THE BULLETIN

Published By

The Vancouver Medical Association

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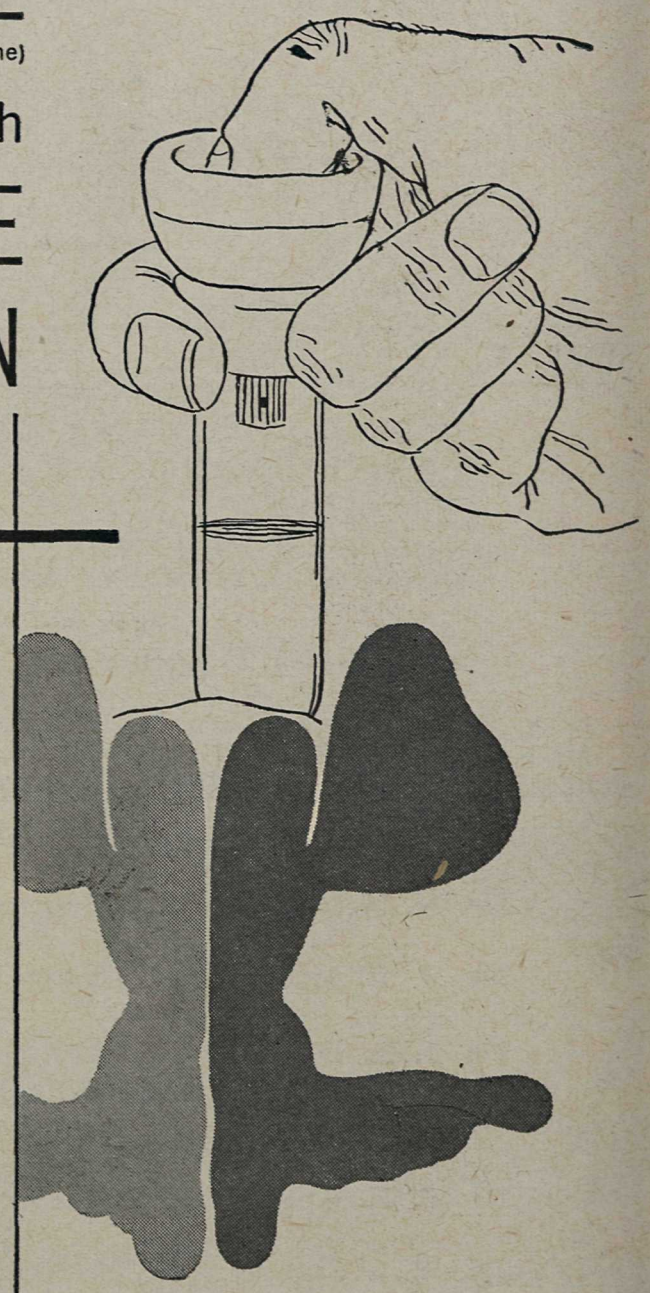

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VANCOUVER MEDICAL ASSOCIATION

Founded 1898; Incorporated 1906

PROGRAMME FOR THE FIFTY SECOND ANNUAL SESSION

(Spring Season)

MARCH 2nd—SPECIAL MEETING Speaker—Professor F. H. Bentley, Department of Surgery, University of Durham Medical School, England.

MARCH 6th—SPECIAL MEETING, Lecture by Sir Reginald Watson-Jones, title: "The New World of Orthopaedic Surgery."

MARCH 8th (WEDNESDAY) "OSLER LECTURE"—Presented by DR. GEORGE A. DAVIDSON, HOTEL VANCOUVER Title: "Men of Osler's Time."

MARCH 28th—SPECIAL MEETING—"Lecture by Dr. Cecil Watson, Professor of Medicine, University of Minnesota, Title: "Some Fundamental and Clinical Aspects of the Problem of Hepatic Cirrhosis."

APRIL 4th—GENERAL MEETING—"The Problem of the Prostate", Dr. L. G. Wood.

MAY 2nd—ANNUAL MEETING.

It has been the practice in the past to hold a meeting of the Clinical Section of the Vancouver Medical Association on the third Tuesday in each month. These meetings were held at alternate hospitals and owing to this fact, often two Clinical meetings were held at a hospital in one month. To overcome this situation a plan has been worked out whereby the members of the Vancouver Medical Association are invited by the various hospital Directors to attend their Clinical Staff meetings. These meetings will be held as follows:

Second Tuesday—Shaughnessy Hospital

Third Tuesday—St. Paul's Hospital

Fourth Tuesday—Vancouver General Hospital

Notice and programme of these meetings will be circularized by the Executive Office of the Vancouver Medical Association.

All special and general meetings will be held in the Tuberculosis Institute Auditorium.

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.

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Advertising Rates on Request.

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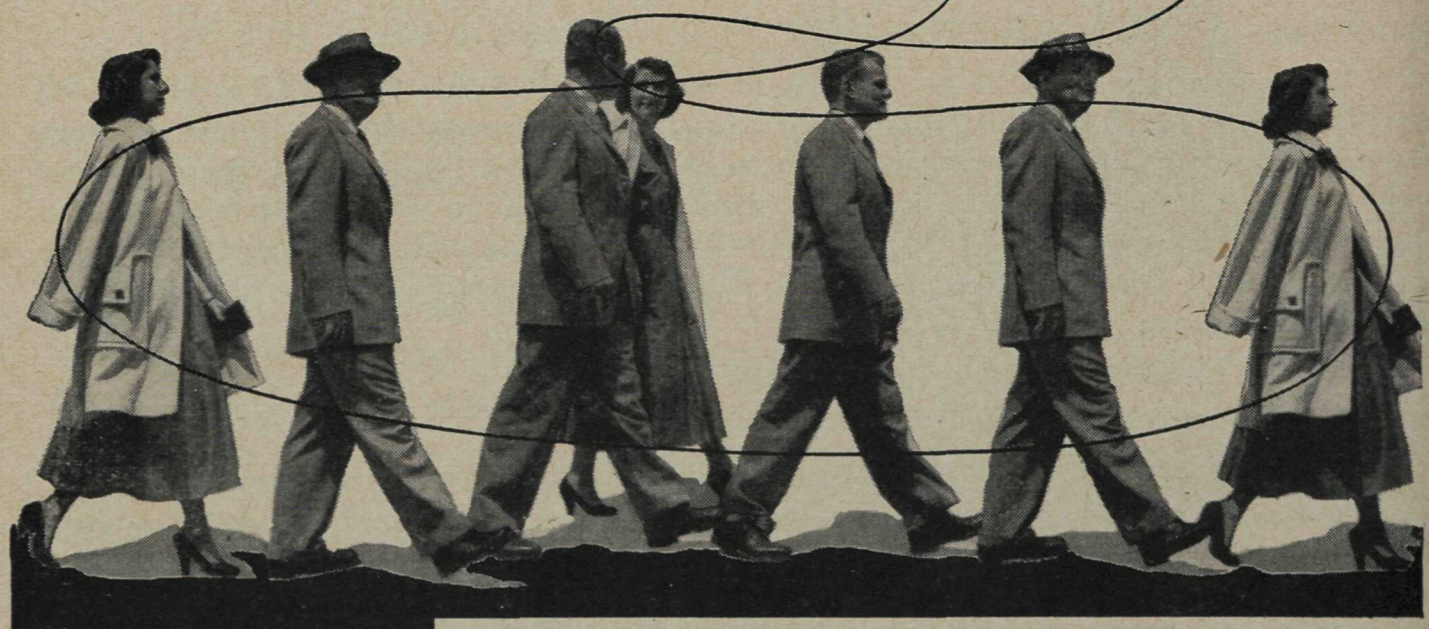

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Perloff, W. H.: Am. J. Obst. & Gynec. 58:684 (Oct.) 1949.

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VANCOUVER HEALTH DEPARTMENT

CASES OF COMMUNICABLE DISEASE REPORTED IN THE CITY

STATISTICS—FEBRUARY, 1950

Total Population—Estimated	385,500
Chinese Population—Estimated	6,877
Hindu Population—Estimated	133

JANUARY, 1950

	Number	Rate per 1000 Pop.
Deaths, residents only	389	12.1
Total deaths (by occurrence)	419	13.0
Chinese deaths	15	26.2

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

	January, 1950	
Male	372	
Female	368	
	740	23.0

INFANT MORTALITY—Residents only:

	January, 1950
Deaths under 1 year of age	11
Death rate per 1000 live birth	21.2
Stillbirths (not included in above item)	15

CASES OF COMMUNICABLE DISEASES REPORTED IN THE CITY

	January, 1950		January, 1949	
	Cases	Deaths	Cases	Deaths
Scarlet Fever	12	0	10	0
Diphtherit	0	0	0	0
Diphtheria Carriers	0	0	0	0
Chicken Pox	154	0	531	0
Measles	117	0	123	0
Rubella	6	0	7	0
Mumps	284	0	17	0
Whooping Cough	14	0	0	0
Typhoid Fever	0	0	0	0
Typhoid Fever Carriers	0	0	0	0
Undulant Fever	0	0	1	0
Poliomyelitis	1	0	0	0
Tuberculosis	49	7	46	14
Erysipelas	5	0	1	0
Meningitis	1	0	0	0
Infectious Jaundice	0	0	0	0
Salmonellosis	3	0	2	0
Salmonellosis Carriers	0	0	0	0
Dysentery	5	0	0	0
Dysentery Carriers	0	0	0	0
Tetanus	0	0	0	0
Syphilis	28	12	31	1
Gonorrhoea	292	0	244	0
Cancer (Reportable)				
Resident	77	49	78	47
Non-resident	13	2	19	3

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The Editor's Page

In a recent issue of the *Vancouver Daily Province*, we noted an advertisement which read as follows:

Physicians' Office Assistants.
Night School Classes in Receptionist Duties, Office Routine and
Medical Laboratory Technique. Apply, etc.

To our mind, this brings up a very disturbing chain of possibilities. We would ask some questions. Who gives the course in laboratory technique? How accurate is it? What sort of diploma do the graduates receive? What is it worth?

There are three persons concerned here. First, the doctor who may employ one of these technicians; second, the patient who is the subject of the tests; thirdly, the person who receives the training.

To our mind, it would seem obvious that each of these people should have some sort of guarantee that the training given conforms to rigid standards of competence and accuracy—standards that would be recognized by some competent authority, such as the University of British Columbia, through its Preventive Diseases and Public Health Department, or the Provincial Health Department of the Province of British Columbia. Otherwise, the doctor cannot rely on the tests made—serious injury and damage may be done to the patient through inaccurate diagnostic tests—and the person who spends his or her money to take the course has been swindled.

In British Columbia, so far as we know, the standard of medical laboratory work is very good. The big hospitals and the Provincial Health Department give adequate courses to those that they train, the time devoted to the course is adequate, and the training is such as to make reliable technicians. But there is, as far as we know, no standard laid down by the governmental authorities which must be reached before any aspirant can practise as a fully-qualified technician—though we understand that the Provincial Department of Education has some responsibility in the matter.

This is a very serious problem, and not one that can be regarded lightly. There is a very comprehensive article (appearing in the April number of the *Women's Home Companion*) dealing with this question. It is entitled "Menace in the Medical Labs," and it contains some very disturbing statements. Cases of faulty diagnosis, due to incorrect laboratory tests, are quoted freely. The article states that "every year from 2½ to 3 million illnesses are wrongly diagnosed because of faulty laboratory tests" (out of some 300,000,000 tests made in the United States).

The Pennsylvania Society of Clinical Pathologists "discovered that the leading cause is poorly trained technicians. . . . Unfortunately, thousands of technicians . . . are too often hastily taught by slipshod methods." "A number of Lab. technicians . . . are turned out by profit-making private diploma mills." (Such as we imagine is the one whose advertisement appears above.)

These quotations apply, of course, to the United States—but we cannot doubt that a survey of Canadian conditions would reveal that there is the same danger threatening us in Canada. The remedies suggested in this article include "proper supervisory control (by the States) over medical Labs." California, for instance, has laws requiring that Lab. technicians and technicologists must pass licensing examinations. In Seattle, we understand, there is a training school, recognized by the King County Medical Society, requiring nine months' training as a minimum, and proper examinations.

We would suggest that it would be well worth the while of our medical authorities to look into this matter, and make recommendations, possibly through the Provincial Health Department, that legislation be instituted which will control the training and licensing of all who wish to do medical laboratory work, with a view to protecting the public at large, the medical profession, and the technical workers themselves.

Vancouver Medical Association

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LIBRARY HOURS:

Monday, Wednesday and Friday	9:00 a.m. to 9:30 p.m.
Tuesday and Thursday	8:00 a.m. to 5.00 p.m.
Saturday	9:00 a.m. to 1:00 p.m.

RECENT ADDITONS:

A Guide to the Diagnosis of Occupational Diseases, Industrial Health Division, Department of National Health and Welfare, 1949. (Gift.)

American Cancer Society and National Cancer Institute of the U. S. Public Health Service—Proceedings of the First National Cancer Conference, 1949. (Gift.)

An Introduction to Essential Hypertension, by R. F. Herndon, 1946.

Association of American Physicians—Transactions of the 62nd Session, 1949. (Gift.)

Canadian Ophthalmological Society—Transactions of the Eleventh Annual Meeting, 1948. (Gift.)

Handbook of Medical Emergencies, by T. B. Fitzpatrick, 1949.

Medical Clinics of North America—Symposium on Pediatrics, January, 1949.

Medical Etymology, by O. H. Perry Pepper, 1949 (Historical and Ultra-Scientific Fund).

Medical Research Council Memorandum No. 23—Nomenclature of Fungi Pathogenic to Man and Animals—Names recommended for use in Great Britain by the Medical Mycology Committee. London: H.M.S.O. 1949.

Medical Research Council Report for the Years 1945-1948. London: H.M.S.O. 1949.

Medical Research Council Report No. 266—Infection and Sepsis in Industrial Wounds of the Hand: A Bacteriological Study of Aetiology and Prophylaxis, by R. E. O. Williams and A. A. Miles. London: H.M.S.O. 1949.

Modern Surgical Technic, by M Thorek, 1938. (Gift from Dr. Watson Dykes.)

Ophthalmological Society of the United Kingdom—Transactions, 1948.

Surgical Clinics of North America—Symposium on Recent Advances in Surgical Physiology, December, 1949.

Surgical Clinics of North America—Symposium on Urology; Obstetrics and Gynecology, February, 1950.

The following is a further list of some of the specialized journals taken in the Library:

DERMATOLOGY AND SYPHILOLGY

American Journal of Syphilis, Gonorrhea and Venereal Diseases.
Archives of Dermatology and Syphilology.
British Journal of Dermatology and Syphilis.
Excerpta Medica—Section XII—Dermatology and Venereology.
Journal of Venereal Disease Information.

ECONOMICS

Medical Economics
Public Health Economics.

RADIOLOGY

Acta Radiologica.
American Journal of Roentgenology
British Journal of Radiology.

TUBERCULOSIS

American Review of Tuberculosis.
British Journal of Tuberculosis.

MISCELLANEOUS

Communicable Disease Center Bulletin (U.S. Public Health Service).
Journal of the History of Medicine.

BOOK REVIEW

AUTHORITY AND THE INDIVIDUAL—The Reith Lectures for 1948-49, by Bertrand Russell. London: George Allen and Unwin Ltd. pp. 125 (portrait frontispiece).

The Reith Lectures, established by the B.B.C. in 1947, are an annual series of broadcast lectures given each year by an acknowledged authority in a particular field, sociology, literature, history, public affairs or economics, who has been invited to undertake some study or original research on a given subject, the results to be presented to B.B.C. listeners. The lectures are named after Lord Reith, the founder of the B.B.C.

Lord Bertrand Russell is known not only to students of philosophy and mathematics, but to the general public as well, for his views and activities have been such as to bring him into conflict with the state on a number of occasions, and his philosophic doctrines have violently opposed large bodies of popular opinion, sentiment and orthodox beliefs. His earlier life was devoted to mathematics, and his first important book was "The Principles of Mathematics", followed later by a joint work with Alfred Whitehead, who became Professor of Philosophy at Harvard, "Principia Mathematica". Stirred from time to time by the rise of British Imperialism at the beginning of the century, his philosophy led him to enter the field of politics. His outspoken opposition to conscription in 1915, and defense of conscientious objectors, won him a fine which cost him his library. When invited to Harvard to give a course of lectures he was denied a passport, and in 1918 he served a six-months term in prison for publishing a pacifistic article.

He lectured in several universities after coming to the United States in 1938, and when his right to teach philosophy in the College of the City of New York was questioned because of his moral beliefs, the legal action which followed was played up on the press as a *cause célèb're*, somewhat similar to the Dayton "monkey trial". His claims were not sustained, and he was then appointed for a five-year term as a lecturer for the Barnes Foundation. This was later cancelled by the director of the Foundation, again not without wide publicity.

Orphaned at the age of three, his father's designs to have him raised as an agnostic were apparently successfully circumvented; nevertheless it would appear that the paternal desires came to eventual fruition. The fundamental characteristic of Russell's philosophic position has been an intensive application to his successively developed views, of the logical mind. By a series of logical progressions he has concluded that "the ultimate constituents of mind and matter are of the same type, the difference between minds and bodies lying in their structure and not in the elements of which thy are composed . . . the difference between physics and psychology lying not in the events which they seek to establish, physics being concerned with structure and psychology with quality." Russell thus identifies sensations and images, which compose a man's mind, with physical events in his brain. It would appear to follow from this therefore that in his theory of knowledge Russell has modified his original rationalistic views in the direction of pragmatism or behaviourism, which he has demonstrated in his work, "An Outline of Philosophy".

The foregoing sketch of Bertrand Russell's life and philosophy has been given at some length in the belief that such a background is necessary to a consideration of what Lord Russell now has to say about the relationships existing between Authority and the Individual. The views of an individual who during more than half of his life has

frequently been in conflict with what is considered as "duly constituted authority" can not be lacking in interest.

On the first page of Lecture One, entitled "Social Cohesion and Human Nature", Russell states briefIfly and clearly the fundamental problem which he proposes to consider in this and the five lectures which follow: "*How can we combine that degree of individual initiative which is necessary for progress with the degree of social cohesion that is necessary for survival?*" This is followed by an analysis of the problem in successive steps, followed by an examination of what can be done towards its solution, and finally a consideration "as a matter of ethics the whole relations of individual thought and effort and imagination to the authority of the community".

The subject of social cohesion, its history, the stage at present reached and its possible future development is first discussed; then the subject of individual initiative is similarly discussed, and finally, as Russell phrases it, "the conflict which modern technique has introduced between organization and human nature, or, to put it in another way, the divorce of the economic motive from the impulses of creation and possession" is considered.

It appears to the reviewer that with the social and economic problems which face the medical profession, individually and collectively, everywhere today, the addition of this small volume to the medical library is most timely. It deserves careful perusal, and the reader will find that its style lends itself to ready comprehension, instruction and even entertainment. Whether the conclusions arrived at are accepted or not, it is stimulating reading, hence profitable. —D. E. H. C.

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THE BRITISH COLUMBIA SURGICAL SOCIETY

THIRD ANNUAL SCIENTIFIC SESSION

GUEST SPEAKER
Dr. Deward O. Ferris,
Mayo Foundation, Rochester, Minnesota.

APRIL 27th and 28th, 1950

EMPRESS HOTEL VICTORIA, B. C.

Reservations may be made by applying directly to the Hotel.

A special invitation is extended to members of the following Societies:

North Pacific Surgical Association.
Seattle Surgical Society.
Spokane Surgical Society.
Portland Surgical Society.
Tacoma Surgical Club.

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Dr. T. M. Jones

THE NEW WORLD OF ORTHOPEDRIC SURGERY

Lecture by Sir Reginald Watson-Jones.

A special meeting of the Vancouver Medical Association at the Chest Clinic Auditorium on March 6 was addressed by Sir Reginald Watson-Jones, F.R.C.S., M.Ch.Orth., B.Sc., M.B., Ch.B., L.R.C.P., Orthopedic Surgeon to the Kings' Household.

Sir Reginald is the first Sims Commonwealth Professor of the Royal College of Surgeons and in this capacity was making his second visit to Canada, his first to Vancouver.

The subject of Sir Reginald's discourse was "The New World of Orthopedic Surgery," and dealt principally with the physical aspects of this new world as exemplified in the realm of traumatic surgery. He is a renowned teacher, an accomplished platform speaker, and nobly carries on the tradition of his illustrious predecessors who have led the field of orthopedics, Sir Owen Thomas and Sir Robert Jones. He spoke clearly and with charm, the accents and modulations of St. James and Mayfair triumphing over a temporary difficulty with phonation owing to laryngitis.

The speaker announced that he brought a special greeting to the medical profession of British Columbia; this came exclusively to this province, the home of the youngest medical school, from the centre of the British Commonwealth, where ancient and honourable institutions of medical teaching of world-wide renown have their seat.

He then briefly reviewed the history of the last fifty years in surgery, and more specifically traumatic surgery starting with reference to the dictum of Sir Owen Thomas, "the mechanical shall not trespass upon the physiological". "Surgery," it was declared, "is the restorer of symmetry; nature if unthwarted, will do the rest." This was beautifully demonstrated by a splendid series of slides which showed how this has proved true in very many cases of formidable disorganizations of bone and soft parts which in by-gone days would have furnished summary indications for amputation. With these were shown some "horrible examples" of what unwarranted and excessive interference may do. To emphasize the broad philosophy of surgery as opposed to mere mechanical dexterity, and give point to the concept that no advance can take place in one branch of science without corresponding advance in others, Arbuthnot Lane's "no touch" technique was referred to as it applies to bone surgery. The work of W. E. Gallie, of Toronto, in bone and fascia transplants was also referred to and many illustrations of successful restorations by such means were shown.

Sir Reginald spoke feelingly and admiringly of the fine work of the late Murray Meekison, of Vancouver, at R.A.F. "crash stations". Many of the best illustrations displayed were from the photographs and roentgenographic records of R.A.F. crash injuries.

That the Air Force injuries and restorations were a subject near to the speaker's heart was evident especially in his remarks, and the attention he has given to the rehabilitation of these young men. The incredible skeletal damage they sustained and the even more incredible restoration which was achieved was elaborately demonstrated in the illustrations of men restored to active and vigorous life who had apparently been destined inevitably for total crippling.

Restoration, the speaker showed, must be psychological and physiological. Both physical and psychological education must be right. This must take place in a succession of four stages: Good surgery, good rehabilitation, good re-education and good "re-settlement". "The job must be fitted to the man, not the man to the job. Instil hope, resolve anxiety, remove fears—only then are we physicians to the body and the soul." In this latter connection a note of admiration was sounded for the conception and working of the Vancouver Rehabilitation Centre. It was evident that his visit to this institution had strongly and favourably impressed the speaker.

Sir Reginald's arresting style and eloquence drew his address to its conclusion as he said: "We do not need better mechanics; we need knowledge that will aid repair."

He ended most appropriately with Wordsworth's lines from "The Prelude", apostrophizing the Age of Youth, "the chosen time",

"Bliss was it in that dawn to be alive,
But to be young was very Heaven."

A very large and enthusiastic audience greeted the eminent speaker and heartily showed its appreciation.

Dr. J. R. Naden, in a few well-chosen words expressed formally the thanks of the Vancouver Medical Association and the pleasure enjoyed by the audience.

—D. E. H. C.

THE BRITISH COLUMBIA MEDICAL ASSOCIATION

1949 - 1950

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SINUSITIS

By PAUL M. OSMUN, M.D.

Delivered before the British Columbia Medical Society's Annual Meeting at Victoria, B. C., 1949.

What is sinusitis? It is a word for the most fashionable conglomeration of symptims suffered by our generation. Only the layman is sure of the diagnosis. To the rhinologist, the divers forms it takes are an endless challenge to his special training.

The past 25 years have seen a swift evolution in our concept of sinusitis. After the First World War, Austria and Germany were considered the centre and fount of this knowledge. The ear, nose and throat specialist had been a curiosity in the allied armies. When the war was ended, hundreds of American doctors flocked to Berlin and Vienna. These ambassadors of the new specialty returned each with a case of beautiful hand-forged instruments, a lifetime of prestige, and a conviction that sinusitis was a disease of the sinuses which could be cured by surgery. Today we think that sinusitis is a local symptom complex, often of systemic disease.

The first book about the physiology of the nose was written less than ten years ago. Since then there have been tremendous advances in enocrinology, nutrition, psychiatry, biochemistry, bacteriology and allergy. Fitting together knowledge from these fields rescued rhinology from the abyss of disrepute into which it has been flung by the purely surgical mind.

For example, in the field of endocrinology are syndromes of sinusitis which are due solely to the lack of estrogenic hormones, yet which present exactly the same symptoms as acute or chronic bacterial sinusitis. The differential diagnosis is not so much concerned with whether sinal symptoms exist, or whether we shall call a syndrome sinusitis, as it is concerned with the underlying etiology. We can no longer think of etiologies in terms of strains of bacteria, but must think also of systemic endocrine imbalances and nutritional deficiencies which may have resulted in vaso-motor swelling of the nasal mucosa, giving rise to symptoms of sinusitis.

Let us consider a not too hypothetical clinical example of vaso-motor rhinitis: It is sundown. (It could be tonight in Victoria). A gentleman stands with a cigarette

dangling from his lips in the corner of a drawing room by an open window. He is wearing silk socks, holds a drink in one hand and a handkerchief in the other. His back is wracked with sneezes, his eyes are watery, his nose is running. He is hawking and spitting into his handkerchief. His throat is raw from coughing and his neck muscles are sore. He has a headache.

Hersh, of New York, in a recent analysis of 1000 headaches, traced 25% to vasomotor swelling of the intra-nasal and sinal mucosa, and ascribed another 25% to reflex cervical muscle spasms, variously termed myositis, fibro-myositis, or the cervico-somatic syndrom. Muscle changes in the neck, particularly of the trapezius, were demonstrated by Wolff using myographic electrical tracings.

Patients of this type present themselves to their physicians complaining of "sinusitis". They are literally holding their heads in their hands and say: "But, Doctor, the headache is worst right back here in the cords of the neck. If you could only do something about that."

It is the older clinical physician who knew, long before Wolff came along with his myographical electrical tracings, that the best treatment was a hot poultice, a liniment and massage, and local heat. He knew that the swelling inside the nose was best controlled by bed-rest, stable room temperature and steam inhalations. He knew there existed a definite relationship between cervical muscle spasm, occipital headache, and so-called sinusitis. The rationale of traditional heat and massage therapy becomes more scientific.

A CLASSIFICATION OF SINUSITIS

A.	Infectious (bacterial or viral)	15%	
B.	Mechanical or Anatomical	10%	
C.	Vasomotor	75%	
50%	1. Allergic	35%	Weather: (Temperature, humidity, light, barometric pressure, fumes, dust, etc.)
50%	2. Physical Agents	15%	Hypo-Thyroid
	3. Endocrine	10%	Hypo-Ovarian
	4. Nutritional	10%	Avitaminosis
	5. Miscellaneous	5%	Malnutrition
			Excess Carbohydrate
			Psychological (emotional)
			Cardio-Nephritic
	Inhalants		Febrile states*
	Intrinsic factors.		Debilitating disease*
	Foods		Neoplasm*
	Vasospastic Drugs		

*Not necessarily vasomotor.

The term "Sinusitis" itself is unfortunate in that it ascribes the notion of inflammation and infection to a phenomenon which is, above all, vasomotor. The incidence of primary bacterial or viral agents in sinusitis is probably less than 15%. What infection we do see is often secondary to intra-nasal oedema, stasis and obstruction. That architectural or mechanical defects within the nose contribute to nasal obstruction and sinal symptoms cannot be denied. The large cystic turbinates, the fracture deformities and pressure-point spurs of the nasal septum are all too common, but that they constitute a surgical challenge in more than 10% of cases no longer holds.

What has happened to our friend in the drawing room? Concerning him we might say that vasomotor responses are commonest in the early morning and at nightfall, when temperature variations and chilling most frequently occur. There are frequently vasomotor responses to the ingestion of alcohol, the presence of excessive smoke, or dust, or humidity. Sinusitis may be complained of when the mucous membrane is

producing either too much or too little moisture. The patient becomes conscious of discharge in the throat when mucous is either abnormally fluid or excessively viscid, when it becomes mechanically or chemically irritating, odorous or obstructive. A dry condition of the nose and throat is often produced by belladonna which reduces humidification and ciliary activity. The same parasympathetic symptoms can be experienced by the unaccustomed public speaker. It often times occurs in circumstances of social stress. We all know the dryness of febrile states, the nasal mucoid stasis in debilitating disease, the vasomotor secretory disturbances in cardio-nephritics and in heart failure. We see the stuffy nose and the accompanying headache that may characterize emotion, recumbency, or simple chilling.

As human beings, we physicians are personally familiar with the relation of recumbency and simple chilling to a stuffy nose. When we arise in the morning, and our feet —unslippered—touch the cold floor, there is a reflex vasomotor flooding of the nose. We frequently sneeze or cough at this time. People like the Finns, for example, who live in an unfriendly cold climate, have evolved in their culture the habit of steam baths followed by a plunge into ice water. They have learned through countless years of previous living the secret of what we modernists call "contrast baths". They now know that thermal conditioning toughens, as it were, our labile vasomotor responses. We have learned that histamine is released in vasomotor responses. It appears likely that in *this* country we may succeed in substituting for the bath—Benadryl.

These disturbances are not diseases of the nose. They cannot be dismissed as rhinitis whether it be intumescent rhinitis, allergic rhinitis, simple rhinitis or catarrhal rhinitis. These complaints are frequently rhino-sinal manifestations of systemic vasomotor phenomena. To repeat, these phenomena may or may not give rise to sinus symptoms, and this depends upon the degree of oedema, the advent of secondary infection, or the local pressures within the nose, aggravated by architectural or mechanical abnormalities.

The nose and sinuses must be thought of as belonging to the respiratory tract. Bronchiectasis and bronchial asthma illustrate very well this unity. We no longer believe that sinusitis causes bronchiectasis. They simply co-exist. Nor is bronchial asthma often seen in the absence of allergic rhinitis. In either case, that the sinusitis should have become bacterial and suppurative is neither more nor less remarkable than that the bronchiectatic lung should suppurate.

The sinus mucous membranes are extended portions of the nasal mucosa. Patholgic changes in the nasal mucous membrane are accompanied by similar changes in the sinal mucosa. During the growth of the face, from babyhood to maturity, the nasal mucosa grows into the scaffolding we call the paranasal sinuses. Hence, whatever happens in the nose can happen also in the sinuses. Vasomotor rhinitis is frequently vasomotor sinusitis. An antrum that is dark today may transilluminate beautifully tomorrow, or it may remain dark in the presence of a deviated septum, because of altered function. It is for precisely these same reasons that X-Rays of the sinuses are so often negative despite the reality of sinal symptoms.

(A) BASIC SYMPTOMS OF SINUSITIS:

1. Wet obstructive nose with or without sneezing.
2. Post nasal discharge.
3. Dull frontal—occipital headache
4. Cervical myalgia
5. Mental depression.

(B) ACCESSORY SYMPTOMS OF SINUSITIS:

1. Cough
2. Sore throat
3. Pain
4. Stuffy ears or deafness

What, then, is sinusitis if it is not a disease? Certainly it is a reality. Articles in the *Readers' Digest*, paragraphs in *Time*, pictures in *Life* all attest to its existence.

Sinusitis is a symptom complex of considerable variability. The basic features are the wet obstructed nose, the post-nasal drip, the dull frontal or occipital headache, the cervical myalgia and the mental depression that every man who practices medicine knows very well indeed. If there should also be present a mechanical or structural defect within the nose or sinuses and a super-imposed infection, one can usually add one or more of the following symptoms: Cough, sore throat, ear complaints and facial pain.

These basic symptoms are those that characterize vasomotor rhinitis. They are secondary to swelling and congestion within the nose. Anyone who has lived through those endless hours of total nasal obstruction in the early phases of a common cold (and I think most of us have) are convinced that irritability and mental depression can be symptoms of intra-nasal oedema and pressure.

Now a word about the accessory symptoms of sinusitis:

1. *Pain*:
   Contrary to lay opinion, severe pain is rarely found as a symptom of sinusitis except in the acute inflammatory type when suddenly the lining membrane swells. Occasionally a slowly evolving empyema will give pain and tenderness. Whereas there is fever in the early inflammatory type, fever is rarely seen in chronic empyemata. However, an elevated sedimentation rate is invariably found when pus is pocketed.
2. *Cough*:
   The drainage openings of the antra are on the top mesial side. This is because the face grows downward as the jaws and maxilla adapt themselves to adult chewing needs. The antrum started its growth as a small bud, high in the nose. This bud becomes the opening of the antrum and remains located high in the nose during adult life. At night when we lie down, the antrum is turned on its side. The outlet then becomes dependent, pus runs into the nose, flows back over the ustachian tubes causing oedema of the tubes and deafness. The exudate continues into the throat causing sore throat; on into the larnyx and trachea causing cough.
3. *Sore Throat*:
   A sore, red throat is sometimes the only symptom and sign of chronic suppurative sinusitis.
4. *Chronic Cough*:
   A chronic cough in the presence of a dark antrum is highly suggestive of suppurative sinusitis.
5. *Ear Symptoms*:
   As a consequence of irritation or oedema at the naso-pharyngeal end of the eustachian tubes, there may be referred to the middle ear fleeting moments of sharp pain. There may be popping and crackling sensations in the middle ear. Occasionally, with persistent tubal oedema, the hearing becomes muffled and dull; episodes of light-headedness may occur. These symptoms of the ear may accompany suppurative sinusitis, or *vasomotor rhinitis*, from whatever cause. Indeed, they may be our only clue to the existence of such traditionally unrelated diagnoses as Sinusitis —or the menopause.

The significant feature of the classification of sinusitis as shown is that 50% of sinal symptoms derive from so-called allergic sources. There is not time to enter the controversial subject of allergic sinusitis. There is not yet an agreement of definition among rhinologists as to just what allergic rhinitis is. There are few reputable criteria of diagnosis. Eosinophiles in the nasal secretion are like skin tests for foods and inhalants —variable and inconclusive. When does a chemical rhinitis, due to the use of Privine, become an allergy? Is it because eosinophiles may appear in the exudate? Eosinophils

may also appear in the early coryza or in the serous rhinitis (to use an outmoded pathologic classification) of chilling and of emotion.

The physical characteristics of the allergic nose, upon inspection, are variable. The mucosa that is very pale, translucent with oedema, in a nose which appears to be full of wet, swollen turbinates, is usually an allergic nose. The symptoms may be indistinguishable from those of any vasomotor, rhino-pan-sinusitis whether due to emotion, chilling or the virus. The antra will usually be either very dull or completely dark upon transillumination. The onset is frequently acute. Vasomotor rhinitis caused by inhalant allergens is the commonest type and usually presents a pale grey nasal mucosa. In general, the degree of paleness is directly proportional to one's ability to make the diagnosis of an allergic nose. In contrast, fumes that are chemically irritating to the nose cause a congestion, which although wet, is more apt to be red than pale grey. The questions whether one wishes to include vasomotor rhinitis which is secondary to physical agents, in the allergic group, is an open question. More important is the realization that allergic or not, the rhinitis is vasomotor. The treatment of the acute vasomotor nose is often one of finding the most effective and well-tolerated anti-histaminic for that particular person. Chlor-Trimeton, 4 mgms. 4 I. D. for adults and 2 mgm 4 I. D. for children, seems to be the drug of current choice. Chlor-Trimeton has a remarkably low incidence of toxicity and a high incidence of efficicy. However, all cases of low-grade perennial rhinitis that do not respond to the anti-histaminics deserve to be skin-tested for the inhalant allergens. Skin tests are about 20% accurate, as we know, especially for foods. A negative skin reaction for milk does not mean that milk may not be the cause of post-nasal drip. Again and again, patients ask: "Is milk a mucus producer?" It seems to be a part of the folklore of rhinology that milk produces mucus. There is scientific evidence that many people are allergic to milk (milk, eggs and chocolate are common foods to which people are allergic). One can be sure only by eliminating particular articles of food from the diet and noting the effect on the patient's symptoms. When tests for inhalants fail, many rhinologists empirically give a course of dust-desensitization. Standard dust extract contains many environmental allergens that occur in the home: hair, dander, feathers, lint, molds and bacteria. It is a shotgun extract which in the absence of specific allergenic evidence is used with surprisingly effective results in many cases of perennial allergic rhinitis—cases that would otherwise be dismissed as hopeless. Dust is relatively harmless to administer and certainly can be undertaken by the general practitioner. The complications of neglected childhood simple allergic rhinitis are seen in adult asthma.

There are a number of interesting and specific causes of sinusitis. One of these comes under the heading of *intra-nasal medicaments*. In medical literature exists too little condemnation of nose drops. They are advertised widely as vaso-constrictors. Most of them are actually vasospastic drugs. This is especially true of the synthetic drops. Those in soluble combination with antiseptics and the sulfonamides are particularly harmful. Napthazoline and a solution of 1.25% sodium sulfathiazole have caused marked fibrosis, permanent ciliary damage, epithelial metaplasia and fixed vascular dilation in experimental rabbits, in a matter of weeks. One per cent of ephedrine in normal saline is a harmless and efficient vaso-constrictor, both topically and orally. Its action is mild and it does not further insult the vasomotor imbalance already present. Many cases of sever pseudo-allergic rhinitis, better termed chemical rhinitis, have been terminated by insisting that the patient *give up all nose drops*. Privine is the most consistent offender in this category. I have seen dozens of cases—cases representing all the symptoms of severe pan-sinusitis. If these patients were using Privine, the cure required 48 hours of complete withdrawal. It was sometimes necessary to hospitalize the patient so that he might be well sedated, and the withdrawal of *Privine* assured. Here, again, Chlor-Trimeton and steam inhalations were allies. In 48 hours these people were well and unbelievably grateful. One case had been using Privine every three hours, day and night, for more than six months following a simple cold!

Penicillin solutions up to 5000 units per cc. have been shown to be quite harmless experimentally and clinically. The calcium salts are thought to be especially well tolerated by the nose. This anti-biotic, however, has a very limited application, but it does combat bacterial ethmoiditis effectively when used with 1% ephedrine. Penicillin powder in the nose seems to be quite effffective in cases of bacterial ethmoiditis. That it might cure vasomotor rhinitis is preposterous.

Penicillin aerosol for office use is effective and seems to be associated with a lowered incidence of irritative reactions in the nose as compared with the powder. In cases of suppurative ethmoiditis, gentle lavage of the nose with warm, normal saline after shrinkage with one per cent cocaine and ephedrine, followed by penicillan aerosol gives gratifying results. (One's nurse can do the treatment.)

ENDOCRINE:

Although endocrine-rhinitis has been touched on previously, the subject merits reiteration because of its common occurrence among females of the climacteric age group. These ladies complain of stuffy nose and frequently stuffy ears, and of excessive mucus in the throat. They frequently also complain of being light-headed and, of course, as we all know, of being mentally depressed and irritable. Physical examination of the nose in these cases reveals a pale indolent cyanotic nasal mucosa throughout—a suggestion of what the textbooks call atrophy. It is amazing how the stuffiness vanishes and the pale cyanotic indolence gives way to pink healthy mucosa when adequate estrogen substitution therapy is employed. The same results can be achieved in cases of hypo-thyroidism. The appearance of vascular atonicity here is not characteristic. These people are not profound hypo-thyroids and often a very short course of thyroid extract is adequate to get them over the vasomotor imbalance which brought them to the rhinologist complaining of a stuffy wet nose. Of these two the menopausal symptom is by far the more commonly seen.

NUTRITIONAL:

Among nutritional causes of vasomotor disturbances in the nose, excessive carbo-hydrate is probably the single greatest offender in children. Sugar, as we all know, has little body-building value. It has energy, caloric, value only, and its oxidation takes place at the expense of essential food elements found in raw vegetables, fruits and meat products. The huge tonsils and adenoids, the deafness, dental caries, and anaemia found in malnuitrition have their expression in the nose. Avitaminosis especially of C is related to tissue oedema. There is growing evidence that the naturally occurring vitamins are preferable.

EMPYEMA:

Among specific bacteria problems in the nose maxillary sinus empyema is the most satisfactorily treated. The concomitant use of systemic triple sulfonamides, or penicillin, with surgical lavage of the antrum renders antral empyemata easily cured without operation.

It is also true that a few suppurating maxillary sinuses harbor penicillin-resistant organisms. In these, the use of streptomycin systemically and one per cent sulfa-mylon locally are often effective. In bacterial sinusitis there is usually associated a vaso-motor disturbance which may have anteceded the bacterial suppuration, or which may have developed because of the infection. Often it is advisable to use one per cent ephedrine in normal saline nose drops four times a day, plus chlor-Trimeton by mouth in order to establish drainage; at the same time combining the systemic use of either penicillin or streptomycin with surgical lavage of the antrum. Despite this regime, a few cases will require naso-antral window. But *rare* today is the need for *radical sinus surgery.*

When a general practitioner advises a patient never to let anyone puncture his sinus, he builds up patient-resistance to proper antral-management. Thereby he does both his patient and the rhinologist an injustice. This bias is a heritage the specialty well deserves because the method employed in the past, in which amid great crunching

of bone and near-shock the deed was done, is anachronistic. Today, using a middle meatus catheter where there is no bone, antral lavage is a simple procedure whether the patient is 7 or 70. Using this method there is no bleeding, no pain, no shock. It is the memory of less felicitous methods that give rise to patient and doctor resistance.

Suppurative frontal sinusitis usually develops during the course of the common cold. It is, as we know, an extension of pyogenic secondary invaders into the frontal sinus. Severe inflammatory oedema of the frontals causes prostrating supra-orbital pain. Penicillin, the sulfonamides, and local ephedrine in normal saline are usually adequate therapy. However, when an empyema develops and is retained, the help of a rhinologist is urgent. Any frontal headache which comes on between two and three hours after arising in the morning and grows worse during the day, is aggravated by bending or stooping, which disappears toward evening only to reproduce this pattern day after day, should be referred to the rhinologist. External trephination for drainage is rarely required. Fracturing the middle turbinate medialwards toward the septum is a minor procedure, and will provide drainage in nine out of ten cases. Neglected, the frontal sinus empyema is potentially the most dangerous of all sinusitis, because the posterior wall leading to the frontal lobes of the brain is much thinner than the anterior sinal walls. Penetration into the silent area of the brain is *fatally insidious*. Any persistent frontal headache that comes on two to three hours after getting up in the morning, that pounds and gets worse during the day only to subside at night and recur the next day is a bombshell.

In conclusion, it is well to re-emphasize the changes that have occurred in our concept of sinusitis, its etiology and treatment, in the past decade. Otorhinology has passed from a surgical to a medical specialty . . . as the same otolarnygologist has expanded his surgical talents toward the fields of broncho-esophagology and rhinoplasty.

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FAT EMBOLISM

D. S. MUNROE, M.D., M.R.C.P. (Lond.), F.R.C.P.(C)
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Read at Meeting of North Pacific Society of Internal Medicine, at Victoria, B.C.
September, 1949.

The subject of fat embolism is one which has occasionally been discussed before medical groups for many years. It has, however, never become a condition which readily crosses the mental threshold of the average doctor when he approaches an acutely ill patient with an apparent chest or brain lesion. Even the presence of a plaster cast on one or more extremities may not be sufficient to force the diagnosis on one's mind. Fat embolism remains one of those many conditions in medicine which is comparatively easy to diagnose if it is considered. It also remains the one non-surgical condition that the orthopedic surgeon will diagnose before his medical consultant even thinks of it.

Since the war, we have had occasion to see several cases of fat embolism. I do not propose to present any statistical review of these cases because I do not think that any new diagnostic or therapeutic point has been brought forward. A brief review of the subject, however, I thought might be of interest.

The frequency of fat embolism seems to be almost impossible to estimate. A few reported figures will serve to exemplify this point. Denman & Gragg (1) in 1948, studied 99 autopsies selected at random. In 32%, fat emboli were present, and 7% died as a result of fat embolism. Vance (2) in 1931 in a study of 246 routine autopsies found fat embolism in 44%, and in 5.7% it was considered to be the cause of death.

These two series suggest that in routine autopsy material, fat embolism is the cause of death in about 6%, and that the condition may be diagnosed as being present in 35 to 40% of cases. These are, to me, surprising figures.

Robb-Smith (3) in 1941 reported the presence of gross fat embolism in 33% of 125 autopsies done on patients in which death was due to accident. In another series of autopsied cases due to injury reported by Mavor (4), 40 cases had a fracture of one or more long bones. Of these in 7 deaths (17%), the cause of death was due to fat embolism. On the other hand, Darrach (5) has reported only two cases of fat embolism among 12,000 fractures in New York.

There are thus big differences in the reported incidence and mortality in fat embolism. Why the discrepancy? Perhaps the greatest factor is the varying index of suspicion of the man doing the autopsy and the required technique to demonstrate the fat. Perhaps fat embolism occurs very commonly with such few or such mild symptoms that no additional entity is suspected. Perhaps many mild cases are passed by as a mild post-operative bronchitis. Many of these would be diagnosed at autopsy by a searching pathologist or missed by one who was not impressed with the possibility of fat embolism.

On the clinical side, no reliable figures are available as to the incidence of fat embolism. Many writers insist that it is present much more frequently than suspected. Robb-Smith (3) may be quoted as saying that "there is little doubt that many of the complications of injury such as traumatic pneumonia, delirium, delayed shock and blast are in reality fat embolism." Newman (6) has added concussion to this list.

Precipitating Factors

Injury, and especially injury with a fracture of a long bone, is by far the commonest precipitating cause. Extensive burns are perhaps the next most common cause, although fat embolism is an uncommon complication of burns. Fat embolism may be found at autopsy in a multitude of other conditions, but these are usually of no import. Finally, I must mention one of our cases in which massive fat embolism occurred following resection of a large lipoma on the medial aspect of each thigh.

Pathogenesis

The pathogenesis of fat embolism has been a source of much argument and considerable investigation. There is unanimity of opinion on only some of the factors involved

and much speculative and often contradictory evidence on others. Harris, Perrett and MacLachlin (7), in 1939, published an interesting paper and listed three things that were necessary for fat embolism to occur.

(1) An accumulation of fluid fat freed from its cellular envelope by trauma.
(2) Open veins, the ends of which do not collapse. Veins normally collapse when they are opened.
(3) The pressure on the accumulation of fat must be greater than the pressure in the veins.

It will thus be evident that in the case of fractures, all these requirements may be met—the fat is freed, the veins are held open in the Haversian canals and the wound exudate may readily accumulate under tension. These authors did some experimental work using human fat. Previous investigators had used vegetable or mineral oils. They were able to establish that hydrolyzed fat (dilute hydrochloric acid added to human fat) was very much more toxic to experimental animals than neutral fat. The pathological picture of human fat embolism could be reproduced and it was their opinion tht the serious effect of fat embolism may be due to alteration in the fat, whereby it becomes much more irritative and toxic. Thus, the chemical as well as the physical characteristics of fat may be important. Proof of this is lacking.

The elimination of fat is said to be by four ways.
(1) Passage through the glomeruli into the urine.
(2) Excretion by the liver cells into the bile.
(3) Absorption by phagocytes.
(4) Solution by lipase in the blood stream.

Pathology (8)

Perhaps the most characteristic gross pathological lesion is acute pulmonary oedema. Petechial haemorrhages may be noted in the brain. Microscopically, fat droplets may be found in almost every tissue in the small vessels and capillaries. The droplets are of irregular shape and varying size and in ordinary paraffin sections appear as vacuoles. The true nature of these vacuoles is readily demonstrated by appropriate fat stains. Considerable degree of coronary embolism can be demonstrated. The fat passes through the alveolar walls into alveoli and may often be recovered in the sputum as early as 36 hours later. It also passes through the glomerular capillaries to appear in the urine.

Clinical Picture

The clinical picture of the victim with fat embolism is often fairly characteristic—often very obscure. The patient is one who has suffered a fracture of one or more bones. His early symptoms are usually related to his traumatic lesion alone. After an interval which varies considerably but which averages about 36 hours, the patient becomes dyspnoeic and begins to cough. The cough is soon productive of watery mucus, which very frequently is heavily stained with red blood. An attempt to bring forth his history may be satisfactory, disappearing, or even useless because of his mental state. This mental state may be quite clear, slightly dulled, or so dulled that the patient superficially would appear to have encephalitis. His memory may be poor, he may be restless, disorientated, irritable, stuporous, or even comatose.

The symptoms are thus either pulmonary or pulmonary and cerebral.

In the lesser grades of the condition, examination may reveal nothing. Usually, however, cyanosis is present, the pulse rate is elevated and the temperature ranges to 104 degrees F. The patient coughs almost continuously and at first glance the picture is one of acute pulmonary oedema or extremely acute bilateral pneumonia. Widespread rales are present throughout both lungs. Vague unrelated and fleeting neurological signs may be present. Petachiae should be sought for, and if they are only few in number, they are most likely to be found in the lower neck, pectoral areas, and in the conjunctivae. If more numerous they may spread over the trunk and even to the legs. Usually, how-

ever, only a few petechiae are to be found and they are easily missed.

Exudate round the macula and following the course of the retinal vessels as well as small haemorrhages may be seen by opthalmoscopic examination (9).

X-ray of the chest gives the picture of pulmonary oedema, or a more irregular picture of blotchy infiltrative lesions. There is nothing characteristic from a radiological point of view. A considerable quantity of fat may be held up in the lungs without producing radiographic changes, although functional impairment may be present. This statement is suggested because fat can often be found in the sputum with a negative radiograph, and also by animal experiments.

Differential Diagnosis:

Several things must be considered and almost any one of them might be diagnosed unless one considers fat embolism. These include broncho-pneumonia, acute heart failure, septicaemia, cerebral concussion or compression, and even alcoholism, if the fat embolism comes on soon after the accident. Each can usually be excluded in time, if not immediately.

The diagnosis can often be confirmed by the use of two laboratory aids.

(1) *Fat in the sputum*: It is said that fat may be obtained in the sputum in normal individuals and especially in those with pulmonary disease. In these instances, however, the fat is said to be in the form of fine globules within the phagocytes. Finding of extra cellular fat globules in a suspected case of fat embolism is strong confirmatory evidence. The larger the size of the globules, the stronger is the evidence. This usually does not occur until at least 36 hours after the onset.

(2) *Fat in the urine*: This occurs later (after 4 to 5 days) but is a more reliable diagnostic aid. Two technical points should be remembered—fat floats on the surface of urine and thus is most likely to be present in the last bit voided, and secondly, a lubricated catheter may be the means of obtaining a false positive result. Some surgical lubricants are water soluble, but it is best in these cases to use water only as a lubricant. All the urine for 24 hours should be saved and allowed to stand. The fat rises to the surface. A capillary tube is then allowed to just touch the surface of the urine and the drop transferred to a slide and stained with a fat stain. The most important thing to differentiate from the fat globules is air bubbles.

The course of the disease is one of acute illness for a period of several days. During this period it is most important to do a daily red blood count and haemoglobin, because a fairly acute anaemia may develop. Harris *et al* (7) were the first to point out this fact and to them it was taken to be a corroborating sign in the diagnosis.

Treatment:

Treatment is usually stated to be of little avail or "symptomatic". I cannot concur with this feeling of futility and I am confident that much can be done to tide the patient over.

The following are the most important things:—

(1) Oxygen—this is well worthwhile and often considerable immediate improvement occurs. It is preferably administered by tent, especially in the "cerebral" cases.

(2) Penicillin—this is of distinct prophylactic value.

(3) Blood transfusions—these are not infrequently required to alleviate the anaemia and should be given if the erythrocyte count falls.

Recovery is not followed by any permanent damage.

As far as prophylaxis is concerned nothing is of much value except early immobilization of fractures.

To summarize, fat embolism is not rare, it is not always or even usually fatal; it exhibits a reasonably uniform clinical picture and therapy is of avail. The most difficult thing about the diagnosis is to remember that such an entity exists.

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GALL BLADDER PERFORATIONS

F. W. GRAUER, M.D.C.M., F.R.C.S. (ED.), F.A.C.S., F.I.C.S.

Perforation of a gall bladder apart from traumatic rupture is usually a complication of pre-existing gall bladder disease. Perforation may occur slowly and quietly or more commonly be the outcome of an acute or subacute exacerbation in a chronically diseased gall bladder. Over 90% of patients with perforated gall bladder give a positive history for previously existing biliary system disease.

Prior to 1924, when Graham and Cole introduced the dye concentration X-Ray test, the diagnosis of gall bladder disease was made largely on a clinical basis. The commonest sign of cholecystitis in those times was, according to Dr. Terence Turner, an appendectomy scar. The discovery of the dye concentration test stimulated much interest in gall bladder disease and as so often happens, this test, for a while at least, was held as the ultimate in finality of diagnosis. Today we realize that it provides only another useful aid in the study of any given patient. By 1934, many surgeons were recommending early surgery for all patients with acute cholecystitis and prophylactic cholecytectomy for those with chronic cholecystitis, particularly those with calculi.

It is difficult to obtain accurate figures to demonstrate the incidence of gall bladder perforation. Many case histories show just the word "cholecystitis" and many diagnoses are in reality clinical impressions. The number of perforations we know, but the number who had acute or chronic cholecystitis we do not know for certain.
To simplify, one uses the term "Total Cases" to include acute, subacute and exacerbations of chronic cholecystitis; in short, all conditions which might lead to perforation.

TABLE I. Incidence of Gall Bladder Perforation

	Total Cases	No. Perforated	% Perforated
Cowley and Harkins	12,915	433	2.8
Bachhuber *et al*	1.699	90	5.3
Vancouver General Hospital	2,493	19	.8

O—Acute, subacute, and chronic cholecystitis.
X—1932 to 1947 inclusive.

TABLE II. Incidence of Gall Bladder Perforation in Post Mortem Room.

	No. of Consecutive Autopsies	No. of Gall Bladder Perforations	%
Johnstone & Ostendorph	12,000	32	.26
X Vancouver General Hospital	10,941	7	.065

X—1932 to 1947 inclusive.

Since perforation must be the end result of pathological process, be it acute, subacute or chronic, in point of time, the pathology of these processes may be of interest. Niemeier's classification appears widely quoted in the literature. He recognizes three clinical types:

Type I. Acute, free perforation into the abdominal cavity with generalized peritonitis. This may be a suppurative or biliary peritonitis depending upon the patency of the cystic dust and presence of infection.

Type II. Pericholecystic abscess with gall bladder perforation and localized peritonitis. The abscess may be in the adjacent liver substance.

Type III. Biliary fistula. This may be external or internal. The external type results from an abscess bursting through the abdominal wall or more commonly following surgery. The internal type applies to perforation of an adherent gall bladder into a neighboring hollow viscus such as the duodenum, stomach or transverse colon.

It will be noted that this classification depends largely on whether adhesions have formed prior to perforation and on whether the gall bladder adhesion has involved another viscus.

In Cowley and Harkins series the pathological laboratory diagnosis in perforated gall bladders was as follows:

Acute exacerbation of chronic cholecystitis	33.6%	62.2%
Chronic cholecystitis	28.6%	
Gangrene	24.0%	38.3%
Acute cholecystitis	14.3%	
Carcinoma	Rarely	

Johnstone and Ostendorph show an incidence of calculi associated with perforation in 85% in a combined series of 265 cases of gall bladder perforation. It would seem fair to assume then that chronic gall bladder disease, particularly with cholelithiasis, is the commonest pathological background leading to perforation.

From an etiological point of view the diseases leading to gall bladder perforation may be listed as follows:

1. Non-specific Inflammations
   (a) With or without calculi;
   (b) With or without cystic duct obstruction.
2. Traumatic rupture of the gall bladder.
3. Specific inflammations, such as tuberculosis, typhoid and syphilis.
4. Carcinoma of the gall bladder.

Gangrene of the gall bladder is included under non-specific inflammations because it is doubtful whether it occurs primarily. It may, however, occur along with perforation following torsion of the gall bladder.

Pursuing the calculus further, we find several methods of mischief. A stone by direct mechanical attrition and ulceration on a softened, inflammed wall or gangrenous wall, or one with intramural abscess, may eventually work its way through to perforation. Since 90% of 433 patients with perforated gall bladders gave a history of previous gall bladder attacks, it would seem reasonable to assume that the majority of calculi required more than exacerbation to complete the penetration.

Another method of penetration is by the nature of the calculus, wherein a small, hard, pointed stone, acting as an arrowhead, finally punctures the gall bladder and leads to free perforation into the abdominal cavity and generalized peritonitis. The opening may be only pin hole in size.

A calculus may lodge at the mouth of or in the cystic duct and give rise to empyema or mucocele, depending upon the presence of obsence of infection. Either of these may result in perforation. The shrivelled up gall bladder containing stones may well have gone through an obstructive phase with final absorption and contraction, or,

more directly, such a gall bladder may reach its contracted, thickened state through repeated inflammation with resulting scar tissue contracture in the absence of obstruction.

Cases have been described in which perforation with stone extrusion into surrounding adhesions has occurred, creating a false diverticulum or sac communicating with the gall bladder and assuming a reservoir function. If there should be an infection in this cavity a localized abscess will form, but suppuration does not necessarily follow upon the perforation of a gall bladder. Stones may be found in adherent masses of omentum with no obvious signs of inflammatory exudate.

All of us are familiar with gall stone ileus wherein usually a large stone lodges in the terminal ileum, having entered the small bowel through either a cholecystoduodenal or other fistula. Patients have been known to vomit biliary calculi as well as pass them *per anum*. Some indeed rare internal fistulae have been reported. Communication between gall bladder and right kidney pelvis with the passage of many biliary calculi into the urinary bladder, as well as bile stained urine, has occurred. Communication between gall bladder and uterus with subsequent passage of calculi *per vaginam* has been reported. Fistula between gall bladder and inferior vena cava with extrusion of calculi into the latter has been described at the post mortem. Communication between gall bladder and bronchus through the intermediary of a subphrenic abscess resulting from gall bladder perforation, the abscess having ruptured spontaneously into the bronchus, has also occurred. A less rare type of fistula is described wherein the gall bladder has perforated into the liver substance. This may result in intrahepatic abscess or in fistula formation with the right intrahepatic biliary tree. Free calculi have been found extruded from the gall bladder into a cavity in the liver substance. The gall bladder may form a fistula with the common duct.

In Cowley and Harkins series of gall bladder perforations the following signs and symptoms were described:

Persisting R.U.Q. pain with nausea and vomiting was usual. 40% had pain radiation to the back. 20% had jaundice. 96% were tender or rigid. 28% had generalized abdominal tenderness. 24% had a palpable mass in the R.U.Q. The white blood cell count varied from normal to 20,000. The temperature varied from subnormal to marked elevation. The diagnosis was correct in 12% of cases.

Johnstone and Ostendorph state that 49% of 32 instances of perforated gall bladder coming to autopsy were undiagnosed clinically.

The mortality rate in gall bladder perforation is strikingly high and may be attributed, in part, to difficulty in diagnosis, resulting delay in surgical treatment and the peak incidence being in the past 50 age group. The following figures speak for themselves:

TABLE III. Mortality in Perforated Gall Bladder.

	No. of Cases	No. of Deaths	%
Heuer (1937)	500	225	45.0
Cowley & Harkins (1943)	443	92	20.8
Bachhuber (1945) (operated on)	54	7	12.9
(Not operated on)	36	36	100.0
Vancouver General Hospital	19	7	36.8

At this point I would like to present briefly 5 cases of perforated gall bladder I have been party to during the past 3 years.

Case I. A white male, aged 45 years, was in a medical ward with deepening jaundice for 10 days. When seen in consultation he was practically moribund, with fever, somnolence, general abdominal resistance and moderate distension. On opening the abdomen there was generalized dark green staining of all serous surfaces. There were no adhesions about the moderately thickened, half-shrivelled gall bladder, and a careful search

revealed no microscopic perforation of the biliary system or bowel. No stones were palpable. The abdomen was closed with a drain in the R.U.Q. The patient died. Post mortem revealed a pinhole perforation of the fundus in the grooved reflection between liver and gall bladder. There was a single, small, pointed stone with its tip in the pinhole. The gall bladder showed evidence of chronic cholecystitis and cholelithiasis.

I recall an article describing primary biliary peritonitis in which several cases were operated upon. No perforations were found in the presence of marked greenish soiling of the peritoneum. There were many theories mentioned, including oozing of bile through microscopic openings in the gall bladder wall, and these are of developmental origin and something akin to the Thebesian foramina in the auricles. One wonders if in reality these might have been pathologically similar to Case I.

Case II. A white female, aged 44 years, was admitted to hospital on December 31, 1947, with a diagnosis of acute appendicitis. Her symptoms began at 3:00 a.m. on the date of admission, with sudden onset of lower abdominal pain, then vomiting and settling of constant pain in the R.L.Q. The W.B.C. was 13,510 with 82% polys. There was a history of previous occasional gaseous indigestion associated with constipation. The diagnosis was not clear and gynaecological consultation was sought. More tenderness was reported in the right adnexal than in the appendiceal area. The sedimentation rate showed moderate activity. Intravenous pyelograms on January 5th, 1948, were normal. The patient was discharged on January 22nd, 1948, having been treated for non-specific salpingitis.

The patient went to bed at home and a day later began to have pains in the R.U.Q. with nausea and vomiting. The pain persisted, but the nausea stopped. She was re-admitted on January 27th, 1948, with a diagnosis of acute cholecystitis. There was no jaundice. The gall bladder area was very tender for the first time, with slight abdominal distension, moderate rigidity in the R.U.Q. She was treated conservatively and a gall bladder X-Ray series on February 6th, 1948, showed a non-functioning gall bladder without radio-opaque stones. On February 13th, 1948, the abdomen was opened and a thickening gray colored gall bladder was stripped from surrounding adhesions and viscera. In Hartman's pouch area a small facetted stone was encountered lying outside the gall bladder. On completion of cholecystectomy, no perforation could be found in the gall bladder wall, which incidentally contained other calculi. Recovery followed.

This was a case in which perforation must have occurred at first admission, with escape of one stone and fluid enough to run down the right paracolic gutter to the pelvis to cause inflammation there. Subsequent sealing off of the perforation took place, and in the absence of infection, no abscess was formed.

Case III. A white male, aged 68 years, was admitted to hospital on May 29th, 1948, with severe, colicky pain in the R.U.Q. radiating to the right shoulder. The pain began on May 23rd, and by May 27th had moved to the R.L.Q. There was a previous history of gall bladder colic on many occasions since 1930. It was sometimes followed by jaundice. On examination, there was tenderness and rigidity over the right half of the abdomen, with restricted movement. The sclerae were faintly icteroid. The W.B.C. was 14,350 with 81% polys. The pulse was 92 and temperature 98.8° F. The urine showed albumen and W.B.C., there being a degree of prostatism.

The abdomen was opened and clear, slightly bile stained, mucoid fluid was encountered. There were no omental adhesions. A thick walled semi-friable, pearly gray gall bladder was removed which contained stones and exhibited a perforation. The common duct appeared dilated. It was opened, and two large stones were removed. The patient made an uneventful recovery. The pathological diagnosis was acute cholecystitis and cholelithiasis.

This case demonstrates the importance of common duct inspection for other calculi. I believe that there are three main causes of jaundice associated with gall bladder perforation which may exist independently or in combination. First, absorption from a

free biliary peritonitis; second, hepatitis from contiguous abscess or inflammation and, third, stones in the common duct.

Case IV. A white male, aged 62 years, was admitted to hospital on April 9th, 1948, with a history of epigastric pain of increasing severity for three days, and vomiting for two days. There was a previous history of gaseous indigestion and fat intolerance of some years standing. The patient had considerable weight loss and mild jaundice for three months prior to admission. On examination, the patient showed emaciation and all the signs of a perforated ulcer, except for the jaundice. The pulse rate was 140, the blood pressure 90/55 and the temperature 102. The pre-operative diagnosis was perforated carcinoma of the stomach though no mass was felt.

At operation a cholecysto-duodenal fistula was found with free perforation into the abdominal cavity at the adhesion line between gall bladder and duodenum. The gall bladder containing calculi was removed and the duodenal perforation was repaired. The common duct contained palpable stones and these were removed. The pathological diagnosis was tuberculosis of the gall bladder. The patient subsequently developed a chronic sinus at the site of the drain. After X-Ray therapy and intra-muscular streptomycin had failed, the sinus healed by daily instillations of streptomycin solution into its opening. The patient is now robust, has a good colour, has gained weight and has shown no evidence of tuberculosis elsewhere in his body.

Case V. A white male, aged 72 years, was admitted to hospital on December 11th, 1948, with severe epigastric pain following a large meal the same day. The pain persisted and radiated to the right shoulder and R.L.Q. and he soon became nauseated and vomited. There was no history of previous attacks of pain, indigestion, flatulence, fat intolerance or jaundice. Examination revealed obesity, tenderness and rigidity in the right half of the abdomen most marked in the R.U.Q. Rebound pain phenomenon was present. There was no evidence of icterus. The pulse rate was 100, the temperature 100.8° and the blood pressure 145/80. The W.B.C. was 17,900 with 11% staffs. The pre-operative diagnosis lay between acute cholecystitis and acute appendicitis. The urine showed sugar.

The abdomen was opened alongside the umbilicus and the appendix was found to be normal. The incision was enlarged upwards and a large pericholecystic abscess was encountered. The pus was suctioned off and the gall bladder was found to be very dark in color and oedematous with surrounding adhesions. The gall bladder was too friable to remove, so a cholecystostomy was done. No stones were seen. A separate drain was placed in Morrison's pouch. The patient was then treated for diabetes mellitus and made an uneventful recovery.

This case demonstrates perforation through a necrotic patch in the gall bladder wall occasioned by either intramural abscess or gangrene, which, literally translated, means ischaemic necrosis. Certainly infection was an essential part of this case and, as with infection elsewhere in the presence of uncontrolled diabetes, the course of events was rapid.

All of us have had the experience of incidentally finding gall stones in the course of investigating a patient perhaps for some other complaint. Very often, such a patient will give an entirely negative symptomatic history for gall bladder disease. The question of the innocuous gall stone has not infrequently been the subject of papers in the medical literature. As early as 1911 W. J. Mayo gave a paper entitled "Innocent Gall Stones A Myth."

By and large physicians take a conservative view regarding silent gall stones, while surgeons take a more positive view. Lahey, for instance, stated in 1938 "There are no harmless gall stones"—and that cholecystectomy should be advised unless special contra-indications exist when the diagnosis is made.

As surgeons, we are aware of the association of gall stones with gall bladder carcinoma in over 95% of instances of the latter; and of the association of gall stones with

perforation of the gall bladder in about 85% of instances. By comparison, we are also aware of the havoc urinary calculi and salivary calculi may cause and the diligence with which they are sought and extricated.

I should like to close with the thought that it might be better surgical practice to remove calculi with their diseased gall bladders prophylictically, rather than wait until some complication arises. Elective cholecystectomy is said to bear a mortality rate of .5%. A compromise could be reached with the conservative point of view, if cholecystectomy were advised in known cases of asymptomatic cholelithiasis only after the onset of early vague symptomatology.

• • • •

NEWS AND NOTES

Dr. J. S. Madill has moved from the North Vancouver General to the Royal Columbian in New Westminster, as associate radiologist.

Dr. C. G. McNeill, president, and *Dr. R. B. Townsley*, secretary-treasurer, are new officers of the North Vancouver Shore Medical Society.

Dr. J. T. Hugill, formerly of Vancouver, is now on the anesthetic staff of the West Coast Hospital at Port Alberni.

Dr. B. F. Green of Vancouver has joined the staff of the Elizabeth Hospital in Prairie Grove, Arkansas.

Apprentice help during the summer months may be obtained by writing the CAMSI executive at the University of Montreal.

Aortic stenosis has been treated in Philadelphia by sliding a bougie down the aorta through an incision behind the clavicle.

Dr. G. H. Manchester, of New Westminster, has had to vacate offices he has used for forty-two years, because of renovations. He still plans to continue as a psychiatric consultant.

Dr. R. G. Walton of the Vancouver General Hospital will continue studies in dermatology at Stanford next year.

Dr. Harold Spiro has begun a private radiology specialty in Vancouver.

Dr. L. C. Kindree of Squamish made a 20-mile mercy trip on jeep and skis, to an injured man on Mount Garibaldi last month.

Dr. David B. Boyd recently arrived in Victoria from Toronto, where for the past two years he has been studying at the Hospital for Sick Children. He has opened a Pediatric office in the city.

Dr. Ernest A. Frejd, formerly of Prince Albert, Sask., is now established in general practice in Victoria.

Drs. D. Murphy, L. T. Maxwell, L. B. MacLaren and A. L. Chambers of Prince George attended the recent convention of the Northern Interior District Medical Association.

BIRTHS

Dr. and *Mrs. L. W. Warcup* of Vancouver, a daughter.
Dr. and *Mrs. J. H. Lindsay* of Britannia Beach, a daughter.
Dr. and *Mrs. B. T. Marteinsson* of Vancouver, a son.
Dr. and *Mrs. R. S. Clarke* of Vancouver, a son.
Dr. and *Mrs. J. A. Porter* of Vancouver, a daughter.
Dr. and *Mrs. F. J. Marshall* of Vancouver, a daughter.
Dr. and *Mrs. M. C. Hay* of Vancouver, a son.
Dr. and *Mrs. A. C. Lang* of Vancouver, a daughter.
Dr. and *Mrs. F. H. Mills* of Ashcroft, a daughter.
Dr. and *Mrs. J. T. Cruise* of Victoria, a son.
Dr. and *Mrs. C. A. Matthews* of Vancouver, a son.
Dr. and *Mrs. F. L. Skinner* of Vancouver, twins.

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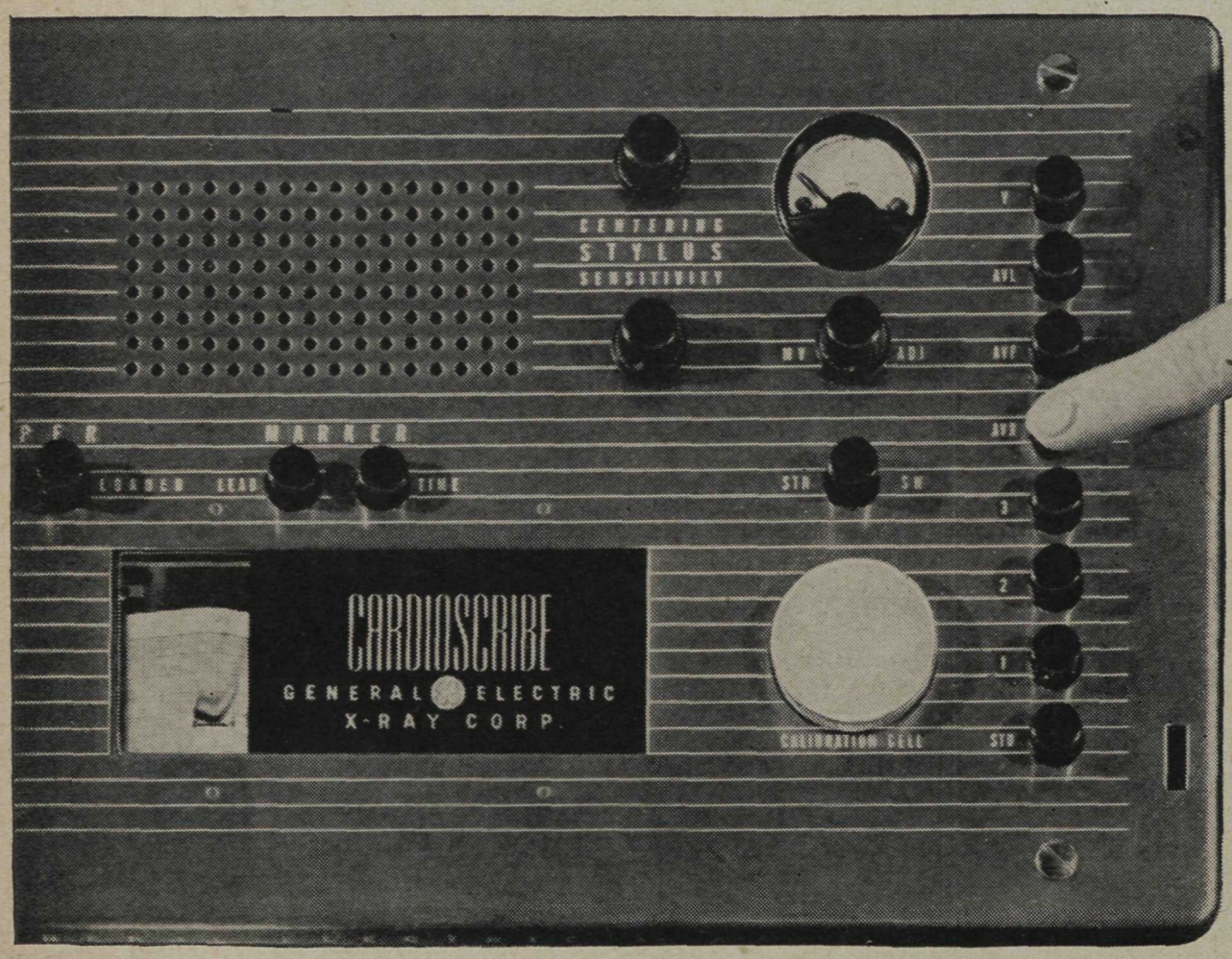

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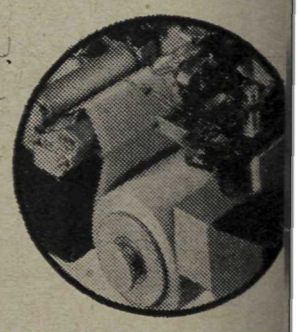

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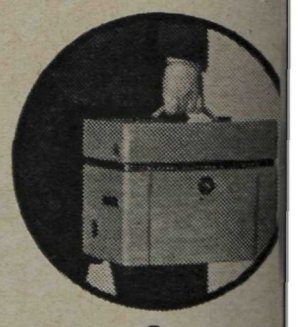

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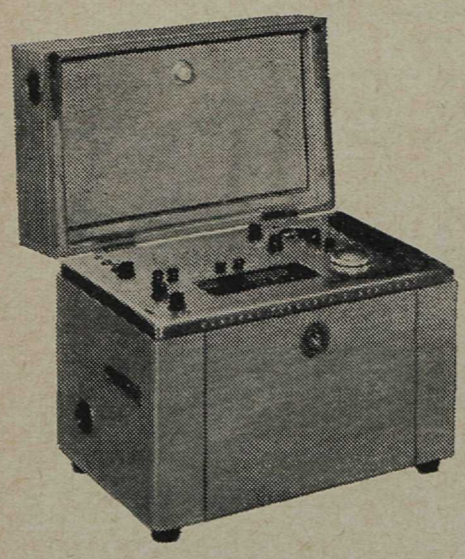

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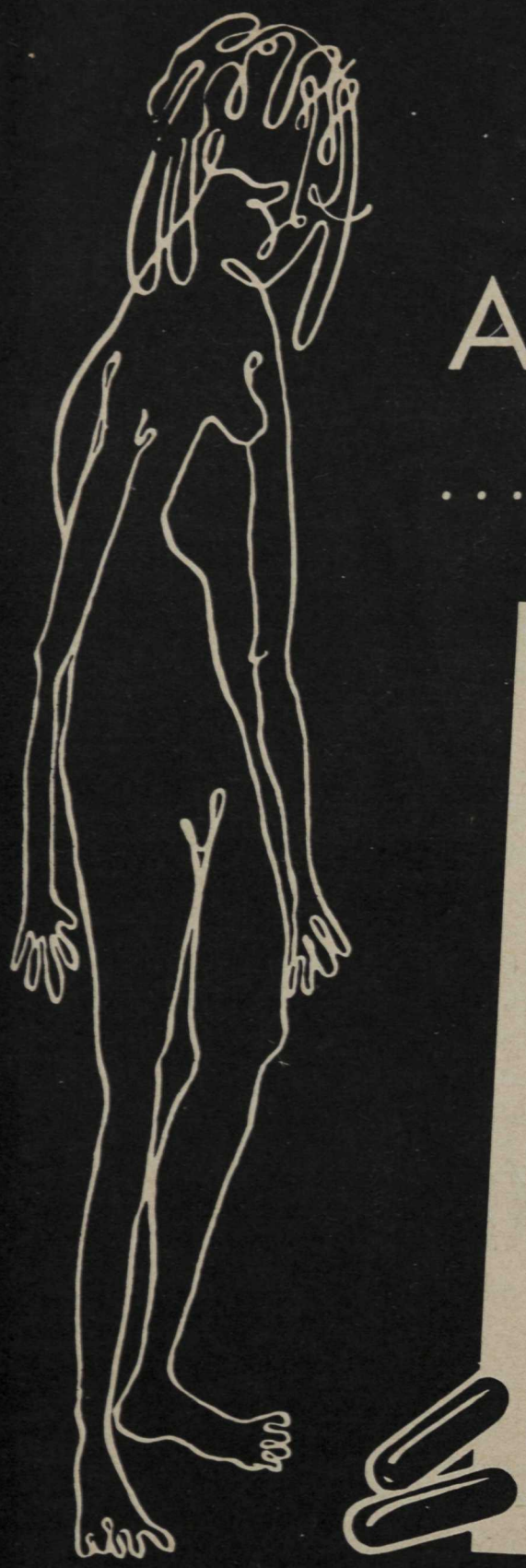

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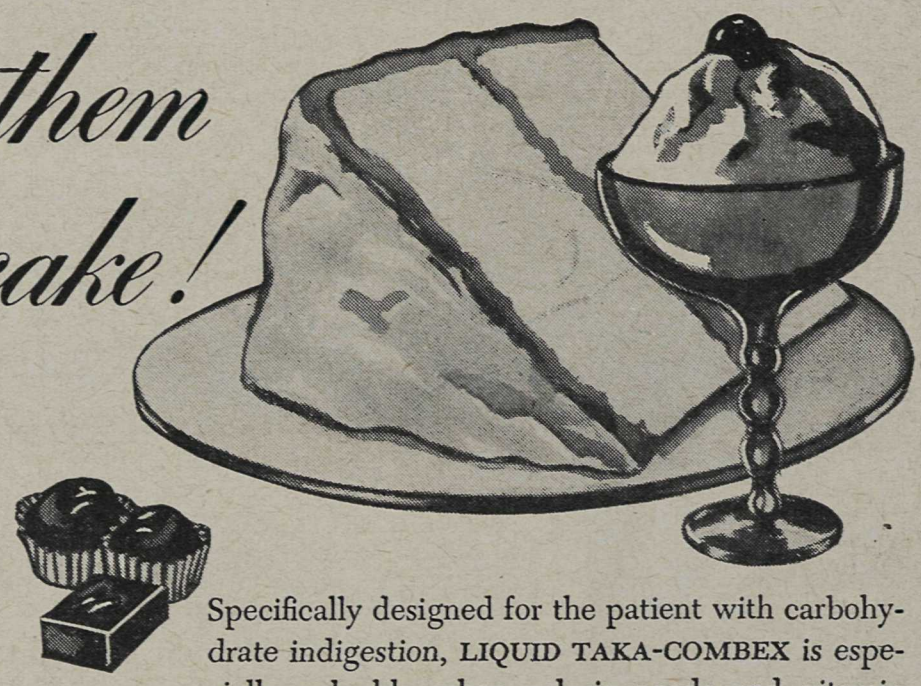

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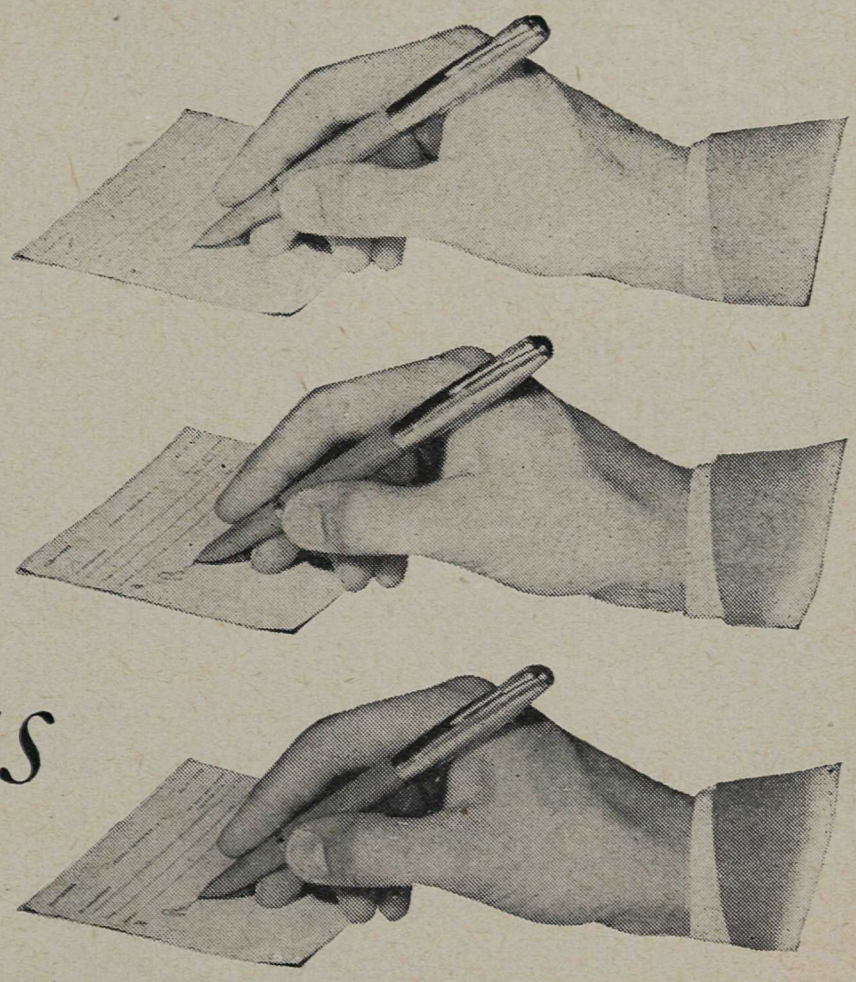

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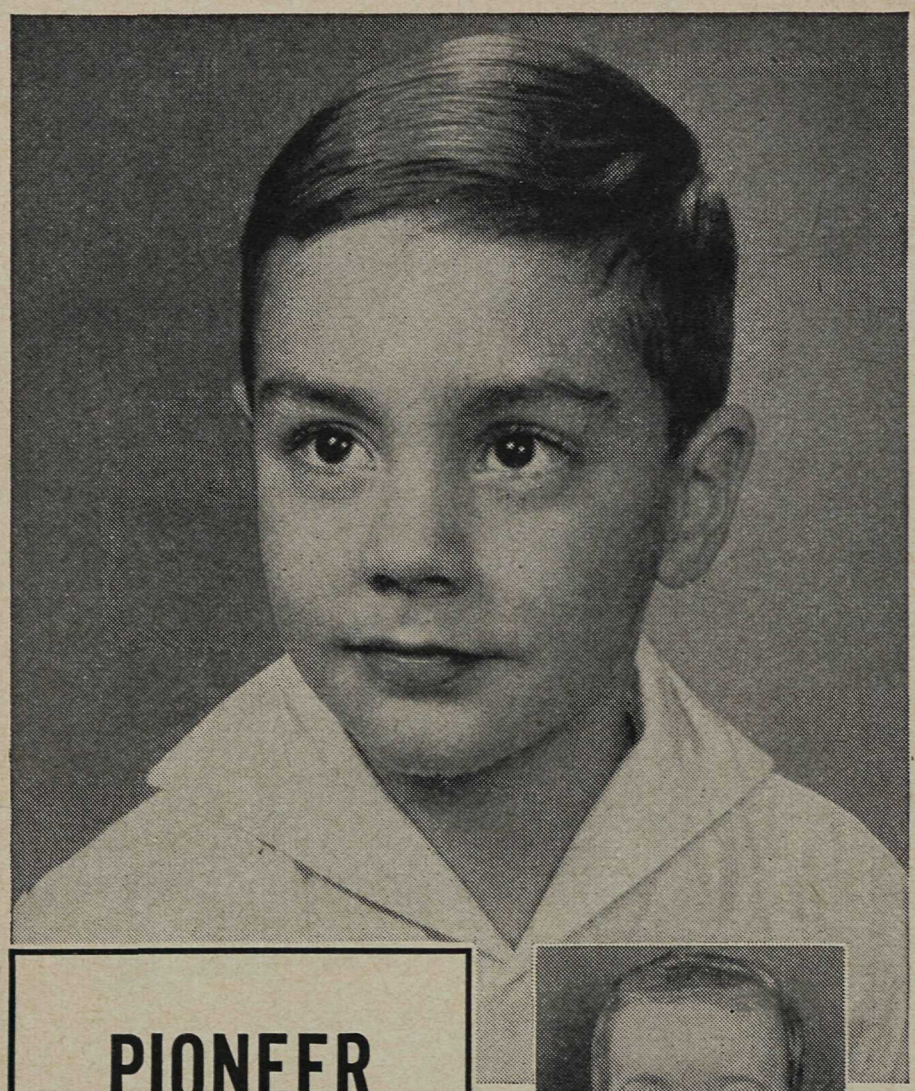

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REPORT No. 2

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In Swift's original test feedings, Kenny Gorman started Swift's Meats for Babies at *three months*—right along with his first solid foods. Contrast this with the general practice five years ago. Before the development of specially prepared Swift's Meats for Babies, the customary age to start meat was *six* or *nine* months. And because home-preparation was so difficult, most babies simply went without!

He's big and husky now

Kenny's mother will verify the fact that he's as "big and husky" as he looks in his photograph. Says Mrs. Gorman, "He can hold his own against my other three children."

Today any baby can have the same right start in life that little Kenny had. Doctors now recommend Swift's Meats for Babies in the early weeks of life—to provide the complete, high-quality proteins and iron every infant needs every day.

Swift's appetizing variety includes: beef, lamb, pork, veal, liver and heart—to help infants form sound eating habits.

Swift's Meats for Babies are expertly trimmed to minimize fat content . . . carefully cooked to preserve a maximum of essential meat nutrients.

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

All nutritional statements made in this advertisement are accepted by the Council on Foods and Nutrition of the American Medical Association.

SWIFT QUALITY FOODS

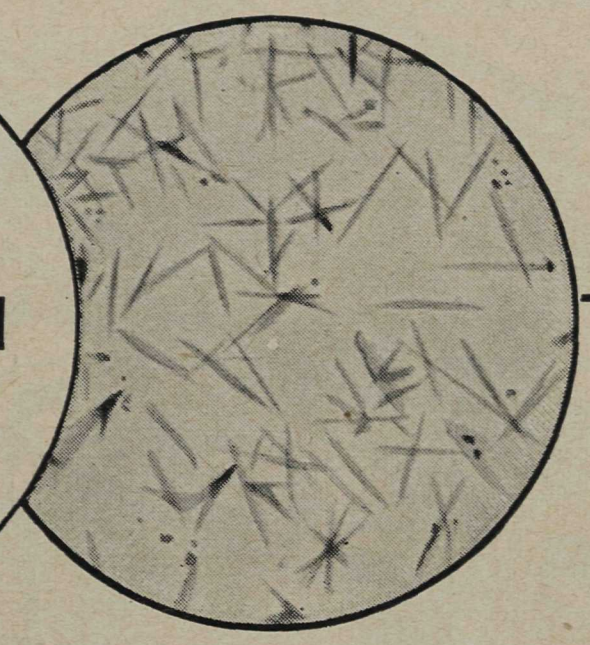
SWIFT

. . . foremost name in meats

. . . first to develop and clinically test 100% Meats for Babies

VITAMIN B_{12}

B. D. H.



An
erythropoietic
principle of liver

ANACOBIN

A solution of pure
crystalline
Vitamin B_{12}

FOR USE When Hypersensitivity To Liver Extracts Exists

It has been observed generally that Vitamin B_{12} has not given rise to reactions nor the development of hypersensitivity. A case of extreme hypersensitivity to liver extracts has been reported (Lancet, 1949, 1:1119) in which Anacobin B.D.H. was used without producing untoward reaction of any kind.

Anacobin is, therefore, recommended especially for the treatment of pernicious anaemia patients who have developed hypersensitivity to liver extracts. It is also used for its general "tonic" effect.

Anacobin is issued in 1 ml. ampoules each containing 15 micrograms of pure crystalline Vitamin B_{12} in saline solution.

Issued in boxes of 6, 25 and 100 ampoules

THE BRITISH DRUG HOUSES (CANADA) LIMITED

TORONTO CANADA

3-50B

THE NEW ANTI-NAUSEANT Gravol

for

PREGNANCY
MOTION SICKNESS
RADIATION SICKNESS

And other conditions where nausea and vomiting are factors

Reports from the literature on beta dimethylaminoethyl benzohydryl ether 8-chlorotheophyllinate (GRAVOL).

GRAVOL IN PREGNANCY

"Out of forty-three women with symptoms from 4 - 6 weeks, thirty-one (72%) were completely relieved within 3 hours after treatment. Twelve women (28%) had no relief." (1)

GRAVOL IN MOTION SICKNESS

98.6% effective in prevention of sea sickness . . . eliminates symptoms in up to 97.6% of cases already developed. (2)

GRAVOL IN RADIATION SICKNESS

Out of 82 patients with moderate to severe radiation sickness, 65 reported good to excellent relief. (3)

FORMULA FOR GRAVOL:

Each scored tablet contains:
Beta dimethylaminoethyl benzohydryl ether 8 - Chlorotheophyllinate .....50 mg.

PACKAGE

Available in vials of 25 and 50 mg. tablets.

NOTE: To date there is no evidence of toxic reactions with Gravol. However, some individuals may become drowsy or confused on high or continuous dosage.

References:

1. Carliner, P.E., Radman, H.M., and Gay, L.H.: Science, 110: 215 (Aug. 26, 1949). 2. Gay, L. H., and Carliner, P.E.: The Prevention and Treatment of Motion Sickness. Bull, Johns Hopkins Hosp., May, 1949. 3. Beeler, J. W., Tillisch, J. H., and Popp, W. C.: Proc. Staff Meet. Mayo Clinic (Sept. 14, 1949).

FRANK W. HORNER LIMITED

MONTREAL CANADA

Effective Esthetic

the most widely
prescribed
vaginal jelly

New

WATER-DISPERSIBLE VITAMIN DROPS

for Infants and Young Children

"Alphamette" Aqueous - No. 929

Each drop contains approximately:

Vitamin A	1,000 I.U.
Vitamin D	500 I.U.

In bottles of 8, 15 and 30 cc. with dropper.
The average dosage is 2 drops daily.

"Supplavite" Drops - No. 931

The average daily dose of 10 drops (0.6 cc.) provides approximately:

Vitamin A	5,000 I.U.
Vitamin D	1,000 I.U.
Ascorbic Acid	50.0 mg.
Thiamine	1.5 mg.
Riboflavin	1.0 mg.
Niacinamide	20.0 mg.
Pyridoxine	1.0 mg.
Calcium d-Pantothenate	5.0 mg.
Mixed Natural Tocopherols (as Antioxidant)	2.0 mg.

In bottles of 8, 15 and 30 cc. with dropper.

"Alphamettes" and "Alphamette" Liquid are also recommended sources of vitamins A and D; "Supplavite" Liquid, "Supplavite" Tablets and "Supplavite" with Minerals provide multivitamin supplementation.

Ayerst, McKenna & Harrison Limited

Biological and Pharmaceutical Chemists

MONTREAL, CANADA

due to factors involved

rectal therapy

is often advantageous over other forms of administration.

Adults and Children

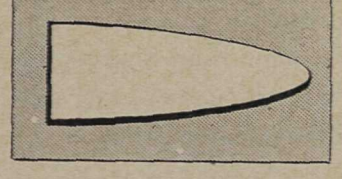
BRONCHIAL INFECTIONS

BRONCHO-RECTAL SUPPOSITORIES

Each ADULT Suppository is equivalent to 15 minims of Creosote.

Each JUNIOR Suppository is equivalent to 5 minims of Creosote.

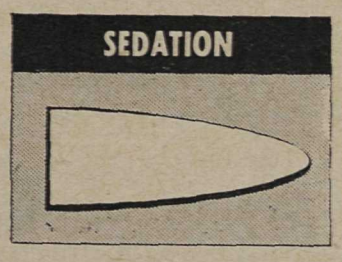
PINWORMS



VERMANCA SUPPOSITORIES

Contain the bitter principles of Quassia (alpha and beta picrasmin) along with Quinine Sulphate.

SEDATION



SEDANCA SUPPOSITORIES

Each Suppository represents:

Chloral Hydrate ..... 1¼ grains
Potassium Bromide ..... 1¼ grains

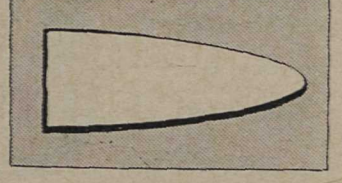
Diuretic & Myocardial Stimulant

AMINOPHYLLINE SUPPOSITORIES

Presented in 3¾ grains and 7½ grains strengths.

Infants and Children

CROUPY COUGHS

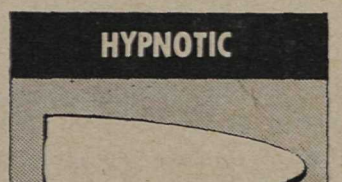


CROUPANCA SUPPOSITORIES

Each Suppository represents:

Antipyrine.....1 grain Camphor.....¼ grain
Ext. Belladonna 1/20 grain Po. Ipecac ⅛ grain
Creosote.....5 minims

HYPNOTIC



CHLORANCA SUPPOSITORIES

Each Suppository represents:

Chloral Hydrate.....5 grains

ANGLO-CANADIAN DRUG COMPANY LTD.

OSHAWA CANADA

ANCA

THE SUFFIX **ANCA** WILL IDENTIFY ANY OF THESE PRODUCTS ON A PRESCRIPTION.

CALGLUCOL E.B.S. is an especially prepared brand of calcium gluconate available in tablet, granule and ampoule form for therapeutic use. Vitamin D is added for its value in promoting the absorption and utilization of Calcium.

Calglucol E.B.S.

advantages of CALGLUCOL E.B.S.

Tasteless
Readily absorbed
Non-irritating when used parenterally
Less upsetting to digestive system than other calcium salts when taken orally

for

rickets
osteomalacia
pregnancy
lactation
calcium deficiencies

use

→ TABLETS

CALGLUCOL D E.B.S. *or*

→ GRANULES

for

colic
intractable pain
lead poisoning
nocturnal muscular cramps
tetany

consider

→ CALGLUCOL E.B.S. INJECTABLE

availability

CALGLUCOL D E.B.S. C.T. No. 149A, 10 gr. calcium gluconate and 500 I.U. Vitamin D per tablet, in bottles of 100, 500 and 1,000.

CALGLUCOL E.B.S. C.T. No. 149, 10 gr. calcium gluconate per tablet, in bottles of 100, 500 and 1,000.

CALGLUCOL D GRANULES E.B.S., each ounce contains 330 gr. calcium gluconate and 1,600 I.U. Vitamin D per oz. in 5-oz. bottles.

CALGLUCOL AMPOULES E.B.S. No. A-31, 10 gr. calcium gluconate per 10 cc. ampoule.

THE E.B. SHUTTLEWORTH CHEMICAL CO. LTD., TORONTO, CANADA

AN ALL CANADIAN COMPANY . . . SINCE 1879

Representatives: Mr. V. Garnham, 3228 West 34th Avenue, Vancouver, B.C.
Mr. F. R. Clayden, 3937 West 34th Avenue, Vancouver, B.C,

the answer to seasonal allergies

NEO-ANTERGAN

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all allergies

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Grippe
Spasmodic coryza
Allergic asthma
Zona, pruritus
Seasickness
Vomiting in pregnancy

ampoules
tablets
25-50-100 mg
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ointment
nasal drops
expectorant
suppositories

PHENERGAN *"the bedtime antibistaminic"*

in
nocturnal or persistent allergies

Asthma
Nocturnal pruritus
Zona

ampoules
tablets
10-25 mg.

POULENC LIMITED MONTREAL

"pioneers in the antihistamine field."

EXCLUSIVE AMBULANCE

LIMITED

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OXYGEN THERAPY SUPPLIED ON YOUR ORDER. 24 HR. SERVICE

J. H. CRELLIN W. L. BERTRAND

Only on your ℞

When prescribing Ergoapiol (Smith) for your gynecologic patients, you have the assurance that it can be obtained only on a written prescription, since this is the only manner in which this ethical preparation can be legally dispensed by the pharmacist. The dispensing of this **uterine tonic,** time-tested ERGOAPIOL (Smith)—only on your prescription—serves the best interests of physician and patient.

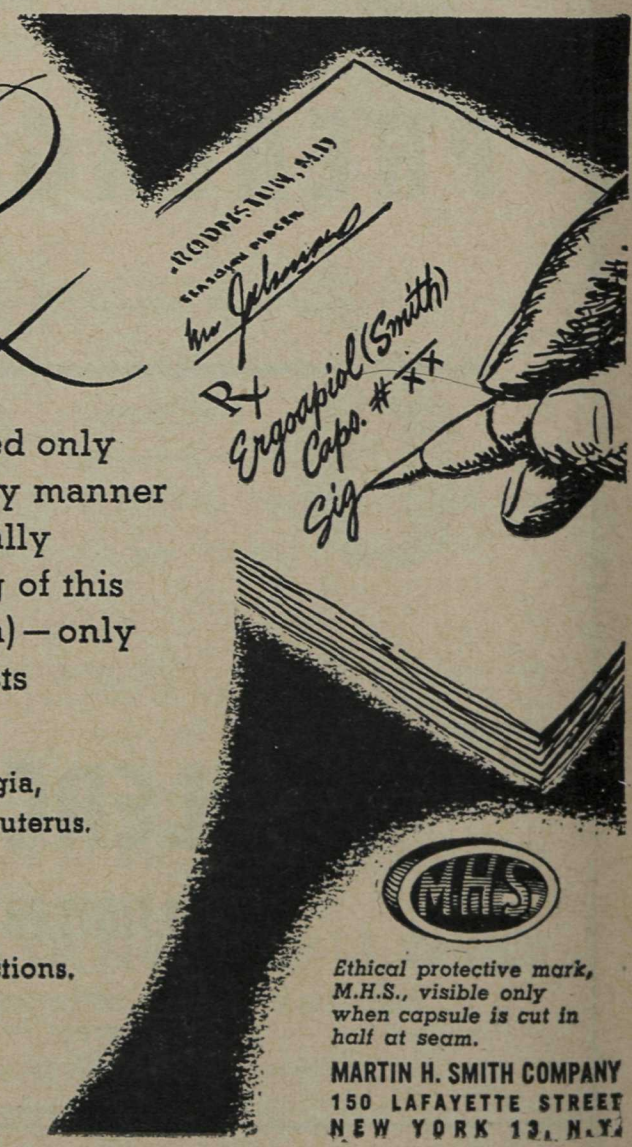
INDICATIONS: Amenorrhea, Dysmenorrhea, Menorrhagia, Metrorrhagia, and to aid involution of the postpartum uterus.

GENERAL DOSAGE: One to two capsules, three to four times daily—as indications warrant.

In ethical packages of 20 capsules each, bearing no directions.

Literature Available to Physicians Only.

ERGOAPIOL (SMITH)



Ethical protective mark, M.H.S., visible only when capsule is cut in half at seam.

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150 LAFAYETTE STREET
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