THE

BULLETIN

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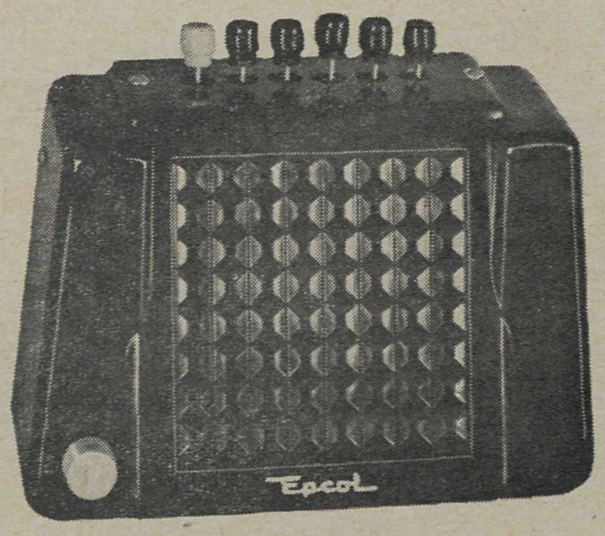

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Sanborn Company

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VANCOUVER HEALTH DEPARTMENT

CASES OF COMMUNICABLE DISEASE REPORTED IN THI CITY

STATISTICS — APRIL, 1949

Total Population—Estimated	376,00
Chinese Population—Estimated	7,45
Hindu Population—Estimated	27

	February		March	
	Number	Rate Per 1000 Pop.	Number	Rate Per 1000 Po
Total Deaths	396	12.7	399	12.7
Chinese Deaths	12	19.3	14	22.5
Deaths, Residents Only	359	12.4	356	11.4

BIRTH REGISTRATIONS—Rehidents and Non-Residents.

	February		March	
Male	350		441	
Female	373		433	
	723	23.1	874	27.9

INFANT MORTALITY—Residents Only.

	February, 1949	March, 1949	March, 1948
Deaths Under 1 Year of Age	4	22	13
Death Rate Per 1000 Live Births	7.6	36.0	21.3
Stillbirths (Not Included in Above Items)	10	9	5

CASES OFCOMMUNICABLE DISEASES REPORTED IN THE CITY

	Number		Rate Per 1,000 Population			
	February, 1949		March, 1949		March, 1948	
	Cases	Deaths	Cases	Deaths	Cases	Death
Scarlet Fever	44	0	9	0	12	0
Diphtheria	0	0	0	0	0	0
Diphtheria Carriers	0	0	0	0	3	0
Chicken Pox	360	0	530	0	68	0
Measles	228	0	243	0	255	0
Rubella	13	0	9	0	8	0
Mumps	17	0	57	0	7	0
Whooping Cough	1	0	0	0	1	0
Typhoid Fever (Carriers)	1	0	0	0	1	0
Undulant Fever	0	0	0	0	0	0
Poliomyelitis	0	0	0	0	2	0
Tuberculosis	44	13	72	15	46	11
Erysipelas	2	0	0	0	2	0
Meningitis	2	1	0	0	0	0
Infectious Jaundice	0	0	0	0	0	0
Salmonellosis	0	0	1	0	0	0
Salmonellosis Carriers	0	0	0	0	0	0
Dysentery	1	0	0	0	0	0
Dysentery (Carriers)	0	0	0	0	0	0
Tetanus	0	0	0	0	0	0
Syphilis	31	4	48	4	75	7
Gonorrhœa	244	0	189	0	206	0
Cancer (Reportable:						
Resident	62	51	74	61	104	78
Non-Resident	21	10	14	11	23	15

The Editor's Page

One sees constantly in the press, and more particularly of late, appeals for bedside nurses, for nurses to staff the wards of the hospitals—in short, for nurses who will nurse. Private duty nurses are becoming harder and harder to secure, and there is no doupbt that there is an acute shortage of a commodity which is one of the most vital necessities to our economy. Yet the list of hospital graduates is continually increasing, and more and more girls graduate every year. It does not seem to be entirely a matter of money, since the fees paid now are, though not perhaps in true consonance with the value of the work done, yet higher than they have ever been—and there need be no lack of continuity in work, where nurses are so badly needed

There is, of course, the undoubted tendency of women who take nursing training, to go into public health, social service, and other ancillary departments of the nursing profession. Presumably, the hours are easier, the work easier and more regular, and there are many advantages, such as opportunities for promotion, and these graduates do splendid work and work that is essential. But it is not nursing, in the fullest sense of the work.

It is true, too, that with the growth and expansion of hospitals, more and more nurses will be needed, and the sources of supply are limited. Also, being a completely feminine occupation, it is subject to big inroads by reason of marriage, and so a change of orientation in the way of life.

Oddly enough, we have no remedy to suggest. It is a huge problem, and many minds are at work endeavouring to solve it, and we hope it will be solved—for, to our mind, there is nothing in this world quite as good as a good nurse. One thinks with undying gratitude of the cases one has had, hopeless and helpless, but for the work of the nurse. If we are honest, we must gladly admit that nursing, not medical care, is in most of our very serious cases the deciding factor between success and failure, between life and death. We see our patient once or twice a day, we issue orders, we consult the better brains and the abler consultant—and the nurse does the work. She gives a devotion and care which, added to the skill acquired by training, eventually pull the patient through, if it is humanly possible, or at least afford us complete assurance that everything possible has been done—and with the good nurse (and we think they are all good) the skill is the least important element of the contribution she makes. We cannot do without the trained nurse, the bedside, clinical nurse—and we must have her, and more and more of her. Sincerely we trust that this vital problem will be solved, and that some way will be found of keeping the ranks full.

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We have been fortunate in obtaining a brief report from Dr. A. W. Bagnall, who has recently been attending a conference in the United States where the subject of Cortisone, referred to so freely of late in the public press as Compound "E", has been under discussion. Dr. Bagnall's note appears in this issue, and will, we are sure, be of great interest to all medical men. The public is very keenly interested also, and we feel that this authoritative statement of Dr. Bagnall's will enable us to give our patients answers to their questions.

—Editor.

Vancouver Medical Association

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Saturday	9:00 a.m. - 1:00 p.m.

ON THE IMPORTANCE OF THE LIBRARY

Two of the important factors that distinguish man from less elevated animals are the ability to reason and the capacity for speech. A refinement that has enabled each generation of mankind to profit most fully from the experience of its predecessors has been the recording of ideas and the preservation of these records, whether carved in stone or scratched in tablets of clay; impressed upon the printed page.

Only by consulting its records has man been able to build adequately upon the experiences of the past; of greater importance to his progress than the invention of the wheel has been his establishment of the library, which contributes to his cultural and spiritual as well as to his technical advance.

Some of the brightest chapters in the story of mankind have been illuminated by the development and preservation of literature. Some of the darkest pages in history have been punctuated by the destruction of libraries and the burning of books, from the conflagration at ancient Alexandria to more recent attempts at the destruction of modern culture.

The library as an institution has been one of man's sacred heritages. This applied to the first Sumerian tablet that was laid aside to tell its story four thousand years later; it applies to the most recent text that has been placed on file. This institution is an urgent public charge, from the Library of Congress, supported by an assessment on the tax payers, to the smallest village library in existence. . . .

From an editorial in the New England "Journal of Medicine", February 17, 1949.

RECENT ACCESSIONS

Bacterial and Mycotic Infections of Man, edited by R. J. Dubos, 1949.

Diseases of Infancy and Childhood, by W. P. H. Sheldon, 5th edition, 1946.

Hematology, George R. Minot Anniversary volume, edited by Dameshek, W. and Taylor, F. H. L., 1949.

Index to Literature of Experimental Cancer Research, 1900-1935, prepared by the Donner Foundation, Inc., 1948 (Gift).

Mechanism and Graphic Registration of the Heart Beat by Sir Thomas Lewis, 3rd edition, 1925.

Medical Clinics of North America, Symposium on Cardiovascular disease: Hypertension—New York Number, May, 1949.

Medical Research Council Memorandum No. 20. The Physique of Young Adult Males by W. J. Martin, H.M.S.O., 1949.

Progress in Clinical Medicine, edited by Daley, R. and Miller, H. G., 1949.

Progress in Gynaecology by Meigs, J. V. and Sturgis, S. H., 1946.

Recent Advances in Obstetrics and Gynaecology, edited by Bourne, A. W. and Williams, H. W., 7th edition, 1948.

Royal College of Physicians, London—List of the Fellows and Members, 1948.

Textbook of Endocrinology by Hans Selye, 1947.

The Medical Register, parts 1 and 2, 1949.

The Salicylates by Gross, M. and Greenberg, L. A., 1948 (Gift).

The Vitamins in Medicine by Bicknell, F. and Prescott, F., 1948.

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RECENT ADVANCES IN THE RHEUMATIC DISEASES

Report from B. C. Medical Committee on Arthritis and Rheumatism.

This summary is presented with the hope that the physician in B. C. will temporarily at least, have the chance to be in command of more knowledge about compound E than his patients, who have been deluged with articles in the popular press on this subject.

Compound E has now been renamed Cortisone, probably to differentiate it more clearly from Vitamin E, about whose therapeutic value in human disease states there remain grave doubts.

The material presented by outstanding medical scientists at the recent International Congress, on the Rheumatic Diseases in New York, leaves no doubt that the daily intramuscular administration of cortisone produces well-nigh incredible reversal, even of advanced cases of rheumatoid arthritis, to normal within one to two weeks. When the injections are stopped, the previous picture of seriously active disease is usually resumed. This means that cortisone neutralizes the pathological process that produces the clinical features of rheumatoid arthritis but has no effect on the ultimate cause of the disordre.

In their original paper, "Hench, Kendall et al" mentioned that A.C.T.H. (Adrenocorticotrophic hormone extracted from the pituitary gland of animals by the Armour Co.) reproduced the beneficial results of cortisone in rheumatoid arthritis, presumably by stimulating the apparently intact adrenal cortex of the rheumatoid arthritic to produce sufficient of its own cortisone to reverse the musculo-skeletal changes.

Up to this time, A.C.T.H. had been used, in the few research centres where it was available,, principally as a test stimulator of the adrenal cortex in diseases such as Addison's or Simmond's disease to see how much functional cortical tissue remained. If the adrenal cortex is intact, an injection of A.C.T.H. stimulates it to a few days' activity following which there is an overswing into lethargy. There are several methods of following this variation in activity of the adrenal cortex, such as counting the total number of circulating eosinophiles, estimating the excretion of breakdown products of adrenal cortical hormones in the urine (17-keto and/or 11 oxysteroids) etc. Fortunately, these methods had become fairly well standardized during the two years prior to the announcement from the Mayo Clinic, and the appropriate research centres were able to train most of their big hormonal guns forthwith on the rheumatic diseases, so that much preliminary information has become available in the short space of a few weeks.

Cortisone and A.C.T.H. have been used with rather dramatic effect on a few cases of acute rheumatic fever. In this instance, however, once the activity has subsided, cessation of hormone therapy does not seem to be followed by exacerbation of the rheumatic fever. This may well be because rheumatic fever is an abnormal connective tissue response to a single, preceding, non-persistent insult to the body, i.e. a haemolytic, strepto-

coccal infection. In contrast, the insult producing rheumatoid arthritis is apparently a persistent one and may, in some instances at least, be psychosomatic via the hypothal amus, pituitary and adrenal cortex.

That the pituitary-adrenal mechanism is not causally specific is further accentuated by the therapeutic activity of A.C.T.H. in other collagen tissue disorders. There are good indications that it arrests the actviity of diffuse lupus erythematosus, up till now considered an almost uniformly fatal, if rare, disease. Another rare and often fatal disease, dissemminated, or generalized, scheroderma also appears to respond favourably. So far, there are reports dealing with periarteritis (or polyarteritis) nodosa, but this disease is a close analogue to the above and frequently accompanid by eosinophilia. Since the injection of A.C.T.H. reduces the number of circulating eosinophiles, further investigations in this field may result in extension of the A.C.T.H.-cortisone therapeutic mechanism to other diseases in which the pathogenesis remains a mystery and eosinophilia is common.

The lack of etiological specificity of the new pituitary-cortisone system is even further stressed by the study of its effect on gout, considered to be a familiar disease of inborn error of metabolism of uric acid or its precursors. One injection of A.C.T.H. will usually clear an acute attack of gout in a few hours—it also induces a doubling in the excretion of uric acid in the urine and a profound fall of the serum uric acid. If colchicine is not continuously administered during the week following the injection, however, an acute attack of gout will recur five days later, apparently produced by the overswing from overactivity of the adrenal cortex to lethargic underfunction. Similarly, an injection of A.C.T.H. to a gouty person who at the time is suffering from no joint symptoms may produce an acute attack of gout five days later when the stimulated adrenal cortex overswings into lethargic underfunction. There appears to a further connection between gout and the adrenal cortex. Wolfson and others at the Michael Reese hospital in Chicago have produced some evidence that in the gouty patient, the male sex hormone manufactured in the adrenal cortex is an *abnormal* one which, while maintaining reasonable sexual control, upsets the metabolism of uric acid. The dearth of clinical gout in the female may be explained by their conclusion that this abnormal androgen is opposed by oestrogen. This may point also to a new avenue of gout therapy, although it would appear to be more and more risky to tamper with the body's hormones without certain knowledge of the effects to be expected.

The pituitary-adrenocortical mechanism therefore seems to be concerned with the production of part or all of the clinical pictures associated with those diseases we know as rheumatoid arthritis, rheumatic fever, diffuse lupus erythematosus and generalized scleroderma. In the rheumatoid group, Marie-Struempell spondylitis responds as favourably as rheumatoid arthritis of peripheral joints but this again does not necessarily infer that there is a common cause.

There are good indications that there would appear to be a complete conflict between the Addisonian and the "collagenoses" hormones of the adrenal cortex. Selye, perhaps, was first to correlate the rheumatic disases with the adrenal cortex by producing, in animals, lesions resembling rheumatic fever in man, by injection of relatively large quantities of desoxycorticosterone (the synthetic electrolyte-controlling cortical hormone). Thorne, working with adrenal-insufficiency of the Addisonian type, showed that Compound "F" had a marked blocking effect on desoxycorticosterone. Compound "F" is closely related physiologically to Compound "E", so that a similar antagonism with cortisone may soon be proved. Furthermore, if the clinical manifestations of rheumatoid arthritis, rheumatic fever, etc. were due to a simple deficiency of cortisone, these manifestations should be a constant concomitant of adrenal insufficiency of the Addisonian type, in which the adrenal cortex is partially or completely destroyed—which they are not. The symptomatology of rheumatoid arthritis may therefore turn out to be due to a predominance of the Addisonian and a relative deficiency of the "collagenosis" factors of the adrenal cortex, possibly secondary to disturbance of pituitary function.

The discovery of dramatic new "cures" often lead the practitioner in desperation to the use of some available kindred compound, particularly when the new cure is expensive and unobtainable (which cortisone is at present). For the above partially hypothetical reasons, however, the present commercial extracts of the adrenal cortex (Percortin, Ciba, Adrenal Cortical extract, Upjohn and Connaught) and D.O.C.A. (Desoxycorticosterone acetate) are absolutely contra-indicated in rheumatoid arthritis.

Man must again marvel at Nature when one looks at the structural formulae of these kindred yet coniflicting substances—cortisone has precisely the same, very complex, basic structure as D.O.C.A., the only difference being the addition of an oxygen atom at the 11-position and an OH group at the 17-position. Not only does this small difference make a tremendous alteration in the biological activities of these two compounds, but it is in the addition of these two oxygen, and one hydrogen, atoms in their proper places that the tremendous difficulty of synthesis of cortisone arises. The present minute supply of cortisone (sufficient in the whole wide world to treat only about five patients at one time) comes from partial synthesis, starting with desoxycholic acid, one of the bile acids. With the present method, it is extremely doubtful if synthesis will become commercially practicable without huge manufacturing plants which would render the product almost as costly as at present, $100.00 per injection, i.e. per diem.). A very great deal of research continues however, and there is real ground for hope that cortisone may be available in 18-24 months for more universal use. At present, A.C.T.H. is in more plentiful supply than cortisone since it is extracted from the pituitaries of animals obtained in slaughter-houses. Because of certain dangers and of the limited supply, the use of A.C.T.H. is confined to special research centres. It is doubtful if it will ever reach the open market because the supply depends on the number of animals slaughtered and thus will hardly increase significantly in the future. Its structural formula is unknown and the possibility of commercial synthesis of A.C.T.H. is thus even more remote than is that of cortisone.

Certain more practical conclusions were reached at the International Congress. It was agreed, almost unanimously, that chrysotherapy was the most effective available agent for rheumatoid arthritis. Most authorities continue to use suspensions in oil of Myochrysine or Solganol B or aqueous solutions of gold sodium thiosulphate. Even with the aqueous solution, preference is felt for I.M. over I.V. injection to avoid sudden high nephrotoxic levels in the blood. Tendency in administration is to reach saturation levels by 10-15 weekly injections of smaller-than-previous dosage and to maintain this level by injections at intervals of 2-4 weeks for months or years. This method has been used by some physicians in B. C. satisfactorily for the past three years or more. Lauron does not appear to have gained much favour. Vaccines and high dosage vitamin D were not mentioned. Forestier advocated the use of organic salts of copper when gold was contraindicated or failed. He received little support from other investigators.

The importance of colchicine in the acute attack of gout was emphasized, particularly in its relation to warding off the delayed exacerbation following A.C.T.H. injection. Its mode of action is not yet elucidated. Intensive oestrogen therapy was advocated on somewhat slim grounds for the two male sex-linked arthritides, Marie Strumpell spondylitis and gout. Some animal experiments were reviewed which suggested that lessened thyrooid and sex hormone production contribute to the advance of degenerative changes of osteoarthritic type. This may be worth remembering when investigating early osteoarthritis in the middle-aged patient.

Rheumatic fever is now almost unanimously considered to be an abnormal pathological response to a haemolytic streptoccal infection in a susceptible individual. The continuous use of small doses of the sulphonamides daily (e.g. ½ gram b.i.d.) is relatively safe and may prevent 5 out of 6 recurrences of rheumatic fever. This is particularly appropriate in the case of children with rheumatic heart disase, the propylactic dose being continued till puberty, when, for some reason, the liability to recurrences lessens very considerably. Penicillin orally, 100,000 units, three-quarters hour before breakfast,

increasing to b.i.d if the hazard of haemolytic streptococcal infection increases, is a more expensive but less risky method of doing the same prevention.

In a person known to be subject to rheumatic fever, adequate parenteral penicillin given from the start of a streptococcal infection is now said to be moderately effective in preventing subsequent exacerbation of acute rheumatic fever, although sulphonamides are powerless to do so. To do this, the penicillin must be started within the first day, if possible. The plan suggested is that the rheumatic patient should notify his physician of a sore throat, without delay—a culture for haemolytic streptococcus is made and I.M. penicillin started at once. If the culture is positive the next day, penicillin is continued for 10 days: if not, further penicillin need not be given.

Parenteral salicylates have no place in rheumatic fever where oral dosage is possible. The proper dose of salicylates is that which just fails to give toxic effects, this quantity varying down from a total daily dosage of one grain for each pound of body weight. In acute cases, Professor Davidson of Edinburgh proposes 20 grains of sodium salicylate every two hours until toxic effects begin to appear, dropping then to a smaller maintenance dose which just avoids tinnitus and gastrointestinal disturbance. Salicylates should not be pressed past the point of toxicity since it is not widely accepted that there is any effect on the lethal aspects of rheumatic fever. From several widely-scattered countries, there are reports that the incidence of rheumatic fever is steadily diminishing.

—A. W. BAGNALL (Chairman).

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TREATMENT OF ARTHRITIS

A Statement re the care which can be provided through the British Columbia Division, Canadian Arthritis and Rheumatism Society.

British Columbia Division
Canadian Arthritis and Rheumatism Society
Vancouver, B. C.

997 West Broadway,
Vancouver, B. C.
8th June, 1949.

Dr. F. L. Whitehead,
925 West Georgia,
Vancouver, B. C.

Dear Doctor Whitehead:

So many requests from doctors have been received, for services we would like, but are not yet equipped to render, that the enclosed statement may clarify our present programme.

Yours sincerely,

MARY PACK,
Organizing Secretary.

"The Canadian Arthritis and Rheumatism Society, B. C. Division, is anxious to co-operate with the doctors in bringing relief to their patients suffering with rheumatic disease.

The Headquarters of the British Columbia Division of the Canadian Arthritis and Rheumatism Society at 997 West Broadway is not a treatment centre but an office from which educational material is circulated.

So far, the patients who can be helped under the Canadian Arthritis and Rheumatism Society programme are:

1. Those who are eligible for Outpatient care—i.e. those of very low income, or pension.

These should be referred in the regular way to the Outpatient Department of the Vancouver General Hospital. From the routine physical examination, they may be referred to the Arthritis Clinic. Here, as prescribed by the doctors in charge, treatment is given gratis by the physical therapists employed by the C. A. & R. S.

2. Those who are physically unable to leave their homes.

Treatment for these patients may be requested by the family doctor. Requisition forms may be obtained from the Divisional Headquarters by telephoning CE. 5114. Upon receipt of this requisition, the physical therapist will visit the patient, giving one or more treatments a week as indicated. Reports on the patients' progress will be made from time to time.

Limited by lack of personnel and facilities, these are the only two groups of patients to whom we can offer practical help at the present.

To all those whose arthritis is not yet crippling enough to keep them within the house, or to those whose income is greater than the maximum allowed for admittance to the O.P.D., the advice we give is as follows—"Ask your family doctor for an examination. If he prescribes physical therapy treatment, it will be possible to obtain treatment similar to that which is given at our centre, from a member of the Canadian Physiotherapy Association practising privately in your own locality." Names and addresses of C.P.A. members on request.

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Correspondence

May 13th, 1949.

Dr. J. H. MacDermot,
Editor,
Vancouver Medical Association Bulletin,
925 W. Georgia Street,
Vancouver, B. C.

Dear Dr. MacDermot:

For the past two years it has been our practice to bring a Visiting Chief of the Department of Medicine to this Hospital for one week in April and again in October. This practice has provided considerable benefit for the staff at the Hospital, both Resident and Attending, and for the local medical profession. The purpose of this letter is to acquaint you with the plans for the future in order that you may publish in the Bulletin the dates on which our future Visiting Chiefs will be at the Hospital. All the clinical sessions given at the Hospital are open to the profession and it may be that some members of the medical profession from outside Vancouver would be interested in taking advantage of these opportunities.

Sir John Parkinson of London, England, will be Visiting Chief of the Department of Medicine during the first week of October, 1949. These sessions will run from Monday, October 3rd, until Friday, October 7th. I am writing today to the Vancouver Medical Association to offer Parkinson's name as a speaker for their meeting on October 4th.

Dr. Cecil J. Watson, Professor of Medicine at the University of Minnesota, has accepted by invitation to be Visiting Chief of the Department of Medicine for the first week of April, 1950. These sessions will be held from Monday, April 3rd, to Friday, April 7th, and I will also offer his name to the Vancouver Medical Association as a speaker for their April meeting.

Yours truly,
G. F. STRONG,
Chief, Department of Medicine.

St. Paul's Hospital

OESOPHAGEAL ATRESIA AND TRACHEO-OESOPHAGEAL FISTULA

Abstract of An Address by HERBERT E. COE, M.D., F.A.C.S.
Lecturer on Surgical Paediatrics, University of Washington, Chief Surgeon Children's Orthopaedic Hospital, Seattle

Delivered at St. Paul's Hospital, February, 1949

This condition has been recognized for many years and until recent times has been universally considered fatal.

The first recorded case was reported nearly 280 years ago by Durston in 1670. Probably the condition was considered so hopeless that reports seemed superfluous, because in 1884, 214 years later, Sir Morrell Mackenzie was able to collect only 62 cases. Early in this century interest was evidently stimulated, and in 1932 A. H. Rosenthal and A. Himmelstein summarized 255 cases, an increase of 193 cases reported in 48 years.

Attempts at surgical treatment were reported by several men from 1913 for over 20 years without a single recovery until W. E. Ladd reported several survivals following the construction of an extra-thoracic œsophagus. This was followed soon by reports of successful intrathoracic anastomosis of the œsophagus with ligation of the accompanying tracheo-œsophageal fistula. The technique has been so improved that the percentage of recoveries is rapidly increasing.

Successful results are absolutely dependent upon early diagnosis and treatment with meticulous technique and highly skilled post-operative care.

Pathology:

The proximal segment of the œsophagus terminates at the level of the third or fourth thoracic vertebra. Five types have been noted:

1. Both the proximal and the distal segments end blindly.
2. There is a fistula between the proximal segment and the trachea.
3. There is a fistula from the distal segment to the trachea above the bifurcation.
4. There is a fistula from the distal segment to the trachea at the bifurcation.
5. There is a fistula from each of the segments.

The third type is the most common.

The fistula may enter a main bronchus instead of the trachea. The atresia varies in length from a mere spur between fistulæ to a complete absence of the distal segment.

Symptoms:

The symptoms are clear, distinctive, and easily recognized when the condition is kept in mind.

Brennemann's description is dramatic. "If attempts are made to encourage nursing or water is given the infant will swallow once or twice without difficulty. With every further attempt at swallowing the fluid will return in jets alongside of the nipple and through both nostrils. The child is now compelled to breathe with the mouth, nose and pharynx full of liquid. It suddenly stops nursing, its eyes become fixed, it rapidly turns blue, and then struggles to empty the lungs by coughing." This sequence is repeated as soon as fluid is taken again or when the œsophageal pouch fills with saliva or mucus. This picture has little variation and is not produced by any other condition. The diagnosis should not be missed.

The infant may drown at once. The fluid may be completely or partially drained through the fistula into the stomach, or aspirated into the bronchial tree. No food

enters the gastrointestinal tract and the weight soon begins to fall. Signs of dehydration appear,—depressed fontanelle, dry skin, loss of tissue turgor, scanty and concentrated urine. The stools show normal meconium at first, and then the mucoid starvation type. Gastric distention soon appears, except in the type without a fistula. Fever occurs early as a result of bronchitis, aspiration pneumonia, dehydration and inanition. Death occurs in from one to 14 days, the average being seven days.

Diagnosis:

The symptoms are so pathognomonic that confirmatory procedures are rarely necessary, and valuable time may be lost by resorting to them.

A catheter may be passed gently down the œsophagus and it will stop 10 or 12 cm from the lips.

Lipiodol,—*not* barium,—may be introduced through the catheter under fluoroscopic control using only enough to outline the bottom of the œsophageal pouch or demonstrate a fistula from it into the respiratory system, and then immediately aspirated.

It is best to use a syringe, leaving it attached to the catheter so that no time will be lost when the aspiration is indicated. The œsophageal pouch should be kept free of fluid constantly.

An X-ray may be taken to show the catheter tip at the bottom of the pouch, to determine pneumonia or atelectasis and to show air in the stomach and intestines.

Bronchoscopy is rarely advisable as it may cause unnecessary laryngeal irritation.

There may be associated anomalies of the heart, gastro-intestinal tract, or other systems, but these are of minor importance compared to the œsophageal atresia.

Treatment:

As soon as the diagnosis is suspected the pharynx and œsophagus should be kept clear of fluid and secretions by frequently repeated aspiration. This requires constant care. Postural drainage is often valuable but must be used with care if there is a fistula from the distal segment through which gastric secretions can enter the bronchial tree. Gastric regurgitation may occur even though the shoulders are elevated.

A high concentration of oxygen is valuable in reducing respiratory motions and in diminishing the intestinal distention.

Parenteral fluids may be given if necessary, but caution is essential to avoid overhydration with resultant pulmonary œdema and salt retention. It is to be remembered that the food and fluid requirements of the neo-natal period are low. If protein support is necessary, small amounts of blood are preferable to plasma as being less apt to cause pulmonary œdema, and saline should be used sparingly, if at all, because there is little or no loss of hydrochloric acid by vomiting.

The child should be transported to the operating centre as soon as possible, preferably within the first 12 hours, and should be under the constant care of a nurse or other person who is trained in the technique of frequent aspiration of the œsophageal pouch and the administration of prophylactic penicillin.

The most frequent cause of post-operative death is the pre-existing pneumonia. This pneumonia can be largely prevented by early diagnosis and expert pre-operative care, and an infant may be maintained in good condition for several days and transported long distances under these conditions.

The post-operative care is also of great importance. It includes high oxygen atmosphere, incubator, special nursing by nurses skilled in this technique, pædiatric supervision, chest and head slightly elevated, parenteral fluids, 1½ ounces per pound per day, sulfadiazine 0.5% solution, 3% to 5% dextrose in water (not saline) intravenously or subcutaneously as necessary for carbohydrate support, blood rather than plasma for protein support, avoidance of excess fluid, feeding by gastrostomy, after the second or third day using one-half to one ounce every two hours. Gradually begin mouth feedings after the eighth day. The gastrostomy should be done at the time of the operation if possible to prevent the regurgitation of gastric contents to the operative area. The

infant's condition may be such that it is inadvisable to prolong the operation and the gastrostomy is then deferred to the next day.

Operation:

Because of the age, size, and condition of the patient, and the delicacy and vital character of the tissues involved, the operation is difficult and the technique exacting. Expert anæsthesia with facilities for positive pressure is essential. Ether or cyclopropane with or without supplementary local anæsthesia may be used. The thorax is opened preferably through a right paravertebral incision, the site of the atresia exposed extra-pleurally, the tracheo-œsophageal fistula isolated and ligated, the distal œsophageal segment mobilized and the two segments united by either a single or a double layer anastomosis. Fifty thousand units of penicillin in 10 cc. of fluid is left in the retropleural space.

When the gap between the segments is long, it may be necessary to exteriorize the end of the proximal segment as the first stage in the construction of an extra-thoracic œsophagus, but by a thorough mobilization of the distal segment and also the stomach if necessary, an immediate anastomosis is possible in nearly all cases.

A high percentage of good results is possible by following this method of surgical treatment but success depends upon prevention of the early aspiration pneumonia, early operation, and skilled operative and post-operative technique.

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RHEUMATIC HEART DISEASE IN CHILDREN

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I. Introduction

In this short review of rheumatic heart disease we shall endeavour to re-confirm the older concepts that have stood the test of time so to speak, but more particularly we shall consider some of the newer trends of thought concerning the disease that already show some promise in their effect on one or another phase of the picture.

II. History

According to Wilson in the report of the Commonwealth fund, Balonius first used the term rheumatism but his first work was not published until 1642, after his death. Sydenham gave the first satisfactory clinical description of the differences between gout and rheumatic fever in 1676. Later, he described chorea, which disease bears his name. From then on various great names are attached to the story of rheumatic infection . . . names like Sir Thomas Watson, Poyntin, Payne, Barlow and others. In 1904 Aschoff described the peculiar bodies now known as Aschoff bodies. As John Parkinson in his Harvian Oration said: "In the 17th century rheumatism was disengaged from gout; in the 18th century it became engaged to the heart and, in the 19th century, wedded to it."

III. Etiology and Pathology

The present concept of the cause of rheumatic infection is that Alpha. Strep. Hæmolyticus, of which there are many sub-types, plays a definite role. The following is the probable chain of events:

> This streptococcus initiates an upper respiratory infection which lasts from one to three days, and then there is a latent period from one to three weeks during which time some phenomenon occurs: allergic or, to use a better term, hypergic or, possibly, immunological. There has been some interesting work done on this and much speculation has arisen. Swift and Cokum have done considerable work in this field

and they also feel that there is a specific alteration in the connective tissue resulting from exposure to bacterial antigens.

The third stage is the acute manifestation of rheumatic disease which may be of varying severity. Following a milk-borne epidemic of Strep. throat infections in Denmark in 1926 there was a great increase in the incidence of rheumatic infection. Likewise during World War II many observers noticed that rheumatic infection was preceded by an upper respiratory streptococcal infection—in some instances in as high as 90 per cent of the cases. It is felt, however, that conversely, although many people do have Strep. throat infections as shown by symptoms, throat culture and an increase in the anti-streptolysin titres, few actually develop rheumatic disease. Familial incidences suggest that it may be "the type of soil" and Wilson believes that this inherited factor is possibly present in 5 per cent of people and he believes this to be on a Mandelian recessive basis which predominates over other factors, such as climate, environment and geographical location.

Pathologically speaking, rheumatic infection and rheumatic heart disease fall into a group known as a diffuse collagen disease. This term represents a very new concept in pathology but it is the outgrowth of fifteen years' study. Klinge in 1933 was the first to re-orient our views concerning the basic pathology of rheumatic infection and, according to our present concept, it is linked in histopathology with disseminated lupus erythematosus, diffuse scleroderma, periarteritis nodosa and thromboangiitis obliterans. The most important histological change is a so-called fibrinoid degeneration of fibrous connective tissue. The collagen fibre becomes swollen and loosened and later granular and may break up into fragments. The architecture may be so loose that all that may remain is a swollen smudgy patch, as Lyman-Duff says. The reason it is called fibrinoid is because early in the process the combination of the increased ground substance and the degenerating collagen fibres resembles fibrin and takes on similar staining qualities. In rheumatic infection, around these areas of fibrinoid degeneration there is found a great proliferation of fibroblasts. These invade the areas of fibrinoid necrosis and replace them. Naturally, new collagen fibres are laid down through areas of fibroblastic proliferations. There is a third component, of course, and that is the inevitable inflammatory reaction from the initial insult to the connective tissue. This varies with the degree of injury and the location in the body. A granulomatous reaction that occurs in the various parts of the body such as the cardiac valves, myocardium, subcutaneous nodules and in other areas, notably in the connective tissue beneath the serosal surfaces of the joints and the subpericardial connective tissue, an inflammatory exudate, is a common accompanying feature. In the other diseases to which rheumatic infection is linked one or another component of this triad may dominate the picture and this counts in the recognition of the various diseases as distinct pathological and clinical entities.

Klinge, with the group, stresses the fact that fibrinoid degeneration is essentially an allergic reaction.

IV. The Acute Attack of Rheumatic Infection

Our attitude towards the treatment of acute rheumatic infection has altered relatively less than our attitude towards other phases in the problem. The diagnosis may be different and clinical observations are of more value than laboratory data. The classical signs and symptoms are well known.

Duckett Jones in his classification of rheumatic fever lists as major manifestations carditis, arthralgia, chorea and subcutaneous nodules. When the latter are demonstrated they are, of course, pathognomonic and imply a severe degree of infection. Minor manifestations are fever, abdominal pain, praecordial pain, rashes such as erythema marginatum, epistaxis and pulmonary and laboratory findings.

Taran made the following observation in the study of a large group in Brooklyn from six to fourteen years of age. Ninety per cent had an increase in white blood cells. They all had a fever for at least one week from onset, although 90 per cent showed

rheumatic activity after the temperature was normal. 85.5 per cent showed prolongation of the P-R interval in E.C.G. although 7.5 per cent had clinical evidence of continuing acute rheumatic infection when the P-R interval had returned to normal. The pulse and the cardiac rate are of tremendous importance, both being increased out of proportion to the temperature. In Taran's series all children had a pulse rate of over 100 until the end of the ninth week from the onset if the acute episode. One hundred per cent had an increase in sedimentation rate for eight weeks following onset and in 32 all sedimentation rates were normal, although 40 per cent showed evidence of mild rheumatic activity.

All children lost weight during the first eight to nine weeks of the active disease and returned to normal weight gain level 7½ months from the onset. All had lowered hæmoglobin at the onset of the illness but all had normal hæmoglobin eight months after the illness. Taran believes fatiguability, pallor, and cardiac symptoms to be important criteria. Cardiac sounds and murmurs change constantly and only become stabilized when the condition becomes quiescent—even an apical diastolic murmur, contrary to common belief, does not necessarily have a bad prognostic significance. There is a tic tac type of rhythm and systole is lengthened. Activity of rheumatic infection is probably best gauged by fever, tachycardia, leucocytosis, high sedimentation rate and electrocardiographic changes. A sleeping pulse over 80 means activity and over 90 probably indicates myocardial damage.

The treatment of the initial attack of rheumatic infection or recurrence consists if absolute bed rest until all signs of activity are over. How long the patient stays in the hospital depends on whether he is going to a convalescent unit or to his home and upon what kind of a home it is. In general, all other signs being favourable, and in the absence of trachycardia, the patient is let up three weeks after the sedimentation rate is back to normal.

Aspirin is universally back in favour again, 1 to 1½ grains per pound of body weight is given. The consensus of opinion is against intravenous use in childhood and many feel that taking it with food or mixing it with fruit juice is better than taking an alkali. Many also feel that capsules are contra-indicated because absorption is poor at the lower level in the gastro-intestinal tract. Aspirin may cause an early hypo-prothrombinæmia but spontaneous recovery is the result. Aspirin probably does not have a specific good effect on the rheumatic process. This controversy was started again in 1935 when salicylates were first used intravenously. It probably does not help to prevent or lessen cardiac damage but it certainly lessens the pain, makes the patient feel better and has a good sedative effect. The blood level of salicylates should be about 35 mg. per 100 cc. Phenobarb. or even morphine in older children, may have to be used to control pain or apprehension.

Oxygen is used when indicated by dyspnœa. More recently it has been used in cases of carditis without dyspnœa and appears to lessen the degree of cardiac damage.

Digitalis is used more at the present time than previously. Its use is indicated by a congested type of heart failure or fibrillation. Subjects with rheumatic heart disease do not respond as well to this drug as in the older degenerative group. The diet should be adequate in the acute phase and fluids not limited. The main thing is to prevent distension, so well-cooked, palatable meals, readily digestible—possibly smaller meals, more often—are advised. In addition to digitalis, when extensive œdema occurs, mercupurin may be used. Paracentesis may have to be used occasionally to relieve embarrassment in pericarditic fusion.

There is the problem of sub-acute rheumatism and sub-clinical rheumatism. These are definite posers and require careful investigation. Careful history, sedimentation rate, white blood counts, cardiograph, X-rays (A.P. and Lateral and oblique) and repeated physical examinations and observation are neressary.

About 50 per cent of so-called growing pains prove to be rheumatic later. Growing pains may be orthopædic in nature and are more likely to occur at night, not during

periods of physical activity. Other signs of rheumatic infection, of course, form a basis for considering the pains to be rheumatic.

Chorea exists to some degree at one time or another in 50 per cent of patients with rheumatic disease. It usually has a definite period of onset, the whole body is involved in writhing movements and it subsides fairly abruptly—in about two months. This distinguishes it from all habit spasm.

Laboratory tests to assist in the diagnosis of acute rheumatic infection are Red Blood Count, Hæmoglobin, Differential, Sedimentation Rate, Electrocardiogram and X-Rays. The latter is not so important in the first attack but it is in recurrent infection to show ventricular enlargement, particularly of the right and also the left auricle as well as pericardial fusion.

The evaluation of the status of the heart when the active stage is over is important. As we have said, no conclusions should be drawn about murmurs during the acute episode. Many are due to dilatation of the valve rings and the effect of the febrile condition on the myocardium. It must be remembered that heart murmurs are very common in childhood and according to Epstein at Cornell, 66 per cent of children have murmurs at one time or another during the age span of from 8-14 years.

Certain views may be helpful. As MacKenzie once said: "Rheumatism leaves more than one sign." Early in mitral valve disease the murmur is blowing and later becomes harsh. Generally speaking, louder murmurs are more likely to be organic murmurs. Whether or not they are propagated to the axilla depends on their loudness rather than their nature. The early murmur in mitral valve disease is accompanied by a disappearance of the first sound, and the murmur is not affected by respiration or changes in posture. An organic murmur may be considered when it is accompanied by a palpable thrill when there is evidence of cardiac enlargement or persistence of the murmur for longer than six months. Pure mitral incompetence is probably relatively uncommon and when it exists it accompanies to some degree a thickening of the mitral valve. A full-blown mitral stenosis takes five to fifteen years to develop. There is an apical diastolic murmur with presystolic accentuation. At this time there is accentuation of the first heart sound and an accentuation of the second pulmonic sound. The latter is always a sign of established mitral disease. Aortic insufficiency is much less common, and the murmur is heard in the upper sternal area and propagated downwords. There is the accompanying high pulse pressure and the typical pistol shot phenomenon. X-rays can help us in the diagnosis. There is left ventricular enlargement in aortic regurgitation; left auricular and right ventricular enlargement in mitral stenosis. An electrocardiogram is helpful, showing the left axis deviation and mitral incompetency and a right axis deviation in mitral stenosis with large and bifid P. waves. Other changes are observed which help to tell us of a damaged myocardium, but these are beyond the scope of this paper.

V. Recurrences

Rheumatic infection is the great disease of recurrence. In fact, the most characteristic tendency outside of its proneness to involve the heart is its tendency to recur. This tendency, with its repeated insults to the heart, leaves a high mortality rate. Only 1 per cent die in the first atack whereas 25-40 per cent die in the ten- to twenty-year period that follows the first attack when there are recurrences. It is not generally realized how serious rheumatic infection is, and few diseases constitute a greater problem as far as death and disability in children and young adults is concerned. This is true in this country as well as in England. Rheumatic fever and rheumatic heart disease account for more deaths in childhood than all forms of tuberculosis, and almost as many as the combined deaths from poliomyelitis, diphtheria, measles and scarlet fever. According to Martin, it is responsible for seven times as many deaths per annum as polio. Again, in World War II, according to many observers, the greatest single cause for rejection in the youngest age group was heart disease, and most of these were rheumatic in origin. It is apparent, then, that prevention of subsequent attacks would seem to be the crux of the whole rheumatic problem. Recurrence may occur in a matter of weeks

following the first attack or longer. Expectancy is high in the first year and 73 per cent of the cases recur in three years, acording to Roth. Frim six to eight years of life is the most common age for its onset and from eleven to thirteen is the peak of recurrence. Following puberty there is a falling off. The earlier the initial attack, the greater is the likelihood of recurrence and the more grave the outliok for the individual.

During the past decade, advances have been made toward the prevention of the recurrence of this dread disease. We will now consider them.

1. Convalescent care: this will be discussed later.
2. Moving to climates where rheumatic infection is less probable. Coburn's work on transporting children from crowded eastern Atlantic cities to Puerto Rico illustrates the importance of this. (Incidentally, the mortality rate for the Pacific area in school children was 4.6 in 1940 as compared to 11.3 in the middle Atlantic cities.) This, of course, when it is practicable and possible, might be applied in the winter months but it will be only possible for a favoured few.
3. Removal of focal infection: opinion is divided here and many say that after the initial infection little can be gained by removal of infected tonsils, teeth and clearing up infection of the sinuses. Others say that it is most effective. We, personally, know of one case that apparently benefited from the removal of grossly infected teeth. Probably a better view is to consider each case on its own merits, and if there is enough infection to merit removal in the interest of general health, then do so: for, as Parkinson says, "improving the health should apply all the more particularly in the case of rheumatic children." The pre- and post-operative periods should be covered by sulfa or penicillin. It is interesting in this regard that Weston, in quoting Kaiser, says that "the tonsils act as a storehouse for Vitamin C and a filtering plant for bacteria that cause systemic disease." Perhaps, in the future, we shall be even more conservative about tonsillectomies than we are now.
4. Prevention of upper respiratory infection: The occurrence of upper respiratory infection is directly related to the standard of living and the hygiene of the environment. More about this will be discussed later.

   Recently, following a better understanding of the pathogenesis of rheumatic infection, drugs have been used to prevent the occurrence of the strep. infections of the upper respiratory tract. Until recently the most important of these drugs were sulfanilamide and sulfadiazine, and still later sulphamerazine became the drug of choice because of its slower rate of excretion and, on this account, the smaller doses required. It was generally agreed that the drug would not stop the recrudescence of strep. infection once it has got underway and it has been likened to a time fuse and a bomb once the fuse has been lighted. Many of the children who have been put on the sulfa drug were kept on it all year around. This prevented summer recurrence and also eliminated the possibility of the first period of administration acting as a sensitizing dose and paving the way for future reactions. Under this regime the child should be free from all rheumatic activity, because the drug will aggravate rheumatic infection if present. Many children started receiving the drug in the hospital before they went home following the acute attack. Periodic examinations including Hæmoglobin, White Blood Count, Differential, Urinalysis to detect any signs of sensitivity to the drug should be done. If possible, sulfa blood level should be determined and kept from 1-3 mgms. to 100 cc.'s of blood. The administration may be continued up until puberty, after which time the recurrence rate drops off. The minimum time should be five years because 80 per cent of recurrences occur in this time. There have been surprisingly few reactions during the time the child is getting the drug. The dose of the drug—either sulfadiazine or sulfamerazine is 1 gram daily, preferably in two doses for those over 9 years and ½ gram for those under 9 years. Penicillin has been used more recently and will probably be used more in the future. Many workers report the development of sulfa-resistant strains of strep. with continued use. Penicillin troches may be used—

500 units per gram over four hours or, according to Duckett Jones in Boston, Aurol tablets in large doses—300,000 to 1,000,000 units may be used daily. Penicillin is probably more effective against strep. hæmolyticus and the work so far is very promising.

VI. Convalescent Care

We have become more conscious of convalescent care of children with rheumatic heart disease of recent years. Lugens-Anderson, Silver on this continent and Slechenger in England have contributed to our knowlewge concerning the importance if this phase of the work. A gap between the acute stage and the quiescent stage is supervised and the regimentation helps the child to adjust his first activity to the functional return of his heart. Taran has shown several interesting things with the group in a convalescing home and he had as a control group Outpatient and a cardiac clinic. The period of observation was close to 9 months and the period of observation after discharge was 14.5 months. He found that gain in weight in the convalescent home was always a favourable sign in rheumatic infection and was 2 to 3 times that in the cintrol group. It was also evident that the incidence of respiratory infection was low in the institutional group as compared with the control group. Regulation of rheumatic fever in the treated group was one-third of that in the control group. Another interesting thing was that prior to convalescing, the acute upsets were less frequent and milder than in the control group. The actual routine at the convalescent home is a study in itself, but good diet and cheerful surroundings to elevate the morale, carefully graded exercises starting with tension exercises early to preserve muscle tone, massage, supporting exercises later and then more active exercises are all part of the programme. School classes can be started soon after the acute stage. Occupational therapy to fill in the idle hours and the use of the sun porch are outstanding features in a regime of this type.

VII. Sociological and Education Factors

The education of a rheumatic child presents many difficulties. Most of them are about three grades behind their more fortunate companions. This makes for possible maladjustment because often they are the oldest and the biggest children in the class. Too often they become conscious of this and this results in their leaving school early. Thus we have children who are handicapped from the educational standpoint as well as physical. This is a double misfortune because often they require a sedentary type of work and their fruitful years may be short. Actually they should be better educated than others.

Sociological aspects are startlingly clear in this disease. No other disease has so high a social incidence. It has a high prevalence where there is overcrowding, dampness, bad housing, absence of sunshine and poor food. Conversely, Jackson and others found that the diet and environment materially benefits the status of a group of rheumatics in Iowa, brought in on account of recurrence of the infection. This rather large group received 1 quart of milk daily, ample proteins in eggs and meat, two kinds of fresh fruit and two fresh vegetables and cod liver oil and apple butter and margarine. The child slept in his own room, or at least in a bed by himself, because of frequent familial incidences of this disease and got at least ten hours' sleep every night. Temperature and humidity in the rooms were controlled when possible and proper clothing worn. More often other members consulted the doctor when they had an intercurrent infection. The recurrence rate in this group differed significantly from the group picked at random in Iowa where there was no supervision.

VIII. Large Scale Control Measures

There is no doubt that large scale and coordinated control measures are as necessary in the case of rheumatic children as in the case of tuberculosis and should be on a provincial or federal level. More convalescent facilities should be available. The disease should be notifiable so that the family can be investigated before the child returns home. When he does, he is to return to optimum conditions for his complete recovery. Educa-

tional and financial assistance are necessary. It is gratifying to know that in some places this is in effect. The Invalid Children's Aid Association and the London County Council in England and the Central Registry in San Francisco where they coordinate an intensive programme of case finding and follow-up work in connection with rheumatic disease stand as examples of this. Good work is done also under supervision of the State University of Iowa through the University Hospital and mobile clinic of the state services for crippled children. Recently a Government grant has been created in Canada to study the needs and improve the conditions pertaining to crippling disease in childhood and the beginning of this important work is already under way.

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THE EYE IN GENERAL MEDICINE

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It is my purpose to discuss this subject briefly, under the following headings: 1. General Considerations; 2. Paralysis of the Extrinsic Muscles; 3. Paralysis of the Intrinsic Muscles; 4. Changes in the Fundus Oculi, and 5. Changes in the Visual Fields.

I. General Considerations

The eyelids, conjunctivæ and cornea may be involved in many skin diseases, such as lupus vulgaris, erysipelas, erythema multiforme, blastomycosis, molluscum contagiosum, acne rosacea, pemphigus, etc. Smallpox used to be an important cause of blindness, from corneal involvement. Chickenpox occasionally causes corneal and conjunctival lesions. Herpes zoster ophthalmicus may give rise to corneal scars or iritis. Rarely severe generalized dermatitis or eczema is accompanied by bilateral cataracts. Leprosy frequently involves the cornea and conjunctiva.

Measles, scarlet fever, influenza and certain cases of pnuemonia commonly cause conjunctival congestion, and may cause iritis or ididocyclitis. The ocular complications of diphtheria are well konwn. Syphilis may involve various structures of the eye at any stage of its course. Tuberculosis likewise may affect the eye in various ways. Gonorrhœa not infrequently is the cause of a purulent conjunctivitis both in the infant and adult. Acute gonococcal iritis may be associated with arthritis of the same ætiology. Undulant fever has been found to underlie certain obscure varieties of ocular inflammation. Animal parasites may infest the eye, but this is rare in this country—trichinosis is perhaps the commonest, the larvæ lodging in the extrinsic muscles, producing localized inflammation with tenderness and pain on moving the eyes. Oedema of the eyelids may be associated.

The fundal changes of anæmia and cardiovascular renal disease will be discussed later.

Jaundice is frequently detected first in the eyes, where it produces characteristic discolouration of the scleræ. The pingueculæ of Gaucher's Disease are almost pathognomonic of the condition, showing up as brownish-yellow wedge-shaped thickenings, first on the nasal, and later on the temporal, side of each eye. Not infrequently, the petechiæ of subacute bacterial endocarditis are to be found in the conjunctivæ.

Diabetes mellitus may be accompanied by changes in refraction, cataract and retinopathy. Hyperthyroidism not uncommonly is attended by exophthalmos, and the other "eye signs" of this disease, which are well known. Paralysis of one or more of the extrinsic muscles, and optic neuritis, may complicate the more severe grades of exophthalmos. Cataracts are seen in hypoparathyroidism. Gout may occasionally cause ocular inflammation of various types.

Inflammatory changes in the orbit may cause proptosis and chemosis, and are sometimes followed by cavernous sinus thrombosis. An orbital new growth commonly gives rise to unilateral exophthalmos without accompanying signs of inflammation. Pulsating exophthalmos results from rupture of the internal carotid artery into the cavernous sinus, and is usually of traumatic origin. Less commonly it is due to atherosclerosis or aneurysm of the artery.

Exophthalmos forms part of the clinical picture of Hand — Schuller — Christian disease in a certain proportion of cases, and along with diabetes insipidus and calvarial defects constitutes the so-called "Christian Triad."

II. Paralysis of the Extrinsic Muscles

In this connection, the fourth cranial nerve is of much less clinical importance than the third and sixth. It is essential to ascertain whether one isolated muscle is paralyzed, or whether the paralysis is in terms of conjugate movements. If the latter proves to be the case, then the lesion is in the nucleus or higher up—in the cerebral hemisphere, etc.

The paretic origin of early squints is supported by many eminent ophthalmologists and birth injuries and prenatal anoxæmia and toxæmia have been invoked to explain them.

Paralysis of lateral movements is the commonest type of conjugate lesion, but paralysis of upward movements can occur in lesions of the upper part of the third nerve nucleus. These are seen commonly in luetic and other vascular conditions. Myasthenia gravis is the only muscular lesion in which this may be found.

The trunk of the third or sixth nerve may be involved in the *orbit* (when there are usually other signs beside muscular palsies—such as proptosis or chemosis of the conjunctivæ). *Complete paralysis of all the muscles of one eye suggests an intraorbital lesion*—such as new growth, cellulitis, orbital periostitis, etc.

The nerve trunks may be involved in their course along the *basis cranii* on the under surface of the brain—the commonest causes of such palsies being tuberculous meningitis, syphilis, aneurysm, tumour, etc.

Lastly, the nerve trunks may be damaged in the brain stem by tumours, vascular lesions, syphilis, etc.

Causes of Ocular Palsies

A. *Acute* — *Poisons,* such as alcohol, lead, botulism.
Infections, notably diphtheria, encephalitis lethargica, syphilis, tuberculosis, etc.
Trauma, tumours, and *aneurysms.*

B. *Chronic*—G.P.I. and tubes dorsalis, rarely disseminated sclerosis causes a permanent, but often a transient strabismus;
occasionally bulbar palsy, myasthenia gravis, and tumours.

The role of syphilis in the causation of ocular palsies deserves special mention. It may take the form of a gummatous meningitis at the base of the brain, or the patient may have tabes dorsalis, general paralysis of the insane, or cerebral thrombosis, the result of syphilitic endarteritis obliterans.

Isolated paralysis of the external rectus muscle occurs for no apparent reason at times, and probably is anaogous to the common Bell's palsy affecting the facial nerve. It is of sudden onset, lasts a few weeks or months and then gets well. One must be careful to exclude other causes, especially syphilis.

It is rare to get ophthalmoplegia in migraine, but when it occurs it is usually in young persons who have been subject to attacks (of migraine) for years. The ocular paralysis comes on after an attack and does not completely clear before the next attack, and is made worse, and this continues until the palsy, e.g. ptosis, is complete.

The ocular manifestations of myasthenia gravis are of great importance and constitute the most frequent and earliest signs of the disease.

In Walsh's paper in the January, 1949, issue of the C.M.A.J. the important ocular findings were enumerated as follows:

1. Usually ptosis is the first sign. Diplopia is the commonest and earliest symptom in most cases. Ocular signs frequently remain predominant, and usually are fluctuant.
2. Purely ocular myasthenia gravis occurs occasionally. In some instances there was a spread of the weakness after months or years.
3. In a very few cases the ocular signs appeared late in the course of the disease.
4. The ocular signs may completely disappear during a remission. (We recently had such a case in Victoria Hospital, with a spontaneous remission lasting about a year.)
5. Oedema of the eyelids is rarely a prodromal sign of myasthenia gravis. No explanation for this is available.
6. Retraction of the eyelids is seen infrequently and usually occurs where there previously has been ptosis.

7. Ptosis may be present in association with opposite-sided lid retraction.
8. Weakness of the orbicularis oculi is always present when ptosis exists. This observation, which is easily determined by asking the patient to close the eyes while the upper lid is held up with the finger, differentiates myasthenic from neurogenic ptosis. Weakness in closure of the eyelids is overlooked more often than any other common ocular sign.
9. The similarity of abnormal associated movements of the eyelids in myasthenia gravis and those resulting from misdirection of regenerated fibres in the third nerve has been observed.
10. Limitation of ocular movements occurs either unilaterally or bilaterally and in all combinations. Strictly unilateral ocular involvement was observed in a single case. In many instances the ocular signs are misinterpreted. Some of the erroneous diagnoses were: hyperthyroidism; postencephalitis; tumour of the brain stem; disseminated sclerosis. Nystagmoid movements occasionally are due to myasthenia gravis affecting the ocular muscles.
11. Changes in accommodation were noted in only one case, but slight changes might have been overlooked.
12. In the cases studied the pupillary responses to light were invariably normal. This observation suggests a useful rule in diagnosis. If the pupillary responses to light are normal and the origin of a ptosis or other extraocular paresis is not crystal clear, myasthenia gravis should be suspected.

In the majority of cases the final proof of a diagnosis of myasthenia gravis rests on the improvement in power that occurs in weak and readily fatigued muscles after the injection of prostigmine. Atropine should be given with the prostigmine to counteract side-effects which may assume serious proportions.

Hereditary and familial ptosis, in which no other lesions have developed, has been reported recently by Amyot, who reviews the literature on this subject, and describes several (9) cases of his own. In marked cases the frontalis is contracted and the head tilted backward in order to get the pupils into line with the narrowed palpebral fissure.

III. Paralysis of the Intrinsic Muscles

There are two sources of nerve supply to the pupil—the parasympathetic (third cranial nerve) constricts it, and the cervical sympathetic dilates it.

Normally, the pupils are round, regular, equal, and centrally-placed, and react briskly to light and upon convergence.

Meiosis, or abnormal smallness of the pupil may be due to paralysis of the cervical sympathetic; minute pupils are sometimes associated with syphilis of the nervous system, particularly tabes; meiosis also occurs with acute lesions of the pons and it may be seen in advanced age without pathological changes. It is also an accompaniment of opium poisoning.

Mydriasis denotes dilatation of the pupil. The sphincter of the pupil is controlled by the small nucleus of Edinger-Westphal, situated in the uppermost part of the third nerve nucleus. Paralysis of this nucleus, or of the third nerve may result in mydriasis. It results also from the action of belladonna, atropine and cocaine, and sometimes is seen in middle meningeal extradural hæmorrhage and subdural hæmatoma.

The importance of careful and repeated observations of the state of the pupils, in cases of head injury, should be stressed. Unilateral dilatation and fixation are of value as localizing signs in extradural and subdural hæmorrhage, but may be found in patients with extensive and intracerebral hæmorrhage as well. Bilateral dilatation and fixation, and contraction and fixation of the pupils, after trauma, are indicative of serious injujry to the brain. Fixation is of more grave significance than inequality.

Inequality, irregularity, and defective reaction to light are commonly seen in syphilis. However, in a series of 500 normal subjects, 17 per cent were stated to have anisocorcia, in a recent publication.

Argyll-Robertson, the distinguished Edinburgh ophthalmic surgeon, described the pupils in tabes as (1) small, (2) unequal, (3) irregular, (4) eccentric, (5) failing to react to light but doing so upon convergence. Before this full-blown picture is reached, they may be sluggish and show a lessened amount of contraction, with hippus, and there are often irregularities on the two sides.

The *Holmes-Adie Syndrome* is seen in young adults without syphilis—one pupil may be affected more than the other. Such a pupil is often dilated, does not react to light when a light is shone on the eye, but if the patient sits for 10 or 15 minutes in a bright diffuse light, the pupil gradually contracts, and if he sits in a dark room it dilates slowly. During accommodation-convergence, contraction of the pupil takes place slowly and continues through an abnormal range of movement, until the myotonic pupil becomes smaller than its fellow of the opposite side. After relaxation of accommodation it slowly dilates. The iris does not show the degenerative changes that are commonly seen in the Argyll-Robertson pupil. In some cases it is associated with absent tendon jerks, or either phenomena may occur alone. It is a benign clinical syndrome of unknown ætiology, and quite compatible with a state of perfect health.

Paralysis of the Cervical Sympathetic

Paralysis of the cervical sympathetic produces narrowing of the palpebral fissure (cervical sympathetic ptosis), and a small pupil. Enophthalmos is also held by many to be part of this picture, though others doubt it.

Cervical sympathetic paralysis occurs in the following conditions:

1) In many lesions of the cervical cord, especially when the last cervical and first dorsal segments or roots are damaged, commonly in syringomyelia.
2) In lesions of the cervical sympathetic trunk by trauma, pressure, growths, etc.
3) It is very common in tabes and neurosyphilis generally, where it appears as partial bilateral ptosis with small pupils, and does not improve with treatment.

IV. Changes in the Fundus Oculi

It should be noted that a certain amount of venous pulsation occurs normally in the fundus oculi. Arteriolar pulsation is much less common as a normal phenomenon, and is seen in certain cases of free aortic regurgitation, arterio-venous shunts, hyperthyroidism, etc.

a) *Arteriosclerotic Retinopathy*—variations in the calibre of the arterioles, and arterio-venous compression are early signs, and are often unilateral at first. Later on, hæmorrhages and areas of localized ischæmia, showing up as white plaques, make their appearance. If the macular region is involved visual acuity is reduced.

b) *Diabetic Retinopathy*—is really only a variant of the preceding type. Typically one finds small round or oval white deposits in the retina, which may have a yellowish or fatty appearance. Hæmorrhages of varying size occur, as well as changes in the vessels.

Lipæmia retinalis occasionally occurs in diabetes mellitus and when present is most striking, the vessels appearing to be filled with milk or cream instead of blood.

c) *Hypertensive Retinopathy (or Neuroretinopathy)*—the so-called "albuminuric retinitis" shows widespread arteriolar constriction, and often marked arteriovenous compression. Oedema of the retina occurs, especially in the macular region, resulting in the characteristic star-shaped white deposits. More extreme disturbances of the circulation result in papillœdema being added to the picture.

d) *Papilloedema* or choked disc is due to swelling of the nerve head as the result of increased intracranial pressure of interference with the venous circulation of the eye.

Space-occupying intracranial lesions, and meningitis are the most common causes. When it occurs in patients with severe hypertension it is often associated with the so-called hypertensive cerebral attacks of hypertensive encephalopathy, in which headache, vomiting, less of consciousness, disturbances of vision, and convulsive phenomena are features.

e) *Post neuritis (secondary, or consecutive) Atrophy* is characterized by blurring of the outline of the disc, and white lines (exudate) often accompany the vessels away from the disc. It is seen when papillœdema or optic neuritis has subsided.

f) *Primary (simple) Optic Atrophy* shows a pale or white nerve head, with well-defined outline. Syphilis is commonly the cause. It may be the result of retrobulbar neuritis or toxic amblyopia. Direct pressure on the optic nerve by a tumour causes this picture. Disturbances in vision and in the visual fields are always present.

g) *Embolism (or Thrombosis) of the Central Artery of the Retina* produces a pale fundus, and is commonly seen in elderly people with arteriosclerosis, or in persons with valvular heart disease. Sudden and almost complete loss of vision in the affected eye is the only symptom noted by the patient. Injections of acetyl choline subconjunctivally, prompt and vigorous massage of the eyeball, or paracentesis of the anterior chamber, designed to reduce intraocular tension, may allow the embolus to pass into a smaller vessel.

h) *Thrombosis of the Central Vein of the Retina*—most commonly due to arteriosclerosis of the accompanying artery, which may compress the vein quite readily, and produces intense venous congestion and severe hæmorrhages in the retina. Vision is reduced, but not as dramatically as in embolism of the central artery, and often takes several hours to reach its maximum. The prompt use of heparin and dicumarol may be of some value in preventing complete thrombosis, and should be kept up for a week or longer using the customary controls.

i) *Primary Optic Atrophy in one fundus and Papilloedema in the other (the Foster Kennedy Syndrome)* is ordinarily due to a tumour pressing directly on the optic nerve on the side of the atrophy, and causing papillœdema in the other eye due to a general increase in intracranial pressure. However, the writer has seen one such patient who developed sudden loss of vision in one eye some three years before the onset of hypertensive encephalopathy. On examination she showed fine attenuated vessels, with optic atrophy, in her left eye, and marked papillœdema in the right, and a systolic blood pressure of 260 mm.Hg. She had undoubtedly suffered an embolism or thrombosis of her left central retinal artery. With bed rest, lumbar puncture, etc., her blood pressure fell, and her papillœdema subsided.

j) *Anaemia*—Pallor and hæmorrhages into the retina are common in the anæmias, particularly in pernicious anæmia, and severe leukæmia. The hæmorrhages, if not marked may be peripherally situated, and missed if the eye grounds are examined hastily, or if the pupils are not dilated. White deposits similar to those seen in arteriosclerotic retinopathy may occur. Papillœdema of mild degree may also be seen in association with severe anæmia.

k) *In polcythemnia vera* and in the type associated with certain forms of congenital hetrt disease the retina may be cyanosed and the veins and arteries may be intensely engorged and tortuous.

V. *Changes in the Visual Fields*

a) *Papilloedema* causes an enlargement of the blind spot in the early stages. Later conrentric contraction occurs as optic atrophy supervenes.

b) *Optic Atrophy* always produces defects of central vision or of the fields. Syphilitic optic atrophy commonly produces a gradual concentric contraction of the visual

fields which may become quite marked before the patient observes it. Central vision is involved later. Complete or nearly complete blindness is the end-result.

c) *Lesions of the Higher Visual Pathways* are commonly due to tumours or vascular lesions. Carefully determined visual fields are of the utmost importance in localization. Lesions between the globe and optic chiasma produce visual damage to only one eye. Lesions at the chiasma affect both visual fields as a rule. A pituitary tumour may result in a bitemporal hemianopsia, or, if it grows asymmetrically, it may cause blindness in one eye and the loss of the temporal field in the other. Should it extend posteriorly and damage one optic tract, an homonymous hemianopsia will be present. Lesions of the visual radiation and occipital cortex likewise produce hemianopsia, in which the macular fibres are commonly spared, thus leaving good central vision. Temporal lobe tumours frequently cause an homonymous quadrant defect, usually of the upper quadrants.

d) *Hysteria* commonly produces a helical type of visual field, a rapidly changing field, or "rifle" or "tunnel" vision, or the colour fields may be reversed.

News and Notes

At the recent Canadian Medical Association meeting in Saskatoon, Dr. Norman H. Gosse of Halifax was named President-Elect for 1950; President, Dr. J. F. C. Anderson of Saskatoon. The Executive Committee of Provincial Representatives to serve with Dr. Anderson will include Dr. F. M. Bryant of Victoria, Dr. Harold Orr, Edmonton, Dr. E. A. McCusker, Regina, and Dr. R. W. Richardson, Winnipeg.

The first national organization of Pathologists was formed in Saskatoon by members of the Canadian Medical Association with tentative headquarters in Saskatoon and Toronto. Officers chosen included two vice-presidents: Dr. D. F. Moore, Saskatoon, and Dr. John Hamilton, Kingston, Ont. A president has not yet been named. Included on the executive are: Dr. Harold Taylor, Vancouver; Dr. John Duffin, Calgary; Dr. D. R. McLatchie, Regina; Dr. S. Lederman, Winnipeg.

Leading physicians and surgeons from 10 countries met in Saskatoon, June 7, for the first annual British Commonwealth Medical Conference. The sessions lasted four days. Delegates from Australia, Canada, Ceylon, Eire, Great Britain, India, Newfoundland, New Zealand, Pakistan, South Africa and Southern Rhodesia have attended and discussed the whole range of Medicine. Canada was represented by Dr. William Magner of St. Michael's Hospital, Toronto, president of the Canadian Medical Association, and Dr. Harris McPhedran, Chairman of the general conference of the C.M.A.

At the C.M.A. meeting in Saskatoon it was reported by the Federation of Medical Women of Canada that there were 700 women doctors in Canada. Dr. Jean Macdonald of Halifax was chosen federation president for 1949-50, succeeding Dr. Anna Nicholson of Saskatoon. Vice-presidents include Dr. Elda Lindenfeld of Vancouver.

Congratulations are extended to the following doctors on their recent good fortune:

Dr. and Mrs. John E. Hill, a son, John Edward, Jr.
Dr. and Mrs. George H. McKee, a son, William Treen.
Dr. and Mrs. Angus MacMillan, a son, Bruce Donald.
Dr. and Mrs. F. E. McNair, a daughter, Lorraine Margaret.
Dr. and Mrs. J. A. Marcellus, a son.
Dr. and Mrs. Eric Webb, a son.

It is also a privilege to extend congratulations to newlyweds, Dr. and Mrs. L. W. Warcup and Dr. and Mrs. Norman C. Chivers. Congratulations to Dr. and Mrs. Howard McEwen on the marriage of their daughter to Mr. James Y. Johnstone.

A number of changes of location have taken place:

Dr. W. D. Love, formerly of Vancouver, moved to the Irving Clinic, Kamloops.

Dr. K. J. Williams is now at Invermere.

Dr. Ralph C. Pronger has been appointed to the staff of Drs. OCallaghan & Haszard, at Kimberley.

Dr. Gordon Wride has been appointed assistant director of Health Insurance for Canada in Ottawa.

Dr. W. C. Stewart has joined the Burris Clinic at Kamloops.

The following doctors have left on post-graduate work:

Dr. H. E. Cannon is leaving Abbotsford for one year's study at Edinburgh in Surgery.

Dr. R. R. Galpin is going to the Henry Ford Hospital, Detroit, for training.

Dr. C. C. Jackson is leaving Shaughnessy Hospital to study Proctology with Dr. Clement L. Martin, Loyola University Clinics for one year, and later, he is going to work with Dr. Harry E. Bacon, Temple University Hospital, Philadelphia, for two years.

Dr. Frank D. Wilson is leaving for a year's study in Surgery at the Crawford Long Memorial Hospital, Atlanta, Ga.

Dr. W. R. McEwen will leave for his study in Obstetrics and Gynaecology, at the University of Alberta.

Dr. Alan Inglis has left the Sechelt Penninsula to take a post-graduate course in Surgery, at the Vancouver General Hospital.

Dr. E. W. Skwarok is leaving to take a course at the Maine General Hospital, Portland, Maine.

Dr. J. H. Moore of Victoria, has been appointed deputy coroner to succeed Dr. Walter Bapty. Dr. Moore will act during the absence or illness of Coroner E. C. Hart.

It is a pleasure to welcome back in our midst Dr. W. C. Gibson, Victoria neurologist.

It is with regret that we record the deaths of the following:

Dr. C. Wesley Prowd, Vancouver, age 66.
Dr. Gordon C. Draeseke, Vancouver, age 68.
Dr. A. E. Archer, Lamont, Alta., age 70.
Dr. Lawrence Broe, New Westminster, age 67.

Deepest sympathy is extended to Dr. McNeill on his recent bereavement.

British Columbia, with the rest of Canada, mourns the loss of Dr. A. E. Archer of Lamont, Alberta, who died recently at the age of 70. As President of the Canadian Medical Association some years ago, he had reached the top of his profession, from the point of view of service to that profession. He had, in his life, many other honours conferred on him, and his life was one of service in the highest degree. He was intensely interested in all matters referring to the well-being of medicine, and especially in Health Insurance, in connection with which he was consultant and adviser to the Canadian Medical Association.

DR. GORDON C. DRAESEKE

Obit., June 13, 1949

The Medical Profession of Vancouver has lost in Dr. Gordon Draeseke, one of its outstanding members. Quiet, unassuming, but a man of great personal charm and an excellent physician. The community at large has lost a man who gave eminent service to his country, both in peace and war: for Draeseke served with distinction in the Canadian forces, and reached high rank in his own department, the Army Medical Corps. He had an intense interest in military medicine, and threw himself heart and soul into the activities of this great branch of the Army; but withal he was first and foremost a practitioner of medicine, and kept always the human sympathy and understanding of human problems ,that make the good physician.

Dr. Draeseke began his practice in Vancouver in 1909, specializing in eye, ear, nose and throat diseases. His proficiency as a specialist was well known, and from 1930 to 1943, he was head of this department at Shaughnessy Hospital and the Vancouver General. In the latter year or two of his life, repeated warnings of the ailment that was to claim his life, (coronary disease) had compelled him to limit his activities.

Gordon Draeseke was a fine man and a good physician. He was, too, a good citizen. No finer epitaph can be written of him. Our sympathies are extended to his wife and family.

DR. W. S. PROWD

Obit., June 9, 1949

The death of Dr. "Wes" Prowd, marks the passing of one of the leaders of medicine in Vancouver, and of a pioneer in modern medicine. When Dr. Prowd came to Vancouver, radiology was in its infancy, and he saw it grow from a rather inadequate, very limited process, to the magnificent thing it is today, one of the special branches of medicine, unique as a diagnostic and exploratory agent, but also of inestimable value through its therapeutic and curative powers. When Dr. Prowd began his long career as a radiologist, the "X-ray man" was a technician with medical knowledge, rather than a specialist, and he saw his chosen field develop and widen, till today, the radiologist can claim to be a specialist and consultant in his own right. He was one of the men, and a very prominent member of the craft, who were responsible for this growth. He became himself, one of our greatest Canadian radiologists, and had in full measure, all the integrity of mind and complete objectiveness, which are the true signs of the good radiologist. The modern radiologist owes a debt to men of Dr. Prowd's type and generation that cannot lightly be reckoned.

He was the head of the fine X-ray department at St. Paul's Hospital, and he made it and kept at at a very high level of excellence. His work with radium, and deep X-ray therapy, led the field, and he was an early leader in this department of radiology.

Personally, he was one of the most charming and kindly of men, a witty speaker, and a man who formed his own opinions and judgments. These always commanded the respect of his fellows—and he was regarded as one of the leaders of our profession in British Columbia. He was at one time, president of the Vancouver Medical Association, and took an active part in the affairs of his profession. He was, of course, in constant contact with medical men and enjoyed their affection as well as their respect and confidence.