THE BULLETIN

OF

The Vancouver Medical Association

EDITOR

DR. J. H. MACDERMOT

EDITORIAL BOARD

DR. D. E. H. CLEVELAND — DR. J. H. B. GRANT
DR. H. A. DESBRISAY — DR. J. L. MCMILLAN

Publisher and Advertising Manager

W. E. G. MACDONALD

VOLUME XXIX. DECEMBER, 1952 NUMBER 3

OFFICERS 1952-53

DR. E. C. MCCOY, *President* — DR. D. S. MUNROE, *Vice-President* — DR. J. C. GRIMSON, *Past President*

DR. GEORGE LANGLEY, *Hon. Treasurer* — DR. J. H. BLACK, *Hon. Secretary*

Additional Members of Executive:

DR. G. R. F. ELLIOT — DR. F. S. HOBBS

TRUSTEES

DR. G. H. CLEMENT — DR. A. C. FROST — DR. MURRAY BLAIR

Auditors: MESSRS. PLOMMER, WHITING & CO.

SECTIONS

Eye, Ear, Nose and Throat

DR. J. A. IRVING.....Chairman — DR. W. M. G. WILSON.....Secretary

Paediatric

DR. J. H. B. GRANT.....Chairman — DR. W. H. S. STOCKTON.....Secretary

Orthopaedic and Traumatic Surgery

DR. A. S. MCCONKEY.....Chairman — DR. W. H. FAHRNI.....Secretary

Neurology and Psychiatry

DR. B. BRYSON.....Chairman — DR. A. J. WARREN.....Secretary

Radiology

DR. R. G. MOFFAT.....Chairman — DR. H. BROOKE.....Secretary

STANDING COMMITTEES

Library

DR. J. L. PARNELL, Chairman; DR. D. W. MOFFAT, Secretary;
DR. A. F. HARDYMENT; DR. W. F. BIE; DR. R. J. COWAN; DR. C. E. G. GOULD

Co-ordination of Medical Meetings Committee

DR. W. M. G. WILSON.....Chairman — DR. B. T. SHALLARD.....Secretary

Summer School

DR. S. L. WILLIAMS, Chairman; DR. J. A. ELLIOT, Secretary;
DR. J. A. IRVINE; DR. E. A. JONES; DR. MAX FROST; DR. E. F. WORD

Medical Economics

DR. E. A. JONES, Chairman; DR. G. H. CLEMENT, DR. W. FOWLER,
DR. F. W. HURLBURT, DR. R. LANGSTON, DR. ROBERT STANLEY, DR. F. B. THOMSON

Credentials

DR. W. J. DORRANCE, DR. HENRY SCOTT, DR. J. C. GRIMSON

V.O.N. Advisory Committee

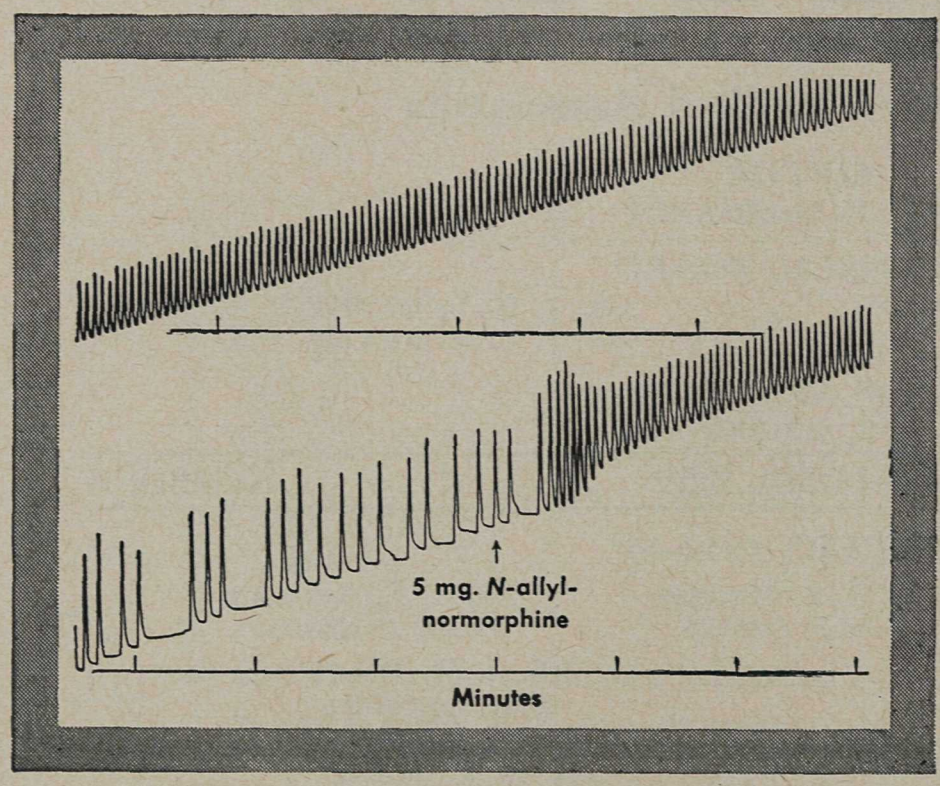
DR. ISABEL DAY, DR. D. M. WHITELAW, DR. R. WHITMAN

Representative to the Vancouver Board of Trade: DR. D. S. MUNROE

Representative to the Greater Vancouver Health League: DR. W. H. COCKCROFT

ublished monthly at Vancouver, Canada. Authorized as second class mail, Post Office Department, Ottawa, Ont.

Announcing a New and Specific Narcotic Antagonist—



Effect of NALLINE *on respiratory depression caused by 57 milligrams of morphine.*[1]

potent and well-tolerated

NALLINE is a specific antidote for poisoning following accidental overdosage with morphine and its derivatives, as well as meperidine and methadone.

This new product, the Merck brand of *N*-Allylnormorphine, rapidly reverses respiratory depression. The respiratory minute volume promptly increases and the rate increases two- or threefold.

A recent study[2] of 270 parturient women indicates that NALLINE may be of value in obstetrics. Onset of breathing occurred significantly sooner in infants from mothers (sedated with meperidine) who were given NALLINE 10 minutes prior to delivery.

Literature available

[1]Eckenhoff, J. E., Elder, J. D., and King, B. D., *Am. J. Med. Scs.* 223:191, February 1952. [2]Eckenhoff, J. E., Hoffman, G. L., and Dripps, R. D., Annual Meeting of the American Society of Anesthesiologists, Washington, D. C., Nov. 8, 1951.

SUPPLIED:
Solution of NALLINE Hydrochloride in 2-cc. ampuls containing 10 mg. of active ingredient, 5 mg./cc.

NALLINE comes within the scope of the Opium and Narcotic Drug Act and regulations made thereunder.

NALLINE

TRADE-MARK

(*N*-ALLYLNORMORPHINE HYDROCHLORIDE, MERCK)

Research and Production for the Nation's Health

MERCK

MERCK & CO. LIM

Manufacturing Chemists

MONTREAL · TORONTO · VANCOUVER · VAL

VANCOUVER MEDICAL ASSOCIATION
PROGRAMME FOR THE FIFTY-THIRD ANNUAL SESSION

Founded 1898; Incorporated 1906

VANCOUVER GENERAL HOSPITAL

Regular Weekly Fixtures in the Lecture Hall

Monday, 8:00 a.m.—Orthopaedic Clinic.
Monday, 12:15 p.m.—Surgical Clinic.
Tuesday—9:00 a.m.—Obstetrics and Gynaecology Conference.
Wednesday, 9:00 a.m.—Clinicopathological Conference.
Thursday, 9:00 a.m.—Medical Clinic.
12:00 noon—Clinicopathological Conference on Newborns.
Friday, 9:00 a.m.—Paediatric Clinic.
Saturday, 9:00 a.m.—Neurosurgery Clinic.

ST. PAUL'S HOSPITAL

Regular Weekly Fixtures

2nd Monday of each month—2 p.m.—Tumour Clinic
Tuesday—9-10 a.m.—Paediatric Conference
Wednesday—9-10 a.m.—Medical Clinic
Wednesday—11-12 a.m.—Obstetrics and Gynaecology Clinic
Alternate Wednesdays—12 noon—Orthopaedic Clinic
Alernate Thursdays—11 a.m.—Pathological Conference (Specimens and Discussion)
Friday—8 a.m.—Clinico-Pathological Conference (Alternating with Surgery)
Alternate Fridays—8 a.m.—Surgical Conference
Friday—9 a.m.—Dr. Appleby's Surgery Clinic
Friday—11 a.m.—Interesting Films Shown in X-ray Department

SHAUGHNESSY HOSPITAL

Regular Weekly Fixtures

Tuesday, 8:30 a.m.—Dermatology.
Wednesday, 10:45 a.m.—General Medicine.
Wednesday, 12:30 p.m.—Pathology.
Monday, 11:00 a.m.—Psychiatry.
Friday, 8:30 a.m.—Chest Conference.
Friday, 1:15 p.m.—Surgery.

BRITISH COLUMBIA CANCER INSTITUTE

685 West Eleventh Avenue,
Vancouver 9, B.C.

SCHEDULE OF WEEKLY CLINICAL MEETINGS

Monday—9 a.m. - 10 a.m.—Ear, Nose and Throat Clinic
Tuesday—9 a.m. - 10 a.m.—Weekly Clinical Meeting of Attending Medical Staff
Tuesday—10:30 a.m. - 11:30 a.m.—Lymphoma Clinic
Daily—11:45 a.m. - 12:15 p.m.—Therapy Conference

B.C. SURGICAL SOCIETY

Spring meeting—April 25th, 26th, 1952.

In "shop talk" about peptic ulcer

TRIMUCOLAN
TRADEMARK

is news

DR. SMITH: *Do you remember that ulcer patient, Trunket?*
DR. HAYES: *How is he doing?*
DR. SMITH: *Better, since we put him on Trimucolan...*

42% recurrence with standard treatment

Studies covering 50 years show 42% recurrence rate in peptic ulcer patients given standard treatment and followed for 2 years. "The overall values, including 64 reports and 13,537 patients, revealed a total recurrence rate of 42% for the immediate to two year study."[1]

18% recurrence mucin-antacid

Hardt and Steigmann treated 125 patients with mucin antacid mixture and found a recurrence in only 18% of patients followed for two years.[2]

---

5 more advantages... TRIMUCOLAN
(MUCIN + REACTIVE ALUMINUM HYDROXIDE + MAGNESIUM TRISILICATE)

1. Prompt pain relief
2. Longer antacid action
3. Better protective coating than antacid alone
4. Fast healing, fast relief of symptoms
5. Less chance of ulcer flare-up

Winthrop-Stearns OF CANADA, LTD.
WINDSOR, ONTARIO

1. Bralow, S. P., Spellberg, M., Kroll, H., and Necheles, H.: *Am. Jour. Digest Dis.*, 17:119, Apr., 1950.
2. Hardt, L. L., and Steigmann, Frederick: Am. Jour. Digest. Dis., 17:195, June, 1950.

443 SANDWICH STREET WEST, WINDSOR, ONTARIO

VANCOUVER HEALTH DEPARTMENT

STATISTICS—JUNE, 1952

Total population (census) 344,833

Chinese population 7,117

	October, 1952	
	Number	Rate per 1000 pop.
Total deaths (by occurrence)	433	15.1
Chinese deaths	21	35.4
Deaths, residents only	368	12.8

Birth Registrations—residents and non-residents
(includes late registrations)

	October, 1952	
Male	553	
Female	518	
	1071	37.3

Infant Mortality—residents only

	October, 1952
Deaths under 1 year of age	13
Stillbirths (not included in above item)	7
Death Rate per 1000 live births	17.7

CASES OF COMMUNICABLE DISEASES REPORTED IN CITY

	October, 1952		October, 1951	
	Cases	Deaths	Cases	Deaths
Scarlet Fever	17	—	60	—
Diphtheria	—	—	—	—
Diphtheria Carriers	—	—	—	—
Chicken Pox	31	—	58	—
Measles	7	—	90	—
Rubella	6	—	5	—
Mumps	71	—	22	—
Whooping Cough	11	—	3	—
Typhoid Fever	1	—	—	—
Typhoid Fever Carriers	—	—	1	—
Undulant Fever	—	—	—	—
Poliomyelitis	17	—	5	—
Tuberculosis	37	12	42	9
Erysipelas	—	—	4	—
Meningitis (meningococcic)	1	—	2	—
Infectious Jaundice	17	—	—	—
Salmonellosis	8	—	4	—
Salmonellosis Carriers	—	—	—	—
Dysentery	—	—	10	—
Dysentery Carriers	—	—	—	—
Tetanus	—	—	—	—
Syphilis	9	1	25	—
Gonorrhoea	106	—	149	—
Cancer (Reportable Resident)	193	70	57	69

CORTICOTROPHIN
(ACTH)

In December 1949, at the invitation and with the financial support of the Federal and Provincial Governments, the Laboratories undertook to produce ACTH in Canada.

Specially collected pituitary glands were obtained across Canada through the helpful co-operation of the Canadian meat-packing industry. The facilities and staff of the Laboratories were applied to the development of methods of production and testing of ACTH, with the result that a product was prepared which met with favourable acceptance. Under the arrangements in effect during the initial period, supplies of ACTH were delivered to the National Research Council of Canada for distribution for research purposes by its Advisory Committee on ACTH and Cortisone.

In January of this year the Laboratories commenced distribution of ACTH direct to Canadian hospitals, physicians and research workers. The product was supplied as a stable, sterile powder, protein in nature and readily soluble in water or saline.

More recently, the Laboratories developed a stable aqueous solution of ACTH (Corticotrophin). This readily injectable form of the product, which is now available in addition to the freeze-dried material, has a potency of 20 International Units per cc. Both forms of the product are free from other pituitary hormones or harmful impurities in clinically significant amounts.

HOW SUPPLIED

Dry Powder	—	10 International Units per vial
	—	25 International Units per vial
Sterile Solution (in 10-cc. vials)	—	20 International Units per cc.

Amounts of ACTH have been expressed in terms of International Units, Provisional U.S.P. Units, and milligrams (of an original house-standard). These various units represent equal amounts of activity as assayed in rats by the ascorbic acid depletion method.

CONNAUGHT MEDICAL RESEARCH LABORATORIES
University of Toronto **Toronto, Canada**

Established in 1914 for Public Service through Medical Research and the development of Products for Prevention or Treatment of Disease.

DEPOT FOR BRITISH COLUMBIA
MACDONALD'S PRESCRIPTIONS LIMITED
MEDICAL-DENTAL BUILDING, VANCOUVER, B.C.

The Editor's Page

We noted with interest the recent publication in the papers of parts of a letter from the B.C. Division of the C.M.A. to the Community Chest of Vancouver. This dealt with the question of narcotic drug addiction, which, as our readers will remember, was dealt with exhaustively in a report by a Special Committee of the Community Chest, headed by Dr. L. E. Ranta, of the University of British Columbia.

This Community Chest report has been the subject of an immense amount of discussion—and of some adverse criticism. It is, therefore, of great interest to read the conclusions to which the B.C. Division has evidently come. This latter body appointed a Committee of its own, which has gone deeply into the question, and its conclusions, with some definite modifications, coincide, on the whole, with those of the Ranta report. These conclusions, it may be noted, are the considered opinions of medical men, including psychiatrists of note: and so are based, not merely on economic or even sociological grounds, but on medical grounds as well, even mainly so.

The problem is admittedly a most difficult one to solve—and will need all our best brains, and our greatest goodwill, to deal with it. Meantime, there are some discouraging, even alarming, features about it which point to the need for immediate, and definite action.

The first of these is the rapid growth of addiction to drugs: there is no doubt about this. Some of the figures we have seen are definitely frightening. And this increase is particularly an evil thing amongst young people.

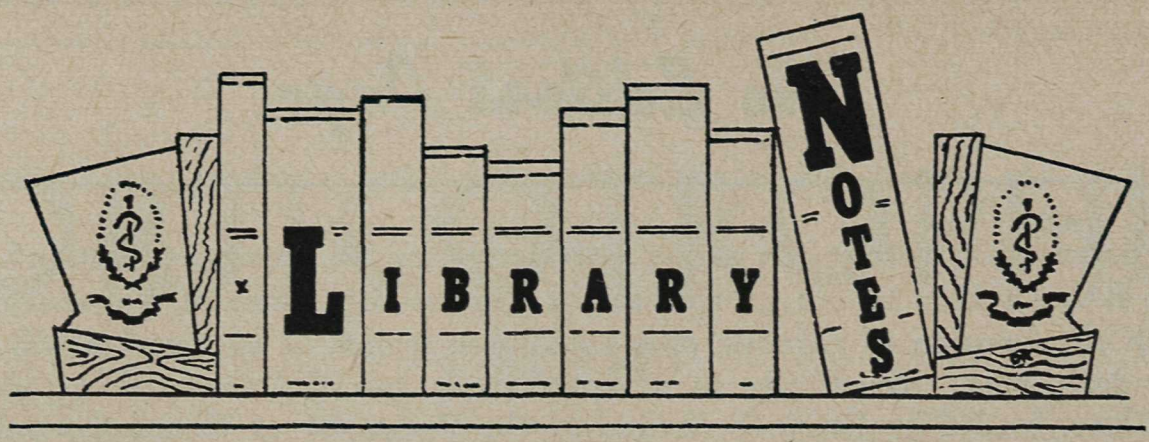
Secondly, we have no provision at all for treatment of addicts. Up to now, society's only action has been at the police level, through the R.C.M.P., operating under the Narcotic Drugs Act. But this is locking the stable door after the horse has got out.

Again, there is no provision for rehabilitation of those who are amenable to cure—and this is one of the most vital needs in any programme that may be devised. Without this, no treatment will do much more than cost a lot of money, for which there will be little or no return.

Lastly, there is no provision for the incurables, who are left to find the drugs they must have, and can only find, in the great majority of cases, through theft and crime. A stay in jail, when they are caught, is only an interlude, till they resume this sort of life when they are let out.

The B.C. Division, like the Community Chest, dealt with all these. One suggestion made by both, is that there should be segregation of addicts in jails and penitentiaries.

There is no doubt that the federal authorities are doing all that they can do, under existing legislation—but there is no doubt, too, that amendments and alterations need to be made in ths legislation, to enable the inauguration of a modern, sociologically and scientifically correct, programme which shall include prevention, treatment (these people are sick people, not criminals alone), rehabilitation where possible, and for the derelicts, some form of control. And time, as the real estate men say, is of the essence of the contract. If this terrible scourge is not effectively handled soon, the numerical growth of addicts in this community will soon make any programme mechanically impossible, through sheer cost in money, institutions and personnel. We applaud the action of the B.C. Division, as of the Community Chest, and feel that they did well to publicise their opinions. It is one more step in the right direction.



Library Hours:

Monday to Friday ..... 9:00 a.m. - 9:30 p.m.
Saturday ..... 9:00 a.m. - 1:00 p.m.

Recent Accessions:

Brain, Sir Russell—Contribution to Medicine to Our Idea of the Mind. Rede Lecture for 1952.

Duncan's Diseases of Metabolism.

Elman, R. Surgical Care—A Practical Physiologic Guide.

Lucas, G. H. W.—Symptoms and Treatment of Acute Poisoning.

Moyer, C. A.—Fluid Balance.

Seletz, E.—Surgery of the Peripheral Nerves.

Duke-Elder, Sir Stewart—Text Book of Ophthalmology, Vol. 5.

Year Book of General Surgery, 1952. Ed. Graham, E. A.

Memorial Volume.

The following book was purchased as a memorial volume to Doctor T. K. McAlpine:

Doctors by Themselves—An Anthology
Compiled by E. F. Griffith

It is intended that a list of current journals received in the Library shall be published each month, and according to subject. This month's list will comprise those journals in the fields of general and special medicine:

Acta Medica Scandinavica
American Journal of Medicine
American Journal of Medical Sciences
American Practitioner and Digest of Treatment
Anales de la Facultad de Medicina de Montevideo
Annals of Internal Medicine
Annals Medicinæ Internæ Fenniæ
Archiva Medica Belgica
Archives of Internal Medicine
British Medical Journal
British Medical Bulletin
Bulletin of the New York Academy of Medicine
Canadian Journal of Medical Technology
Canadian Medical Association Journal
California Medicine
Cancer
Circulation
Clinical Science
Diabetes
Diabetic Abstracts

GP
Gaceta Medica de Lima
Gastroenterology
Indian Journal of Medical Research
Journal of Allergy
Journal of the American Medical Association
Journal of the American Medical Women's Association
Journal of Clinical Endocrinology and Metabolism
Journal of Clinical Investigation
Journal of Experimental Medicine

The rest of the journals in the field of general and special medicine will be published n the next issue of the Bulletin.

THIRD ANNUAL MEDICAL BALL

The Medical Undergradute Society of the University of British Columbia is happy to announce that plans are well advanced for the Third Annual Medical Ball to be held on Friday, March 27, 1953, in the Ballroom of the Hotel Vancouver. The Society hopes that the success of the previous years will encourage their many friends in the medical profession to lend again their splendid support, so that the bursaries established from the proceeds of the preceding Medical Balls can be maintained.

REMEMBER — "THE DOCTORS VIEWPOINT"

Your Radio Programme, every Tuesday at 10:15 p.m.—Station CJOR, 600 on your Dial. Please ask your patients to listen, and do so yourself.

GROWTH IN CHILDREN

H. BAKER, M.D.

"For of all words of tongue or pen,
The saddest are these: "It might have been!"

I can match Whittier's with words equally sad—"In the short time allotted me what can I say about Growth in Children?"

The word growth as used in the above title is synonymous with development. Growth or development in a child is the unfolding of the potentialities of that child changed as they may be by circumstances prenatal or post natal. We must not think that development goes on only in the child. Rather think of it as a process that starts with the fertilized egg and ends with the death of the aged body. We must grasp this monistic concept and realize that we are only talking about part of a large process.

Five factors influence the development of any individual.

1. His inheritance from the human race and all that it represents.
2. The ethnic group to which he belongs.
3. His ancestors remote and immediate.
4. His constantly changing environment.
5. Himself, because the whole represented by him is greater than the sum total of the parts.

The various speakers tonight have chosen different facets of this whole vast problem which they want to discuss. Some of these represent norms. Others represent factors outside normal limits.

In addition to this brief introduction I want to give you a fleeting glimpse into an important part of the neuromuscular development of the child. We take for granted that any well child can pick up things with his hands and lay them down. We seldom stop to think that this is a very complex process which passes through many phases in its development before it can be used.

On fertilization the egg is a very complex unit. As the organism takes on a more mature form it becomes more and more prepared for extra-uterine life. The neuro-muscular system develops as do the rest of the systems. By the 28th week the fetal infant presents 3 main sets of postural reflexes.

(a) Attitudinal.

(b) Righting—These return the creature to the right attitude on the loss of normal orientation.

(c) Stato-Kinetic reflexes are produced by movements designed to keep the fetus moving in the right direction.

We will talk about a very small part of the first one—The attitudinal reflexes. They keep the individual in the right attitude. They determine the basic pattern of orientation to the stimulus of gravity.

The head and neck, very early, exert a controlling influence upon the attitudes assumed by the limbs and torso producing the tonic neck reflex stance. In the classic full-blown t-n-r pattern the faceward arm and leg are in marked extension; the opposite arm and leg are in partial flexion. There is a sensitive interaction between the position of the eyes and the posturing of the limbs and trunk. The eyes assume compensatory poses with changes in stance. In the fetal infant (i.e. the premature) the face but not the gaze is directed toward the extended arm. Even in the newborn post-fetal infant, visual fixation may be rudimentary.

As the full-term infant matures he is able to look at near objects and later adjusts to both near and far objects. The supported seated infant goes though the following stages in the use of his hands in relation to one or more cubes 1" in all dimensions. At 16 weeks he looks at the cube and then at his hand and then at the cube. Later at 20 weeks if the cube is placed in his hand it is held between the tip of the finger or fingers

and the heel palm. He holds it the same way should he grasp it spontaneously. At 24 weeks the fingers close about the cube when it is in the palm. The cube is held more to the radial side of the palm at 28 weeks, with the thumb opposing the fingers. At this time one may even place a second cube in the other hand and he holds them both more than momentarily. This ability to hang onto a cube in each hand is strengthened at 32 weeks, even when a 3rd cube is presented. The growing sensitivity of the fingers is increased further so that at 36 weeks he can hold a cube between end of first and second fingers at thumb. The increased complexity of the neuro-muscular system is reflected in the hand at 40 weeks when he is able to deliberately, albeit clumsily, let the cube go. It is not an accidental dropping. At 48 weeks he utilizes this ability as he moves the cubes, picking them up and letting them drop. By 52 weeks he is not quite able to release one cube on top of another to make a stable 2-cube tower. He does this well at 15 months.

Similarly other facets of the neuro-muscular activities of the developing child have been charted, e.g. When a pellet the size of an aspirin tablet is placed before the seated supported infant at 28 weeks he rakes it with his whole hand. By 32 weeks the raking motion is better oriented and the radial side of the hand takes the lead. At 36 weeks the pellet is picked up between thumb and side of the curled index finger.

At 40 weeks he is able to pick up the pellet between thumb and index or middle finger. At this stage he has to still rest his hand on table surface. By 48 weeks this action will be much better. At 52 weeks he brings the pellet between thumb and forefinger to a presented bottle but cannot drop it in. He accomplishes this well at 15 months.

The human fetus, in his various developmental stages reflects the evolution of the race. The t-n-r harkens to the dim past when one of the ancestral creatures of necessity developed that reflex posture. The t-n-r also looks to the future when it will evolve and change. The primitive posture will grow many highly skilled hand to eye co-ordinations.

By 16 weeks the t-n-r is not easy to elicit and should not be seen at all by 20 weeks. A persistence of the t-n-r after this time is more than suggestive of a brain injured child.

BIBLIOGRAPHY

Developmental Diagnosis—Gesell and Amatruda.
The Embryology of Behaviour—Arnold Gesell.

● ● ● ●

OVERWEIGHT IN CHILDREN

By K. A. CAMPBELL, M.D.

No one who has had any dealings with children and their troubles can have failed to come up with the problem of overweight. The vast majority of these children are seen because of some complaint other than obesity, or are seen on routine check-ups. The parents often are not aware that their child is fat, or won't admit it. If they do become alarmed about the weight, they go to the doctor to find out what gland is not working properly or to get an injection for it.

As my time is limited, I do not propose to discuss obesity due to the endogenous factors, that is, the disorders of the endocrine glands or disorders that are intracranial.

In the light of our present knowledge, endogenous disorders constitute a good deal less than one percent of cases. Hilda Bruch found only one case in one hundred and forty children, most of whom had been referred as endocrine problems.

Before I go any further, I would like to try and clarify what is meant by a "fat child". One author stated it was any child who was more then ten percent overweight, using the standard height and weight charts as a guide. I do not agree with this statement. I think that if the build of the parents, muscular development and

constitutional type of the child are taken into consideraion, he can exceed the ten percent and still not be considered fat. The only good way that I know of making a diagnosis is to examine a child without his clothes—if there is an excess of adipose tissue, then the patient can be called fat.

It is relatively rare that a fat baby or toddler is considered to be an endocrine disorder. This is not the case in the pre-puberty group, however. I am certain that the diagnosis of Fröhlich's Syndrome is made less commonly now than formerly, but this label is still being put on the obese child far too frequently. In obesity of adolescence, the child is usually above normal in skeletal size and the genitalia are normal. Because of the boy's size and the suprapubic fat, the genitalia may appear abnormally small, but this is not the case. Puberty may be a little slow in boys, but is completely normal in girls. The child's skin is fine and moist, and the hair of normal texture. The bone age is normal or slightly advanced; the blood cholesterol and 17-ketosteroids are within normal range. The basal metabolic rate and blood pressure are, if anything, elevated. Add to these points the fact that the Fröhlich Syndrome child is probably suffering from increased intracranial pressure with the resulting symptoms, and the differential diagnosis should not be hard.

A word about the B.M.R. These children do not have normal body proportions, so using weight and surface area in computing the B.M.R., gives you an abnormally low reading. This probably accounts for the misconception that these children lack thyroid. Talbot claims that the most accurate method in a child who does not have normal proportions, as juged by standard age, height and weight charts, is to estimate the muscle mass. This is done by taking twenty-four hour creatinine levels, estimating the muscle mass from charts and using the results to compute the B.M.R. If this method is used, it is found that these children have a normal or slightly elevated metabolism. If the muscle mass method is not available, it is probably more accurate to use the height and disregard the weight in computing a B.M.R.

Incidence of Obesity

(1) The two age groups where you are most likely to meet obesity are under three years and between eight and twelve years of age.
(2) It occurs almost equally often in girls as in boys.
(3) It is more frequent in the slightly below average income group.
(4) The intelligence quotient of the parents and children have very little bearing on the situation.
(5) Heredity seems to play a role, and there seems to be a fair number of obese pre-puberty children in whom fatness at this age is claimed as a family trait.

This angle needs clarifying. Some authors claim that it is over-anxiety on the part of the mother to prevent the child suffering as she did that actually perpetuates the condition, instead of helping it.

Etiology

The simple laws of physics apply here as elsewhere. The child that has gained weight must take in more than he puts out. This means that either the intake is in excess, or the output below normal. There are, therefore, two questions. First, can an obese child eat a normal amount and utilize it in such a way as to get more calories out of it than a normal child? Experiments indicate that he can not. Both types absorb the same percentage of food and utilize it in the same way. Therefore, the only way for the intake of calories to be excessive is to eat excessively. Secondly, does an obese child utilize less energy in taking a normal amount of exercise? The only way that this could happen to an appreciable extent would be for the B.M.R. to be lower.

There are other factors that have to be considered, such as the dynamic action of food, but it is felt by most authors that they couldn't play a significant role. For a long time, it was felt that the B.M.R. was below normal and it played an important role. But recent work, notably by Talbot, as I mentioned before, indicates on the

contrary that it is elevated. Answer to the second question as to whether the obese child uses less energy is, therefore, "No." So far, the problem has been relatively simple. The obese child you see in your office, in the vast majority of cases, is not suffering from an endocrine or intracranial lesion, but is a child that has been eating more than he can metabolize and storing the excess as fat.

The next big question is why does the patient overeat? Several possible answers have been put forward.

(1) To satisfy an emotional need. According to most authorities, this accounts for the majority of cases.
(2) Faulty feeding habits caused by mothers forcing food.
(3) Faulty feeding habits of the family setting a poor example.
(4) Less output due to illness, but same intake due to habit.
(5) Abnormal gratification obtained by flavour. For example, the so-called "sweet tooth".
(6) Feeling of comfort only obtained by very full stomach. This may account for some of the obesity in very small babies. Several other possibilities have been given, but they start to get pretty far-fetched, and I don't think need to be seriously considered.

Practically all the literature deals with the psychological basis for overeating, and no more than mentions the other possible causes. Kanner claims a psychogenic basis in all cases. In his series, 50 percent of children were frankly unwanted. He states "the insecure and unloved child seeks relief from feeling of anxiety and helplessness by overeating". Kanner, however, is a psychiatrist and probably only sees the serious problems. His case histories show pretty clear-cut psychogenic bases. The obesity I see in my office is certainly not nearly so clearly psychogenic. Hilda Bruch claims that with the rejected child, food stands for security, love and satisfaction, while muscular activity stands for insecurity, danger and threat. So the child increases the food intake and cuts down the exercise. The psychogenic theory seems to be gaining in popularity. In doing any reading on the subject, one forever runs across the words "insecurity" and "feeling of rejection". However, there are still some authors, notably Joseph Warkany, who feels that emotional problems involved are largely the result of obesity, and not the cause of it. Certainly, there is a vicious circle there. The fatter the child gets, the more he is teased and rejected by playmates, and the more he withdraws, seeking solace in food.

Treatment

In treating these patients, one should attempt to find out why there is an increased intake of food before one prescribes a diet, etc. In the severe cases, when it is quite apparent that there is a definite psychogenic background, I feel that the help of a psychiatrist or a child guidance clinic should be obtained. But if you don't get help, you must proceed slowly and with caution. Parental rejection may be quite apparent early in the interviews, however, unless a good deal of groundwork has been laid and the parents have a fair amount of insight, the mere mention of the word "rejection" may be just enough to terminate the interviews. In the average case, the one you will see by far the most frequently, diet, reassurance and encouragement will probably be enough.

The diet is usually 1,000 to 1,500 calories with as much bulk as possible, high in protein and with all the necessary vitamins. I don't feel one should attempt to take off too much or take it off too quickly. Even if the weight is held steady, the appearance will improve as the child grows. Encouragement is very important, and constant supervision is necessary by the doctor—at least every two weeks. The child should be encouraged to develop outside interests, clubs, hobbies, etc. Too much fresh air and exercise should probably be discouraged until the child has got used to the diet, as these will tend to increase the appetite. In some cases, little can be done until the child reaches puberty and thus develops new interests and a new awareness of himself.

The anorexigenic drugs may have a place in getting the child started but shouldn't be carried on too long. There has been very little written about their use in pediatrics, and I, personally, have had no experience with them.

Summary:

(1) An obese child gets that way by eating more than he can metabolize and storing the excess as fat.

(2) The vast majority of obese children are not suffering from an intracranial or endocrine disorder.

(3) An emotional factor is felt to be the main cause of overeating.

(4) In sever cases, psychiatric treatment is necessary, but in moderate cases, diet and outside interests are all that are needed.

● ● ● ●

ENDOCRINE THERAPY IN CHILDHOOD

By JOSEPHINE MALLEK, M.D.

Whenever a child grows too tall or too short, too fat or too lean, or when his genitalia develops to early or too late, he is brought to his physician for the treatment of an endocrine disorder, for both the general public and the medical profession are inclined to believe that every disorder of growth is associated with some endocrine abnormality. This is not necessarily true, and it is essential that we recognize those disorders which are not produced by the endocrine glands and which will not respond to endocrine therapy. I call to mind an achondroplastic child three years of age who has received an injection of some pituitary extract every day for the past two years. This therapy can have no influence upon the achondroplasia, it may cause considerable damage to the child's metabolism and ultimate development and has been the financial ruin of the family.

Of the non-endocrine factors controlling growth the first to be considered is the genetic inheritance. The importance of this factor can best be explained by drawing an analogy from dogs. If a chihuahua, a dachshund and a Great Dane were taken as young puppies and were brought up under ideal conditions, with a diet of chicken livers and filet mignon, the chihuahua would still grow up to be a shrimp, the dachshund would remain close to the ground, and the Great Dane would probably become too large to keep in the house. No care, no feeding and no endocrines could make a Great Dane out of a chihuahua. As a result of his genetic inheritance he has a very limited capacity for growth. To return to the human, if one's father and grandfather have been famous jockeys, one's chance of reaching six feet in height is very poor. The best one can do is to buy a horse and learn to ride.

Another very important non-endocrine factor is the end organ which must be capable of normal growth. In certain congenital malformations classed as achondrodystrophies, the growing portions of the bones have been so altered as to be incapable of growing normally. No therapy can alter this situation.

Assuming that one has an average genetic inheritance and that no abnormality of the end organs exists, growth should proceed along average lines. However, this growth can be interfered with at any time during the life of the individual by a series of external or internal factors. The external factors are concerned with nutrition. Adequate nutrition implies that the essential amino-acids, the vitamins and minerals and oxygen are available to the individual in sufficient amounts. In times of war and famine children will be stunted owing to starvation. The mere availability of food, however, does not necessarily imply adequate nutrition. In anorexia nervosa the person refuses the food. With chronic pyloric stenosis the food is vomited. With celiac

disease it is not absorbed. In metabolic disorders such as liver disease, diabetes or chronic renal disease, it is not metabolized normally, and is therefore not available to the organism for growth.

Adequate oxygenation of the tissues is an important factor in influencing growth. In congenital heart disease, with mixing of the arterial and venous streams, producing a low oxygen saturation in the tissues, growth may be markedly impaired. This may also occur in lung diseases, such as polycystic disease or severe asthma. The oxygen lack resulting in dwarfism.

The internal factors influencing growth are under the control of the endocrine glands of which the pituitary is the master gland. Only those functions of the pituitary will be considered here which have an effect upon growth. With one exception, the pituitary gland secretes a number of tropic substances which regulate the activity of various target glands, which in turn furnish hormones to the body tissues or end organs. The one exception is pituitary growth hormone which has a direct action upon the end organ without the intervention of a target gland.

The effects of a hormone can best be studied by observing clinical syndromes in which the hormone is known to be lacking, and those in which it is known to be present in excess. In the case of growth hormone we are familiar with the condition of giantism resulting from excessive production of growth hormone by an eosinophil tumor of the pituitary. The tumor, by compression or invasion, eventually destroys all the normally functioning pituitary tissue. The giant, unlike the hero of our fairy tales, soons suffers from multiple pituitary deficiencies with lowered metabolism, diabetic blood picture and later hypoglycemia and failure of the gonads. He finally collapses unheroically in a heap.

Lack of pituitary growth hormone should produce an individual in miniature who is normal in every other way, with normal mental development, ossification time and sexual maturation. The result would be a tiny adult or human chihuahua. Such an individual should be capable of accelerated growth, if treated early with potent growth hormone. Unfortunately, growth hormone preparations, although highly purified are found to be species specific, and there is no growth hormone available at the present time which has the slightest effect in stimulating growth in man. In the absence of a therapeutic test the etiology of the above condition can not be proven. Some authorities believe that the diminished capacity of the tissues for growth depends on a hereditary genetic factor, the effects of which are not mediated through the pituitary gland, and have called the condition primordial or genetic dwarfism.

Of the tropic substances produced by the pituitary the thyrotropic, adrenotropic and gonadotropic factors will be considered. The thyrotropic principle acts upon the thyroid gland and causes it to secrete thyroid hormone which in its turn accelerates the metabolism of all the tissues in the body. A lack of thyrotropic factor results in a lack of stimulation of the thyroid gland to secrete, and produces a thyroid deficiency state. A similar deficiency can result from congenital lack of thyroid tissue or from destruction of the gland by disease, surgery or radiation. Cretinism occurs where the thyroid deficiency is present from birth. The thyroid has its most notable effect on growth and maturation of tissues in the early years, so that the cretin is characterized by defective growth, delayed bony age and impaired mental development.

The pituitary adrenotropic factor stimulates the secretion of three different groups of hormones by the adrenal. The first controls sodium and potassium metabolism and has no direct effect upon growth. The second, or corticosteroid, promotes gluconeogenesis. It increased the utilization of amino acids and fatty acids for the formation of carbohydrate. Hypersecretion tends to diminish growth and may lead to obesity. The picture of the moonfaced, hirsute, obese individual with ravenous appetite has become very familiar since the clinical use of cortisone and A.C.T.H. in the treatment of numerous chronic diseases.

The third group of hormones secreted by the adrenals are the androgens. Two-thirds of the androgen produced by the male and all of the androgen produced by the

female are derived from this source. They stimulate nitrogen retention with protein synthesis and are responsible for the accelerated growth and muscular development which occurs during adolescence in both sexes. Sexual hair in the female depends upon adrenal androgen and this hormone is important in the sexual development of bth sexes. With excessive secretion, as occurs in adrenal hyperplasia or adrenal tumor, the male child shows an accelerated growth, excessive muscular development and precocious puberty. The female child presents a spectacular picture with rapid growth, very early closure of the epiphyses, acne, hirsutism, voice change, pubic hair and an enlarged clitoris which may reach the proportion of an adult penis.

The gonadotropic factors stimulate the ovaries in the female, and the testis in the male to secrete their respective hormones. These factors are responsible for the onset of puberty in both sexes with the development of secondary sex characters. Puberty is accompanied by a rapid spurt of growth and a muscular development which is most marked in the male. This growth soon comes to an end, for the sex hormones, and particularly the estrogens bring about closure of the epiphyses. Failure of pituitary gonadotropic factor can be demonstrated by a lack of follicle stimulating hormone (F.S.H.) in the urine. The clinical picture is that of infantilism—a failure of the secondary sex characters to develop. This may be accompanied by excessive growth owing to a failure of the epiphyses to close at the expected time. Primary failure of the gonads may produce a similar picture, but can be distinguished from pituitary failure in that follicle stimulating hormone will be present in normal or excessive amounts in the blood and urine.

A syndrome has been described in recent years, called ovarian agenesis. It is being recognized with increasing frequency and consists of dwarfism with a primary failure of the genital ridge to develop. In spite of the absence of ovarian hormones these children are very feminine. They lack breast development and have infantile genitalia, but show normal pubic and axillary hair. The dwarfism can not be considered to result from the ovarian failure but is an associated congenital abnormality. Other defects are frequently noted, such as webbed neck and coarctation of the aorta.

Finally, the pituitary may fail to secrete any of its factors, producing the condition of panhypopituitary dwarfism. In this syndrome, the dwarfism is accompanied by a lack of thyroid factor, adrenal factor and sex factor. The metabolism is therefore low, hypoglycemia is present, sexual maturity is not attained, and the bony age is delayed. The lack of growth in these cases is not entirely due to a failure of the thyroid function. For these children do not look like cretins, they do not behave like cretins and they fail to respond to thyroid therapy.

INFORMATION WANTED

The Bulletin has a request for information as to the whereabouts of Dr. Bill Poul (or Powell) formerly of Vienna. He is about 60 years of age and is believed to be somewhere in British Columbia. Any information might be forwarded to the Bulletin, 675 Davie Street, MArine 7729.

REMEMBER — "THE DOCTORS VIEWPOINT"

Your Radio Programme, every Tuesday at 10:15 p.m.—Station CJOR, 600 on your Dial. Please ask your patients to listen, and do so yourself.

CRETINS—The relations of the Hypothyroid State to Growth and Development

By HAROLD KRIVEL, M.D.

About four-fifths of the patients who have hypothyroidism during childhood present definite evidences that the deficiency has existed from birth. The term cretinism is applied to these cases of congenital hypothyroidism. The most marked changes in growth and development occur in this group as opposed to acquired hypothyroid patients who apparently have been enirely normal children during their earlier years.

There is no diagnostic sign or test for hypothyroidism. The entire clinical picture must be evaluated to determine whether there are physiologic and biochemical evidences compatible with disturbed thyroid function and whether these are accompanied by the signs of retarded growth and development which results from hypothyroidism in early childhood. Our chief interest tonight is the relations of the hypothyroid state to growth and development.

(1) *Stunted Growth*

Growth is always slowed in hypothyroid children. In the first few months of life the cretin may not be much shorter than the normal infant of the same age. However, as he grows older, his height lags further and further from normal. Long standing hypothyroidism dating from early childhood results in some of the most extreme forms of dwarfism encountered. If the deficiency does not occur until late childhood, the patient already will have attained the normal height for age, but growth may become markedly slowed from then on.

(2) *Skeletal Proportions*

The hypothyroid dwarf remains infantile in his build, having a ratio of upper/lower skeletal segments, as measured from the symphysis pubis, more nearly commensurate with his height than with his age. The facial expression is characteristic because failure of naso-orbital development causes the bridge of the nose to be flat and broad, so that the eyes are set far apart.

(3) *Retardation of Osseous Development*

The rate of osseous developement is always retarded in hypothyroidism. In the first three or four years of life when there is normally a rapid appearance of many new epiphyseal centers, the delay in osseous development soon becomes obvious. Unless thyroid failure has occurred only recently or the patient has been treated previously, absence of osseous retardation excludes the diagnosis of hypothyroidism. Delay in epiphyseal ossification alone does not establish a diagnosis of hypothyroidism, as marked retardation may occur in pituitary dwarfism with no thyroid deficiency in cases of delayed adolescence and in children stunted from nutritional and other causes. When bone age is retarded because of thyroid deficiency, substitution therapy with thyroid causes a rapid acceleration of osseous development; when retardation is due to other causes, thyroid treatment has little effect.

Epiphyseal dysgenesis: When hypothyroidism exists there occurs a disorder in the cartilages of the epiphyses and round bones leading subsequently to irregularities of ossification which can be visualized in roentgenograms as "styled epiphyses." In normal ossification there develops in the center of the epiphyseal cartilage, an area in which the cartilage cells become more closely arranged, swollen and vacuolated, while the matrix becomes more basophilic. In the center of this zone of altered cartilage, preliminary calcification and then ossification follows rapidly in the same manner as at the epiphyseal lines of the long bones. Normally ossification extends peripherally from this central area in an orderly manner, being preceded by a narrow zone of the altered, swollen cells. Experimentally in thyroidectomized kittens, it has been shown that the preparatory alteration of the cartilage in the centre of the epiphyses occurs normally, but is not followed promptly by calcification or ossification. The area of change

becomes gradually longer until it may occupy a considerable portion of the epiphyses. When eventually ossification begins, it arises from multiple irregular foci scattered throughout the area of altered cartilage. These changes probably account for the fact that in hypothyroid children, not only is the ossification of the epiphyses delayed, but it appears in the roentgenogram as small irregular foci scattered over a considerable area of the cartilage. These grow longer and coalesce to form a single irregular centre. According to the stage of process, the roentgenogram may show multiple small centers of ossification or a single centre may appear either stippled, porous, fluffy or fragmented. By means of frequent serial roentgenograms taken before and during thyroid treatment, it has been shown that these changes occur in all centres which normally would have ossified during the span of life when thyroid deficiency existed. The occurence of this type of epiphyseal dysgenesis is one of the most characteristic evidences of hypothyroidism because it seldom is found in other dwarfs or stunted children, even though they may have marked retardation in their osseous development.

(4) *Retardation in Dental Development*

The development of the teeth is retarded in hypothyroidism and the delay usually parallels that of endochondrial ossification. The position and shape of the teeth is very irregular. Roentgenographic evidences of delay of the formation of the tooth buds is of more significance than merely retarded eruption of the teeth. The latter may be due to lack of development of the alveolar processes and may occur in other conditions of stunted growth.

(5) *Retardation and Defect in the Development of the Brain*

There is evidence that in early life, thyroid deficiency may cause—

(a) Stunting of mental reactivity and responsiveness

(b) Retardation of the development of the brain

(c) In some cases—permanent damage with histological changes in the brain cells.

In addition it is possible that in some cases, there may be a congenital malformation of the brain coincidental with the embryonic defect responsible for the lack of development of the thyroid. Before treatment, almost all cases show some degree of mental retardation, often to a severe degree (imbeciles). If thyroid deficiency does not develop until later childhood when the maturation of the nervous system is complete, no retardation or defects in brain development may occur, and the patient may have a normal intelligence quotient. However, even in these cases prior to treatment, the mental reactions may be sluggish and the response slow.

One must observe caution in predicting the intellectual future of cretins, irrespective of both the time at which treatment is first instituted and the adequacy of the treatment as judged by physical criteria. Information regarding the rate of spontaneous functional progress before treatment supplies a fairly reliable basis for prediction. Too vigorous use of thyroid can cause a deterioration in the ability to perform and in the behaviour of a long standing case of cretinism.

Treatment

The treatment is specific. An established brand of fresh V.S.B. thyroid should be employed. It must be remembered that hypothyroid patients show a marked sensitivity and respond to small doses of thyroid. It is safe to begin with an initial dose of 1/4 gr. daily in very young cretins and 1/2 gr. in older children. With a uniform daily intake there is an accumulative effect, so that maximum results from a fixed dose are attained after about 20 days. It is therefore unwise to increase the dose more frequently than every three to four weeks. The average case requires one to three gr. daily. With adequate treatment, the results from the standpoint of growth and development are good. The characteristic signs of the cretin disappear and there is rapid growth and osseous development demonstrated after two to four months. The earliest clinical signs of improvement are —increased warmth and colour of the skin, and a marked increase in the patient's activities and responsiveness. Ultimately these patients cannot be distinguished from normal individuals, frequently however, they do not attain average height before their epiphyses close.

CHONDRODYSTROPHY

By G. A. GAYMAN, M.D.

One cause of shortening of the skeleton is to be found in the group of entities embraced by the term "Chondrodystrophia," or decelerated growth of cartilage.

Three subgroups of chondrodystrophy will be presented: the first and by far most common is that formerly termed "Achondroplasia"; the second is Morquio's disease or osteochondrodystrophy and finally Hurler's Syndrome, lipochondrodystrophy or gargoylism.

The common pathogenetic factor in these diseases is retarded or irregular growth of endochondral bone—i.e., bone laid down in the calcified cartilage matrix of the epiphyseal plate. Also this type of bone growth ceases at the normal age of the patient or earlier; hence due to shortening of the trunk, extremities or both, when final adult maturation is achieved, a dwarfed individual results.

Another common factor is that these malformations are supposed to be determined on a genetic as opposed to those acquired diseases which may result in dwarfism. Among those—such examples as Pott's disease of the spine, untreated rickets and the end result of severe poliomyelitis might be cited.

We shall now present brief case historie of examples of the three types of chondrodystrophy, show their photographs or present them in person, and finally Dr. Fraser will demonstrate the salient radiographic features exhibited by each group.

Note: Dr. Moore at Children's Hospital has the Case histories.

● ● ● ●

CASE REPORT

The following two cases were presented at a recent meeting at the Children's Hospital.

By H. STOCKTON, M.D.

CASE (1)—B. A. Male, born on the 18th of November, 1948.

He was brought in originally for his failure to grow normally.

He was a full term baby and weighed five pounds. His neonatal history was essentially negative.

Family history—negative. He has a healthy, normal brother who weighed eight pounds at birth.

Development history. He sat at six months and walked at one year. His speech at three and a half years, when first seen, was rather retarded, although he speaks much better now. Some of this may have been due to a considerable amount of family discord at home which ended in a divorce, and consequently the boy was ignored a good bit of the time. At one year he weighed thirteen pounds and his weight when first seen, at the age of three and a half years, was twenty and a half pounds. In other words, definitely below that of an average year old child. His present weight is thirty and three-quarter pounds, which is roughly normal for a year old child.

Past illnesses—essentially negative.

Nutritional history is favourable. He has always received adequate nutrition and adequate supplementary food substances.

Physical examination revealed a small but normally proportioned little boy who in every way appeared to be about a one year old child. He had a rather delicate build and muscular development was rather below par. His head appeared small uniformly, and by measurement, was smaller than one might expect in reference to the chest and other measurements. However, he was not a microcephalic and this was supposed to be an inherent condition with no bearing on his general condition. Otherwise, physical

examination was quite negative. Ears, eyes, nose, throat—negative. Heart and lungs—negative. His cranial sutures were closed as well as his fontanelles. A flat plate of the skull was done which revealed a normal sella turcica and his eye grounds were quite normal.

All the pertinent laboratory work was done. His general blood picture was normal, as well as his blood chemistry, including serum calcium, phosphorus, alkaline phosphates, potassium, sodium, sodium chloride, cholesterol and also a fasting blood sugar. Glucose tolerance test was negative. Sedimentation rate was normal. Routine urinalysis was normal on several occasions and his urinary 17-ketosteroids were normal. Tuberculin test was negative. Kahn negative. Serum protein was normal. Fat content of the stool was within normal limits. X-ray investigation showed a completely normal age.

Most of the causes of dwarfism were ruled out by this investigation. The 17-ketosteroids and serum sodium chloride were normal, as were the potassium and sodium; and the fact that both testicles were descended, would certainly rule out a pituitary cause for his dwarfism, because it is exceedingly doubtful that the pituitary gland has a discriminatory selective effect on growth alone, without affecting sexual development or certain aspects of the blood chemistry. Normal bone age and the closing of sutures would further bear this out. These facts would rule out the possibility of a pituitary tumour. The fact that he was born small and that his rate of development has been consistent would tend to rule out other causes in dwarfism. Rickets, heart disease, celiac disease, hypothyroidism, would all be ruled out and we are left with rather an apparent diagnosis of primordial dwarfism. This can be a purely constitutional condition, inherent in the individual but not inherited, but in this boy's case, the fact that his grandfather was a dwarf, appears to be significant. It may be a Mendelian recessive characteristic and this might be the case here. There is no known successful type of therapy beyond good nutrition and general attention to hygiene.

CASE (2)—J. M. Male.

This boy is a dwarf in that his stature is short. The only other point of similarity to the above case is that it has a constitutional background. Otherwise ,these two cases are poles apart.

This boy was born November 17, 1947. He was a full term delivery with a normal birth history but immediately after birth it was perfectly apparent that there were grossly abnormal features present. His head was large and broad. He had a short neck. His chest was deformed in a characteristic manner. It was inclined to be broad, with a shortened antero-posterior diameter, and there were nodular swellings seen which were apparently deformities of the ribs. These were numerous and situated near the sternum. His extremities were short and deformed in a very characteristic way, in that they were bowed antero-posteriorly and the mid-shaft areas of the arms and legs were thickened considerably. Radiological studies showed some thirty-odd fractures to be present, including the long bones, the ribs, the clavicles and the vertebrae. It was interesting that the callus formation appeared to be about three weeks old, so the stress resulting in these fractures was sustained in utero about three weeks before term. His skull showed typical wide sutures and the Wormian formation of skull bones.

During the intervening years, this boy was admitted on numerous occasions for fractures involving the long bones and other bones of stress, such as the clavicles. Recently, this tendency to spontaneous fracture has been less obvious and he has not required as much orthopedic care.

This is a case of *osteogenesis imperfecta,* to which we may add the term congenitalis, because it was present at birth. In his case there was no family history. The other type in which they acquire this picture later in infancy or early adulthood, is on an obvious familial basis. There is probably a relationship between the two and they are caused by inherent defects in the mesenchyme, because in the latter type they often show otosclerosis and other bony defects. Blue scleræ, which is due to abnormally

thin scleræ as seen in this case, is very typical. The callus formation is prompt in healing and early but it gives rise to defective bone, the nature of which we are not sure. It is like a workman building a brick wall, who has proper tools, good type of brick and mortar, but owing to incompetency is not building a strong structure. Complete investigations on an endocrinological basis, blood chemistry including calcium, phosphorus, phosphates, all significant and pertinent labratory findings, are completely negative, as was in this case and there is no known treatment.

● ● ● ●

TWO CASES OF DWARFISM IN CHILDHOOD

By B. SHUMAN, M.D.

Case (1) Ovarian Agenesis.

Case (2) Pituitary Dwarfism.

CASE (1)—I. K. Female. Age 14 years.

This child was first seen by the Children's Hospital Travelling Clinic in April, 1952. She was brought in for consideration of growth failure and lack of sexual development. The patient was admitted to the Children's Hospital in July, 1952, for investigation.

Birth History: Normal pregnancy and labor. Full term. Birth weight 5 lbs. 9 oz. The mother stated that the infant's length was 15 inches at birth. The baby gained well and ate well in its early months and subsequently.

Developmental: Dentition occurred normally but the deciduous teeth were poor were lost early. Permanent dentition, normal. The baby began to talk at 1 year and walked at 15 months. Her school work has been exceptionally good and she is now in grade IX.

According to the mother, the child's growth has always lagged behind those of other children of comparable age. At present, she has attained a height of 4′ 5½″ as compared to an average of 5′ 3″ among her girl classmates.

The patient has not menstruated as yet and breast development has not yet started.

Past Illnesses: Severe pertussis, age 12 months.

Family History: No incidence of dwarfism in family. Mother is 5′ 2½″, father 5′ 10½″. Two male siblings, age 19 years, 5′ 11″, and age 16 years, 5′ 9½″.

Physical Examination: The patient presented a bright and animated appearance. She was stocky in build, her arms and legs seeming too short for the muscular trunk. Her hands and feet were small and her fingers short and thick. Her measurments were as follows:

Ht. 53½″. Wt. 85 lbs. Head to pubes 29½″. Pubes to floor 24″. Span 52″. Head circumference 21½″. Pulse 92. B.P. 105/75.

There was no breast development and the nipples were small and preadolescent in appearance. There was prominent light coloured hair on the forearm, sparse axillary hair and moderate pubic hair, the latter having been present for about 1 year.

Gynecological examination revealed underdeveloped labia and a prominent urethral meatus. A tiny cervical os was seen and on pelvic examination no uterus or adnexæ could be palpated.

X-rays for bone age showed that the epiphysis for the olecranon, which unites at 14 years, was still present. Ossification of the external epicondyle and radial head was also not yet complete. This indicated some retardation in bone development. There were no distinctive anomalies of the sella turcica in the skull x-ray.

This patient falls into the category of cases described by Turner in 1938, characterized by shortness of stature and sexual infantilism consisting of failure of development of secondary sex characteristics and primary amenorrhea.

Allbright in 1942 pointed out that this condition was primarily an ovarian deficiency and not due to pituitary dysfunction. Non-development of the ovaries alone was responsible for the sexual infantilism. He described these cases as presenting the following features:

(1) Shortness of stature, but not the dwarfism which is found in panhypopituitarism.
(2) The presence of axillary and pubic hair indicating normal adrenal function.
(3) Infantile breasts, uterus, vagina and labia.
(4) The patients are strong and well nourished, unlike the frail, easily tired pituitary dwarf.
(5) Moderate retardation of skeletal maturation. Epiphyseal closure is delayed but occurs eventually. In hypopituitary dwarfs epiphyseal union is often delayed indefinitely.
(6) The frequent presence of other congenital defects such as webbing of the neck, cubitus valgus, coarctation of the aorta, ocular changes and Madelung's deformity.
(7) Hormone assays reveal an increase of the follicle stimulating hormone of the pituitary and the presence of moderately reduced amounts of 17-ketosteroids in the urine.

The treatment of this condition consists entirely of substitution therapy with estrogen substances. This brings on the sexual maturation which has hitherto failed to develop. Estrogen and progesterone therapy produces a secretory type of endometrium and menstruation is produced. Marriage and marital relationship are entirely possible but the patient should be made aware that gestation cannot occur.

Dwarfism in these cases is considered to be a genetic defect as are the other congenital defects often found concomitantly. Quite the reverse might be expected in the growth pattern in these cases since in hypogonadism with onset before puberty normal or increased stature is the rule because of delayed epiphyseal closure.

CASE (2)—Pituitary Dwarfism. D. B. Female. Age 5 years.

This patient was presented at the Children's Hospital Travelling Clinic in April, 1952, for consideration of growth failure which had ceased at the age of 10 months.

Birth History: Normal pregnancy and labor. Full term infant. Birth weight 7 lbs.

Development History: Dentition began at 6 months. She walked at 15 months and talked well at 2 years. Her mother noted a tendency to alopecia up to 3½ years of age.

The mother stated that from the age of 10 months the child had ceased to grow in length. The child's weight at 2 years was eleven pounds and at 3½ years was 15 pounds. When seen in April, 1952, the child's weight was 15 lbs. and on admission in July, 1952, was 17 lbs.

Family History: There was no incidence of dwarfism in the family of either parent. The mother is 5′ 6½″ and the father is 5′9″. One male sibling age 6 years is normal in height.

Physical Examination: The patient presented a bright, friendly appearance. She was 28″ tall and appeared frail and doll-like. She spoke animatedly and intelligently as one might expect of a normal 5 year old. Her build was proportionate. The measurements were as follows:

Height 28″. Head circumference 18¼″. Head to pubes 16″. Pubes to floor 12″.

Physical examination revealed no other abnormalities of note. Her anterior fontanelle was closed.

X-ray of skull and wrists for bone age:

The skull diameters were normal. The sella turcica was normal. There were three centres of ossification at the wrist indicating a retardation of bone development at the 3-year level.

This case falls into the category of dwarfs called Pituitary or True dwarf. It is primarily due to a failure of the growth factor of the anterior pituitary gland. Pituitary dwarfism can be due to an accidental disturbance of the anterior lobe or it may be genetically determined. Congenital hypoplasia of the anterior lobe of the hypophysis is said to cause the hereditary form of pituitary dwarfism as we feel is the cause in this case. The mode of inheritance is recessive in most of the cases. In non-hereditary cases of pituitary dwarfism congenital malformation, new growths of progressive pathological processes near or in the pituitary must be considered etiologic possibilities.

Many pituitary dwarfs have panhypopituitarism. They lack not only growth hormone but also gonadotropic and adrenotropic hormones and possibly thyrotropic hormones. In partial hypopituitarism there may be a deficiency of one hormone while the others are normally secreted. This explains the existence of pituitary dwarfs who are sexually normal.

Pituitary dwarfs are usually of normal size and weight at birth. Growth is slow and usually occurs in spurts but always below normal. Epiphyseal closure is retarded indefinitely. The proportionate growth in childhood is continued into adult life giving the adult a childlike appearance.

The prognosis in pituitary dwarfism is naturally dependent on the severity of pituitary deficiency. In the severe cases infantile cachexia similar to Simmond's disease in the adult may result. In milder forms the patient may reach old age.

Prognosis for growth is difficult since an increase in height is possible long after the age of adolescence because of the persistance of epiphyseal clefts. Sexual maturation may also take place 10-20 years later than in normal persons. The adult dwarf usually does not exceed 130 cm.

The effects of treatment are difficult to evaluate since spontaneous growth is possible at any age. Crude anterior pituitary extracts now available are thought to have little significant effect upon growth in doses that are now practical. Contradictory results concerning treatment with extracts may be due to differences in the preparations used.

This patient will be kept under observation for one year and her growth observed before any treatment will be contemplated. Methyltestosterone is currently considered to be successful for growth promotion in such cases. Estrogen is used to offset virilization in females. Some workers feel that thyroid and methyltestosterone simultaneously produce the best results.

POSITION WANTED

Physician certified in internal medicine desires to do general medicine in an established practice. Will gladly discuss any opening. Write: The Bulletin of the Vancouver Medical Association, 675 Davie Street.

REMEMBER — "THE DOCTORS VIEWPOINT"

Your Radio Programme, every Tuesday at 10:15 p.m.—Station CJOR, 600 on your Dial. Please ask your patients to listen, and do so yourself.

British Columbia Division
Canadian Medical Association

1807 West 10th Ave., Vancouver, B.C. Dr. G. Gordon Ferguson, Exec. Secy

OFFICERS—1952-1952

President—Dr. J. A. Ganshorn ..... Vancouver
President-elect—Dr. R. G. Large ..... Prince Rupert
Vice-President and Chairman of General Assembly—Dr. F. A. Turnbull ..... Vancouver
Hon. Secretary-Treasurer—Dr. W. R. Brewster ..... New Westminster

Members of the Board of Directors

Victoria
Dr. G. Chisholm
Dr. E. W. Boak

Nanaimo
Dr. C. C. Browne

Prince Rupert and Cariboo
Dr. R. G. Large

New Westminster
Dr. J. A. Sinclair
Dr. W. R. Brewster

Yale
Dr. A. S. Underhill
Dr. C. J. M. Willoughby

Vancouver
Dr. F. A. Turnbull
Dr. A. W. Bagnall
Dr. F. P. Patterson
Dr. P. O. Lehmann
Dr. G. C. Johnston
Dr. Ross Robertson
Dr. R. A. Gilchrist
Dr. J. Ross Davidson
Dr. R. A. Palmer

Kootenay
Dr. J. McMurchy

Standing Committees	*Chairmen*
Constitution and By-Laws	Dr. R. A. Stanley, Vancouver
Finance	Dr. W. R. Brewster, New Westminster
Legislation	Dr. J. C. Thomas, Vancouver
Medical Economics	Dr. P. O. Lehman, Vancouver
Medical Education	Dr. T. R. Sarjeant, Vancouver
Nominations	Dr. J. A. Ganshorn, Vancouver
Programme and Arrangements	Dr. Harold Taylor, Vancouver
Public Health	Dr. G. F. Kincade, Vancouver

Special Committees

Arthritis and Rheumatism	Dr. F. W. Hurlburt, Vancouver
Cancer	Dr. Roger Wilson, Vancouver
Civil Defence	Dr. John Sturdy, Vancouver
Hospital Service	Dr. J. C. Moscovich, Vancouver
Industrial Medicine	Dr. J. S. Daly, Trail
Maternal Welfare	Dr. A. M. Agnew, Vancouver
Membership	Dr. E. C. McCoy, Vancouver
Pharmacy	Dr. D. M. Whitelaw, Vancouver
Public Relations	Dr. G. C. Johnston, Vancouver

OPERATION SECRET

That is what the B.C. Division has determined to eliminate. In accordance with the general principles behind the renaissance of this association, your executive are determined to do everything possible to keep all members fully informed on all developments. Following this policy, please note the article by Dr. Peter Lehmann, chairman of the committee of Economics, giving some of their problems and ideas.

Your new Board of Directors are utilizing the Public Relations committee and this Bulletin to keep you informed. It is felt by all of them that our Division must be the active voice to express the ideas of a closely knit profession. By all means let us have our differences of opinion and discussions amongst ourselves, but let us resolve

them into what is the best for the profession as a whole, and then all get behind it. For the Association to have any power, we must have the greatest possible membership, for when dealing with the government or any other body, the first question asked is—How many doctors do you represent? Let's all get behind this revived B.C. Medical, for the old cliché is still true—"We must all hang together or we will certainly hang separately." This support means not only joining and paying annual dues, which should be a routine, but also being as active as one can in the affairs of the group. If you have any ideas, make them known. Bring them up at your local medical association, write letters to me c/o The Bulletin, or direct to the Board of Directors.

One of the dangers at the present time of course, is this problem of Health Insurance. Not that we are necessarily going to fight Health Insurance of itself, but unless we are very active in trying to guide the politicians, some form of State Medicine will be foisted upon us, which will be disastrous for the health of the nation, unpleasant for ourselves, but glorious for an army of petty bureaucrats, at a cost which could be an economic catastrophy. We must have someone in Ottawa with his ear to government opinion and able to give advice in the proper development of this service. This, in a way, is one phase of Public Relations.

At the last meeting of the Public Relations committee, we were discussing the patients we have seen who have been complaining about some doctor or other. We realize that many of these complaints are fictitious or due to misunderstanding, but we do want to stress to all of you, that one of the most important parts of Public Relations, is that between you and your patients. The goodwill, or otherwise, which you generate helps greatly in the public viewpoint of the doctor.

Recently, I had occasion to ask the library of the Vancouver Medical Association for help in looking up a particular point in which I was interested. I phoned them one day and the following day had quite a collection of articles in the most recent journals. This was certainly most helpful. When returning these books I had a chat with the librarian, and found that as well as a very good selection of books, they subscribe to about 75 journals, and through inter-library loans have almost unlimited books available. These services are intended not only for those who wish to write papers, but if you have some case and are interested in the latest treatments or special aids in diagnosis, you can utilize your membership in the V.M.A., and have these articles put at your disposal. I was informed that quite a few Vancouver doctors make use of this, and that there are several associate members throughout the province to whom requested books or journals are being sent most all the time.

We are hoping to set up a short section in this Bulletin about post-graduate courses. Unfortunately most of the information for the spring will not be available until after this issue goes to press. It is hoped however, to maintain a list, and if you would write to me c/o The Bulletin, requesting any information you want on courses, we will try to give you whatever is available.

—F. L. SKINNER.

THE ECONOMIC COMMITTEE of CANADIAN MEDICAL DIVISION—B.C. DIVISION

DR. P. O. LEHMANN, Chairman

Since your Association took over economic problems most of the decisions made and actions taken were, if somewhat overdue, at least popular. Most of the doctors favored the changes and they were essentially non-contentious issues.

This year your economics committee has before it problems of perhaps less immediate demand but never-the-less of equal import. They have however much less popular demand and they are for the most part contentious in nature.

We are endeavouring to establish the value of a doctor and what remuneration a salaried physician should receive. A subcommittee with Dr. Ross Davidson as chairman, has been set up to gather information, assess data and determine many of the hidden components to the problem. It will undoubtedly be necessary to obtain actuarial assistance and a professional auditor's guidance. The study will be an extension of, and projection of, the White Report, delivered to the Council of the College in 1950.

The S.A.M.S. problems are still actively before us. A decision must be determined as to policy with regard to medical services provided in hospital. It has been suggested that full fees be paid for home and office visits and to prorate the hospital work in an attempt to make the essential home and office care attractive. The problem of an equitable policy with regard to teaching hospital staffs and many current problems have kept Dr. Ross Robertson's committee very active to date.

The subcommittee to study prepaid medical care plans with Dr. Gordon Francis as chairman, is this year having to deal with the consequences of extra billing, and the rather critical financial problems of some of the approved plans other than M.S.A. There are difficulties which have become manifest consequent to the reduction of allowable discount from 25% to 10%, and the separate increases in tariff, all occurring over the past two years.

It is planned that a revision of the 1952 schedule of fees will be published in April, 1953, and that a policy will be established for a comprehensive preamble to the schedule.

This year the C.M.A. has established an economics committee and your chairman has already attended one meeting of the committee, which has served to orient the various divisions of C.M.A. as to the national and regional problems of medical economics. Of local interest in particular, the question of treatment of R.C.M.P. personnel (formerly B.C. Police force) was aired. From the experience of other divisions this problem can be expected to solve itself within a few years, as the R.C.M.P. personnel can be expected to move extensively and freely from centre to centre. The police officers will no longer be permanent residents in any community and the local physician will probably welcome the responsibility of the D.V.A. for their medical service to R.C.M.P.

Another local problem aired at the meeting was the Sick Mariner's Fund. From the discussion it was manifestly evident that none of the coastal division was satisfied and the C.M.A. will be taking action to clarify the problems.

This year an attempt will be made to re-define the position of the profession with regard to a national health scheme. This, of course, is a momentous matter, and has many facets of a contentious nature.

The new members of the economics committe of the B.C. Division are Dr. Arthur McConkey and Dr. Gordon Francis of Vancouver, Dr. John McMurchie of Nelson, who, together with Dr. H. F. P. Grafton of Kamloops, Dr. J. Tysoe of Victoria, and Drs. Ross Robertson, Ross Davidson, Russel Palmer of Vancouver, serve with Drs. John Ganshorn, Frank Turnbull and Walter Brewster, as ex-officio members of the committee, making a group of twelve.

● ● ● ●

REMEMBER — "THE DOCTORS VIEWPOINT"

Your Radio Programme, every Tuesday at 10:15 p.m.—Station CJOR, 600 on your Dial. Please ask your patients to listen, and do so yourself.

PUBLIC HEALTH AND MENTAL HEALTH NEWS

G. F. AMYOT, M.D., D.P.H.,
Deputy Minister of Health, Province of British Columbia

A. M. GEE, M.D.,
Director, Mental Health Services, Province of British Columbia

B.C.G. VACCINATION

Considerable interest has been shown by the medical profession in the use of B.C.G. in private practice. This vaccination is very similar to that used for smallpox and for that reason should not present any difficulty so that its use as an office procedure is to be recommended.

Supplies of vaccine may be obtained by the medical profession free of charge through any Unit of the Division of Tuberculosis Control or any Health Unit.

The following outlines the policy and procedure of the Division of Tuberculosis Control in the use of this method of vaccination.

PURPOSE—to provide protection against tuberculosis through vaccination of those not already infected.

PROVINCIAL POLICY—to vaccinate all contacts of known cases of tuberculosis who are negative tuberculin reactors. This includes particularly those in contact in the home, all hospital personnel, and others exposed to tuberculosis in their occupation.

VACCINATION PROCEDURE—

1. *Age groups*—when vaccination is indicated all ages may be done safely. Infants even in the first two weeks of life are frequently vaccinated.

2. *Pre-vaccinal Tuberculin Testing*—
   a. All children ten years old and under—patch test. If negative give 1 mgm. O.T. If still negative, B.C.G. vaccination to be given within one month.
   b. Older children and adults—1/10 mgm. O.T. If negative give 1 mgm. O.T. If still negative, B.C.G. vaccination to be given within one month.

3. *Method of Administration*
   An area of skin about three inches square is cleansed with either alcohol or soap and water and allowed to dry.
   The contents of one or two capillary tubes (60 mgm. per c.c. strength) of vaccine are placed on the area, *making sure to get all the organisms off the wall of the capillary tube and suspension.*
   The vaccine is spread over a square inch (with the sterile needle provided) and three scratches 2 cm. long and 1 cm. apart are made through it. The scratches should cause a slight flow of serum and very little blood.
   Note: Children under six months should have two scratches instead of three scratches.
   A sterile gauze pad soaked in half the contents of a vial of oral vaccine (5 mgm. per c.c. strength) is placed over the scratches and held in place with one or two strips of adhesive. The pad is left without washing for 24 hours.
   The sites commonly used are deltoid region, thigh, lower back. Any site may be used but one which is not rubbed by clothing and not too conspicuous is preferred, as the scratches may be visible for six months and at one stage may be itchy.

4. *Post-vaccinal Tuberculin Testing*
   Eight weeks after vaccination, repeat the above tuberculin tests and if negative, re-vaccinate. If still negative after an additional eight weeks, further attempts

will probably be unsuccessful but vaccination may be tried again at two year intervals.

5. *Follow-up*
Successful B.C.G. vaccinations to be tuberculin tested at two year intervals. Vaccinated persons are to be X-rayed at the same intervals as other similar contacts.

COMPLICATIONS OF VACCINATION

1. *Local*—the scratches become raised and reddened in two to four weeks. At that time they may be itchy and in some cases, pin head sized papules containing white fluid occur along the scratch line. The scratches persist two to five months and then usually disappear but may leave a faint scar for a year or more.
2. *Regional Lymph Glands*—The regional lymph glands are always involved and in rare instances are palpable. In one case in approximately 4,000, there may be a cold abscess of a regional lymph gland with suppuration. The duration of this complication is shortened by aspiration. One aspiration at the time of softening of a gland is usually sufficient.
3. *Constitutional*—There are no general or constitutional signs or symptoms caused by B.C.G. vaccination. Therefore if unusual signs and symptoms appear at this time, a thorough search should be made for another cause.

STAFF NOTES

The following physicians are taking further training in preventive medicine during the academic year 1952-53 at the University of Toronto:

Dr. A. F. Balkany, Peace River Health Unit.
Dr. H. T. Lowe, formerly Cariboo Health Unit.
Dr. E. W. Wylde, Simon Fraser Health Unit.
Dr. J. C. White, Metropolitan Health Committee of Greater Vancouver.

Also taking postgraduate training in preventive medicine are Dr. D. A. Clarke, South Okanagan Health Unit, who is attending Harvard University, and Dr. E. C. Mahaffy, Victoria Esquimalt Board of Health, who is attending the University of California at Berkeley.

Dr. Donald Black, formerly in private practice in Kelowna, has joined the staff of the provincial Health Branch as Director of the South Okanagan Health Unit.

Dr. Paul F. Dubois has been appointed Director of the Peace River Health Unit. Dr. Dubois graduated from the University of Amsterdam in 1947, and for the past year has been interning at the Royal Alexandra Hospital, Edmonton.

● ● ● ●

ASSISTANT WANTED

Assistant required in an established practice in West Vancouver. For further details please contact the Publisher, Bulletin of the Vancouver Medical Association, 675 Davie Street, MArine 7729.

DR. J. C. FARISH

Obiit November 28, 1952
act. 87

There is an old adage which says "It is better to live well than to live long." Better still is when a man does both: and this is particularly true of our old friend Dr. J. C. Farish, who lived an exceptionally good, as well as an exceptionally long, life. Few men have led such full and active lives.

Dr. Farish came by his doctoring honestly: it was in his family—and two sons are carrying on the tradition. He was born in Liverpool, N.S., where his father practised medicine, and graduated from King's College and the University of Pennsylvania. He did postgraduate work abroad, was a ship's doctor during the Klondike gold rush, and practised in New Westminster as a general practitioner: coming to Vancouver in 1901. He soon became one of the leading eye, ear, nose and throat specialists in that city, and carried on this specialty till 1941. Since then he had more or less retired—but was active in many other fields.

He was a man of hobbies, and of the outdoors. Skating, riding, tennis were his favourite sports, and woodworking gave him an indoor hobby in which he excelled.

He was very popular with his confrères, who trusted him and consulted him freely. His personality was genial and generous, and he had many friends. He was one of the old school, whose numbers are so fast diminishing.

To his wife and family, we extend our most sincere condolences.

News and Notes

Dr. F. W. Arber is now practising in Kemano.

Dr. A. M. Barrera, formerly with the W.C.B. at Vancouver, is now practising at Kaslo.

Dr. G. B. B. Buffman is now taking postgraduate work in Philadelphia.

Dr. A. A. Gordas is now practising in Vancouver after a long illness.

Dr. Elliott Harrison has opened new offices in the Broadway Medical district.

Dr. E. A. Johnson has a new office in the Point Grey District of Vancouver.

Dr. E. G. Lapp is now practising in Campbell River.

Dr. D. M. Miller is now associated with *Dr. H. A. Mooney* of Courtenay.

Dr. E. G. Word is now doing postgraduate work in obstetrics at the Vancouver General Hospital, after 3 years in Saskatchewan.

Dr. E. A. Boyd is now associated with *Dr. Grant Gould* in Vancouver.

Dr. L. E. Wood is leaving Vancouver to go to London on a Research Fellowship to get research training in otolaryngology. On his return he will be connected with the Department of Physiology at the University of British Columbia. He will later resume practice and hopes to start some research work at U.B.C.

Dr. S. P. Findlay, formerly of Fraser Lake, is now at Aldergrove.

Dr. Gilbert Gordon has moved from Chilliwack to Vancouver.

Dr. C. P. Holmes, formerly of Vernon, is now associated with *Dr. Wilson* at Kelowna.

HAROLD EDWARD RIDEWOOD—1878-1952

On November 8th, 1952, the death occurred in London, England, of Dr. H. E. Ridewood of Victoria, B.C. Through him, medical practice in Victoria has been interwoven with that of the great London Hospital for a period of forty years.

This was a time of magnificent and spectacular development in all fields of medical practice and Dr. Ridewood was singularly well equipped, through natural ability and aptitude, and an excellent and prolonged training, to integrate most successfully all the new advances in his daily work and to make them a vital force in Canada—the land of his adoption. At the same time an elder brother established an international reputation in research in tropical diseases. Dr. Ridewood brought to the practice of medicine a rare intellect and exceptional training so that when these were combined with his high sense of professional responsibility and idealism, he fulfilled the high tribute paid to the medical profession by Robert Louis Stevenson in his dedication to "Underwoods"—

> "There are men and classes of men that stand above the common herd; the soldier, the sailor and the shepherd not infrequently; the artist rarely; rarelier still the clergyman; the physician almost as a rule. He is the flower (such as it is) of our civilization. Generosity he has such as is possible to those who practice an art—never to those who drive a trade; discretion tested by a hundred secrets; tact, tried in a thousand embarrassments; and what are more important, Herculean cheerfulness and courage."

Dr. Ridewood's father was a brilliant Headmaster in Enfield Grammar School in Middlesex, so as a child he grew up in an atmosphere of books and culture. It is of passing interest that he was born in a part of that old institution known as "Queen Elizabeth's Stables" which was not a misnomer in any sense of the word. That was the purpose for which it was originally built.

Commencing his medical studies in the London Hospital Medical College and at the University of London on a scholarship, he qualified for the degrees of M.D. and B.S. in 1903. His brilliance was further emphasized by the winning at this time of the gold medal. A second gold medal was won when he took his M.S. in 1909. During this period, he filled several important hospital posts in various London hospitals including that of Surgical Registrar at the London Hospital from 1908 to 1910.

Dr. Ridewood came to Canada in 1911 and became associated with Dr. O. M. Jones, then senior surgeon in Victoria. During the first Great War he served with the Canadian Forces from 1915 until 1918 when on the death of Dr. Jones he was recalled to Canada as Consulting Surgeon to Military District No. 11. He was retired with the rank of Lt.-Colonel and began independent private practice in Victoria in 1920.

Since that time he has been actively identified with hospital development both in the Royal Jubilee Hospital and St. Joseph's Hospital. He has been Chief of Staff in both institutions as well as President of the Victoria Medical Society and the British Columbia Medical Association. He was, in 1913, one of the Founders of the American College of Surgeons. He was married in 1919 and his wife and four children survive.

It is easy to summarize in this fashion the milestones in a busy life. It is a much more difficult task to attempt to appraise the various factors, beyond a restless and versatile intellect combined with exceptional training, which made Dr. Didewood the Dean of Medicine in Victoria—beloved by the profession which appreciated his skill and his outstanding surgical work; and respected and beloved by his patients whose devotion and confidence in him were well attested by the large congregation which filled St. John's Church when Canon Biddle conducted a moving memorial service on November 19th. His ashes will be placed in the new Columbarium in Christ Church Cathedral.

To those who watched his practice over the years, the outstanding explanation of his tremendous power of accomplishment was that he knew the value of time, and while never hurried in his actual work, there was no waste of precious minutes in futile conversation and banter which often fills so large a part of the daily routine. To some this may have made him seem abrupt, but he had unlimited time—almost leisure—for the sick patient who consulted him and the young physician who sought his aid. While his health was impaired for some years before his death, he took up the organization of a Cancer Clinic in Victoria with enthusiasm and his very valuable work in this field will not be forgotten.

Another possible explanation of his success may lie in the fact that he was always a student and as a close friend of his said: "He read always for knowledge and not for temporary entertainment." His hobbies were such as you might expect—photography and physics. In the latter field he found endless satisfaction and it is quite clear that had he taken training in this subject, he would have been one of the outstanding physicists of his time. This is obvious when we learn that as far back as 1919, he was thinking of and studying the possibilities of nuclear fission. For many years he corresponded with well known physicists on this and other subjects. Furthermore, he had that greatest of assets—an orderly mind and an excellent and well stocked memory where difficult surgical problems of the past were stored away as in a filing cabinet—always ready for reference and application.

He found his greatest relaxation in his home and with his family, and the friends who knew him in that setting, realized the warmth of his affection and the fullness of his life, and his singularly fine basic character. The old Roman poet who wrote "Non omnis moriar" gave the Canadian poet, Bliss Carman, the title for one of his imperishable poems beginning:

"And here in the November dusk
There comes an echo faint and far
Of that gay, gallant careless voice
That cried—'Non omnis moriar'."
"Behind the mask of lore and creed
There dwells an instinct strong and blind
Refuting sorrow, bidding grief
Be something better than resigned."

Yes—something of Dr. Ridewood lives in each one of us who knew and loved him. His work goes on in the medical practice of every physician and surgeon who has followed his work and known his devotion to medical science. It is our responsibility to so live and practice our profession that this heritage may not perish.

—H. H. MURPHY.

Dr. C. H. Moore has left Victoria to take post graduate studies at the Mayo Clinic.

Dr. C. A. Cawker has opened a practise in urology at 2205 West Broadway in Vancouver.

Dr. Donald M. Black is Acting Director of the South Okanagan Health Unit for nine months.

Dr. G. E. Singer has moved from Queen Charlotte City to Vancouver where he is a Teaching Fellow at the University of British Columbia.

Dr. Ian S. Kenning has moved from Victoria to Essondale, with the Provincial Mental Health Services.

Dr. A. H. Moore has returned to Vancouver from England and has opened an office at 2695 West Broadway.

DOCTOR'S OFFICE

Available after Dec. 15th. Bright, modern Doctor's suite at 1538 Marine Drive, the most central section of West Vancouver. Heated. Reasonable rent. For complete information apply J. P. MacDonald, phone West 1655 or KErr. 3985M.

A FREE 1953 Copy is Yours

of the most widely read authority on Canadian Gas and Oil.

"Oil in Canada"

This is a service to those who are interested in having the latest news on Western Canada fields.

WESTERN PETROCHEMICAL CORPORATION LIMITED

William Buckner, Pres. and Gen. Mgr., with Head Offices in Edmonton. Holdings in eight areas in Alberta and British Columbia.

Vancouver Offices:

126-129 Merchants' Exchange Bldg., 815 West Hastings St. TAtlow 8431

Write, wire or mail this coupon for Free Copy

Western Petrochemical Corporation Ltd.,
126-129 Merchants' Exchange Bldg.,
815 West Hastings St., Vancouver 1. B.C.

Without any obligation on my part, please send me a copy of "Oil in Canada."

Name.....

Address.....

.....

Mount Pleasant Undertaking Co. Ltd.

KINGSWAY at 11th AVE. Telephone: EMerald 2161 VANCOUVER, B.C.

KINDLINESS :: UNDERSTANDING :: DEPENDABILITY