital.

Finally, I shall make a few brief remarks concerning our use of group methods here. Since June, 1949, I have been conducting twice-weekly sessions with the male alcoholics in this hospital. Psychotic patients were, of course, excluded, but otherwise selection was controlled by those fortuitous factors that determine which patients come to hospital and stay long enough to receive such treatment. The majority of the patients were admitted voluntarily, usually brought in by one or two members of the A. A.; their mean age was about 42 years; very few were married and living with their wives; their educational level was primary school; their socio-economic status largely skilled or semi-skilled, lower-middle and upper-lower class. Although, as I intimated, a majority had a personality problem of the sort described, many other kinds of conflicts and resolutions were noted, and in addition the severity of the disturbance varied tremendously.

The group was of the "moving picture theatre" type; membership was continuously changing as patients entered or left the hospital. This was, I feel, extremely undesirable as it prevented the adequate exploitation of all relevant topics, interfered with the

development of group feeling, old members being bored when new ones introduced subjects already discussed.

The therapist's frame of reference was roughly analytical and non-directive, developed particularly from the works of Sullivan, Fromm, Horney, and Alexander. The aims of therapy have already been discussed.

Meetings were of hour-length and were held twice weekly, at first in one of the dormitories where we took seats at random in a circle, and more recently in the study room in the Crease Clinic where we sat around a table. The number participating varied from five to twenty.

In conducting the meetings, the therapist attempted to be non-authoritarian and rather detached. Repressive-inspirational techniques were avoided as being the legitimate property of A. A., and an attempt was made to limit the therapist's participation to a somewhat passive role in which clarification and interpretation were the chief techniques. Accepting, non-judgmental attitudes are extremely important because of the alcoholic ambivalence to critical authoritarian figures.

The presentation of topics for discussion was largely in the hands of the members themselves. It would have been impossible with a shifting population to make specific plans, even if it had been considered desirable. I also found it impossible to make adequate records of what went on and still function effectively as a member of the group. A non-participating observer is required for this.

The results were assessed chiefly by information obtained from A. A., both as to sobriety and increased social effectiveness, which were the criteria of success.

Because of the absence of a suitable control group, and because no truly valid information concerning the success of treatment is available, I cannot present statistical results. Parenthetically, it is extraordinary how few workers in this field are similarly constrained. Such information as I have would tend to indicate that the treatment was moderately effective. A few patients improved remarkably, and I am definitely convinced that their improvement was due primarily to this treatment. A large number are doing quite well, but I suspect that membership in A. A. is the most significant factor in their success. About half are completely unimproved.

With regard to future problems; a controlled research study would, of course, be of immense value. As to the use of therapy, I should like, speaking personally, to see alcoholic patients continue with out-patient meetings avoiding the three factors which I found most destructive.

(1) The unwillingness of patients to stay in hospital for prolonged periods.

(2) The shifting membership.

(3) The heterogeneous population.

It would be necessary to make a careful selection of patients with a view to their homogenity and carry on with this same group over a period of six months to one year. I would anticipate that group therapy carried on in this way would prove to be extremely effective in the treatment ofalcoholism.

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Vancouver General Hospital Section

HAEMORRHAGIC DISEASES

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The patient presents signs and symptoms of an increased tendency to bleed, as shown by petechiae, purpura, subcutaneous haemorrhage, bleeding from mucous membrane surfaces, haemorrhages into body structures or organs. Diagnosis is a matter of establishing, as well as possible, the mechanism of the bleeding, since prognosis and treatment in the haemorrhagic diatheses vary widely. A series of basic and screening examinations are done first in an attempt to establish a diagnosis of the primary illness, (which may or may not be that of a primary haemorrhagic disease), and there are available a series of special examinations to help evaluate abnormalities of the vascular defense of the body and the process of clot formation.

Normally, there are three factors involved in the mechanism of haemostasis.

1. *Extravascular,* having to do with the location of the vessel, and tissue, and tissue tone. Bleeding causes a fall in pressure within the vessel and a rise in pressure in surrounding tissues. Normal tissue tone plus the increased pressure from extravasated blood tends to reduce blood flow through the vessel. In old people whose extravascular tissue tone is reduced, along with elasticity of vessels themselves, petechial haemorrhages are liable to occur.
2. *Vascular,* the size, structure and tone of the vessel. Following vascular rupture there is prompt vasoconstriction and contracture, lasting a few minutes or longer. This is one factor involved in the initially normal bleeding time in haemophilia.
3. *Clot formation,* platelets and other factors. Blood from the damaged vessel mixes with tissue fluids and factors that enhance clot formation. Platelets adhere to the broken intima and agglutinate. Fibrin forms about disintegrated platelets, and a clot forms, retracts, and occludes the vessel lumen.

You will notice in the table (Figure 1), that the classification of the haemorrhagic diseases is in three groups.

1. Those associated with apparent abnormalities of the vascular bed but with normal platelets and essentially normal blood coagulation.
2. Those associated with thrombocytopenia, poor clot retraction and increased capillary fragility.
3. Those associated with manifest abnormalities of clot formation.

In the first group the most notable feature is the increased capillary fragility as shown by the tourniquet test. The proper procedure for this test is worth while noting, since a significant increase in capillary fragility may be missed by employing either the wrong pressure or the wrong time interval. The usual procedure has been to apply a pressure midway between systolic and diastolic level, for five minutes. Actually, it is recommended that a pressure equivalent to 100 mm. Hg. be employed for an interval of TEN minutes. If the systolic level is at or below 100 *then* a point may be chosen midway between the systolic and diastolic levels and the pressure applied at that level. Five minutes after the cuff is removed, petechiae are counted on an area previously marked off, usually about 5 cm. in diameter and a few cm. distal to the anter-cubital fossa. Ten to fifteen spots are considered as the upper level of normal.

1. ABNORMALITIES OF VASC. BED (NORMAL PLATELETS, ESSENTIALLY NORMAL COAGN.)

Disease	Bleeding Time	Coag. Time	Clot Retractn.	Platelet Count	Tourniquet Test	Prothr. Concn.	Prothr. Consumptn.
Hereditary Haemorrhagic Telangiectasia	N	N	N	N	N	N	N
"Thrombasthenia"	☆ ☆ ☆ ☆ or N	☆ ☆ Seom Cases	May be —N	N	☆ ☆ in 50%	N	No Studies Reported
Scurvy	N	N	N	N	☆ ☆ ☆ ☆ ☆ Vit. C N	N	N
Senile Purpura	May be ☆	N	N	N	May be ☆	N	N
Sepsis	N	N	N	N	May be ☆ ☆	N	N
Allergic Purpura	N	N	N	N	N ☆ ☆	N	N

II. THROMBOCYTOPENIA, POOR CLOT RETRACTION, INCREASED CAPILLARY FRAGILITY

Idiopathic Thrombocytopenic Purpura	Usually ☆ ☆	N	Poor	—N	☆ ☆ ☆ ☆	N	Poor
"Secondary" Thrombocytopenic Purpura	Usually ☆ ☆ (Variable)	N	Poor	—N (Variable)	☆ ☆ ☆ ☆	N	Poor (Variable)

III. ABNORMAL CLOT FORMATION

Hypo-Prothrombinaemia	☆ ☆ (Severe)	☆ ☆ (Severe)	N	N	☆ ☆ (Severe)	—N	N
Circulating Anticoagulants	N	☆ ☆	N	N	N	N	No Studies Reported
Haemophilia	N Initially	☆ to ☆ ☆ ☆ ☆	N	N	N	N	Poor
Deficiency of Fibrinogen	N	☆ ☆	May be N	N	N	N	N
Para-Haemophilia	N	☆ ☆	N	N	N	N	Poor

In the second group, increased capillary fragility, the mechanism of which is poorly understood, is again a feature; there is a decreased number of platelets with a poor performance on clot retraction and prothrombin consumption. (Bleeding time is usually increased.) The prothrombin consumption test is rather involved to describe and I would ask Dr. Eden to elaborate on the principle and interpretation later if possible. Quick has confirmed the fact that platelets are concerned with the total conversion of prothrombin to thrombin, and to a lesser extent with the speed of this conversion. Quick describes a "platelet enzyme", derived presumably from platelet disintegration, which is necessary for the formation of thromboplastin from its inactive precursor, thromboplastinogen. It is known that platelets are directly concerned with clot retraction, and decreased clot retraction is usually roughly proportional to the drop in the platelet count. However, false impressions may be given on this test by changes in fibrinogen level, changes in the number of red cells in the blood, or by elevated plasma proteins, and this must be born in mind when interpreting results.

In Idiopathic Thrombocytopenic Purpura, it is thought that because of the deficiency of platelets there is a resultant deficiency of "platelet enzyme" and apparent failure to form a platelet plug in the damaged vessel.

Prothrombin consumption is low in those diseases where something interferes with the production of thrombin from prothrombin. Thus, in haemophilia, where the plasma thromboplastin concentration is considered to be extremely low, only small amounts of prothrombin will be able to be converted into thrombin. In Thrombocytopenia, the serum thromboplastin is produced only in small amounts, due to either insufficient numbers of platelets, or alteration in platelet function, or both, resulting in a reduction in the amount of "platelet enzyme" which is necessary in the activation of thromboplastinogen.

Very recently different investigators have discovered substances which may be grouped as "prothrombin accelerators" and which also seem concerned with conversion of prothrombin to thrombin. In the disease Owron describes, and calls "parahaemophilia", these substances are not present in high enough concentration; with a resultant prolongation of coagulation time, poor prothrombin consumption, but a normal bleeding time. Administration of whole blood in this disease does away with all clinical and laboratory abnormalities.

In Haemophilia the coagulation time is also characteristically prolonged, not, this time, owing to lack of "prothrombin accelerators", but presumably owing to a deficiency of a plasma factor, probably thromboplastic in nature, or also, or else to a peculior stability of the paltelets as compared with normal platelets. The role of platelets in Haemophilia is still in question.

The test for Prothrombin activity is important in following Dicumarol therapy and may also be used in following vitamin K therapy.

There has recently been a good deal of investigation concerning the use of vitamin P in conditions where capillary defects are present. Opinion differs as to whether the compounds grouped under this name function as vitamins or not. The many types of vitamin P have a similar structure and may be regarded as derivatives of Flavone. Rutin is only one derivative. Flavones occur widely in nature, in citrus fruits, tobacco plants, buckwheat, etc. Not all of them have vitamin P activity however.

Little is known about the metabolism of these substances in the body. Pharmacologically, they differ in action, some are inert intravenously, others have a depressor effect credited to arteriolar dilatation, and one particular group seems to have a specific effect as a stimulator of vasoconstriction. Toxicity appears low. Rutin has been given in doses up to 500 mg. daily without toxic effect.

There are several theories about the mechanism of action of these substances in exxerting a stabilizing effect on capillaries. Actually, little is known. The simplest theory is based on the finding that blocks of agar immersed in a vitamin P solution absorb less fluid than those in an ordinary medium. It is therefore postsulated that vitamin P

acts to decrease capillary permeability by decreasing the permeability of the ground substances binding the endothelial cells together. There is a great gap in our knowledge between the administering of vitamin P and our observation of the effect.

Several types of vitamin P have been clinically employed in diseases where capiliary fragility has been demonstrated or suspected. In *allergic states,* it is generally agreed that there is a capillary weakness that may be corrected by administration of vitamin P. The cause of the capillary dysfunction is unknown; several theories can be postulated: it may be that there is excessive activity of trypsin, which is said to be capable of digesting the ground substance binding the endothelial cells together, or it may be due to liberation of hyaluronidase with resultant increased permeability of that ground substance. The liberation of hyaluronidase has been previously cited as a factor in allergic purpuras, Henoch's or Schönlein's purpura, rheumatic fever, etc.

In *Haemorrhagic diathesis.*

In Thrombocytopenic Purpura, Leukemia, and Aplastic Anaemia, although coagulation disturbances are generally considered to be the prime cause of bleeding, several workers have regarded the platelet factor as secondary to a capillary defect. Even in haemophilia it has been suggested that a capillary disturbance, the exact nature of which is not known, may be of importance. McFarlane has described structural changes in the capillaries of adults suffering from vascular and thromocytopenic purpura. Normally, as previously mentioned, capillary contraction follows on injury; in the above conditions, this contraction fails to occur.

In vascular purpura, and Hereditary Haemorrhagic Telangiectasia, vitamin P has been able to arrest bleeding. A simple mechanical weakness seems to operate in these conditions, and may be reinforced by vitamin P.

But in *Thrombocytopenic Purpura,* where a multiplicity of factors seems involved, vitamin P has proved to be of no value. The compounds used so far, do not stimulate vascular contraction; in view of McFarlane's findings, it would be interesting if the group of vitamin P substances with that particular action be tried.

In a single case of *haemophilia,* Raunert claimed that two i/v injections of vitamin P, given several hours apart, were followed within twenty-four hours by cessation of bleeding; but as on all occasions on which multiple therapy is given, the question of which agent produced the clinical response, is raised.

In hemorrhage following *radiation,* where capillary damage appears to be one factor in the causative role, vitamin P therapy with Rutin seems to reduce the danger of hemorrhage and has been suggested as an adjunct, in a prophylactic role, to radiation therapy.

In *Diabetic retinitis,* several types of vitamin P have been used to control the retinopathy, but the results have not been dramatic. Objective improvement has not been obtained, but it appears that the degenerative process has been arrested in patients whose capillary fragility returned to normal. According to some workers, this may not be achieved until many months of intensive therapy have elapsed. It appears, then, that in diabetic retinopathy, little can be expected from vitamin P therapy unless large doses are administered indefinitely.

In reference to *Dicumarol therapy,* it has been shown that even when Dicumarol produces haemorrhage, there is no increase in capillary fragility; and bleeding does not necessarily parallel prolongation of coagulation time. Quick and others have stressed a vascular component in the institution of bleeding; and other investigators have found, in dogs given Dicumarol experimentally, capillary dilatation. Rutin and Esculin, both types of vitamine P, have failed to prevent hemorrhage in this connection and the use of the vitamin P group which has been shown to effect vascular contractility, would be interesting, again in this case.

In case of *bleeding of obscure origin,* Rutin gave a favourable response where the capillary fragility was found to be the only abnormal finding.

With regard to treatment in *Haemophilia*, an antihaemophilic globulin has been under study by a Boston group, who found it to act as a local haemostatic when applied in powdered form to the bleeding point. Another globulin preparation from human, bovine, rabbit, or swine plasma has a strong haemostatic effect and is superior to the other globulin in that its effect is more immediate. Apparently the rabbit haemostatic globulin does not cause untoward local or systemic reactions.

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THE CANADIAN MEDICAL PROTECTIVE ASSOCIATION

The following impressions were gained by your Executive Secretary at the Annual Meeting. The general calibre of work done by the profession does not allow much hope of successful claims against Doctors, especially if the following cardinal points are observed in all cases:—

(1) Carefully written history and notes.

(2) Adequate X-Ray examination where indicated.

(3) Consultation when in doubt.

During the past year, there have been an increasing number of legal actions. Several cases involved the recovery of gauze sponges following a previous operation. The old excuse that a sponge count is not routine in a hospital no longer can be accepted. The surgeon can demand that there be one routine only at his operations; he must insist that the record of the count be written in ink, to be signed by the nurses reporting to him, and that the record be kept where it will be readily available if needed.

Court action has been taken against the estate of deceased medical men. It was, therefore, suggested that all members have a clause included in their will instructing the estate trustees to pay the Canadian Medical Protective Association fee for one year after death. The Statute of Limitations at the end of this period will then protect the estate against claims.

The same situation applies to medical men who retire from practice; they should retain their membership in the Association for a year after retiring from active practice.

With regard to men who are doing post-graduate work in the British Isles, and who take out medical protective insurance while overseas, this should be continued with the Insurance Company for a year following their return to this Country.

During the past year, assistance has been extended to one hundred and twenty-seven members of the Protective Association. This, together with rising costs of doing business, greater court costs, and legal fees, necessitates an increased fee. It was therefore recommended that, effective January 1st, 1951, the Annual Fee be increased to $10.00.

The Association is in no financial danger at present, and its indemnity reserve is barely touched, but the changing conditions must be recognized and appropriate action taken.

LYNN GUNN, M.D.
Executive Secretary.

OFFICE AVAILABLE

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BLOOD DERIVATIVES

FREDERIC C. MOLL, M.D.

Presented at Paedriatic Refresher Course

With the War came the impetus to obtain a stable, concentrated blood substitute for use in traumatic shock. As a result of this, the blood fractionation program was developed and opened up an entirely new field in regard to therapy with blood derivatives. The primary objective of the investigation was to obtain human serum albumin which would be easier and quicker to use on the battle field than whole blood or plasma, and it is only recently that the other derivatives of blood have received extensive attention.

It is of interest to review the background of the development of these plasma products before discussing their therapeutic uses. Blood is a much more complicated material than the homogenous looking fluid which is drawn from patients' veins. It can be separated easily into three layers by the centrifuge, where one obtains plasma, a layer of white cells and platelets, and the red cells. The plasma from the blood can be further separated into the three main fractions: fibrinogen, which supplies the matrix in a blood clot; the globulin fractions, which have interesting and specialized uses; and the albumin, which makes up at least half of the plasma protein in the normal individual and which controls, for the most part, the balance of fluid between the tissues and the circulation. This simple fractionation is done in most hospital laboratories using saturated solutions of salt. This is, indeed, the most commonly used method of fractionation. It was found to be an undesirable method for obtaining albumin for therapeutic purposes, as it must be done at room temperatures which allows extensive bacterial contamination, and also because the concentration of salt require dialysis before the albumin can be used for therapeutic purposes. To obviate the difficulties involved in salting out proteins, a method was devised by Cohn[1] and his associates at the Pilot Laboratory at Harvard University, which fractionated serum proteins in large quantities by using different alcohol concentrations at very low temperatures and varying the pH Fractionation in this manner permitted large quantities of pure albumin as well as other blood fractions to be obtained.

The electrophore apparatus, which consist fundamentally of determining the relative mobility of plasma proteins in an electric field, produces a pattern which is a representative of the protein fractions in the serum[2]. This electrophoretic pattern permits a qualitative and quantitative evaluation of a particular serum protein but is not a method which is used in the production of large quantities of blood fractions. The method of Cohn allows for large scale production of those fractions demonstrated electrophoretically by Tiselius.

With this as a background of the various plasma proteins and their separation, what functions do they have and of what use are they? The first derivative to be separated is called Fraction I. This consists primarily of fibrinogen and antihemophilic globulin. The latter is presumably the substance which is missing in the hemophiliac and when added in vitro to the blood from the hemophiliac patient, a clot rapidly occurs. Therefore, it was immediately tried on hemophiliacs and found to be usually effective in producing a normal clotting time[3]. This apparently is as effective as fresh blood in treatment, but because fresh blood is not always easily obtainable, and a large quantity is necessary, Fraction I can be given in a concentrated form and therefore in small volume. It must be realized, however, that it is not at present one hundred percent effective. Fraction I also contains fibrinogen, which, with the addition of thrombin from Fraction III, forms fibrin film or fibrin foam. Fibrin foam may be used as an absorbable tampon for hemostasis. Fibrin film is used as a dural substitute in neurosurgery as described by Ingraham and Bailey[4].

The next fraction precipitated in Fraction II which contains the gamma globulin. The gamma globulin contains most of the antibodies in the blood. If one compares the antibodies present in the gamma globulin to that in plasma or even in convelescent serum from a particular disease, one is able to demonstrate a much higher concentration

of antibodies. For example, the complement fixing antibodies of mumps is somewhat higher in gamma globulin than it is in plasma and many times higher in the gamma globulin from convalescent serum than in convalescent serum itself. The same is true with streptococcus antitoxin, typhoid antibodies, pertussis antibodies, influenza antibodies, diphtheria, and so on[5]. Therefore, it is perfectly obvious that the most efficient way of administering antibodies for passive protection is through the gamma globulin.

Pediatricians believe that the infant is protected from a number of infections during the first six months of life by the passive transfer of maternal antibodies. Therefore, one would expect the gamma globulin to protect not only against measles but against other diseases which do not occur in the first six months of life. Although there seems to be an immunity to mumps in the first six months, there is no protection with the gamma globulin. There certainly is prevention of infectious hepatitis with gamma globulin, but whether or not the disease occurs in the first six months of life is not known. At present, German measles and chickenpox are under investigation and no conclusions have been obtained. Obviously, the immunological problem is more complicated than at at first appeared.

Gamma globulin is used mostly against measles, and it is rather interesting to note that the results vary directly with the time at which it is given and the amount of the globulin which is administered. If a large dose is given early after exposure, there is complete prevention, and a smaller dose, at the same time, would cause modifications. Whereas, if a large dose is given at a later time, around five to eight days after exposure, there will be modification only, and a smaller dose will produce no alteration at all. It is most useful in the modification of measles so that a very mild disease will result but the permanent immunity will be established[6]. In general, 0.1 c.c. per pound is given for prevention and .02 c.c. per pound is given for modification. The possibility that this latter dose is too large for modification is now under investigation and perhaps there will be more information soon[7].

The last derivative of blood which is to be discussed is albumin, which is obtained from Fraction III. This contains nearly half of the plasma protein. This was the original end for which the plasma fractionation program was established. It is extremely useful in maintaining blood pressure in individuals who have shock from blood loss as in wounds or burns, but it is also useful in those individuals who have hypoproteinemia, either idiopathic or from the loss of serum through such things as severe eczema[6]. One thing should be mentioned and that is the use of albumin in the nephrotic syndrome whicn is of great interest to all pediatricians. It is probably the one specific diuretic agent which can be used with some expectancy of success. Although not all individuals respond, especially those with definite evidence of chronic nephritis, many of them do, but the response terminates when the albumin is stopped. It is useful for temporary mobilization of the individual but it should not be used in the presence of hypertension, azotemia, or considerable hematuria. As the effect is transient and the material expensive, it is not widely used except in cases of extreme urgency.

The only other portion of the blood which should be mentioned is the red cell. If the plasma is removed, red cells are left. Are these to be thrown away or do they have a specific purpose? Patients with anemia but no hypoproteinemia can be treated more efficiently by giving them merely the red cell rather than loading them with plasma protein. This is illustrated in the treatment of the anemia which develops with rheumatic fever when one is hesitant to overload the circulatory system.

Certainly, one should use specific therapy for specific deficiencies. If this can be worked out satisfactorily, many individuals can profit from a single unit of blood, whereby, under ordinary circumstances if whole blood is given for only one purpose, it eliminates using the other products of that unit of blood for any other individual.

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NON-SPECIFIC MESENTERIC LYMPHADENITIS

By DOUGLAS TELFORD, M.D., F.R.C.S. (EDIN.)
and
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Former Resident in Paediatrics, Children's Hospital.

INTRODUCTION:

Mesenteric lymphadenitis in children was first described by Sydenham in 1723. Little, however, was done to differentiate the non-specific lymphadenitis from the specific until Wilensky (1) in 1920, and Brennemann (2) in 1921 described in detail the non-specific variety. In recent years many authors have emphasized the importance of non-specific mesenteric lymphadenitis and have urged that the disease be kept in mind whenever pain is encountered in children.

INCIDENCE:

The incidence of non-specific mesenteric lymphadenitis is best appreciated when compared with that of appendicitis. Neilson (3) maintains at the age of 4 to 6 the incidence of each is equal, and that before these years, lymphadenitis occurs more frequently and after these years appendicitis is the disease most frequently encountered. The condition is seldom seen past 20 years. The sexes are equally affected.

ANATOMY:

The lacteals of the small intestine drain into 100 to 200 lymphatic glands scattered throughout its mesentery. These glands are arranged in three groups: (1) the juxtaintestinal close to the walls of the intestine; (2) the middle, in relation to the loops and primary branches of the ileal and jejunal arteries; (3) the central glands at the root of the mesentery.

The lymphatic drainage of the appendix and caecum is independent of that from the small intestine, passing as it does along an ileocolic chain of lymphatics.

ETIOLOGY:

No comment will be made on the various theories which have been put forth to explain the etiological basis of non-specific mesenteric lymphadenitis. A generally accepted premise is that some unknown bacteria or toxin passes from the wall of the intestine via the lacteals to inflame the mesenteric lymph glands. A few investigators believe the appendix is the portal of entry of the unknown bacteria or toxin but authorities such as Wilensky and Hahn (4), Goldberg and Nathanson (5), and Aird (6), find little or no evidence anatomically or pathologically to justify this opinion. The authors of this paper believe that the appendix, normal or diseased, has little or nothing to do with the non-specific variety of mesenteric lymphadenitis.

CLASSIFICATION:

Cases of non-specific mesenteric lymphadenitis may be chronic or acute. Many of the chronic cases have acute exacerbations and many of the acute pass through a prolonged phase before resolution is complete. A classification, nevertheless, is desirable and the following is both simple and convenient.

(1) Chronic mesenteric lymphadenitis.
(2) Acute mesenteric lymphadenitis.
(3) Acute fulminating mesenteric lymphadenitis.
(4) Acute suppurative mesenteric lymphadenitis.

The admissions at the Children's Hospital include many examples of the chronic and simple acute but no examples of the last two varieties. This is not surprising since fortunately the acute fulminating and the acute suppurative are rarely encountered.

CHRONIC NON-SPECIFIC MESENTERIC LYMPHADENITIS

Chronic non-specific mesenteric lymphadenitis is more often than not mis-diagnosed as chronic appendicitis. The most outstanding symptom of the disease is mild recurring pain which is vague in distribution or localized to the right lower quadrant of the abdomen. The pain tends to be colicky rather than aching. The mild pain on occasion is replaced by an acute exacerbation often related to an infection of the upper respiratory tract. Gastrointestinal symptoms other than pain are as a rule minimal.

Physical examination in the chronic stage of the disease may reveal little, but certain signs are helpful in establishing a diagnosis. Tenderness may be restricted to the right lower quadrant of the abdomen higher and more medially than the usual tenderness of appendicitis. When the central glands at the root of the mesentery are involved tenderness along a line corresponding to the mesenteric attachment may be present. This line extends from the right lower quadrant upward and to the left just above the umbilicus. When the juxtaintestinal glands along the terminal few feet of the ileum are involved, the area of tenderness may be found to shift to the left when the child assumes the left lateral position.

Chronic cases, however, are difficult to diagnose by history and physical examination alone. They require, as a rule, investigation and observation over a period of weeks or months to confirm the diagnosis and rule out associated conditions. Foci of infection are searched for. The stool is examined for intestinal parasites and occult blood. The abdomen is examined roentgenologically by scout film and barium enema technique and the kidneys and ureters are investigated by intravenous pyelogram.

The outstanding pathological feature of the abdominal disease in the chronic stage is the enlargement of the lymphatic glands of the small bowel mesentery. The glands vary in size from a pea to a cherry. They are white in colour and from soft elastic to fairly firm consistency. Occasionally there is a small amount of serous fluid free in the abdomen. Histologically the glands present a non-specific hyperaemia and cellular hyperplasia. They and the free fluid are sterile on culture.

Case 1:

J. B. was a female patient age 3 years. She was admitted April 28th, 1948. The salient feature of the history was 12 months of bouts of colicky abdominal pain. Examinations repeated over a period of two months were often negative. Occasinally, however, slight tenderness over a line corresponding to the root of the mesentery was present. Special investigation such as barium enema and intravenous pyelogram were negative. Laparotomy was performed and no abnormalities were encountered except for grossly enlarged mesenteric lymph glands, the largest being the size of a small walnut. No gland was removed for biopsy but appendectomy was performed. The mother was informed of the appendectomy and was told the true nature of the disease. She was not surprised when bouts of abdominal pain recurred for two months post-operatively. For the past twelve months, however, the child has been entirely well.

ACUTE NON-SPECIFIC MESENTERIC LYMPHADENITIS:

The acute variety may or may not give an antecedent history of an acute attack. It is difficult, as a rule, to differentiate it from acute abdominal disease such as gastroenteritis, Meckel's diverticulitis, volvulus, intussusception and appendicitis. There are certain features which help in the differential diagnosis, however. The pain is essentially a general abdominal colic and more intense than that of acute appendicitis. The child moves freely during the pain and in between attacks, when there is complete freedom from pain, he looks well and happy. If asked to point to the area of pain, he points to the navel or an ill-defined area in the right lower quadrant. Nausea is usually present and vomiting may occur.

On examination, the patient appears only moderately ill. The temperature is 99 to 102 degrees and the pulse is correspondingly elevated. The nasopharynx may be moderately inflamed but more often than not it shows little of diagnostic significance. The abdominal tenderness is more pronounced but otherwise similar to that described in the

chronic variety of the disease. Mild spasticity and mild rebound tenderness may be present. The white blood cell count is 15,000 to 20,000, 80% or more polymorphs. Higher counts are not unusual.

The outstanding pathological findings at this stage of the disease are a small bowel mesentery studded with swollen lymph glands reaching the size of a small walnut, periadenitis causing an edema of the mesentery and serous fluid free in the abdominal cavity. Occasionally the terminal ileum, caecum and appendix show a lymphoedema or an acute inflammation. The Peyer's patches of the ileum may be markedly enlarged. This involvement of the intestine in many cases of the acute variety of non-specific mesenteric lymphadenitis has led Crohn (8) to consider this disease as either a precursor of, or the same thing as regional ileitis.

Case 2:

J. G. was a female patient, age 7 years. She was admitted on April 19th, 1949. The history was 24 hours in duration and was suggestive enough of acute appendicitis that her family doctor sent her to Vancouver by speed boat. Pain and vomiting were the salient features with her temperature rising to 102°. Examination revealed slight inflammation of the tonsils. The abdomen was tender over the root of the mesentery and in the right lower quadrant. Tenderness shifted when the child assumed the left lateral position. The white blood cell count was 22,000, with 85% polymorphs. Paediatric consultation confirmed the impression that this case was in reality acute non-specific lymphadenitis. Within three days the child had apparently recovered, and on her fifth day she left for her home 30 miles from Vancouver.

ACUTE FULMINATING MESENTERIC LYMPHADENITIS:

The third type, the acute fulminating non-specific mesenteric lypmhadenitis cannot be differentiated from a rapidly advancing appendicitis. Toxicity is evident, the temperature is 101 to 103 degrees and the pulse is rapid and bounding. The abdomen contains a clear to slightly cloudy fluid which is negative on culture. The small and large intestines are diffusely involved in an inflammatory process especially in the ileocecal region. The mesentery is studded with acutely swollen glands.

ACUTE SUPPURATIVE MESENTERIC LYMPHADENITIS:

The fourth or acute suppurative type is probably an end stage of one of the previous three types, and may proceed to abscess formation or diffuse peritonitis. A microscopic examination of the pus reveals the secondary invader.

TREATMENT:

The treatment of non-specific mesenteric lymphadenitis is primarily medical and not surgical. Frequently, however, laparotomy is undertaken to confirm or establish the diagnosis. Acute cases which cannot be differentiated from surgical lesions of the abdomen are surgical emergencies. Those acute cases, however, which are diagnosed with confidence are treated conservatively unless the need for surgery becomes definitely established.

Acute cases submitted to sugery must be thoroughly explored to rule out those conditions which enter into the differential diagnosis of acute non-specific mesenteric lymphadenitis. The small bowel mesentery and its lymph glands are carefully inspected and palpated. The cecum and the terminal ileum with its Peyer's patches are examined. The appendix is removed and any coincidental abnormalities such as intestinal polyp, Meckel's diverticulum, congenital band or unduly mobile cecum are attended to.

Acute cases conservatively treated should be hospitalized for investigation, observation and treatment. Confirmation of the tentative diagnosis must be made by frequent check-ups to make certain that the case is not in reality acute appendicitis or some other surdgical emergency. This regime requires care, patience, and the occasional loss of a night's sound sleep but it is the one of choice if an increasing number of cases of acute non-specific mesenteric lymphadenitis are to be spared a useless laparatomy and appendectomy.

Chronic cases demand weeks or months of conservative regime. Foci of infection and intestinal parasites are eliminated; malnutrition is corrected; and regular habits of rest, exercise and elimination are established. If this regime fails to bring about a cessation of pain, then surgery becomes necessary. Laparotomy is carried out as in the acute variety.

Special attention is given to those cases tentatively diagnosed as non-specific mesenteric lymphadenitis showing a positive Mantoux reaction. If the child is under ten years of age and more especially if he is under six years of age, the conservative regime outlined above is modified to include complete bed rest. Exploratory laparotomy is indicated in these cases unless the immediate response to conservative therapy makes the possibility of a tuberculous abdominal lesion remote.

PROGNOSIS:

The prognosis of non-specific mesenteric lymphadenitis is, as a rule, excellent. The simple acute cases recover in a few days or weeks, and chronic cases in a few months or years. The acute fulminating cases very rarely are fatal and the acute suppurative occasionally are.

SUMMARY:

(1) Non-specific mesenteric lymphadenitis is an important disease of childhood and adolescence and must be kept in mind whenever abdominal pain is encountered in children.

(2) The etiology of the disease is still unknown and although some investigators believe the appendix is the portal of entry of some unknown bacteria or toxin, little or no conclusive evidence has been produced to support this opinion.

(3) The outstanding pathological feature is the inflammation of the lymphatic glands of the mesentery of the small bowel. Additional features which may or may not be present are lymphoedema or inflammation of the terminal ileum or caecum, enlargement of Peyer's patches, oedema of the mesentery, and fere fluid in the abdomen.

(4) A convenient classification is chronic, acute, acute fulminating and acute suppurative.

(5) A clinical diagnosis, on occasion, may be made with reasonable certainty, but more often than not non-specific mesenteric lymphadenitis cannot be differentiated with confidence from other abdominal disease such as appendicitis. Chronic cases are particularly difficult to diagnose and require a thorough investigation and prolonged observation.

(6) The treatment is primarily medical and not surgical. Frequently, however, exploratory laparotomy is necessary to conform or establish the diagnosis and when such is undertaken, appendectomy is performed and any coincidental abnormality such as Meckel's diverticulum or congenital band is attended to.

(7) The prognosis, although varied as to time, is excellent as to ultimate recovery in the chronic and simple acute varieties.

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INTERPRETATIONS OF SEROLOGY IN THE DIAGNOSIS OF SYPHILIS

by C. L. Hunt, M.R.C.S. (Eng.), L.R.C.P. (Lond.), L.M.C.C.

The diagnosis of syphilis is strangely complicated by many factors, one of which is the paradoxical fact that the specific blood test employed is, in fact, not entirely specific.

When Wassermann, Neisser and others first devised it in 1906, they used as their antigen an extract of foetal syphilitic liver, and no doubt they were considerably gratified when they discovered that their serological diagnosis by this means agreed very closely with their findings by clinical methods. It was not until a year or so later that their complacency was somewhat shattered by the finding that *normal* liver extract—and indeed extracts of other normal tissues—seemed to work equally well as an antigen.

Did this mean that man has some close affinity with a spirochatete?

That discovery occurred over 40 years ago, and we are still using normal tissue extract as an antigen in our Complement Fixation and Flocculation tests for Syphilis. We still appreciate that the tests are essentially non-specific, and we are still unable to explain the phenomenon on a satisfactory basis.

This very non-specificity is one of the factors which tends to lead us into pitfalls in diagnosis, largely because there are many who lose sight of the fact, or forget, that these reactions are liable to be affected by conditions other than syphilis.

The antigen used in our tests today consist of an alcoholic extract of normal tissue, usually beef heart, to which cholesterol and lecithin have been added. Why does this extract of normal tissue react in apparently the same way as we should expect syphilitic antigenic material to act? The possible theories that have been brought forward to account for this are many, among which the following appear to claim current popularity:

1. Possibly the destruction of large numbers of spirochaetes liberates lipid substances which bear a close resemblance to those present in alcoholic extracts of normal tissue. Or in other words, death of the human or animal organism, or possibly the actual process of tissue extraction, may liberate or bring about the formation of breakdown products which are themselves closely allied to the breakdown products of disintegrating spirochaetes. It might be feasible to explain many of the false positive reactions on this basis, if we assume that some other types of infection or tissue damage could cause similar destruction of tissue in the living body.
   This might conceivably then liberate sufficient antigenic material to give rise to a positive Kahn or Complement Fixation reaction.
2. Possibly it is the selective desruction of human tissue in spirochaetal infection, and in some other types of damage or infection, which liberates antigenic substances, rather than the destruction of spirochaetes themselves, for we know that there is very little true antibody formed against the spirochaete, unless the infection has been present over a very long period.
3. We know from the work of Cavelti[3] and others that normal tissue extracts can be so altered by bacterial action that they become antigenic as a result of such action.

It may be that the death of normal tissue or the process of extraction can so alter the nature of these extracts as to render them closely akin to substances produces *in vivo* by the action of spirochaetes, and by certain other disease factors on human tissue.

Whatever the explanation might be, we have very definite evidence that there are other types of tissue damage besides syphilis which will bring about a positive serological test. In spite of this, however, the presence of the spirochaete seems to be of fundamental importance in the production of most positive blood tests *in the living person,* for we do know that, in practically all instances where this is the infecting agent, the Kahn

test will in time be positive, whereas in only a fairly small proportion of the remaining types of tissue injury will this be so.

We shall, in all probability continue to remain in doubt as to the nature of the antigenic factor in syphilis until we can culture the organisms on artificial media in sufficient quantities to enable us to carry out further analysis upon them.

The question is frequently asked: "Is there any association between a positive Kahn test and immunity to syphilis?" It will be recalled that the "Reagin" which is responsible for the positive Kahn and Complement Fixation tests first makes its appearance some six weeks after infection, and the titre becomes progressively higher during the secondary stage, the most highly infectious stage of the disease[4]. In the course of years, in an untreated person, the titre gradually falls again to a comparatively low level. It is believed by many that in these later stages, a slight but real immunity does develop,[5] but that it bears no relationship to the titre of the blood Kahn, i.e. to the presence or absence of reagin circulating in the blood. According to Kolle and Prigg[6] and others however, this immunity is more apparent than real, and constitute a symptom-immunity or tissue-immunity rather than a true immunity in the bacteriological sense.

In experiments with rabbits it has been found that fresh spirochaetes do apparently enter the body, but they do not often give rise to new primary chancres; nor is the spread of the infection so rapid as in a previously uninfected animal[7].

Try as we may to explain the true nature of our tests for syphilis, the fact remains that false positive reactions do occur, and they do tend at times to complicate the true interpretation of our findings in individual cases.

False positive reactions (apart from laboratory errors) are due to the interaction of two factors:—

1. Certain virus infectionns—Smallpox vaccinations.
2. The patient's pattern of reaction[8].

The damaging or infecting agent is usually one which gives a high degree of stimulation to the antibody-forming mechanism of the body[9], such, for example, as:

1. Certain Virus infections—Smallpox vaccinations.
   Respiratory tract infections, Virus pneumonia, Glandular fever, etc.
2. Some bacterial infections, e.g. Scarlet Fever, Leprosy.
3. Other Spirochaetal diseases.
4. Sporozoite-induced Malaria.
5. Diseases of doubtful aetiology, such as Disseminated Lupus Erythematosus, in which an excessive immunity response to some unknown factor may play some part.
6. Conditions in which there is blood loss or blood-destruction, with resulting active replacement of the serum globulins. Certain types of *tissue reaction* also seem to be commonly associated with false positive reactions, and they all seem to be those in which there is a marked reticuloendothelial reaction. In these instances there is often evidence of generalized enlargement of lymph nodes, possibly of spleen and liver, and frequently indirect evidence of bone-marrow involvement, as shown by a tendency to depression of all those blood cellular elements produced in the bone marrow.

There is also, in many of these cases, a tendency for the blood globulin fraction, and particularly of the gamma-globulin, to be increased[2]. A false positive Kahn reaction is therefore occasionally found in those diseases characterized by a rise in serum globulin, irrespective of the nature of the underlying condition.

Many efforts have been made from time to time to eliminate false positive reactions, working on the assumption that the so-called antibodies in the blood of a syphilitic patient and those in other diseases giving a positive reaction are not identical, but are cross-reactive with the same antigen[9]. It is know that only a small fraction of the gamma-globulin is serologically active, and therefore attempts have been made, with some degree of success of carrying out a preliminary euglobulin-inhibition technique.

Efforts have also been made to increase the purity of the tissue extracts used as an antigen in these serological reactions. Cardiolipin[10] is a substance recently produced as a result of these experiments, and reports on its use appear to be highly encouraging. The combined use of cardiolipin with the euglobulin-inhibition technique appears to give promise of a definite diminution in false positive reactions when they ultimately become adopted as standard technique.

In view of all that has so far been said, it will be appreciated that the interpretation of serological findings is not always as simple as it would appear.

Some instances of false positive reactions occur in serious and possibly fatal illnesses. These problems are usuallly resolved on general medical lines sooner or later.

Most false positive reactions occur, however, in diseases which are self-limiting and transient in nature, so that patients so affected, if followed for a period of one to four months, will usually show a gradual return to a negative serology. In some instances, the blood titre will fall more slowly, and may take 10 to 12 months before it ultimately becomes negative. Finally, there is the rare instance of the patient who always shows a positive reaction in low titre, and yet who gives no history or other evidence of ever having had syphilis, congenital or acquired.

From these observations it will be obvious that a single positive blood test, especially if it is present in low titre is not diagnostic of syphilis. There may be a fault in the laboratory testing materials or in the recording. The test should therefore be repeated in a week to reduce the chances of faults in this direction. In the meantime careful history-making and a clinical examination may bring out supportive evidence, as also will an examniation of the cerebro-spinal fluid for cells, protein, complement-fixation and colloidal gold curve. Contacts should also be brought in for investigtion, this to include marital partners and children (if any). If all these investigations prove negative, then a positive blood serology of low titre may have to be followed monthly for several months before definite diagnosis can be made one way or the other.

Where the dignosis of syphilis has been established and treatment given, the Kahn titre may be expected gradually to diminish. If the case is an early one, the blood may be expected to become negative in the course of a few months and to remain so, provided that no subsequent relapse occurs. When, however, treatment is given late in the infection, the blood Kahn may take any period up to two years to become negative. Indeed, it may remain a weak positive for the rest of the patient's life. Provided these patients show no subsequent rise in blood titre, and no evidence of changes suggestive of active syphilis on careful physical and cardiovascular examination or in the cerebro-spinal fluid, then there is no need for any further treatment to be given, except, possibly, in women, during subsequent pregnancies.

Finally, reference may here be made to the Kahn test in infants born in syphilitic mothers. It is to be hoped that all cases of syphilis in pregnancy are being discovered early in pregnancy and adequately treated. In many of these cases, however, the syphilitic "reagin" from the blood of the mother will pass the placental barrier and circulate in the blood of the foetus. At birth, therefore, a cord Kahn should always be done as a routine. A negative result is encouraging, and should be confirmed by another blood test in 6 or 8 weeks if the mother has ever been treated for syphilis. A positive cord Kahn is, in itself, not diagnostic of prenatal syphilis, especially if the mother has been adequately treated.

Careful examination should be made, including an X-ray of the long bones, and the infant kept under observation for any clinical evidence of congenital syphilis. In the absence of such evidence, treatment should be withheld unless further quantitative blood tests (taken at monthly intervals) show a rising titre.

In those instances where the mother has been adequately treated the infant's blood Kahn titre will gradually fall and become negative in the course of three or four months. If, however, the Kahn titre rises during the period of observation, then it must be assumed that prenatal infection has occured and early treatment should be commenced.

Where any doubt still exists in the mind of the attending physician on any problem relating to syphilis, it is strongly recommended that full use be made of the consultative service provided by the Division of V.D. Control, bearing in mind the necessity of giving as much information as possible, to cover:—

History—including results of previous blood tests, and details of previous treatment.

Complete physical examination.

Cardio-vascular findings—including screening and E. C. G. if possible.

Neurological and cerebro-spinal fluid findings.

The matter will then be referred to the appropriate consultant for his opinion, and every possible assistance will be given to the physician in charge of the case.

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News and Notes

BIRTHS

Dr. and Mrs. G. E. Little, of Vancouver, a daughter.
Dr. and Mrs. A. K. Mathisen, of Vancouver, a son.
Dr. and Mrs. Frank Crofton, of Vancouver, a daughter.
Dr. and Mrs. G. J. Kirkpatrick, of Vancouver, a daughter.
Dr. and Mrs. Ross MMcNeely, of Victoria, a daughter.

Dr. A. M. Evans, of the B.C.C.I., has spent the last month in Europe studying advances in cancer therapy.

Dr. Austin Dobrey, of Vancouver, has been elected head of Marpole Rotary.

Dr. J. C. Sullivan, of Vancouver, has gone to Des Moines, Iowa, for post-graduate surgery.

Dr. H. W. McIntosh has been named to investigate results of ACTH therapy in the province. *Dr. Bryan Holmes* will assist him in this work at the Shaughnessy D.V.A. hospital in Vancouver.

Dr. Carl J. Reich has returned from a final year in internal medicine in California and is now on a locum for *Dr. W. S. Kergin,* of Prince Rupert prior to certification.

Dr. J. B. Fenwick is spending a year in London, England, on a Canadian Arthritis and Rheumatism Society Fellowship.

DR. C. H. VROOMAN

Obit.

Another of our elder statesmen of B. C. Medicine has gone, with the passing of Dr. Charles Vrooman, of Vancouver. For many years, Charlie Vrooman has been an outstanding figure in the profession of B. C.—not only for his professional attainments, which were very high, but as one of the leaders of his profession in the province.

Dr. Vrooman was a graduate of the Medical Faculty of the University of Manitoba. Early in his career, he developed an intense interest in tuberculosis: an interest which grew steadily with the years, till he became one of the recognized Canadian authorities in this disese. He was associated for many years with the B. C. Tuberculosis Society, and worked with Dr. C. J. Fagan when the latter was Provincial Medical Health Officer. He was Medical Superintendent of Tranquille Sanatorium, and gradually developed this institution into a great centre for treatment and research. His work at this hospital was outstanding.

Moving to Vancouver, he gradually moved more and more to private practice—and became a recognized leader in diagnosis. He was a court of last appeal, and was constantly in demand as a consultant and diagnostician. He had a judicial mind, and weighed carefully all the evidence in a case before arriving at a decision.

Vrooman did great work in the matter of Silicosis, and his published papers and articles on this subject gained wide recognition. Shortly before his death, he published a most comprehensive report on Silicosis in B. C., in the Bulletin. He was sent to Australia last Fall as a representative of the B. C. Workmen's Compensation Board, and of the Canadian Government, to attend the International Conference on Silicosis. We cannot but be pleased that this high honour of his winning did not come too late.

Amongst his fellows, Charlie Vrooman stood out as a wise counsellor, a man of high integrity, and a most loyal member of his profession, which he served in many capacities, notably as President of the Vancouver Medical Association, and in other offices. He was always ready to serve, and his careful, thoughtful judgment and wisdom were of the greatest value. We shall miss him very greatly.

To his wife and family we extend our deepest sympathy.

DR. A. J. MacLACHLAN

Obit.

For those of us who have been in practice for more than twenty-five or thirty years, one of the most disconcerting things is that whereas one used to know, by name at least, all his fellow-practitioners, nowadays, with the enormous influx of men into the province, it has come to a point where, at a medical meeting or the like, one is lucky if he knows a quarter or a third of those present.

But there was one man, our friend of so many years, Jack MacLachlan, who knew every one of them—and came into constant contact with many of them. In his capacity as registrar, he met every man that came here to practise and we are sure that every man in the province thought of him as a friend, and a helpful friend at that.

It was a big and important job that Jack had to fill—and for many years he has filled it well. Councils come and Councils go—but he stayed on—gathering experience and wisdom which helped these Councils greatly in their deliberations.

Jack MacLachlan was born in Blenheim, Ontario, 69 years ago—took his medical degree at Queen's University, in 1905. He was ship's surgeon with the C.P.R. Empress boats for a year, interned at St. Paul's Hospital and the Vancouver General Hospital; was C.P.R. surgeon for 9 years. He gradually limited his practice to proctology.

He served on the Council of the College of Physicians and Surgeons for some years, and on the death of Dr. A. P. Proctor, in 1934, became Registrar of the College, remaining in this position till his death. It will be very hard to fill his place.

His funeral gave some faint idea of the real affection and esteem we all felt for him. It was almost impossible to get into the chapel, and the long lines of cars for blocks, in every direction, were witness to the anxiety that every one felt to say farewell to their departed friend. His pallbearers included men from Victoria, Cloverdale and other centres, and all the members of Council He was a good man to know, a good companion, a good physician, and a good servant of his profession; that is the best epitaph anyone of us could hope to have.

To all the members of his family we extend our deepest sympathy and condolences.

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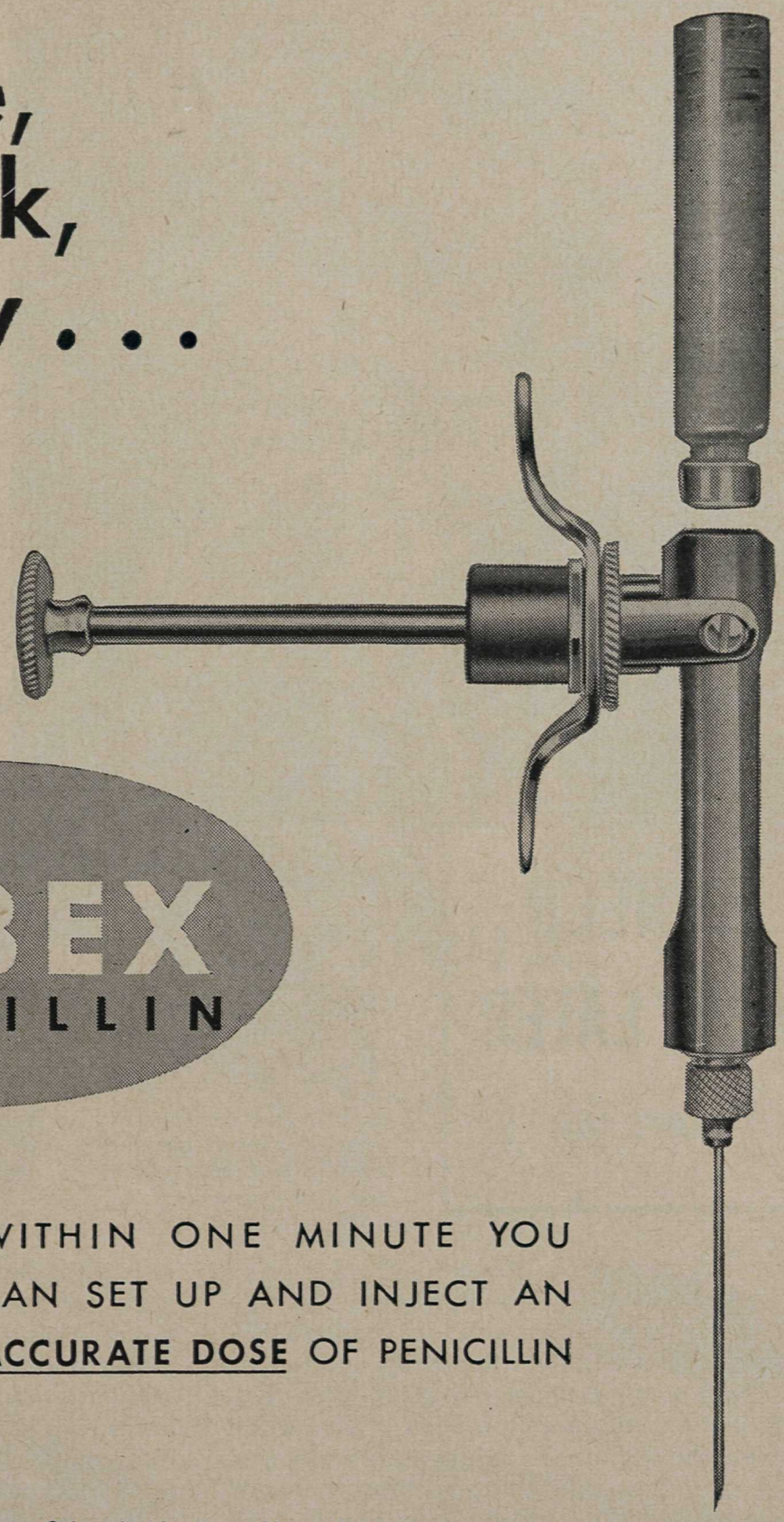

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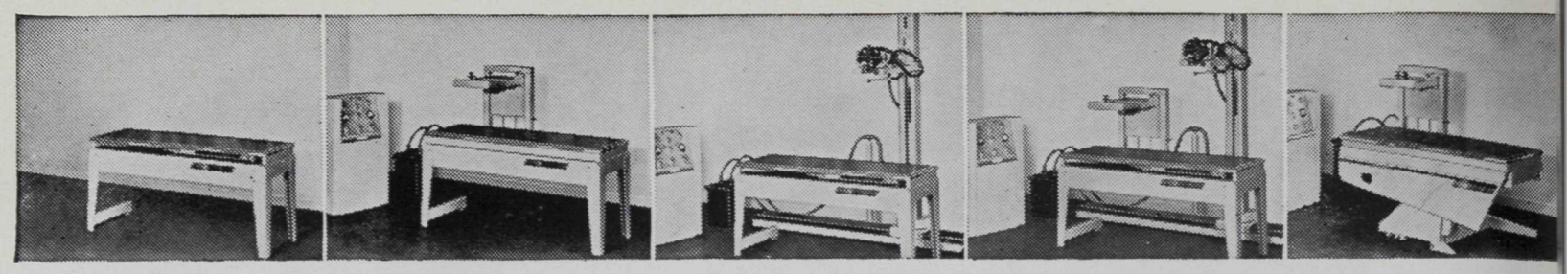

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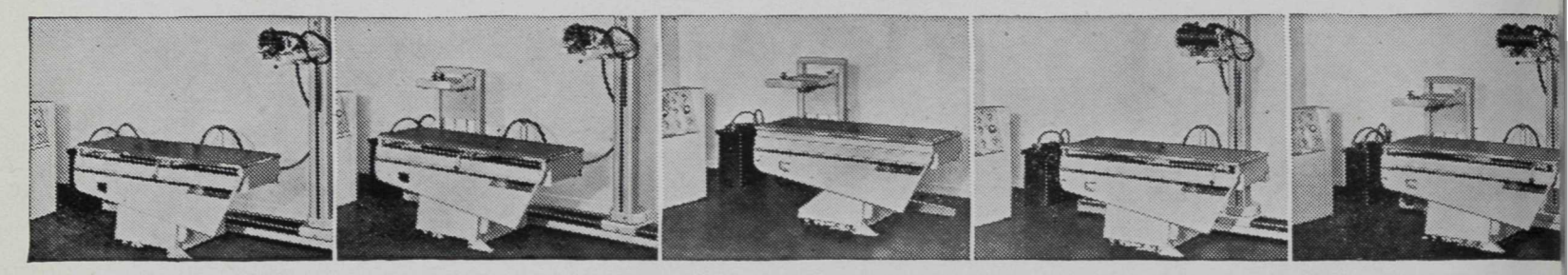

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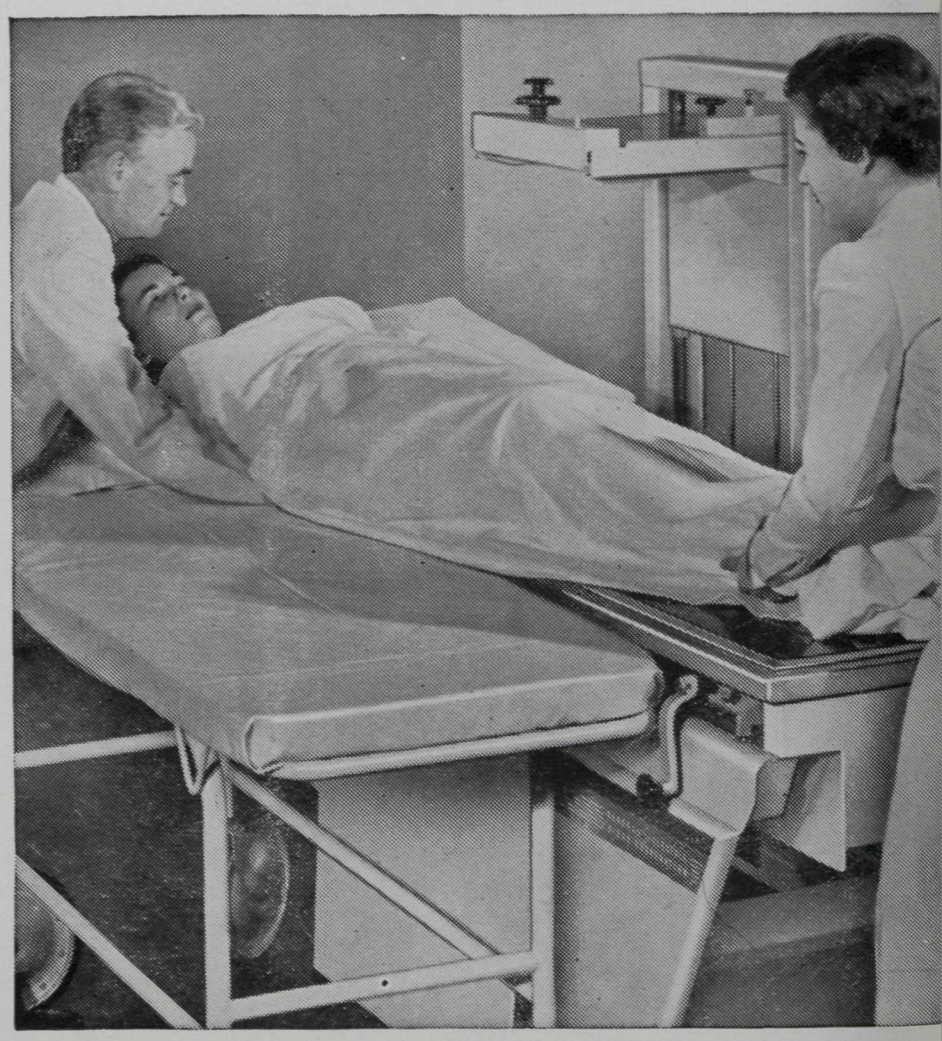

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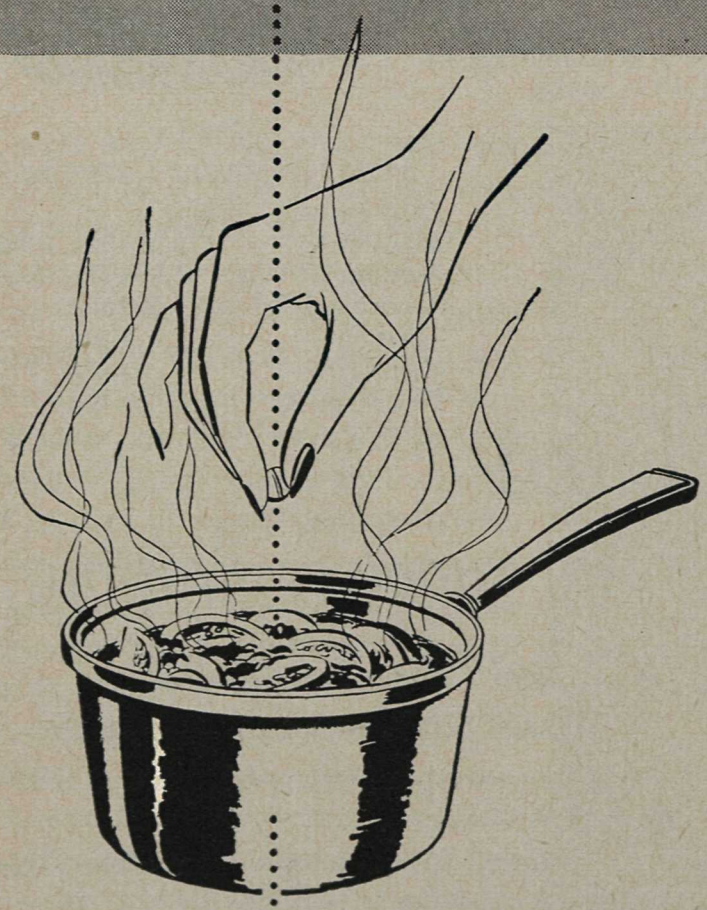

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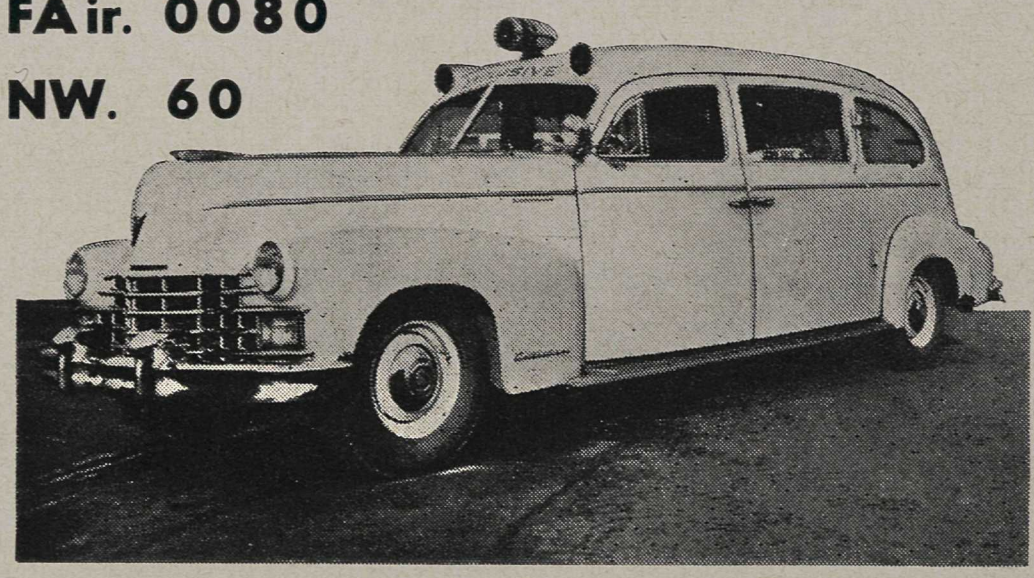

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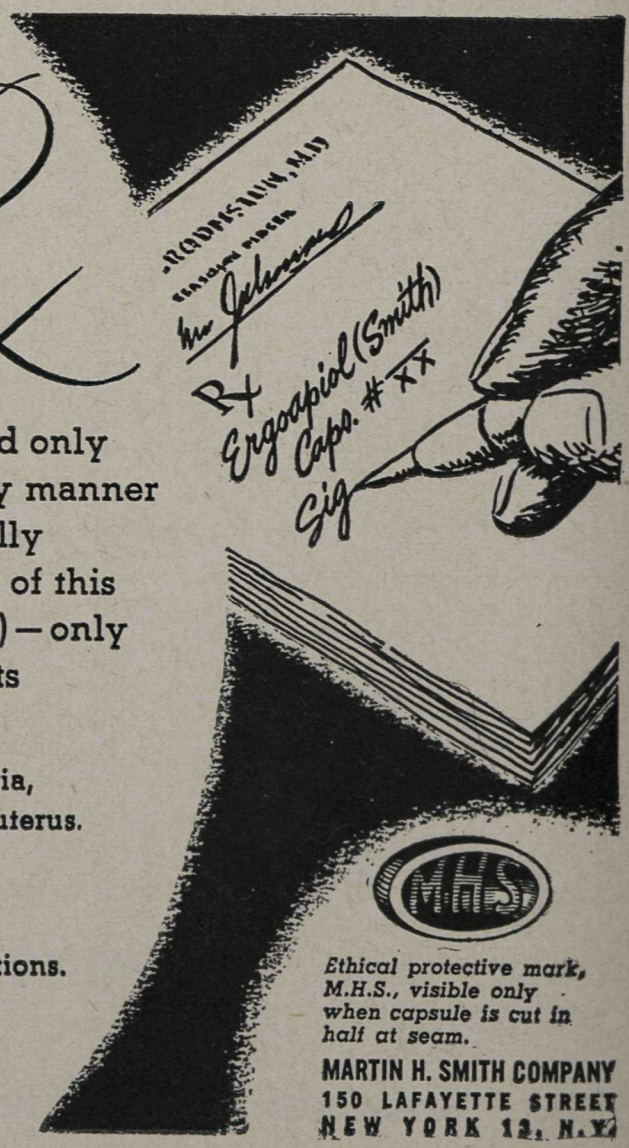

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