THE

BULLETIN

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VANCOUVER MEDICAL ASSOCIATION

Founded 1898 : Incorporated 1906.

Programme for Fiftieth Annual Session

(Spring Session)

February 17th CLINICAL MEETING—St. Paul's Hospital, Nurses' Auditorium.

March 5th (Friday) OSLER DINNER AND LECTURE—Hotel Vancouver, Banquet Room.
Osler Lecturer—Dr. Murray Blair.

March 16th CLINICAL MEETING—Children's Hospital.

April 6th GENERAL MEETING—Auditorium, Medical-Dental Building.
Speaker—to be announced.

April 20th CLINICAL MEETING—Place of meeting to be announced.

May 4th ANNUAL MEETING—Auditorium, Medical-Dental Building.

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1. "Recommended Dietary Allowances Revised 1945"; Reprint and Circular Series No. 122, August, 1945, Food and Nutrition Board, National Research Council, 2101 Constitution Ave., Washington 25, D. C.

For Literature write

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VANCOUVER HEALTH DEPARTMENT

CASES OF COMMUNICABLE DISEASE REPORTED IN THE CITY

STATISTICS—OCTOBER, 1947

Total Population—Estimated	339,350
Chinese Population—Estimated	5,980
Hindu Population—Estimated	118

	Number	Rate Per 1000 Population
Total deaths	379	13.1
Chinese deaths	21	41.3
Deaths, residents only	379	13.1

BIRTH REGISTRATIONS:

Male	487	
Female	454	
	941	32.4

INFANT MORTALITY:

	October, 1947	October, 1946
Deaths under 1 year of age	15	25
Death rate per 1000 live births	21.1	31.4
Stillbirths (not included above)	10	10

CASES OF COMMUNICABLE DISEASE REPORTED IN THE CITY

	October, 1947		November, 1947	
	Cases	Deaths	Cases	Deaths
Scarlet Fever	8	0	8	0
Diphtheria	1	0	1	0
Diphtheria Carrier	6	0	0	0
Chicken Pox	26	0	25	0
Measles	39	0	98	0
Rubella	0	0	3	0
Mumps	13	0	21	0
Whooping Cough	3	0	3	0
Typhoid Fever	0	0	2	1
Typhoid Fever Carrier	0	0	1	0
Undulant Fever	8	0	5	0
Poliomyelitis	11	1	0	0
Tuberculosis	55	14	45	0
Erysipelas	1	0	6	0
Meningococcus (Meningitis)	0	0	2	2
Infectious Jaundice	0	0	0	0
Salmonellosis	2	0	1	0
Salmonellosis (Carrier)	0	0	0	0
Dysentery	0	0	1	0
Dysentery (Carriers)	0	0	0	0
Tetanus	0	0	0	0
Syphilis	0	2	0	0
Gonorrhœa	0	0	0	0
Cancer (Reportable):				
Resident	78	0	0	0
Non-Resident	22	0	0	0

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The Editor's Page

Every doctor in British Columbia will by now have received through the mail a copy of the News Letter sent out by the Committee on Economics of the B.C. Medical Association. We urge our readers to study this News Letter carefully. It is a very laudable move on the part of this Committee, and we note with pleasure that this is only the first of a regular series that is to be sent from time to time. One receives a great many official-looking envelopes in the mail—and it is to be feared that a good many of these are not as carefully read as they should be. This is one that should be kept, and filed for reference.

It will be noted that it opens with a statement as to the M.S.A. which, under its constitution and bylaws, agreed to by the medical profession of British Columbia, has certain rules, laid down in Paragraph 20, 1947 reprint. These rules, we understand, are going to be enforced in the future, more rigorously than they have so far. We think that no fair-minded medical man can object to this enforcement. After all, it is exactly similar to the rules of the Workmen's Compensation Board, and since the M.S.A. is expected to adhere to its promises, and to pay our bills, we cannot deny it the right to insist that we keep to our side of the bargain too. Emergencies are allowed for—and it is only in elective work that the M.S.A. is insisting that authorisation be obtained from the Directors of Medical Services before major surgery, X-rays and son on be carried out. It is certain that interference will be at a minimum, and that every reasonable latitude will be given to doctors.

The rising costs of auxilliary services, of hospitalisation, etc., and the increase in advanced procedures, are a very heavy tax on the M.S.A. Its charges to beneficiaries cannot be increased indefinitely: and the medical profession of B.C. cannot afford to allow conditions to proceed to a point where the very existence of the M.S.A. and its continuance may be threatened. This applies, too, to the other approved plans of prepaid medical services now in force. We all know that there are elements in the community which would be only too glad to see these efforts fail—since then they would feel justified in insisting that the government step in and introduce a system of Health Insurance, which would, if our past experience has taught us anything, be far worse from our point of view. Here we have a system or systems which are in accord with the methods of practice which we prefer. It is true that they involve a reduction on our part of a percentage of our regular scale of fees. But they are only applicable to the lower income brackets, and to families from whom we could not hope, especially under present financial conditions, with the prospect of even leaner times to come, to collect anything like 75% of our fees: not to mention the fact that under the prepaid plans, much work comes to us which should be done, but which would not be done and would not come to us if these people had to pay their own bills in full. All of us who have practised medicine for more than twenty years, let us say, know this to be true. Many of them feel that the income ceiling might well be raised—it has even been suggested that it be abolished—but this is probably too radical a step.

We must all feel one thing at least to be true—and that is that we owe a great debt of gratitude to this Committee on Economics. It has done an unbelievable amount of work—this news-letter only deals with one of its tasks. The scale of fees is another: the question of income tax another—and there are many other problems that they have tackled, and are considering. The medical profession of British Columbia is extremely fortunate in having such a committee. It will be to the interest of us all to consider very thoroughly their carefully prepared statements and recommendations.

LIBRARY NOTES

HOURS—

Monday, Wednesday and Friday	9.00 a.m. to 9.30 p.m.
Tuesday and Friday	9.00 a.m. to 5.00 p.m.
Saturday	9.00 a.m. to 1.00 p.m.

SPECIAL SERVICES—

Messenger Service:

Members in Vancouver whose offices are outside the Medical Dental Building are entitled to free delivery service. Requirements should be telephoned in before 10.00 a.m. if books are needed the same day. It is requested that books being returned to the Library be wrapped in the same or similar covering.

Mailing Service:

Members outside Vancouver may request loans by mail. Packages are sent with return carriage prepaid.

Microfilm Service:

Microfilms or photostatic copies of articles may be obtained through the Library from the Army Medical Library in Washington. Delivery is usually made in from two to three weeks and the cost is absorbed by the Association.

RECENT ACCESSIONS TO LIBRARY—

Psychotherapy in General Practice, 1947, by Maurice Levine.

Pharmacology and Therapeutics (Cushny), 13th ed., 1947, by Grollman and Slaughter.

Diseases of Metabolism, 2nd ed. 1947, by Garfield G. Duncan.

Surgical Clinics of North America, Symposium on Modern Trends in Surgery, Philadelphia Number, December, 1947.

Surgical Pathology, 6th -d. 1947, by William Boyd.

Textbook of Medicine, 7th ed. 1947, by Russell L. Cecil.

Gynecological and Obstetrical Pathology, 2nd ed. 1947, by Emil Novak.

Diseases of Women, 9th ed. 1947, by Crossen and Crossen.

CORRESPONDENCE

January 15, 1948.

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

Dear Dr. MacDermot:

The qualified internists of British Columbia have held meetings and decided to organize the "British Columbia Society of Internal Medicine." The objects of the society are:

(a) To promote the study and advance the knowledge of medicine in the province Medicine.
of British Columbia, with particular reference to the speciality of Internal

(b) To act in an advisory capacity to the established medical organizations in matters relating to the practice of Internal Medicine.

(c) To help young doctors who wish to specialize to achieve the necessary training.

At an organizational meeting the following board of Directors were elected until 31st August, 1948: President, Dr. C. H. Vrooman; Vice-President, Dr. A. W. Bagnall; Secretary-Treasurer, Dr. F. L. Skinner; Directors, Dr. D. S. Munroe and Dr. S. G. Kenning. The board were instructed to offer charter membership to all fellows of the Royal College of Physicians of Canada, and those holding certificates of Internal Medicine or Internal Medicine (Tuberculosis). There are 51 of these in the province. Forty-seven are duly paid members.

It is requested that you give notice to the medical profession, to whom your publication serves, of the formation of this society so that all those interested may communicate with the society, and if there have been any names overlooked such corrections can be made.

The society will be holding clinical meetings, and will be interested in all factors dealing with the welfare of medicine in this province.

Thank you for your help.

Yours sincerely,
F. L. Skinner, M.D.,
Secretary-Treasurer.

* * * *

January 16th, 1948.

Dear Sir:

I am directed by the Directors of the British Columbia Surgical Society to advise you of the forthcoming Annual Session.

This Session will be held in the Hotel Vancouver, March 18th and 19th next. The distinguished guest speaker is Dr. R. M. Janes, Professor of Surgery of the University of Toronto.

There will be a series of sixteen, twenty-minute papers, followed by discussion of a wide range of surgical subjects. These include general gynaecology, thoracic, orthopaedic and urological branches of surgery.

All scientific sessions are open to the members of the medical profession at large. Every practitioner will be made most welcome.

A limited number of rooms have been reserved at the Hotel Vancouver for out of town visitors. These may be had by direct application to the Hotel.

May I repeat that all members of the profession will be made most welcome.

Registration fee is $5.00 for the session.

I wonder if you would be good enough to give this prominent publicity in the Bulletin.

Yours very truly,

Roy Huggard.

British Columbia Medical Association

(Canadian Medical Association, Britsih Columbia Division)

President	Dr. Lavell H. Leeson
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Vice-President	Dr. W. Laishley
Honorary Secretary-Treasurer	Dr. J. C. Thomas
Immediate Past President	Dr. Ethlyn Trapp

BOARD OF DIRECTORS MEETING—JANUARY 7th, 1948

The regular meeting of the Board of Directors of The British Columbia Medical Association was held at the Georgia Hotel, Vancouver, on Wednesday, January 7th. Twenty-nine doctors attended, representative of all parts of the Province.

Many items of business were dealt with and the following is a digest of some of these.

Dr. Wm. Laishley. The President, Dr. L. H. Leeson, referred to the serious illness of our Vice-President, Dr. Wm. Laishley of Nelson. All members of The British Columbia Medical Association will extend sincerest wishes that Dr. Laishley will be restored to health.

Advisory Committee. At the Annual Meeting in September, 1947, it was decided to form a committee of past presidents of The British Columbia Medical Association, to act in an advisory capacity. The aim is to ensure that the benefit of their experience in the ramifications of organized medicine will not be lost. Action is being taken to implement this idea.

Resolution to The Royal College of Physicians and Surgeons of Canada. It will be remembered that a strongly worded resolution, re the arrangements for examination of candidates for certification with the Royal College of Canada, was presented at the Annual Meeting. Dr. J. E. Plunkett, secretary of the Royal College, has replied that the whole question is being explored, that they sympathize with the views expressed by us and will do their best to comply with our wishes.

The Manitoba Medical Association has backed our views and we hope that in a reasonable period of time it will be found possible to modify the regulations so that candidates may take their orals with less expense than is necessary now.

Penal Reform. The C.M.A. is actively interested in and associated with a very strong representative group who are dealing with the question of penal reform. Our Association has begun its study into the matter. The section of Neurology and Psychiatry of the Vancouver Medical Association has arranged a 'round table' for January 16th, 1948, at which interested parties will express their views. In addition to the doctors there will be representatives of the bar, the police and the R.C.M.P. After this meeting the problem will be presented to the District Societies. It is hoped that we in British Columbia will be able to produce some worthwhile assistance in this important study.

Adverse Criticism of C.M.A. In the December, 1947, issue of the "Canadian Doctor," pages 80 etseq., there is a stream of adverse comment directed at the Canadian Medical Association and its General Secretary, Dr. T. C. Routley. The subject of the publicity is the question of the admission to Canada of refugee doctors.

The members of the Board of Directors, present at the meeting, had not read the particular edition in question and it would appear therefore that the adverse publicity had not registered as deeply as one might assume.

Reports. Dr. Ethlyn Trapp reported on the activities of the Executive Committee of the Canadian Medical Association.

Dr. G. F. Kincade presented a report recommending quite a number of changes for our Annual Meeting of 1948. His committee was given authority to proceed and it is anticipated that the changes suggested by him will result in improved facilities all round for our next annual meeting.

The report of the Committee on Medical Education contained the up-to-date position regarding the establishment of the medical school.

Chairmen of the other committees presented interim reports.

Annuities. Some years ago the question of doctors being able to buy annuities from the Dominion Government and the money used for this purpose being exempt from Income Tax was explored. It has never been agreed to and it was felt that the whole question should be re-opened and pressed forward to a satisfactory conclusion if possible.

This, if it could be started, would be a great boon to many doctors. Appropriate action will be taken by the Committee on Medical Economics.

Specialists' Societies. A 'free for all' discussion was entered into on the formation of various specialists' societies in British Columbia. These proposed societies are separate from the B.C. Division of the national specialists' groups, and separate from the clinical division of the Vancouver Medical Association. The general feeling was that, in the main, this was not a god venture, and that it would be preferable to build up the B.C. Division of the national body in each case rather than to further divide the groups.

There is no provision, and rightly so, in the Constitution of The British Columbia Medical Association for the setting up of specialists' societies, and their value as bodies for reference purposes could not be considered final as there would be no assurance that all doctors of a particular speciality would become members.

Shaughnessy Post Graduate Course. The expenditure involved in the printing and distribution of the programme of this course was approved. The hospital authorities report that already thirty paid applications have been received and they are very pleased with the response. We join with them in the hope that this present course will be a marked success and that it will be a forerunner to courses of a similar nature in future years.

Annual Meeting—1948

The Annual Meeting of The British Columbia Medical Association will be held in Vancouver, headquarters at the Hotel Vancouver, during the week of September 27th, 1948.

POST-GRADUATE COURSE FOR GENERAL

All doctors will now have received the programme of lectures and clinics being offered by the staff of Shaughnessy Hospital, March 1st to 12th, 1948.

This is a new venture and the men who have arranged the programme deserve our thanks for undertaking what must have been a time-consuming task.

The course was offered to all doctors through The British Columbia Medical Association. The Committee on Medical Education endorsed the project and recommended that we support it in every way possible.

The programme appears to be well balanced, topical, and of high quality throughout. It is available to any doctor in god standing, at a minimum of expense and waste of travel time, and timed for the first two weeks in March it should attract a number of medical men anxious for a break after an arduous winter. It is anticipated that the maximum enrolment (for this year) of thirty will be achieved easily.

We wish it all success for its first year and may we express a hope that it will be the first of many annual Post Graduate courses, of a similar nature, in this Province.

AMBULATION OR REST FOR THE POST-OPERATIVE PATIENT?

By FRANK B. THOMSON, M.D., Vancouver, B.C.

The Bulletin takes pleasure in publishing this paper, submitted by Dr. F. B. Thomson of Vancouver. It is an attempt to evaluate impartically the relative advantages and disadvantages of early ambulation and sets forth the latter in full, where these are frequently overlooked by the more earnest advocates of the procedure. For this reason, the paper deserves consideration.—Ed.

Rest became an accepted principle of postoperative care because it was thought to facilitate the healing of wounds. It is of great interest that is should be falling into disrepute at a time when many another so called surgical principle is being discarded or having its face lifted as the result of recent advances in medical knowledge and surgical technique. It is true that, in the presence of poor surgical technique, postoperative rest will reduce the number of disastrous complications in the healing of abdominal incisions. No doubt it was poor surgical technique that resulted in the high incidence of evisceration that accompanied the first widespread trial of postoperative ambulation in Europe and America between 1905 and 1915.

Modern surgical teaching by stressing gentleness, fine suture material, accurate haemostasis and the avoidance of mass ligature and tension has greatly reduced the amount of dead tissue, free blood, and bacterial contamination in the operative wound. I believe that this is the fundamental advance which has resulted in the many reports of normal wound healing in the presence of postoperative ambulation. There has been no increase in the incidence of wound infection, haematoma, evisceration, incisional, or recurrent hernia. It would seem to be a general experience that ambulation in no way interferes with normal celiotomy wound healing. Since 1944 I have had the opportunity to treat and observe a group of 173 public ward and service patients with celiotomy wounds. Early postoperative ambulation was practiced in every case with no demonstrable adverse influence on wound healing.

Many of the physical, psychological and economic complications arising during the postoperative period have been attributed to enforced bed rest. The physical complications are numerous, frequent, and at times very serious. From the insignificant "gas pain," to fatal pulmonary embolism, they have plagued the surgeon relentlessly, each seeming to demand its accepted percentage of postoperative patients whenever a sufficiently large number of cases have been studied. The influence of ambulation on the incidence of these physical complications is as follows:

(1) *Abdominal Cramps*: These are less frequent and less severe. Very little is heard about "gas pains" on a ward where early ambulation is the rule.

(2) *Wound Pain*. While undoubtedly moderate the first few times out of bed it is never as severe as anticipated, and is not sufficient to discourage further ambulation. After the second or third postoperative day pain in the wound is surprisingly reduced in the ambulant celiotomy patient, and wound tenderness is reduced throughout the convalescent period.

(3) *Bladder Dysfunction*. Ambulation almost eliminates the need for postoperative catheterization, even after pelvic procedures fewer catheterizations are required. It follows that urinary tract infections secondary to urinary stasis or catheterization are less frequent.

(4) *Constipation*: Ambulation, allowing as it does the use of the bathroom instead of the bed pan, usually results in spontaneous defaecation by the fourth postoperative day. Enemata are rarely needed and repeated enemata almost never.

(5) *Asthenia*: The weakness of the lower extremities, loss of appetite, and weight loss so characteristic of prolonged postoperative bed rest are largely eliminated by ambulation. Muscle tone is maintained, muscle wasting prevented, and a normal diet is requested and tolerated much earlier in the postoperative period. This is particularly im-

portant to the elderly postoperative patient, whose preoperative strength is non too great without having it considerably reduced by disuse atrophy.

(6) *Pulmonary Atelectasis*; Powers and Leithauser demonstrated the return of vital capacity to the preoperative level in four days with ambulation, as compared to ten days with bed rest. Blodgett and Beattie of Boston studied 443 cases and reported that ambulation only reduced the incidence of atelectasis from 6.3% to 4.6%. Similar results have been reported by several others. It would seem therefore that, while ambulation is beneficial, it is far from the complete answer to the prevntion of postoperative pulmonary atelectasis. It should be emphasized however that, because atelectasis occurs during the very early postoperative period, the earlier ambulation is started the more beneficial it is in this regard. After the first postoperative day the influence on the incidence of atelectasis is negligible.

(7) *Phlebothrombosis and Pulmonary Embolisms*: Despite expectations to the contrary, careful clinical studies by several authorities have failed to show a reduction in postoperative venous thrombosis by ambulation. No adequate explanation has been given but it seems likely that the bed exercises of the lower extremities now routinely carried out, are as efficient as ambulation in the prevention of this complication. As for pulmonary embolism there has not been a sufficiently large well controlled series of patients studied to give a statistically significant answer as to the influence of ambulation on the incidence of this comparatively infrequent complication. Despite favourable clinical impressions the final answer remains to be determined.

Ambulation not only decreases the physical postoperative complications but also improves the postoperative psychological reaction. The morale on a surgical ward is surprisingly improved by early ambulation. The partial or complete elimination of "gas pains," catheterization, enemata, bed pans, and asthenia, together with release from the boredom of prolonged bed rest and shorter period of hospitalization, all result in a happier postoperative patient. Patients that have been kept in bed after a previous operation are impressed by the smooth and more rapid convalescence that early ambulation affords, and those that may require further operative proceedures are less apprehensive and more cooperative. It must be admitted however that the occasional patient with a low pain threshold will resist ambulation on the first postoperative day and will resent being forced to do so. It is better not to ambulate these patients too early and to instruct them to get up just as soon as they feel they can. Most of these will be ambulant by the third or fourth postoperative day. Unfortunately in private practice what the patient likes sometimes takes precedence over what is best for him.

Finally what are the advantages of early ambulation from the economic standpoint? The decrease in hospital days not only saves the patient money but also increases the patient turnover of the hospital, and so improves the services of the hospital to the community and helps overcome hospital bed shortage. Doctors are able to get more patients into the hospital and the resident interne staff sees a greater number of cases. Less nursing care is required; an important factor at least for the present. Patient, hospital, community and doctors all benefit from early postoperative ambulation.

Despite the advantages of ambulation and its value in most cases there are certain contriandications to its use. These are severe bacterial peritonitis, severe debility, serious haemorrhage, recent coronary theombosis and non-fatal pulmonary embolism. Fever, pulmonary atelectasis, a draining sinus, or a draining wound if supported, are not necessarily contraindications to early ambulation. Consideration should be given each case before early ambulation is ordered.

In conclusion therefore early postoperative ambulation allows normal wound healing, overcomes many of the physical, mental and economic disadvantages of postoperative bed rest and, when wisely practiced, has no serious disadvantages. Ambulation or rest for the postoperative patient? I believe that early postoperative ambulation, as a result of improved surgical technique, is, and will continue to be, an accepted surgical principle.

VANCOUVER MEDICAL ASSOCIATION
(SYMPOSIUM ON PERIPHERAL VASCULAR DISEASE)

OBSTRUCTIVE VASCULAR DISEASE

By T. R. SARJEANT, F.R.C.S. (Eng.)

For the clinical discussion of peripheral arterial disease we decided to deal with the subject under the two main headings of: A. Obstructive Vascular Disease, and B. Spastic Vascular Disease. The reason for this division is that each group involves different physiological principles, and therefore the treatment of each group is different.

The Obstructive Vascular Diseases are:

(1) Arteriosclerosis, obliterans of senile or diabetic origin.
(2) End-stage of Buerger's Disease.
(3) Embolism and Thrombosis.

Senile arteriosclerosis is obviously the result of the effects of advancing age and hence its final stage of obstruction and gangrene is commonly seen between the ages of 60 and 70. On the other hand, diabetes is in time invariably accompanied by arteriosclerosis and the results of arterial occlusion must be watched for even in the middle-aged patient. At one time the common cause of death in the diabetic was coma; now, however, the commonest cause of death and morbidity is arteriosclerotic gangrene, which is usually of the wet or infected type.

Buerger's Disease, or Thrombo-Angiitis Obliterans, in its end-stage is a truly organic obstructive vascular disease, but in its earlier stages the spastic element of the disease is the predominating feature. In this respect it differs from arteriosclerosis which has no vasospasm associated with it, except in rare cases. Dr. Robertson will therefore discuss Buerger's Disease along with the other spastic disease.

Arterial occlusion in a leg or an arm by an embolus may occur at almost any age as the result of bacterial endocarditis, auricular fibrillation or the floating off of a thrombus which has formed on an area of atheromatous degeneration of the aorta or iliac arteries.

To complete the etiology of obstructive vascular disease I should mention syphilis and also the acute infectious fevers which may cause arterial thrombosis.

If the devastating calamity of gangrene and the loss of a leg is to be prevented, we must recognize obstructive arterial disease in its early stages when beneficial treatment can still be instituted. With a quick glance, anyone can recognize established gangrene, but it requires a careful history, detailed inspection, accurate examination, and a great deal of thought to recognize the early signs of a failing peripheral circulation. Too frequently there is made a wrong diagnosis of weak arches, painful bunions, arthritis or sciatica.

In the time at our disposal it is impossible to deal with all the aspects of this tremendous subject. It would be a waste of time to cover it with broad statements and generalizations. Therefore, I have chosen to discuss in detail the method of diagnosis of obstructive vascular disease as it can be practised in your office or the patient's home. To make the diagnosis is the important thing, the methods of treatment are many and varied and I will make just a few suggestions regarding them.

The symptoms of a failing peripheral circulation may be of slow evolution over a long time period, getting progressively more severe; or they may arise suddenly, or be of an intermittent charcter. The symptoms can be divided into two groups. The first includes those related to muscular function—i.e. tiring, weakness, cramping—referred to as "intermittent claudication." With rest, there is often sufficient blood supply to allow further muscular work. Claudication may involve widely different areas in a limb, depending on the degree of deficiency in blood supply to the various muscles. The second group of symptoms includes changes in sensation, particularly numbness, coldness, tingling, and actual pain, usually of a burning character. This pain may or may not be associted with activity. There is an especially characteristic pain—"rest pain"—when the patient is recumbent for a considerable time. This is probably dependent on hydro-

static factors, due largely to removal of the weight of the column of blood and the absence of compression on the vessels by musclar activity. Rest pain in arteriosclerosis is not as frequent as the pain of intermittent claudication.

Unfortunately, the symptoms of a failing peripheral circulation follow no hard and fast rules. In the early stages of the disease, pain may manifest itself in many bizarre ways, while in advanced cases, on the verge of gangrene, it may be almost entirely absent. Therefore, a differential diagnosis cannot be based on symptoms alone.

On examination of the extremities, one should look for musclar atrophy and trophic disturbances of the skin, nails and hair. The skin may be unusually dry and parchment-like in texture. It may be transparent, glossy and as thin as tissue paper. There may be an absence of hair growth on the dorsum of the toes. The toenails may be very dry, brittle and dull gray or brown in colour. They stop growing at their normal rate and they may curve inward at their distal margins.

All the main arteries of the limbs should be palpated and the characters of their pulsations and of their walls should be noted. Of course, the condition of these major arteries is only one factor in the circulatory efficiency. Absence of pulsation in one of them may be associated with no symptoms, whereas serious circulatory impairment may occur with good pulses in all of them. In other words, occlusion may be present in the digital arteries or extensive arteriolar obliteration may have occurred even in the presence of a pulsation in the dorsalis pedis artery. For this reason colour changes in the distal part of the extremity are very important signs. These may be extreme and obvious; or brought out only by careful examination in certain positions of the limb. In general, they are of two types—namely, pallor due to absence of blood, and cyanosis or rubor due to an accumulation of stagnant blood.

Frequently, plantar ischaemic pallor may be detected by observing the patient's feet while they are in a resting position on the examining table. In early cases, particularly those with palpable pulsation of the dorsalis pedis and posterior tibial arteries, plantar ischaemia if not visible in the horizontal position can be easily elicited by raising the patient's extremities to an angle of about 45 degrees. Exercising the muscles of the foot in this position will then bring out cadaveric pallor in a striking way, even in cases with a minimal degree of arterial occlusion. In the upper extremity the same test can be carried out with the hands elevated above the head.

"Dependent rubor" is a reddish plum-coloured appearance which is brought out by the dependent position, disappearing on elevation and involving chiefly the toes and distal part of the foot. In advanced cases of organic occlusion, rubor appears after a few seconds of dependency, but in the early stages it may be absent. Its diagnostic value is therefore limited. Plantar ischaemia, on the other hand, is of much greater significance because of its constant presence in all stages. It may be erroneously believed by some that rubor is characteristic only of Buerger's Disease. Though it is more commonly seen in Buerger's, it is most certainly also seen in arteriosclerosis.

Cyanosis of the extremities in organic arterial disease is usually of grave significance. It may be present in any position of the limb. It usually indicates a severe upset in the circulation, particularly when it does not disappear on pressure of the examining finger. If it is associated with unusual coldness and ischaemia it is an almost certain sign of impeinding gangrene. The cyanosis is due to thrombosis of the veins draining the part. If this colour can be made to disappear by elevation or pressure, the prognosis need not be so grave.

Decreased surface temperature is a fairly constant indication of diminished arterial circulation because the temperature of a limb is directly proportional to the amount of blood flowing through it. For routine clinical purposes, temperature differences may be easily detected by palpation. It should be remembered that objective coldness does not necessarily coincide with the subjective feeling of coldness.

The facts obtained from a careful history and physical examination as just outlined, usually suffice to establish the presence of arterial disease, and the amount of circulatory impairment can be roughly gauged.

There are a number of special tests and procedures which may be called upon to aid in confirming the diagnosis. An X-ray will show calcification of the vessels and atrophy of the bones of the feet. Arteriograms will demonstrate the patency of the main and collateral vessels. Oscillometric readings of the pulsation at different levels in a limb can be a useful investigation, because it gives information which aids in the prognosis of a limb in which a minor degree of gangrene has occurred. Intradermal saline wheals in my experience are a most useful method of determining the level of viability of a leg. The histamine flare test is another similar method.

However, all the information thus obtained proves only whether or not there is arterial disease; and when this is present, it indicates how seriously the blood supply to the part is diminished. This knowledge is important but is not sufficient for rational treatment of these diseases. It does not tell whether the circulatory impairment is due only to mechanical obliteration or arterial channels or is dependent on spasm of the arteries. In order that our treatment be based on the correct physiological principles, we must determine the presence and degree of abnormal vasoconstriction in the arterial circulation. Dr. Robertson will discuss this in detail.

DIAGNOSIS:

A provisional diagnosis of obstructive vascular disease due to arteriolsclerosis may be made when the patient is *beyond* middle age and *has had* a slowly progressing failure of peripheral circulation. This diagnosis will be *quite* probable *if*, in addition, there is only feeble pulsation or no pulsation obtained in the major arteries; if there is definite hardening of the arteries to palpation; and if the X-rays show calcification in the major arterial channels. When gangrene is present, it must be determined whether it is due to simple circulatory failure from narrowed channels; or to an embolus into a distal artery; or to a combination of diminished circulation and infection, as so often happens in diabetes. All of these distinctions can be made from the history and examination. The presence of diabetes or syphilis should be determined, of course, as soon as possible in every case of failing peripheral circulation and the necessary treatment immediately instituted.

TREAMENT:

The basic principles of treatment of Arteriosclerosis Obliterans are protection of the limb from injury, encouragement of the collateral circulation, relief of pain, healing of ulcers, and amputation when other methods of treatment fail.

The patients should be instructed in the selection of well-fitting shoes and warm stockings. Warning should be given against excessive walking or exposure to cold. The danger of ordinary trifling infections should be emphasized. Minor injuries or skin fissures due to epidermophytosis are often the starting point of an ulcer or a disastrous case of gangrene. Corns and calluses should not be pared except by a chiropodist or the physician. Surgical treatment of ingrown toenails is extremely dangerous and should not be done. It should be remembered that arteriosclerotic legs are hyposensitive, easily injured, and slow to heal, therefore hot water bottles, electric heating pads and particularly electric cradles should never be used on these patients.

The collateral circulation can be definitely encouraged by hot Sitz baths taken once or twice a day. The temperature of the water should not exceed 105 degrees F. The patient remains sitting in the tub for 10 minutes with the level of the water no higher than the hips. Buerger's postural exercises should be carried out at least two to four times a day depending on the ability of the patient to endure the slight exertion entailed. These consist of elevation of the limbs, then dependency, followed by laying horizontally, ofr about two minutes in each position. This cycle should be repeated four or five times in succession. Neither Sitz baths nor postural exercises should be used in the presence of ulceration or gangrene, of course. Short wave diathermy, the pavex boot, intermittent venous occlusion and other mechanical devices designed to improve the collateral circulation, all have their advocates, but the results are disappointing.

Smoking must be absolutely discontinued because it causes arteriolar vasoconstriction.

Alcohol, on the other hand, has a vaso-dilating effect and is also helpful for the relief of pain and for sedation. Vasodilating drugs are practically without effect in arteriosclerosis obliterans and anything which produces a profound fall in blood pressure is extremely dangerous in these patients.

The control of pain may be difficult and may tax the ingenuity of the physician. Severe intermittent claudication and rest pain are strikingly relieved by a preliminary period of bed rest for two to three weeks. Exercise should then be curtailed to just that amount necessary for the patient's occupation. Tissue extracts (such as deinsulinized pancreatic tissue) do relieve the pain of intermittent claudication, but scarcely enough to warrant their daily injection. The combination of alcohol and a barbiturate will be effective in most cases, but the opiates may be necessary in some.

Typhoid vaccine and intravenous hypertonic saline are dangerous and contraindicated in arteriosclerosis.

Theoretically, the use of heparin and dicumarol to prevent thrombosis would be ideal if they could be given over a long period of time. This is, however, impractical. But they should be used in cases of sudden arterial occlusion, both to prevent extension of the thrombus and to prevent subsequent thrombosis in arteries distal to the point of occlusion in which acute spasm has occurred.

Sympathectomy has practically no place in the treatment of arteriosclerosis, except in middle aged patients who may show a definite degree of vasospasm.

The treatment of ulcers and gangrene is too complicated to discuss this evening. Suffice it to say that the first principle in their treatment is to avoid any procedure or any type of application which may do harm.

It must be remembered that the tissues in arteriosclerosis obliterans, particularly at the base of ulcers or gangrene, where the blood supply is markedly diminished, are extremely vulnerable to injury. Attempt to prevent secondary infection, but if it develops it must be adequately drained and combatted with sulpha therapy and penicillin. The decision whether to allow a gangrenous digit to separate or to do a major amputation is based on very definite indications which must be studied in each case.

In diabetic arteriosclerosis, infection and gangrene become much more serious surgical emergencies because of the alarming rapidity with which they may spread. There was much wisdom in the old saying that in diabetic gangrene, if you are going to amputate, "do it early and do it high."

SUDDEN ARTERIAL OCCLUSION

Sudden arterial occlusion in the extremities due to an embolus is one of the most urgent medical and surgical emergencies. If it results in the loss of a limb or a life, the attending physician must hold himself responsible unless he has instituted treatment within the first hour or at least the second.

Determination of the exact cause of sudden arterial occlusion is not always easy. When disease of the heart is present, particularly if it is associated with disturbances in rhythm, sudden occlusion of the arteries can usually be attributed to an embolus from the heart. When sudden occlusion occurs in the presence of characteristic evidence of thrombo angiitis obliterans or arteriosclerosis obliterans, it can be attributed to thrombosis occurring as a part of these disease. Among other causes of thrombosis are trauma, acute infectious fevers and polycythaemia. It is when no obvious cause exists that difficulty is encountered in explaining the situation. Fortunately, the determination of the exact cause is secondary to rational treatment of the diminished blood supply resulting from occlusion.

The symptoms of sudden occlusion are variable, but in most cases are characterized by their suddenness of onset. In only about half of the cases is sudden excruciating pain the first symptom, though it is common belief that this is the most typical symptom.

Numbness, coldness and tingling are important symptoms. Loss of muscle power may be present. The chief findings are lowered surface temperature, collapsed superficial veins, pallor and the loss or diminution of reflexes, sensation and muscular strength and absence of pulsation in some of the arteries of the involved extremity in which pulsation were previously present. The absence of pulsations know or assumed to have been previously present with lowered surface temperature and pallor of an unusual degree is pathognomonic of arterial occlusion.

The localization of an embolus is usually not difficult, for we know that it lodges at a point of rapid narrowing of the arterial lumen where a large branch is given off. The commonest site in the leg is in the common femoral where the *profunda femoris* is given off; in this case the temperature and colour changes will extend up to the junction of the lower and middle thirds of the thigh. If the embolus lodges at the bifurcation of the common iliac, the changes will occur at the junction of the middle and upper thirds. If the block is in the popliteal artery, the changes will be just above the ankle. Obviously these levels conform to the collateral circulation, and the same is true in the arm.

Prior to the discovery of heparin, the results of treatment of sudden occlusion by embolus or thrombosis were disappointing, 50% of the limbs became gangrenous. The anticoagulants have altered this picture, and now many cases of embolism do not even require embolectomy. The explanation of this is that though the collateral circulation might dilate sufficiently in 6 to 8 hours to maintain the circulation, the damage done to the intima of the occluded vessels during the initial period of ischemia causes widespread thrombosis when the circulation returns. The anticoagulants prevent this secondary thrombosis.

For this reason treatment must be instituted without delay. The limb should be kept in the horizontal position, never elevated. It should be loosely wrapped in absorbent cotton or wool to preserve its natural warmth. Heating the limb increases its metabolism, thus demanding more blood. Furthermore, this type of limb is much more easily burned than a normal limb. Whiskey should be given by mouth and papaverine hydrochloride (½ grain) intravenously. If the papaverine is effective, improvement will be seen in a few minutes and if this is so, it should be repeated whenever evidence of failing circulation to the extremity is present. A paravertebral sympathetic block should be done to obtain maximum dilatation of the collaterals. Heparin should be given immediately and dicumarol started by mouth.

In a case of arterial thrombosis this treatment is simply continued and everyone hopes for the best. Anticoagulant therapy is continued for about 10 days or until definite gangrene has developed.

In a case of embolism, however, if the medical treatment does not produce rapid improvement in the circulation within 3 or 4 hours, embolectomy should be performed. Removal of the embolus is most successful if done within 6 hours of its occurrence, much less successful ofter 10 hours and useless after 24 hours.

In conclusion—I have tried to point out simple, practical methods of diagnosis and to give a few suggestions regarding the treatment of obstructive vascular disease.

Arteriosclerosis obliterans is by far the commonest peripheral vascular disease. If we are constantly on the watch for its early signs we can prevent much suffering and the loss of many limbs.

VASOSPASM

By DR. H. ROCKE ROBERTSON

Not everyone realizes that blood vessels of all types, except possibly the smallest capillaries, are capable of an intense spasm which is sufficient to prevent the flow of blood through their lumen. The pallor of the skin in response to fear and anger is an outward manifestation of this function; the cringing of a vein when one attempts to put a needle into it, is spasm; surgeons operating upon veins, such as varicose veins, have an opportunity to see a vein, dilated and filled with blood at one moment, suddenly in response to some stimulus, contract and become a cord-like structure whose lumen is virtually obliterated. Those who have had to do with the treatment of wounds will have seen cases in which major arteries have become spastic as a result of a nearby wound and they will have been impressed by the fact that this spasm can persist for many hours, indeed, until such time as the tissues to which it is carrying blood have become gangrenous. It is not surprising, therefore, to find that vasospasm plays, on occasion, a prominent part in the symptoms and signs of some of the vascular diseases. When, upon examination, one discovers that a patient has cold extremities, but that the major arterial channels are open and that these cold extremities will warm up upon exposure to warmth or following the administration of a spinal anaesthetic or some vasodilator drug, one is justified in saying that the coldness is due to spasm of the vessels. It must be appreciated that in the normal individual there is a certain tone to the vessels which can be released by exposure to warmth, spinal anaesthetics, or vasodilator drugs and one is only justified in calling this case one of vasospasm if the difference between the basal state and the state induced by vasodilatation is greater than in the normal individual. For instance, if the skin temperature of an extremity of an apparently normal individual is taken and compared with the temperature following the administration of spinal anaesthetic, it will usually be seen that there will be something in the nature of a 10°F. rise in temperature. Thus, if one is to make a diagnosis of vasospasm, one should see a greater rise than this. Table 1 shows a classification of cases in which vasospasm may be present.

TABLE ONE

PERIPHERAL VASOSPASM

1. Post traumatic
   - Acute
   - Chronic
2. Post Phlebitic.
3. Raynaud's Disease.
4. Buerger's Disease.
5. Obliterative Arteriosclerotic.

1. *Post Traumatic.*

A. *Acute.*

Not uncommonly, following an injury, there is marked vasospasm which under certain circumstances may progress to gangrene of the extremity. If the main blood vessel to the extremity is lacerated, it is not uncommon for the collateral vessels to become spastic. But even if the main vessel is not severely injured, if for example it is just contused, arteriospasm of the main channel and of the collateral channels may occur and may be severe enough to bring about gangrene of the extremity. In some instances, there is no evidence at all, upon gross examination of the vessel, of injury to the vessel itself, and yet in these cases gangrene has resulted. This type of acute and sometimes relentless vasospasm can thus occur after any type of injury, be it a penetrating wound or a non-penetrating wound, such as a blow on the extremity or a fracture. It should be realized that the spasm of the vessels may occur, not only immediately following the injury, but some time later. One has seen a case in which there was no evidence of vasospasm until

approximately twenty-four hours after the fracture had been sustained. The importance thus, of examining the peripheral circulation in any case of injury is apparent, for only if this arterial disturbance is recognized early, can it be corrected.

B. *Chronic.*

Not infrequently, following fractures, sprains or even direct blows to an extremity, the patient will complain of discomfort and cold and perhaps, pain in the extremity. The most dramatic example of this type of disorder, is the so-called Sudeck's atrophy of bones in which, usually following a fracture, there is acute pain in the region associated with coldness, sweating, and oedema. Compared to the acute type of vasospasm following injury, this chronic type of vasospasm is frequent. It is well worth bearing in mind, in any case in which the complaints at some time following an injury which may have been minor in the first instance, are more vehement than they should be. In the vast majority of cases, these symptoms will subside with ordinary physiotherapeutic measures or even with no treatment at all. But in a few, the symptoms persist for long enough or are severe enough to warrant more active intervention.

2. *Post-Phlebitis Vasospasm*

A vasospastic state following an attack of acute phlebitis is occasionally seen and is worth bearing in mind for, if it is recognized, something may be done to relieve the patient of some of the discomfort of this most distressing state. In a few cases, following phlebitis, it has been observed that one of their complaints is a coldness of the extremity and associated with this is a dull aching pain in the calf. Skin temperature observations on such a case may reveal a rise in temperature following the administration of some anti-spastic agent, such as a spinal anaesthetic or tetraethylammonium chloride. If such a finding is made, it is worthwhile to consider applying some form of therapy to overcome this spasm, and it has turned out in a few cases that considerable relief was obtained either by sympathectomy or by the therapeutic administration of tetraethylammonium chloride.

3. *Raynaud's Disease.*

Raynaud's Disease is a vasospastic disease occurring almost exclusively in women, involving the extremities, symmetrically, as a rule, characterized by the exhibition of what is known as Raynaud's phenomenon, which consists of certain changes in the extremity produced by exposure to cold or by some emotional stimulus. Characteristically, the colour changes consist of an initial stage of pallor, followed by a stage of cyanosis which gives over to a stage of lividity, following which the colour of the extremity returns to a normal pink. It should be emphasized that these very same colour changes may be seen in many conditions which are not primarily Raynaud's disease. This Raynaud's phenomenon may be found in any of the conditions listed in Table II. It is therefore of the utmost importance to determine in any case exhibiting Raynaud's phenomenon whether or not there is any primary condition. If careful history taking and physical examination establish that there is no primary condition, then a diagnosis of Raynaud's disease may be considered. The criteria for making a diagnosis of Raynaud's disease may be summarized briefly as follows:

1. Episodes of Raynaud's phenomenon excited by cold or emotion.
2. Bilaterality of the Raynaud's phenomenon.
3. Absence of gangrene or if present, its limitation to minimal grades of cutaneous gangrene.
4. Absence of any other primary disease which might be causal, such as occlusive arterial disease, cervical rib or organic disease of the nervous system.
5. History of symptoms being noticed for two years or longer.

The important point in making the diagnosis is the elimination of any possible

secondary factors which, within reason, may be the cause of the Raynaud's phenomenon. It should be remembered that secondary Raynaud's phemonenon is much more common than Raynaud's disease.

4. *Buerger's Disease.*

Buerger's disease or thrombo-angiitis obliterans is a segmental, inflammatory, obliteraative disease of the arteries and veins which occurs almost exclusively in young men, involves the extremities, and rarely the viscera, also, and produces ischaemia of tissues and frequently gangrene. This disease which was, at one time, thought to occur only amongst members of the Jewish race is now known to occur in all races, but is slightly more common among Jews. The disease usually commences in persons who are between the ages of 25 and 40 years. It may arise at an earlier age, but very rarely does it make its first appearance after the age of 50. Very rarely does it occur in females. Of the etiology, little or nothing is known. It has not been possible to reproduce the disease regularly in animals by any means. One fact, however, seems to stand out and that is that there is some relationship between a sensitivity to tobacco and the development of Buerger's disease for while the disease has been known to occur in non-smokers, nearly all these patients are found to be heavy smokers. Further evidence in favour of the theory of relationship between tobacco and the disease is the almost universal observation that if a patient who has Buerger's disease discontinues smoking his disease will be arrested, while if he should continue to smoke, his disease will in all likelihood progress. Silbert writes that in his personal experience with some 1,100 cases he has never seen a case fail to become arrested once smoking is discontinued, but that in those patients who have continued to smoke the disease has continued relentlessly. The outstanding symptom of the disease is pain, and there are several types of pain; the intermittent claudication, which is perhaps the commonest complaint and may be described as an ache or a dull pain in the arch of the foot or the calf of the leg coming on during exercise. This pain is usually relieved when exercise stops and will return again when activity is resumed. Another type of pain is the rest pain which is usually localized in the digits or adjacent regions. It is usually described as a severe ache or a numb gnawing type of pain which may last for hours and is usually most severe at night. This is an evidence of severe ischaemia and may be a premonitory sign of ulceration or gangrene. Thirdly, there may be the pain of ischaemic neuritis which usually occurs late in the course of thrombo-angiitis obliterans and usually only if the disease is extensive and has included large arteries. It is apparently due to the involvement in scar tissue of the nerve lying alongside the blood vessels. The pain is dull in character and is usually associated with various paresthesias. Fourthly, there is the pain of ulceration and gangrene resulting from local swelling, inflammation, necrosis of tissue and irritation and death of segments of small sensory nerves. Fifthly, the pain of osteoporosis and atrophy, occurring in the foot after long disuse and after prolonged arterial insufficiency. The pain is associated with weight bearing or pressure. Finally, there is the pain from inflammatory lesions of the blood vessels. The lesions of superficial thrombophlebitis, which occurr in thrombo-angiitis obliterans, are usually painful, but the pain is not severe. It may be aggravated by use of the limb or any pressure on the lesion. It usually persists until the lesion involutes, that is from seven to fourteen days and then gradually disappears. Superficial phlebitis occurs in some 40% of cases of Buerger's disease. It is said by some that superficial phlebitis developing in a young male is an indication that Buerger's disease is present or will declare itself before long. This is probably an exaggeration and while it is important to consider the possibility in any case of superficial phlebitis in such a person, it does not seem that the presence of this common condition is necessarily to be associated with Buerger's disease.

The clinical course of the disease is variable. Four main types are described, in the first, there is the non-progressive type of clinical course. After one or two episodes of arterial occlusion, further episodes fail to develop and a period of circulatory compensation follows which may last for many years or even the remainder of the pa-

tient's life. The second type, a slowly progressive type of clinical course is seen most commonly. Episodes of mild to moderate arterial occlusion occur for a number of years. Between episodes are quiescent periods of weeks to months, even a few years, when there is some improvement in the circulation as a result of the development of arterial anastomoses. However, as time goes on, the arterial circulation of the extremities becomes more and more seriously impaired. Finally, active lesions may cease to develop and then the patient is left with a moderately severely damaged arterial tree. Owing to this variable course in this common type, it is most difficult to assess the value of any particular form of treatment. The third type is that in which there is a sudden arterial occlusion of large vessels. In these cases, the damage produced by the sudden occlusion may be severe and result in extensive gangrene. Even if gangrene does not develop, recovery is slow and severe arterial insufficiency usually persists. Finally, the last and, fortunately, the least common type of case is that in which there is a fulminating progressive type of clinical course in which, within a period of months, a person may go on to develop gangrene of several extremities. It is important to repeat that the lesions in Buerger's disease are organic and produce permanent arterial occlusion. The involved vessels never resume normal function either, spontaneously or as a result of treatment. The disease is episodic. It is characterized by exacerbation and periods of quiescence, and there is some tendency to slow improvement of symptoms and manifestations between episodes. The lesions are segmental. They may be extensive or they may occur only in isolated short segments. Much of the arterial tree may be left intact and essentially normal. There are many arterial anastomoses in the extremities which can be developed and enlarged to carry blood around the segmental occlusion. The course of the disease thus depends, to some extent, on the rapidity of development and extent of the arterial lesion versus the development and capacity of blood flow in the anastomosing artery. In most cases the disease ultimately become inactive and episodes of new occlusion finally cease. The tendency towards self limitation is the hopeful aspect of the disease and the basis for persistent conservation treatment. Intimately associated with the organic changes in this condition is the vasospastic element which is invariably present in the earlier stages. Proper management of this element may result in the saving of a limb from gangrene or the alleviation of troublesome symptoms.

5. The final group is that in which vasospasm is associated with obliterative arteriosclerotic disease. Strangely enough, in some individuals in the older age group who have established obliterative arterial disease, there is an element of vasospasm which may be important. It is often difficult to establish from the history whether or not there is any vasospasm, for the complaints of the obliterative disease, namely, the coldness of the foot and the intermittent claudication can always be well explained by the arterial obstruction. Sometimes, however, and particularly in persons who develop the disease at an earlier age than is customary, it is worthwhile to investigate the arterial supply to the extremity by means of skin temperature, and in a few instances it may be found that there is a considerable vasospastic element. Such cases may be improved by such procedures as sympathectomy, but this procedure cannot be advised with any degree of assurance. We have seen several cases within the past year in whom there appeared to be a considerable degree of vasospasm. The dramatic relief in their symptoms following sympathectomy was, however, short-lived and within the space of a few weeks their original pain had returned in its full intensity. Nevertheless, there are numerous cases now reported in the literature of long-lasting results following sympathectomy in this group.

It is not the purpose of this discussion to enter into a description of the treatment of these various disorders, but it might be worthwhile to review the methods that can be used to overcome spasm of the vessels.

1. The application of heat.

   This time-honoured method still has a prominent place in the treatment of vasospasm of any origin. It has long been observed that the application of heat, either

to the involved extremity or to the other parts of the body usually results in a relaxation of the spasm. This observation is used routinely in the management of these cases. In the acute cases, the application of hot water bottles, heat cradles and so on may be sufficient to overcome the spasm in any extremity. In the chronic case the patient is advised to dress himself warmly and to avoid exposure to cold.

2. Artificially produced fever.

   The use of typhoid vaccine and other forms of foreign protein therapy has been widespread and has a considerable value. If finds, perhaps, its best place in the management of Buerger's disease or recurrent thrombophlebitis which may be a manifestation of Buerger's disease.

3. Use of Drugs.

   Many drugs have been used in arterial disease. Papaverin is of use in the acute spastic condition following arterial occlusion or pulmonary embolism. It is administered intravenously in a dose of 1/3 to 2/3 of a grain. It has little or no effect in chronic arterial disease. Mecholyl is supposed to have a vasodilator effect. Theobromin and theophyllin have been used considerably, but appear to be valueless for improving peripheral arterial circulation. Alcohol has a real place in the management of many of the vascular conditions. It is well established, physiologically, that alcohol produces a peripheral vasodilatation, and clinically it has been very frequently observed that it is as useful as anything in controlling the pain of ischemia. Finally, a new drug, tetraethylammonium chloride, should be mentioned, and this substance, which has been used clinically only for about three years, has excited considerable interest because of its property of paralyzing to a considerable extent sympathetic activity in the body. It has been stated that its effects upon skin temperature are comparable in every way to the effects of spinal anaesthesia or local nerve block. If this is true, then the value of the drug, which appears to be non-toxic and without danger, would be very great. Our experience with the drug is limited to a small number of cases, namely; 43 observed over a fairly short period of time, some 7 months. We have not been able to confirm the findings of others in several regards. In our cases there has been no correlation between the skin temperature response to spinal anaesthesia and the response to tetratethylammonium chloride. For instance, of 16 cases showing a good temperature response to spinal anaesthesia, that is a rise in skin temperature of 10°F. or more, 7 showed a good response to tetraethylammonium chloride and 9 showed little or no response to tetraethylammonium chloride. In only 1 case was there a good response to the drug and a poor response to spinal anaesthesia. In other words, so far as we can determine, the response of skin temperature to spinal anaesthesia is much greater than the response to tetraethylammonium chloride in the majority of cases. The clinical effect of the drug has been interesting. This effect has been noted in 37 cases with various types of diseases. In 18 of these cases improvement in their clinical condition was noted and in only 7 of these was there a marked response in skin temperature observed. 19 of the cases noticed no effect on the drug upon their symptoms and in 6 of these the skin temperature response was recorded as good. 23 of our patients have had conditions which we thought might be benefited by the use of this drug. In 16 of them an apparently beneficial effect was obtained. In the remainder little or no effect was noted. We are obviously not in a position to state just what place this drug will take in the management of peripheral vascular disease. It would seem, however, that its effects are sufficiently great to warrant its use in certain situations, which might be:

(1) Any acute arterial lesion, such as arterial thrombosis, or embolus or arterial injury.

(2) Any acute phlebitis. In a small experience of the use of this drug in phlebitis ,it has had some remarkable good effects.

(3) In cases exhibiting minor and chronic vasospastic disorders. We have had experience with two cases now in which the drug, when used over a period of time, has had an apparently beneficial effect.

(4) As a diagnostic procedure to determine the presence or absence of vasospasm. Our experience to date, however, would indicate that this method is not as accurate as is spinal anaesthesia.

4. Interruption of the nervous pathways, by:

A. Paravertebral block. The injection of novocaine into the region of the paravertebral sympathetic chain has been widely used, and usually results in a temporary inhibition of sympathetic stimuli. This method has a place in the management of both the acute and chronic forms of vascular disease. Its position may be taken by tetraethylammonium chloride or some similar drug.

B. Sympathectomy. An operation, which, if completely performed, will abolish vasospasm in the extremity involved. It has been widely used in the management of all types of disease characterized by vasospasm and in some its effects have been dramatic and lasting. The indications for sympathectomy are difficult to define accurately, but in general terms might be described as follows: Sympathectomy should be considered in any condition which produces a real disability and in which it appears that the disability is in large part due to vasospasm which cannot be controlled adequately by non-operative measures.

PERIPHERAL VASCULAR DISEASE

Classification and Pathology

By DR. H. H. PITTS

The subject matter of this symposum dealing with peripheral vascular disease has been divided into four groups:- (A) Classification of peripheral vascular disease and surgical pathology of the various types; (B) Obstructive vascular disease; (C) Spastic vasuclar disease and (D) Phlebitis, pulmonary embolism, etc., my particular role dealing with the classification, etc., and therefore I will attempt to deal with it under the three last named headings only, disregarding such lesions as specific luetic and rheumatic arteritis and periarterities nodosa, the first two at any rate being less likely to involve peripheral vessels while the latter may involve both peripheral and visceral the peripheral however, less frequently.

A fairly convenient method of classifying arterial lesions follows:- (A) Non-specific acute arteritis and specific arteritis (luetic and rheumatic; (B) Obliterative or obstructive including Thromboangiitis obliterans or Buerger's disease and endarteritis obliterans; (C) the Degenerative or arteriosclerotic group—atherosclerosis, Monckeberg's medial sclerosis; diffuse hyperplastic sclerosis and (D) the Angiospastic group with Raynaud's disease as the prototype.

As previously stated Group A of this classification will be by-passed for the purpose of this presentation and we will pass on to Group B the obstructive lesions. Thromboangiitis was originally thought to be a degenerative process with resultant thickening of the intima somewhat akin to arteriosclerosis until Buerger, by whose name it is so frequently known, recognized its inflammatory nature. A definite aetiological factor is uncertain but bacteria of the non-haemolytic streptococcal group have been cultured from the blood of these patients and portions of diseased vessels embedded close to the femoral vessels in rabbits, produce changes in these femoral arteries and veins identical with the lesions of Buerger's disease. However the bacterial origin is still inconclusive.

The time honored or dishonored role of tobacco, especially in the form of cigarettes,

is considered a very probable factor, probably as an allergic manifestation, not to the nicotine itself, but to tobacco proteins.

Sulzberger showed by the use of patch tests with denicotinized tobacco extracts that while only 16% of healthy non-smokers and 36% of healthy smokers were allergic 78% of patients with Buerger's disease were. It is a known fact that allergic reactions are capable of producing necrosis such as caseation in tuberculosis, this resulting in an aseptic inflammation such as the lesion in Buerger's is.

There is an almost exclusive predilection for the male although a few reports of Buerger's in the female have recently been published. Young Russian and Polish Jews are more commonly affected but Gentiles are no exception. Young adult life is the most frequent period which fact rather substantiates the inflammatory as against the degenerative nature of the process. The lower extremities are almost always involved but on rare occasions the upper.

The acute stage is rather suggestive of a more or less migrating phlebitis in the more superficial veins of the legs due to infiltration of the arterial and venous walls by polymorphs, the perivascular tissues being similarly infiltrated. Thrombosis occurs at this site which may be a fairly long or relatively short portion of the vessel. This thrombus gradually becomes organized, recanalized and larger and smaller endothelial-lined canaliculi eventually communicate with the main channels proximal and distal to the organized thrombus. This recanalization is apparently insufficient and other collateral branches become thrombosed with the final picture of gangrene, trophic ulcers, etc. In addition the adjacent veins are involved, the perivascular infiltrate undergoes more or less organization so that ultimately a neurovascular fibrosis occurs involving the nerve, artery and vein in a mantle of fibrous tissue. Medial calcification is never seen in true Buerger's disease.

Intermittent claudication, one of the hallmarks of the symptomatology of Buerger's disease, was first used by a veterinarian named Bouley in 1831 to describe a condition of limping in the horse, developing after a short period of exercise and recovering rapidly with rest, only to recur again on repetition of the exercise, and it was found to be due to an obliterative disease in the main artery of the leg. It and the cramp-like pains that occur in the leg muscles are due to the ischaemia of these muscles incident to the narrowing of the lumina of the nutrient arteries. The erythralgia of the foot in the dependent position and its abnormal blanching on elevation are due to loss of vasomotor control resulting from the perineurovascular fibrosis. The loss of pulsation in the distal vessels is, of course, the result of the occlusion of the lumen by thrombus formation. The advent of gangrene may be long delayed and this appears to be dependent on the degree of involvement of the collateral circulation and recanalization of the thrombus. Lewis paints a concise verbal picture of the process in his monograph as follows:- 'The affection of the arteries in advanced cases is remarkable for its extensiveness, involving the main artery and all its chief branches throughout the leg. It is the rule for the arteries of both legs to be involved, though to different extents; the arteries of the arms are frequently affected too, but generally in less degree; the visceral arteries participate rarely. There is no other disease which brings such universal ruin to the large and small arteries of a limb; and there is none in which the extent of vascular obliteration is so disproportionate to the symptoms, or in which the obliteration of all the chief arteries of the leg may persist for many years without gangrene of toes threatening. This is due to the gradualness of the process and to the development of an extraordinary meshwork of impalpably small artterial anastomoses in which the blood now flows throughout the length of the limb.'

Endarteritis obliterans is also included in the obstructing or obliterating lesions of the arteries but is seen chiefly in the visceral vessels—ovaries, breasts, uterus—probably as an involuntary or physiological atrophic change incident to advancing age and also frequently in the walls of chronic gastric ulcers and here one wonders whether or not this is cause or effect. At any rate it appears to be neither a degenerative nor an inflam-

matory change and is characterized by marked thickening of the intima with narrowing or obliteration of the lumen but without calcification or thrombus formation.

ARTERIOSCLEROSIS

The term conveys to most of us the mental picture of sclerotic, cord-like, tortuous arteries with varying degrees of calcification and intimal thickening and ulceration, but actually it is by way of being a blanket term covering atherosclerosis, Monckeberg's medial sclerosis and diffuse hyperplastic sclerosis. Atherosclerosis is concerned with the larger arterial trunks, the aorta and its larger branches and the cerebral and coronary arteries, diffuse hyperplastic sclerosis with the renal interlobular arteries and efferent arterioles of the glomeruli, and since we are concerned here only with peripheral vascular lesions we will deal only with medial sclerosis which involves chiefly the muscular branches and arteries of the extremities, although in old age typical examples are seen in the arteries of the uterus and ovaries, probably as an additional degenerative process superimposed on the involuntary endarteritis obliterans.

Medial sclerosis is almost without exception a condition met with only in the older age group and is a purely degenerative process in which the media or muscularis undergoes fatty degeneration with deposition of lime salts to a greater or lesser degree either in a parchy or fairly diffuse fashion with thickening, tortuosity and nondularity of the vessel wall. The intima may be compressed toward the lumina in wrinkled folds, thus narrowing its diameter although its histological and biochemical structure may not be altered. However atheromatous change may supervene with marked thickening and degenerative change in the intima and subsequent thrombosis, this being a frequent accompaniment of diabetes and of senility without concurrent diabetes, usually in a somewhat younger age group in the former. The frequency of thrombosis and gangrene in elderly diabetics, chiefly males, may be due to some effect of the high sugar content of the blood on the probably already sub-par intima overlying the calcific plaques, the hyperglycaemic blood also furnishing an excellent medium for secondary bacterial invasion.

There is, in general, a longer interval between the probable initiation of well advanced arteriosclerosis and the first symptomatology as compared with Buerger's. Intermittent claudication is frequent but probably not as severe; there may be sensations of numbness and coldness in the toes or feet, and varying degrees of discoloration apparently due to faulty nutrition of the part by the diminished blood supply. Then too, the age of the patient is a further link in forging the chain of diagnostic data.

In the April, 1947 issue of the Canadian Medical Association Journal Luke of Montreal presented a most interesting report of 25 cases of obliterative arterial disease from the Vascular Service of the Royal Victoria Hospital, 6 being true early arteriosclerotics, 4 Buerger's and 4 from miscellaneous causes. The remaining 11 with no clinical or laboratory evidence of arteriosclerosis showed no histological evidence of Buerger's disease in the biopsy sections of the dorsalis pedis arteries. All were males between 43 and 62 years of age whose chief complaint was intermittent claudication gradually increasing over a two year period. All were moderate to heavy smokers; 5 had unilateral leg involvement, 6 bilateral. Dorsalis pedis, posterior tibial and popliteal artery pulsations were absent and all showed a moderate to good response to procaine lumbar sympathetic block and were consequently treated by lumbar sympathectomy. The biopsies of the dorsalis pedis arteries showed a chronic degenerative change that was not typical of arteriosclerosis of the medial or intimal type and the inflammatory changes of Buerger's were lacking. He contends that this change may be due to an unrecognized thrombus higher in the vascular tree and that it is actually a very early manifestation of what would eventually develop into a true arteriosclerotic process. He also strongly advocates arterial biopsy both as a diagnostic measure and in assessing possible benefits from sympathectomy.

Mention should be made here, merely in passing, of gangrene or at least impending gangrene, in sudden arterial embolic occlusion resulting from three main causes:- thrombi detached from valvular lesions of aortic or mitral valves; from areas of thrombus formation on the basis of atherosclerotic ulcers probably most common in the abdominal aorta

and from mural thrombosis in recent cardiac infarcts chiefly involving the left ventricle. The results are dependent on the vessels involved, condition of the parts supplied prior to the vascular occlusion and the degree of collateral circulation.

SPASTIC VASCULAR DISEASE

The term Raynaud's disease or phenomenon implies a process characterized by intermittent spasms of the digital arteries with or without local nutritional change varying from discoloration to gangrene. It is more common in women usually of a thin type in the 20 and 30 year age periods. The fingers are most frequently involved but the thumb may be included and indeed the whole hand even on rare occasions involving the wrist and lower forearm. There is a familial tendency and symmetrical involvement. The toes and feet may occasionally be involved.

Exposure to cold either of weather or bathing in cold water provokes the attack which is characterized by loss or diminution of circulation to the fingers or toes which assume a white to greyish and finally waxy appearance which, after about 30 minutes, become numb. As the attack wanes the fingers gradually become redder from base to tips usually deeper than normal, even cyanotic and accompanied by tingling. There may be gradual atrophy of the tissues, the fingers become thinner than normal, the skin covering them tighter and tense, and the bone of the phalanges may undergo rarefaction in the long standing cases. Ultimate necrosis and gangrene of small areas may occur with the tips of the fingers becoming gangrenous. Occasionally a diffuse scleroderma may occur. Some disturbance in the vasomotor mechanism due to some undetermined cause, is thought to be responsible for the condition and no pathological changes such as medial or intimal are apparent. It would also appear that Raynaud's disease exemplifies the physiological principle that dilatation occurs in arterioles in spasm distal to the point of constriction and this primary ischaemia followed by the engorgement of the dilated arterioles and consequent sluggish circulation leads to the dry gangrene.

DISEASE OF VEINS

For the purpose of this symposium inflammatory diseases—phlebitis (suppurative and non-suppurative); primary idiopathic thrombophlebitis; phlebosclerosis, phlebothrombosis and pulmonary embolism will be considered.

Suppurative phlebitis is seen most frequently in veins adjacent to areas of extensive inflammatory processes such as ulcers, abscesses and cellulitis, where the relatively thin venous wall is infiltrated by the pyogenic bacteria while the thicker arterial wall will remain impervious. Generally speaking thrombosis occurs at this site and thus a thrombophlebitis ensues. This may become septic depending on the invading organism, undergo disintegration with the formation of multiples septic emboli with resultant pyaemia;—pylephebitis, lateral sinus thrombosis, thrombophlebitis of the facial veins from furuncles or carbuncles of nose and upper lip are a few examples.

Non-septic phlebitis where no evidence of any inflammatory process is apparent, occurs at times in scarlet fever, typhoid and pneumonia, and also following surgical ligation of veins, with gradually organizing thrombus formation distal to the point of ligation.

Primary idiopathic thrombophlebitis or thrombophlebitis migrans occurs in apparently healthy persons and is characterized by recurring attacks of thrombophlebitis in short segments of veins in rather widely separated areas of the venous system usually in young or middle aged men and apparently due to a low grade phlebitis in the small and medium sized veins and somewhat similar to that seen in the early stages of Buerger's.

PHLEBOSCLEROSIS

This is apparently a degenerative process occuring in young males 20-30 years of age, affecting the superficial and deep veins of the legs chiefly, bilaterally. The vein wall is thickened by a marked fibrosis in the media with atrophy of the muscle fibres, loss of the endothelial lining of the intima and increased fibrosis of this coat, the lumen

being correspondingly narrowed the veins having a firm, whipcord-like feel to palpatation.

THROMBOPHLEBITIS AND PHLEBOTHROMBOSIS

Thrombosis results from three main causes:- (1) phlebitis; (2) trauma to the vein wall and (3) slowing or stagnation of the venous stream, and while it does occur in the arterial lumina it is commonest in veins. In thrombophlebitis the thrombus becomes adherent to the intima by inflammatory reaction, while in venous thrombosis or phlebothrombosis, which is the usual precursor of pulmonary emboli, either post-operative or in ordinarily well persons who are suddenly confined to bed for either medical or surgical therapy, here no inflammatory process accompanies or causes the thrombus formation, consequently it is much less adherent and perforce more readily detached in toto or in piecemeal.

Frykholm and his associates have shown that the most frequent initial sites of venous thrombosis are—(1) plantar veins; (2) veins of calf muscles; (3) veins of adductor muscles and visceral pelvic veins. The plantar veins are apparently more frequently involved in young persons and in all ages, in Frykholm's material, the calf and adductor muscle veins were predominantly affected. It would appear that three factors are essential in the production of thrombus formation:- slowing of the venous stream, change in the chemical composition of the blood and injury to the intima. This latter factor may, of itself, cause thrombosis. Post-operatively there may be both stasis and variation in the composition of the blood from normal, which, however, does not satisfactorily explain why thrombosis occurs at these previously mentioned sites. In Frykholm's opinion injury to the intima of the vein is of paramount importance, since (because of the collapse of the veins, with the patient recumbent, by pressure of mattress on calves, thigh against thigh, absence of the long column of venous blod from the plantar veins(there is apposition of intima to intima and probable changes in the lining endothelium from pressure and deficient nourishment from the blood usually bathig it, with consequent release of thromboplastic substances which, acting in a favorable setting of sluggish blood flow gradually produce a thrombus. This is apparently of the red or coagulation type in which red and white blood cells and platelets are enmeshed in a fibrin matrix which is gradually built up and extends proximally and on reaching a large vein, as for example the femoral where blood flow is accelerated, platelets are deposited on its surface producing a white thrombus—this being the least firmly attached portion is most likely to become detached and floating free in the venous stream ultimately finds its destiny in the larger or smaller radicles of the pulmonary artery. If the main pulmonary artery is occluded or a primary branch, the whole lung or one or more lobes are deprived of their blood supply and acute diffuse oedema without infarction occurs with a mortality of approximately 90%. If moderate in size it will pass through the pulmonary artery and the primary branches to occlude secondary branches producing a haemorrhagic infarct with a mortality of 15-20%. Very small emboli pass on into the tertiary or even smaller branches and, while productive of the usual characteristic symptoms may present very little clinical evidence of their presence, and the mortality rate is practically negligible unless as the proverbial added straw to other pathological processes.

It is generally conceded that the most likely period for pulmonary emboli is during the second and third weeks of convalescence and as Boyd states in his Surgical Pathology—'A useful generalization regarding the onset of complications after trauma is as follows:- Shock—3 hours; Fat emboli—3 days; Pulmonary embolus—3 weeks. However it may occur during the first 2 or 3 days. There is one sign which may put the surgeon on his guard—the second week of convalescence should be afebrile, but cases which are going to develop infarction show a slight but persistent rise of evening temperature during that period.'

Henderson found that medical patients dying of pulmonary embolus were, on the average, older than the surgical patients, were overweight and had either a normal or subnormal blood pressure.

I fear I have been overlong in setting the stage for the more important therapeutic phase of this symposium for which I hasten to apologize and make my exit.

PHLEBITIS

(DR. R. HUGGARD)

There is some considerable responsibility in introducing the subject of phlebitis for discussion. During the last several years the literature has been more than full of information and analyses concerning this most troublesome disease.

In a recent series of 25,000 necropsies done at the University of Minnesota over a period of 19 years, it was most remarkable that in 107 of the cases embolism had already occured, or a thrombus had occurred which could have produced a fatal embolism. Venous clotting is of importance, not only because of the symptoms that are associated with certain varieties, but because of the possible sequelæ such as continuing symptoms subsequent to thrombus or even the occurrence of death from pulmonary embolism. There is a wide confusion of thought upon the subject, and it is with that point in view that we have considered it fitting and proper to deal with a few controversial ideas. There may be many who do not agree with this suggested line of thought, but at any event if there is disagreement, at least there is an orderly system upon which these cases can be classed.

I do not believe that one can consider all venous thrombosis the same type of lesion, but that it is necessary to differentiate between two types which are different etiologically, symptomatically, prognostically and therapeutically. It is upon the differentiation of these two types that satisfactory therapy is dependent. It was emphasized eight or nine years ago by DeBakey and Ochsner that the importance of this differentiation between the true inflammatory thrombosis on the one hand and the simple thrombus on the other occurring where there is no inflammatory lesion, was the most important feature in management. They emphasized that consideration must be given to phlebothrombosis which may be defined as a partial or complete venous occlusion by an intravascular clot unassociated with inflammation, and may I emphasize the following point—that the clot is loosely attached to the vein wall. Many subsequent publications have emphasized this condition and indeed most are inclined to accept this as being true. However, there are many who do not agree. Jensen does not think that phlebothrombosis occurs except after trauma and is only to be found in incised or lacerated wounds. On the other hand, Hunter and his associates believe that true phlebothrombosis and thrombophlebitis are two distinct entities. This conclusion of theirs is based upon a very comprehensive investigation, namely 350 unselected autopsies of which in 209 instances, the veins of the calf muscles were examined; venous thrombosis was found in 185 cases and in only 17 instances, or less than 10 per cent, was there any evidence of phlebitis. Their conclusion was that as far as thrombus of the veins in the calf muscles was concerned, inflammation of the vein or thrombophlebitis had nothing or little to do with the production of the clot. The mechanism of the clot formation in the two, thrombophlebitis and phlebothrombosis, is very different. In the former, resulting from inflammatory process of the vein wall, changes occur in the endothelium predisposing to clotting. This is not dissimilar to the process that occurs following the use of sclerosing agents in the treatment of varicose viens. When thrombophlebitis occurs spontaneously, the inflammation usually is the result of involvement of the vein secondary to some perivenous lymphangitis, micro-organisms or toxins being carried to the vein through the lymphatic system. Furthermore, in thrombophlebitis, unless it is associated with an acute suppurative process as we see in sinus thrombophlebitis or involvement of the portal vein, the femoral and iliac veins are most often involved. Since it is a process involving changes in the vein wall, particularly the endothelium, the resultant clot tends to be of a white or mixed variety and here I believe an important point is made, *it does not easily become detached*. There are two exceptions to this, however; the first is that there may be formation proximal to the area of the thrombophlebitis, of a bland thrombus or a simple coagulation thrombus which is not firmly attached and may easily become loosened. This is not of any great significance because it is a relatively short

clot, and if it did become detached it would produce only a small infarction. The formation of such a clot should not be permitted, and it usually can be prevented by the mobilization of the involved extremity during the treatment of the thrombophlebitis. Another exception is that of suppurative thrombophlebitis in which the liberation of proteolytic ferments occurs. The fixed clot becomes liquefied and permits breaking off of fragments to produce septic emboli. This condition, of course, is seen in severe pelvic infections and we have all seen the subsequent course in criminal abortion. Contrasting with the above, the formation of thrombus in phlebothrombosis in considerably different. There is no inflammatory process of the vein wall and the clot may be said to result from two factors, namely increase in the coagulability of the blood and marked slowing of the blood stream. This clot is called red or coagulation thrombus, not dissimilar to that which occurs in the test tube when blood is taken from the vascular system. It is loosely attached to the vein wall and can become free very easily. These thrombi, as has been repeatedly shown at post mortem, for the most part originate in the veins of the calf muscles and might I emphasize: in the veins on the plantar aspect of the foot, while upon occasion it has extended into the veins of the thigh and pelvis. They are usually extensive in scope and of real danger because they will become detached and be carried to the heart and lungs, thereby producing varied degrees of infarction from relatively insignificant peripheral infaction to total occlusion of both pulmonary arteries with sudden death. It is possible, through good fortune, that a detachment does not occur, that sufficient inflammatory symptoms occur in the intima, and firm attachment will ultimately take place. Some authors believe that this process may begin soon after the formation of the thrombus. There are others, however, who believe that this is a late phenomenon and cannot be anticipated with any degree of accuracy. It is easy to understand the development of the thrombus in thrombophlebitis if what we have said above is true, but the development of coagulation thrombus in phlebothrombosis is not quite so clear. For some peculiar reason there seems to be increased coagulability of the blood which results from changes in the blood constituents because of trauma. It has been shown by Shafiroff that over 90 per cent of the patients with post-operative venous thrombosis and acute thrombophlebitis had hypercoagulability of their blood and the average acceleration of blood coagulation was four times that of normal subjects. The localization of the clot in the lower extremity is no doubt due to deterioration and slowing of the blood stream in that part of the body. It would seem to be a reasonable conclusion, upon consideration of all evidence, that body trauma and tissue injury contribute some factor to increase coagulability. Clotting tendency being the underlying or predisposing factor in phlebothrombosis it can then be said that circulatory stasis may well be the precipitating factor, and it is reasonable to conclude that this is responsible for the development of thrombi in the veins of the calf muscles and on the plantar aspect of the foot rather than in the veins of the upper extremity. Increasing age is probably a contributing factor with cardiovascular disturbance and alterations of the vascular system which are often associated with venous thrombosis. Once again Hunter and his associates found at autopsy that involvement of the veins of the leg in middle aged and old persons who had been in bed for variable periods of time was as high as 52 per cent. It was bilateral in 110 instances and unilateral in 75 cases. Allen has said that over 80 per cent of his patients with venous thrombosis were over the age of 40 and other investigators have confirmed these findings. There is undoubtedly a relationship between venous thrombosis and the seasons. Many investigators have observed this. Allen, Linton and Donaldson noted that 32 per cent of their cases occurred in the winter, 21 per cent in the spring, 18 per cent in the summer and 20 per cent in the autumn, in a series of over 300 cases. It may well be that the increased incidence of thrombosis during the winter months is due to the vasospastic factors involved with cold weather. It is also interesting to note that venous thrombosis in northern centres is almost double that occurring in southern climes. Other predisposing factors that should be mentioned are obesity, debility, anæmia, polycythæmia and excessive smoking. As has

been mentioned, the differentiation between phlebothrombosis and thrombophlebitis should be made not only from an etiological standpoint, but from the standpoint of the clinical picture. In frank thrombophlebitis the clinical symptomology is well defined. There is usually pain in the involved extremity, some fever and, with few exceptions swelling. The colour changes are interesting. If the deep veins are involved, and this type is more common, there is a peculiar whiteness of the extremity and the term "phlegmasia alba dolens" has been used. Skin temperature is cool, which is most peculiar in view of the increased surface temperature of the rest of the body. When superficial veins are involved, the skin overlying the affected vein, as you know, is red and warm. The fever is due to the inflammatory process in the involved extremity and the pain can be said to be due to ischæmia, being relieved by vasodilatation. Concerning the whiteness, there has been considerable controversy in the past and I should think it is, in all likelihood, due to vasospasm which is, of course, relieved by vasodilatation. The œdema in thrombophlebitis was at one time thought to be caused by increased venous pressure resulting from venous obstruction. The fact, however, that a large vein such as the femoral, iliac or vena cava can be ligated with the production of only transitory œdema does not support this hypothesis. In all likelihood, as has been shown by Ochsner, the œdema is due to ischæmia resulting from arteriolar spasm, and is relieved by vasodilatation. The patient with thrombophlebitis is ill, whereas the patient with phlebothrombosis may well have no symptoms at all. Under these circumstances, thtrefore, the diagnosis must be made before symptoms are manifest because usually the symptoms are those of a complication such as infarction or a beginning inflammatory process resulting from the retained thrombus. Many of these patients complain of a sense of impending disaster and this should be taken note of and is of great significance in anyone who has suffered any tissue injury because of the likelihood of existing phlebothrombosis. There is usually no fever, the pulse rate may be elevated out of all proportion to any elevation of temperature present, because of the latter if present this is insignificant. The most constant observation is an increased erythrocytic sedimentation rate. Because there are few or no symptoms, diagnosis is dependent upon careful routine examinations of the lower extremities in patients suffering from tissue damage. This policy should be followed routinely—palpating the calf muscles, the plantar aspect of the foot and the forceful dorsiflexion manœuvre (Honan's sign). In all patients in bed, having suffered any tissue injury, the finding of tenderness in the calf or plantar aspect of the foot or the production of pain in the calf or popliteal space when the foot is forcefully dorsiflexed is indicative of phlebothrombosis. Some confirm these findings with phlebography, but many do not consider this necessary.

The prognosis in these two entities varies considerably in the untreated patient, although the outlook need not be bad in either instance if appropriate treatment is undertaken early. In thrombophlebitis, unless there be suppuration, most patients get along well and do not succumb to the thrombotic process. These patients will likely have post-phlebitic symptoms which may last for many years, consisting of swelling, pain, possible ulceration and infection in the involved extremity. Indeed the patient may be incapacitated for many years or for the rest of his life. On the other hand, in phlebothrombosis the patient may well be a potential fatality unless active measures are taken, this being due to the fact that the thrombus is not firmly attached. Non-fatal infarction does occur, but the possibility of a fatal pulmonary embolism is very likely.

Preventative measures in pre-operative management are most important and more so in the older persons with cardiovascular disease. Abstinence from heavy smoking for a period of several days is of value. This point has been emphasized by many observers. The application of compression bandages to the extremities in patients over 40 years of age is likewise important. These bandages are applied to both extremities from toes to the groin before the patient leaves the operating room. The compression of the superficial veins by means of bandages increases the flow of blood through the deep veins. In patients with extensive varicosities this is doubly important. Obesity

being a factor, correction of overweight before elective surgical procedures is to be recommended. The prevention of circulatory collapse during the operation and immediate post-operative period by the administration of fluids and particularly by the re-establishment of blood volume are of great importance. Avoiding abdominal distension by means of indwelling suction is to be recommended. Early ambulation decreases the incidence of venous thrombosis, but this must be undertaken under proper management. Allowing a patient to sit in a chair with his legs dependent produces greater circulatory stasis in the veins of the lower extremity than were he to lie in bed and contract his muscles in the horizontal position, but if by ambulation you mean that the patient walks, this undoubtedly decreases the incidence of venous thrombosis. The use of anticoagulants as a routine measure is not to be recommended and there are many potent dangers in continuing this method. In a person who is known to have had previous thrombosis judicious use of anticoagulants might be undertaken and many observers recommend that only in those cases should anticoagulants be employed in the immediate post-operative period. It has been pointed out by De Takats that if anticoagulants are used in conjunction with sulfonamides, great caution should be exercised because sulfonamides increase heparin sensitivity. When diagnosis has been made and active treatment is to be undertaken, this varies according to the urgency and type. In the open and shut case of thrombophlebitis, treatment consists of vasodilatation secured by sympathetic block. The pain is relieved instantly as a rule, the temperature falls and the swelling subsides within a few days. The response in most cases is dramatic. The patient is not only relieved of his symptoms, but is also spared the persistent manifestations such as œdema, ulceration and recurrent infection. In most instances the relief of pain is complete and permanent (90 per cent of cases), whereas in 10 per cent a second block may be necessary. Of recent months we have been using in some of these cases, tetræthylammonium chloride. The response has been good and in most instances no second dose had to be administered. You are familiar with the technique of sympathetic block which is widely available in the literature and involves the first, second, third and fourth lumbar ganglions. If this procedure is employed, it should be performed every 24 hours as long as the patient has fever. Physiological effect of the block lasts considerably longer than the pharmacological effect. In all uncomplicated cases of thrombophlebitis, conservative therapy is usually sufficient as exemplified by the results seen in the literature and in one's own experience. There are very rare cases of suppurative disease where radical therapy may be imperative. It is sufficient to say that unless the involved vessel is ligated proximally to the site of suppurative phlebitis, septic infarction will continue. There is much controversy among the gynæcologists concerning the advisability of venous ligation in such instances. The mortality rates have been very high. It has been recently shown that the mortality rate in puerperal sepsis after criminal abortion can be greatly reduced by the early ligation of the vena cava and the ovarian veins. However, good results are obtained only if operation is undertaken early before sepsis has developed. On the other hand, in the treatment of phlebothrombosis where the thrombus is not intimately attached and could readily become detached on the slightest exertion, it would seem that radical therapy is in order to prevent fatal embolism. I am well aware that there is a wide difference of opinion on this statement. There are many distinguished observers who believe that all forms of venous thrombosis can be treated by means of anticoagulants, but we subscribe to the point of view that while anticoagulants will prevent the formation of additional thrombi, they will not prevent detachment of those thrombi already present. We do not agree that when a thrombus is recognized clinically all danger of the possibility of an embolus has vanished. The work of Hunter indicates beyond all measure of doubt the proof that bland thrombus can occur not associated wth inflammatory change and not being fixed to the vein wall. I should like to emphasize again that although the administration of anticoagulants will prevent the formation of new thrombi, they cannot prevent thrombi which are already in existence from becoming detached. This false sense of security that everything possible is being done by

the use of anticoagulants may lull one into the belief that the patient is not in any danger. We place ourselves on record at this time, which, of course, may be altered in the future, that immediate surgical intervention should be undertaken as soon as the diagnosis of phlebothrombosis is made. Recently I have had an experience where the intervention was postponed until the morning at the time of regular surgical schedule, but fatal pulmonary embolism occurred that night. The ligation of the vein above the thrombus or opening the vein and aspirating the thrombus proximal to the opening with ligation below that site is imperative. This should be a bilateral procedure for bilateral involvement occurs in a high percentage of cases. Allen of Boston has had the greater experience with curative and prophylactic ligation. It is his opinion that bilateral ligation is the method of choice. We do not believe that ligation is essential in thrombophlebitis except in the rare case of suppurative thrombophlebitis and that opinion is not in agreement with the Boston school.

OPERATION

The treatment of phlebothrombosis consists of exposure of the femoral canal under local anæsthesia. The vein is exposed for approximately 6 cm. below the deep femoral junction and an incision is made in the vein beneath the opening of the deep femoral. If there is free bleeding from the vein proximally, the superficial femoral distal to the profunda is ligated. If free bleeding does not occur, a glass cannula is inserted proximally. In this way the thrombus which extends proximally and may be in the iliacs is aspirated. Aspiration is continued until there is free bleeding. Subsequently, the vein is ligated just above and below the opening. In most instances involvement is limited to the femoral vein below the profunda, however, should there be involvement of the profunda as well as the superficial femoral, then the deep and superficial veins should be ligated. There are some who prefer ligation of the common iliac to that of ligation of the femoral because they consider there is less subsequent œdema.

May I sum up. Venous thrombosis is a very serious complication in both medical and surgical patients. I believe that one should differentiate between the two types as outlined.

Thrombophlebitis has organic changes in the vessel wall resulting from perivenous lymphangitis psoduced by bacteria, toxins, or an allergy. The thrombus usually is well fixed to the vein wall. In phlebothrombosis clotting results from two major causes, the predisposing factor of increased blood coagulability resulting from tissue injury and the precipitating factor of circulatory stasis. The clinical pictures are different. The patient with thrombophlebitis is ill and has many manifestations such as pain, swelling, fever and colour changes. The prognosis for life is good, but unless adequate therapy is instituted, post-phlebitic sequelæ such as œdema, pain, ulceration and streptococci infection are most likely. On the other hand, the patient with phlebothrombosis with few or no symptoms is a potential fatality. The treatment of thrombophlebitis is conservative, using sympathetic block. Rarely is ligation required and then only in septic types. With phlebothrohbosis, the treatment should be radical by means of venous ligation as described or a thrombectomy. Anticoagulation therapy may be used in a person known to have had a thrombosing tendency. It will prevent the formation of additional thrombi but it will not prevent the detachment of a thrombus whirh already exists.

POSTURAL DEFECTS

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Read at Staff Clinical Meeting, Vancouver General Hospital, December 27, 1947.

Posture is a state of muscular and skeletal balance. Good posture suggests a state which will protect the supporting structures of the body against strain and progressive deformity, irrespective of the attitude, whether erect, sitting, squatting or stooping. Under such conditions the muscles will function most efficiently, and the optimum positions are afforded for the function of the thoracic and abdominal organs. If poor posture is maintained, an increasing strain is thrown upon the supporting ligamentous structures; there is less efficient balance of the body over its base of support.

As all parts of the body are inter-related, any displacement of one part leads to a change of other parts above and below this part. Nearly all orthopaedic deformities eventually lead to an impairment in the general posture of the body as a whole, and produce secondary disabilities. For example, flexion deformity of the hip is followed by forward inclination of the pelvis, with compensatory changes in the spine: increased lumbar lordosis and dorsal kyphosis.

The appearance of good posture varies in the different types of individuals; the three common types are the slender, the stocky, and the intermediate types. Malposture occurs in all types, but in youth is commonest in the slender type, with thin bones, small muscles, and relaxed ligaments. There is usually hyperextension of knee and hip; relaxed feet; relaxed abdomen; flat chest, the head and chin forward and down, with increased lordosis of the neck. In the stock type, with firm ligaments, the strong short musceles, the front of the pelvis is held down, the back of the pelvis is held up, leading to lumbar lordosis as the primary postural fault. The intermediate type shows postural deformities between these extremes.

In good posture the head should be squarely balanced over the shoulders, in both planes. The three anteroposterier curves of the spine (cervical lordosis, dorsal kyphosis, and lumbar lordosis) should be barely appreciated when standing or sitting. The chest should be high, and the abdomen flat. The shoulders should be level, and there should be no lateral curvature. The lower extremities in standing should bear weight with the joints in a neutral position, that is, not hyperflexed or hyperextended. The feet should not show the pronation of relaxation.

Malposture is very common; it occurs as high as 80% of individuals. It is common in children, and if uncorrected often progresses to serious deformity in later life. There is an increase in the number of persons with malposture in middle life and in old age, while very poor posture is most common after 40 years. The smallest incidence is in the second decade. Poor posture tends to be more common where nutrition and hygiene are less good. Contrary to what might be expected, malposture is quite common amongst athletes of either sex.

Mechanics of poor posture

The erect posture of man is the result of prolonged evolutionary changes, with visceral and muscular adjustments, as the trunk assumed a more erect carriage. The lumbar spine became shortened; the pelvis became broader to give better attachment to the lower abdominal and pelvic muscles. The cervical and upper dorsal spine became straighter, with an increase in height. The knees and thighs became straightened, and the pelvis tilted downward in relation to the lumbar spine. The pull of gravity in the erect human body is resisted chiefly by the anti-gravity group of muscles: the gastrocnemius, the quadriceps, the gluteus maximi and sacro-spinalis. The variation in man's ability to counteract gravitational stresses gives rise to the various postural deformities. The erect position is resisted also by the downward pull of the hip flexors, and the abdominal muscles. When standing the sacrospinalis muscles remain in almost constant contraction while the abdominal muscles are seldom strongly contracted and have poorer tone. The hip flexors tend to pull the front of the pelvis down, while the sacro-spinalis muscles tend to pull the back of the pelvis up, both working against the pull of the ab-

dominal muscles. The latter are helped by the gluteal muscles which tend to pull the back of the pelvis down. The weakening and stretching of the abdominal muscles, and strengthening and shortening of the sacrospinalis, gradually lead to less effective balance of the body against gravity. The degree of pelvic inclination is probably the most important single factor determining posture, as the muscles attached to the pelvis work much less efficiently when the pelvis is tipped forward markedly.

Poor posture is often the result of numerous occupations and habits which require the frequent use of the hands at waist level or lower, leading to contraction of the depressor muscles of the shoulder girdle (principally the pectoralis major) and to weakness of the shoulder elevators: the levator scapulae, rhomboids, and trapezii.

Effects of Faulty Posture

The immediate effects are increased fatigue and impaired function. The group of muscles which is resisting the full of gravity will be in strong contraction while the opposing muscular group will be in relative relaxation. An example of this may be found in the individual who drives in a modern automobile seat for long periods of time; the cervical spine is held in a position of lordosis, and fatigue and pain are experienced in the posterior muscles of the neck.

In any part of the body the arrangement of supporting structures is so placed that it will provide effective stability for normal use. These supporting ligaments are ineffective when a deformity occurs. The action of muscles is less effective when their origin and insertion are brought closer together or further apart. Energy is wasted and fatigue appears much sooner.

The early effects of malposture are seen in growing children. The scapulae are bowed as a result of the rounded shoulders, and there is an increase in the curves of the clavicles. Increased dorsal kyphosis is accompanied by anterior wedging of the vertebral bodies, the chest is flat, and the ribs are flared. In adult life the postural deformities are most commonly fixed contractures of muscular and ligamentous structures. A common deformity is the excessive lordosis at the lumbosacral junction, with forward inclination of the pelvis; this is accompanied by degeneration and narrowing of the intervertebral disc. The most serious symptoms of malposture appear in older persons who show decalcification of the skeleton with accompanying degenerative changes in the supporting soft tissue structures. When malposture is present disability from trauma or disease tends to be prolonged; this is noticed particularly in fractures of the lower extremity, and in lesions of the low back. In faulty posture the low back is frequently used habitually at the extreme of extension, a markedly increased lordosis. In this position further extension leads to overstretching of supporting ligaments and irritation of the lateral articulations. Healing following trauma will be prolonged in these ligaments and articulations if they remain in an over-stretched position. The Boston group believes many visceral symptoms disappear when faulty posture is corrected.

Treatment

Treatment should be directed chiefly to the prevention of faulty posture in childhood. The teaching profession should be acquainted with the rudiments of good posture and with the relatively simple methods of correction. Most modern city schools have on their staff a gymnasium instructor who should recognize and correct malposture throughout his classes. The medical profession should correct the postural deformities and disabilities of those who have passed the peak of physical fitness. Treatment in this group of individuals must often be palliative, with partial relief of symptoms, since permanent degenerative changes have frequently developed. Cure is frequently impossible, but improvement can be obtained, supporting measures can be instituted, and further progression of deformity can be prevented.

The principles of postural correction can be of aid to the orthopaedic surgeon in the treatment of almost every orthopaedic disability, since deformities of the spine or extremities usually lead to some disturbance in the balance and alignment of the body as a whole. The patients can be taught to develop compensation for their deformities.

In the older age group efforts towards restoring better muscular balance and co-ordination will often be disappointing. However, fairly good results can often be obtained by the additional use of some simple and light external support, such as the full-length, snug-fitting, re-inforced corset, in the treatment of postural low-back pain.

Postural Exercises

The aims of exercise are:

1. To develop co-ordination in the groups of muscles which maintain the erect posture, in order that the body may be balanced against gravity most easily and efficiently.

2. To stretch shortened muscles, in order to lessen deformity and as far as possible restore the normal range of motion in the joints.

3. To strengthen weakened muscles, and permit a restoration of normal muscular balance. This latter occurs very slowly. The chief muscular group which becomes shortened and requires stretching is the sacro-spinalis, or erector spinae. The muscles which most commonly need strengthening are the rectus abdominis and gluteal muscles.

Stretching, co-ordinating and strengthening exercises are given lying; then exercises are given in the sitting and standing positions. A simple first series of exercises for co-ordinating muscular pulls and regaining muscular balance is given. In persons who cannot or will not carry out corrective exercises, and in older persons, palliative measures supplant corrective ones. Light supports and occasionally operative procedures are necessary.

It is assumed that a medical study of the individual will be made to exclude excessive fatigue, malnutrition, or chronic illness.

DOCTORS FARM TOO

A leading Canadian publication declared in a recent story that the country's most desirable climate is that enjoyed in the valley of the Campbell River which enters the Pacific near the border at White Rock.

The other day I drove out from smog-bound, saturated Vancouver and stood in the glorious sunshine on an 80 acre farm divided by that stream. The bottom land was rich chocolate loam and the balance Whatcom silt.

After inspecting the neat dairy and efficient barn with 22 steel stanchions, automatic water bowls and all the latest features, I was quite sure that this, of all hobby farms, would delight a doctor's heart: would fascinate the few momentary lags on his appointment schedule with weekend memories and visions of a future on an estate of his own out where the air fairly sparkles.

The ranch dwelling in its natural locale is an artistic tribute to the aristocrats who built it about four years ago. Picture full length logs burning in the massive granite fireplace, around which is designed the room for living 28′x17′. The master bedroom has private bathrooms in the latest design. Another three-piece Pembroke bathroom serves two more bedrooms and nursery in the west wing.

Special features, too numerous to mention, include dining room designed for entertaining, two guest rooms upstairs and rumpus room 22′x14′ with fireplace, eastern maple floors, knotty cedar wainscoting in some rooms, drive-in basement, complete insulation.

At the full price of $21,000 on terms, the seller will realize a fraction of his investment. The buyer will realize the fulfillment of his leisure dreams in the reality of a sound investment for future happiness.

For an appointment call George G. Slough at W. H. Gallagher & Co., TAtlow 1448, 448 W. Pender St.