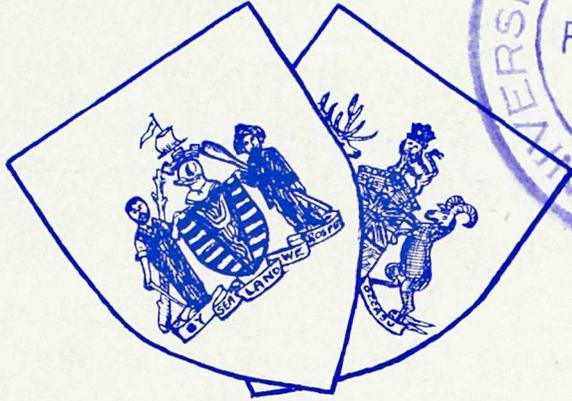




THE BULLETIN



OF THE VANCOUVER MEDICAL ASSOCIATION

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JUNE 22—26, 1931
VANCOUVER, B. C.



Vol. VII.

FEBRUARY, 1931

No. 5



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THE VANCOUVER MEDICAL ASSOCIATION

BULLETIN

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FEBRUARY, 1931

No. 5

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VANCOUVER HEALTH DEPARTMENT

STATISTICS, DECEMBER, 1930

Total Population (estimated)		240,421
Asiatic Population (estimated)		9,335
	Rate Per 1,000 of Population	
Total Deaths	191	9.4
Asiatic Deaths	11	13.9
Deaths—Residents only	174	8.5
Birth Registrations	342	16.7
Male 175		
Female 167		

INFANTILE MORTALITY—

Deaths under one year of age		14
Death Rate—per 1,000 Births		40.9
Stillbirths (not included in above)		7

CASES OF CONTAGIOUS DISEASES REPORTED IN CITY

	November, 1930		December, 1930		January 1 to 15, 1931	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Smallpox	0	0	0	0	0	0
Scarlet Fever	28	0	33	0	16	0
Diphtheria	7	0	10	2	2	0
Chicken-pox	155	0	121	0	109	0
Measles	6	0	2	0	3	0
Mumps	0	0	21	0	19	0
Whooping-cough	8	0	10	0	6	0
Typhoid Fever	8	1	9	3	2	0
Paratyphoid	0	0	0	0	0	0
Tuberculosis	20	14	7	13	10	0
Poliomyelitis	3	0	0	0	0	0
Meningitis (Epidemic)	0	0	0	0	0	0
Erysipelas	4	0	3	0	3	0
Encephalitis Lethargica	1	1	0	0	0	0

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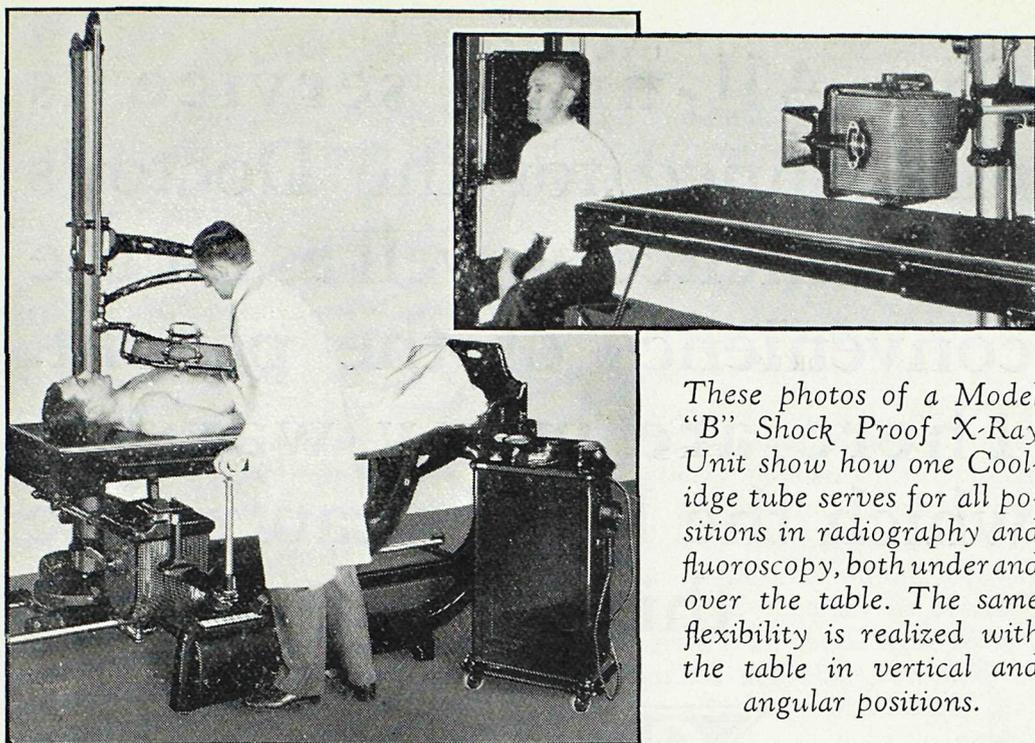
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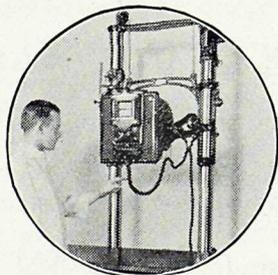
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EDITORIAL

The approaching session of the British Columbia Legislature brings up again the now annual question of the licensure of various cults to treat the sick. Once more we are faced with the question—shall we oppose this or not?

There is much to be said on either side, and we confess to a feeling of sympathy with those who argue that we should leave the responsibility where it belongs, with the Legislature. Why, it may be asked, and with reason, should we spend an enormous amount of energy and a great deal of money, in doing what it is the obvious duty of the Legislature to do, namely, to protect the public against untrained and ill-trained people, who wish to be allowed to employ methods and treatment, which are not based on any scientific knowledge? We are not, after all, the guardians of public health—this is the function of the Legislature. Our opposition puts us in an invidious position with the public, which naturally considers it due entirely to fear lest our incomes be injured, and our work taken away from us, if these gentry be given legal recognition. We know, of course, that this is not so, and that our motives are not interested ones: we see, more clearly than any others can see, the danger that such recognition of the uneducated would be to public health. Through many years there has been built up a structure of defense against disease, not yet complete, but soundly conceived, and based on firm scientific foundations. This is endangered greatly by anything which lowers the standard of education and training demanded of those who treat the sick; and it is the knowledge of this that has made us spend our time and our money fighting those who seek to undermine our work.

But it is a wearisome, and as one often feels, a thankless struggle; and, in one sense, we are not the ones who should be conducting it. We have placed our knowledge and our advice at the disposal of the authorities; we have made all these things clear, *ad nauseam*. Should we rest on this with a feeling that we have done our duty, and that no man can do more?

We feel that we cannot. It is true that our position is unpleasant and hateful—that we are weary of the misrepresentation and the misunderstanding that we meet—that this fight is not entirely ours. But even so, we must continue it, to ensure that those whose duty it is to protect the public adequately, fulfil this duty. For with our superior privilege, of knowledge, comes a responsibility which we cannot evade. We cannot simply sit tranquilly by as onlookers, saying that we are not our brother's keepers. For after all, to some extent, we are. We owe to the community a duty, a moral obligation, which we cannot negatively discharge. We must with all the power that lies in us, protest and keep on protesting against the passage of such legislation. We need not, in this journal, labour the reasons for such opposition—every medical man, every scientifically trained man, knows them well—but we must seize every opportunity to urge them on those whose ultimate responsibility it is to see that only those who have adequate education and scientific training shall be allowed to tamper with the human body.

We should demand and keep on demanding, one of two things—adherence to the present standard of scientific training for all who claim to treat sickness, or the appointment of a Judicial Commission, which shall make an unbiased and full enquiry into this whole affair, as was done in Ontario. This latter was suggested at the last Session, and why this simple method of solving the difficulty was not adopted, is one of the mysteries of political life. Meantime, we must as individuals, and as organized units, get behind our representatives, the B. C. Medical Council, and support and strengthen them in every way. Above all we must never give up the fight—our conscience will not allow us to do so. Wearisome and expensive as it has been, it has been well worth while, and it must go on till some finality is reached, some sane conclusion, whereby, while no injustice is done to any, assurance may be given that public health be not endangered. We cannot compromise or weaken in this regard. The B. C. Medical Council may feel assured of the firm support of every medical man in the province in this; they are our representatives, and we look to them to carry on the fight without faltering.

* * *

It has been brought to our attention that certain practitioners are giving certificates of health for a smaller fee than that laid down in the Schedule of Fees adopted by the Vancouver Medical Association. This is very unfair to other practitioners and should not be done.

OBITUARY

Through the death of Dr. Thomas Alexander Wilson, which took place at his home in Vancouver on Friday, January 16th, the Vancouver Medical Association has lost one of its oldest members.

Born in 1861, at Ottawa, Dr. Wilson was educated in the public schools of Ontario where he later taught for some years. Deciding to enter the practice of medicine, he went to Kentucky and graduated in 1895 from the Hospital Medical College. On the advice of friends he proceeded, in 1900, to take his M.D.C.M. from Queen's University.

Dr. Wilson was associated with Indian affairs from the time he came to B. C. over thirty years ago. He spent six years working among the Indians at Port Simpson, later moving to Vancouver, where he continued his connection with the Indian Department and was their medical officer in the Vancouver district, until his death.

Dr. Wilson married, in 1888, Dr. Belle Holland, who survives him. He leaves two sons, one, Dr. Ray H. Wilson, who practises dentistry in San Diego, and Dr. P. M. Wilson of Britannia Beach.

MEETINGS

The first meeting of the Vancouver Medical Association for 1931 was held in the Auditorium on January 6th, and was exceptionally well attended.

The report of the Dinner Committee was presented by the Chairman, Dr. L. H. Webster, and showed a substantial balance on the right side.

A motion was introduced by Dr. Pearson for the purpose of securing the approval of the Association of the proposed action of the Publications Committee in engaging a business manager. It was explained by Dr. Pearson in his introductory remarks that, while the Bulletin was doing very nicely, thank you, there was a chance of its thriving much more rapidly if it received proper sustenance in the form of additional advertising. The bulk of that now carried had come almost unsought and the surface of this field and source of additional revenue had hardly been scratched. Increased advertising matter would enable the Bulletin to increase its space for scientific matter, thereby increasing its circle of interested readers and completing a virtuous circle (hitherto unknown in medical parlance) would secure still more advertising. A brief discussion followed, all of which was indicative of the accord of the Association with the Committee's project. The motion was seconded by Dr. J. H. MacDermot, and carried.

A motion was then brought before the Association by Dr. J. J. Mason to provide for the formation of a Cancer Committee. The increase in cancer incidence and mortality, especially in British Columbia, of which all are aware, was making it imperative that steps be taken to systematize our observations and collection and arrangement of data on cancer, which would then prove a source from which further knowledge and advances could be gained. Dr. Mason's proposal was that what would be in essence a Nominating Committee of about six members, representative of the various branches of medicine and surgery, should be assembled who in turn should select a committee—the Cancer Committee, who should arrange a two year programme of work in study and assembly of data.

Among those who in speaking to the motion expressed their hearty approval was Dr. J. W. McIntosh, the newly appointed Medical Health Officer for Vancouver, who had shortly before been welcomed back to the Association meetings by the Chairman.

Dr. Mason's motion was carried unanimously.

The Chairman then introduced the guest speaker of the evening, the Hon. Chief Justice Auley Morrison of the Supreme Court. In a most pleasing manner His Lordship gave a very lucid exposition of medico-legal relations, dwelling in particular upon the duties and responsibilities of the expert medical witness and the interpretation and application of the term "insane" in reference to criminal acts and other matters.

WASSERMANN AND KAHN REACTIONS FUNDAMENTALLY IDENTICAL

(From four articles by EAGLE in the Johns Hopkins Hospital Bulletin, November, 1930, and the Journ. Expl. Med., November, 1930).

With comments by H. W. HILL, M.D., Director, V.G.H. Laboratories, Vancouver, B. C.

Of intense interest to physician, to serologist, and to immunologist, the above articles indicate long steps forward in the understanding of the Kahn and the Wassermann tests.

The conclusions reached, shorn of technicalities and detailed proof (for which see the articles above listed) may be stated thus:

Syphilitic serum is distinguishable from "normal" (i.e. non-syphilitic) serum in that the former contains "reagin"—a hypothetical substance (somewhat of the order of the "amboceptors" and "agglutinins" of Ehrlich's familiar nomenclature) having definitely recognizable features, based on its striking effects upon certain lipid suspensions (the "antigens" of the Kahn and Wassermann reactions.)

Normal serum, since it lacks this reagin, fails to produce these effects.

A lipid suspension prepared *secundum artem*, "antigen," when treated with *normal* serum is affected thus—each individual lipid particle, suspended in the saline liquid, becomes irregularly coated with a film of serum-globulin. Before the addition of the normal serum, each lipid particle presents to the surrounding liquid lipid surface only. After the addition of the normal serum, each lipid particle presents to the surrounding liquid but a fraction of its total lipoidal surface, the rest of its lipoidal surface being now coated with a film of normal globulin which, of course, separates that part of the lipoidal surface so covered from the liquid. The lipid particle therefore now offers to the surrounding liquid a "mosaic" surface, part original naked lipid, part film of normal serum globulin.

The normal serum globulin thus adsorbed on the lipid particle is unchanged; but the suspension as a whole no longer consists of *lipoid* particles + saline liquid, but of *lipoid-globulin* particles + saline liquid; and its colloidal properties now show changes corresponding with this change in certain directions (cataphoresis, electric potential). But there is no tendency to agglutination, precipitation, or flocculation in the presence of small quantities of electrolytes (the basis of the Kahn test); nor to complement fixation in the presence of fresh serum (the basis of the Wassermann test).

Now, the original lipid suspension, when treated, not with normal but with *syphilitic* serum, is effected exactly similarly so far as the irregular coating of each lipid particle with globulin is concerned. But this film differs from that formed by the normal serum in that it is not globulin only, but reagin-globulin. Whereas the film of normal globulin was unchanged and therefore remained water-soluble, this film

of reagin-globulin is water-insoluble. This suspension of *lipoid-reagin-globulin* is subject to agglutination in the presence of small quantities of electrolytes (the Kahn test), and absorbs complement from fresh serum (the Wassermann test)—these reactions being due to the *reagin-globulin film*, not to the *lipoid particle*, which acts only as the now inert carrier of the film.

Thus, the complexities of the Kahn test, and the greater complexities of the Wassermann test, all tend to but one end—the demonstration, rather direct in the Kahn test, by agglutination and precipitation, rather round-about in the Wassermann test, by complement fixation, of the presence (and amounts), or absence, of syphilitic reagin-globulin. Eagle suggests that since both tests thus determine a specific syphilitic antibody, reagin, both reactions are after all “specific,” not non-specific as usually considered. (It remains true, however, that the *specific antibody*, reagin, is *detected* by the use of a *non-specific* substance, lipoid (“antigen”); and to this extent these tests *do* depart from those more or less parallel reactions where a specific antibody is detected by its corresponding *specific* antigen, as in the Widal reaction and its analogues.)

The action of cholesterin in increasing the sensitivity of the Kahn test and the Wassermann test is shown by Eagle to be due merely to the fact that the cholesterin adsorbs upon itself the lipoid particles, aggregating them in relatively large masses which then combine with the reagin-globulin in greater quantities than when the particles remain in a finer state of division; thus increasing agglutination in the Kahn test and the absorption of complement in the Wasserman test.

COMMENTS

In view of the above conclusions from such clear cut experimental evidence, it would now appear that the significances of the Kahn test and the Wassermann test are not those of two different substances but merely of two different techniques for determining, more or less accurately, the *same* substance, syphilitic reagin-globulin.

Hence it is worth considering whether or not the demand made for performing both tests on all cases is now as logical as it seemed in the past. The two tests, supposed obscurely to determine two different phases—to supply two different angles of view, so to put it—now appear to determine only one. Both tests, it would now seem, arrive at the same point—a more or less accurate determination of the reagin present.

It would appear now more clearly that the Kahn test is more simple and tends to be more nearly correct in this regard than most Wassermann tests—and that the best Wassermann test can only hope to duplicate or approximate the accuracy of a good Kahn.

Then why do both? If one has two pocket tape measures, one more accurate than the other (and also more easily applied), why insist on using both accurate and inaccurate tapes on all occasions? If a check is needed, why not use the more accurate and simpler one twice, rather than the more accurate first and then the less accurate and more diffi-

cult one as a check upon it? What information can be gained in this way regarding the actual measurements of the object? If the two pocket measuring tapes are equally accurate, neither has the advantage in accuracy—and every one would use the most easily applied.

It would seem that the wide-spread Kahn-Wassermann debate was after all chiefly concerned with a non-essential question—namely, which test gives the *higher percentage* of positives; not with the really important issue,—which test best corresponds with the clinical condition of the patient. That and that only can be the real concern of the physician. That and that only is the real field for investigation.

Since both tests apparently estimate the same factor, reagin-globulin, the Kahn being the superior in this determination, the real problem of the future would now appear to be a careful study of the presence and amounts—or absence—of reagin-globulin, and the clinical significance (if any) of its fluctuations, when present. From this point of view, the estimation of reagin-globulin at various stages in the progress of a case of suspected syphilis might be thought of as more or less parallel (*mutatis mutandis*) with the similar estimation of a Widal reaction or even of a tuberculin reaction.

We are pretty well assured that a Widal reaction lasts long after the germs responsible for it have disappeared from the patient's body. We are inclined to believe that the tuberculin reaction and the syphilitic reagin-globulin reaction usually disappear promptly, if and when the germs have disappeared.

It would seem that Eagle has made a real contribution to further progress in the study of syphilis by his illuminating researches.

ARTERIAL BLOOD PRESSURE, HIGH AND LOW

By PROF. ROBERT D. RUDOLF, Toronto

The subject of blood pressure has been so much before the public, lay and medical, in recent years, that it almost seems as if an apology were necessary for again discussing it.

I will not waste time debating the factors that normally keep the arterial blood pressure at its *optimum* point for the given individual, (and, by the way, this point varies considerably in different healthy people), but will merely enumerate them. They are, of course, the systole of the heart, the elasticity of the arterial walls, the tonus and degree of construction of the muscular coat of these walls, especially of the arterioles, the total bulk of blood and the viscosity of the same. If any of these factors increase the pressure tends to rise. Thus, when the heart beats more rapidly or more powerfully the pressure rises, and if the arterioles contract and so hamper the escape of the blood into the capillary bed it also does so.

1. When we consider what may bring about any of these fundamental changes so that the pressure rises we find a maze of possibilities, but it seems to me that the subject of aetiology of *hyperpiesis*, and so of its treatment may be simplified if all cases of raised pressure be grouped into four classes, as follows: (1) nervous; (2) toxic; (3)

organic; and (4) essential. This last is the group into which are put all those cases that cannot be accounted for, in fact, in which the cause is not evident.

(a) NERVOUS HYPERPIESIS. The blood pressure is very much under the influence of the emotions. Most forms of nervous excitement tend to raise it, either, probably, by increasing the action of the heart, or by causing vaso-constriction, or by both. Possibly this is brought about by an increase in the internal secretion of the adrenal glands. Even a medical examination acts in this way and every physician knows that a single reading of the pressure is quite unreliable and hence will leave the cuff on and try the pressure a number of times or even on several different occasions before coming to the conclusion that the pressure is abnormal. Not long ago I was seeing an old gentleman with his son who was a physician. The pressure during the hour that I was there read at about 180 mm. systolic and I might have concluded that it was really high if the son had not found it that morning to be 125 mm. But not only will emotion raise the pressure but long-continued nervous tension will keep it up indefinitely, in fact as long as the stress lasts and in the long run will produce organic changes in the vessel walls and heart which may eventually cause death. This is no doubt the sequence of events in many people who have for long lived at high tension and eventually die of cerebral haemorrhage, heart failure or kidney involvement.

Most cases of so-called essential hyperpiesis probably really belong to this class although many are probably toxic.

(b). TOXIC HYPERPIESIS. Various toxins can raise the arterial blood pressure. It is usual to see it up in Graves' disease and in toxic adenoma of the thyroid gland. M. C. Pincoffs recorded a most interesting case at the Association of American Physicians last year where a young woman suffered from periodic attacks of high blood pressure when the systolic readings would often be above 300 mm. A tumour of one of the adrenal glands was discovered and upon its removal the attacks of hyperpiesis completely disappeared.

In renal insufficiency there is a retention of products that should normally be got rid of and the blood pressure tends to be high. Exactly what these retained toxins are is not yet settled but Major has produced much evidence that at least one of them is guanidin. R. G. Walter recently concluded after much experimental work that hypertension may be due to the presence in excess of a specific amino-acid. This excess may be due to retention resulting from renal disease, to over-production, or more probably to some impairment of hepatic function whereby this body is not sufficiently destroyed. It would seem that the toxins may be ones that are normally present but are efficiently got rid of before they become excessive or may be produced by abnormal changes in protein digestion, so that the normal amino-acids are broken down into monamines and diamines and some of the former are pressor in action.

There appear to be three ways in which the body is protected from the undue accumulation of toxins in the blood: (1) The mucous mem-

brane of the bowel is normally resistant to their absorption; (2) the liver rejects them after absorption; (3) and lastly, the kidneys can excrete such as reach the blood. If any of these factors is in abeyance toxaemia tends to occur, and hence the importance of attending to such functions; also the need of lessening the protein intake when any of them are faulty.

In the opinion of many, and I share it, alimentary toxaemia is one of the commonest causes of raised blood pressure, and in every case, even when some other cause, such as nerve tension, is evident, it is well to lessen this probable source of trouble.

The late Professor Huchard, of Paris, used to teach that arterio-sclerosis commences as a toxaemia, continues as a toxaemia and ends as a toxaemia, and there was much truth in this, although it is not the whole story. We must remember that in a toxaemia not only may the blood pressure rise, but the toxins themselves will directly poison the tissues of the circulatory apparatus as well as those of all the organs, and hence may produce symptoms which are synchronous but not due to the hyperpiesis.

(c). ORGANIC HYPERPIESIS. In true arterio-sclerosis the blood pressure may or may not be high. It takes a very widespread sclerosis to bring about such a rise. Some say that unless the splanchnic vessels are involved it does not occur. Undoubtedly, if the arterial tree be sufficiently obstructed, a rise must occur on the proximal side of the lesion as is seen experimentally when the abdominal aorta of an animal is ligatured or compressed. Some years ago a young girl came to the Children's Hospital complaining of cardiac distress. We found the heart to be enlarged, and a routine examination showed that there was a loud systolic murmur at the base of the chest posteriorly, and that no pulse could be felt in the abdominal aorta or its branches. The systolic blood pressure was nearly 200 mm. An X-ray examination showed that there was a large mass of tuberculous glands in the posterior mediastinum, which was evidently compressing the descending aorta. Such a case shows how a sufficient obstruction will raise the blood pressure, but a degree of arterio-sclerosis sufficient for this seldom exists. If, however, thickening occurs in some artery supplying a vital centre a general rise in pressure of a compensatory nature quickly occurs. The late Professor Starling thought that such compensatory raising of the pressure was a common cause. The vital centres must have sufficient blood for the maintenance of life. Often when it is found to be hard to lower a pressure by therapeutic means the case is of this nature.

(d). ESSENTIAL OR IDIOPATHIC HYPERPIESIS. As already mentioned, into this class are put all those cases in which the cause of the rise is not apparent. These cases are mostly "benign," are not characterized by any symptoms, and the high pressure is often discovered by accident in the course of a general examination, say for life insurance.

PROGNOSIS. When a rise in blood pressure is found the question arises as to its significance in regard to the probable duration of life.

This outlook depends largely on the cause of the condition. A rise in the diastolic pressure is worse than when the rise is chiefly in the systolic. The state of the kidneys, heart and retina all bear on the prognosis. The dangers are heart failure, uraemia, cerebral or other haemorrhage and acute oedema of the lungs, and the frequency is in this order. While a persistent rise in arterial blood pressure is a serious thing, and insurance companies fight shy of them as "risks," it is wonderful how long and comfortably many of the systolic cases last, and it does not do to take too grave a view of their condition. Especially is it important not to terrify the sufferer, which will only make matters worse, and if the anxious patient insists on knowing what the reading is then it is justifiable for the practitioner to deduct considerably from his findings. I saw only the other day an elderly lady who was living in a state of terror of a "stroke" (which, by the way was a family failing), since a doctor when taking her pressure had looked very grave, but would not tell her the result.

TREATMENT. In the treatment of any ailment, or rather ailing person, it is well to have some system of procedure, as in this way important measures are less apt to be overlooked than if the therapy be conducted in a haphazard manner. The routine that I always follow is the following: environment, diet, specific treatment, and finally symptomatic therapy.

Of course in every case a diagnosis must be made of the condition in so far as this is possible. In the case of hyperpiesis this has already been partially done, but much more than a mere recognition that the pressure is too high remains to be done. Is the rise due to nervous, toxic or organic causes? Is it compensatory in type and hence a protective one for the damaged individual? How are the heart and the blood vessels standing the strain, and of course what is the state of the kidneys? Such questions all come under the heading of diagnosis and should be as fully as possible answered before going further. When no cause can be discovered the case is usually labelled "essential hyperpiesis," which of course is only a cloak for our ignorance.

ENVIRONMENT. Rest is of great importance in all cases, but the degree of it depends upon the urgency. If the case be a bad one, and especially if cardiac distress be present, complete rest in bed for several weeks is necessary, and not only physical but mental rest, which is often quite as important, and let it be repeated that apprehension is a great source of nerve tension and hence the doctor should make as light as possible of his findings with the sphygmomanometer. When an active business man is merely put to bed and allowed to conduct his affairs from there he is apt to fidget and gain no benefit from the physical rest. All his worries should for a time be kept from him, which of course is often a counsel of perfection. In patients who do not settle down to rest well the free use of bromides is often useful.

Massage has its place in partially taking the function of exercise.

During sleep the blood pressure is usually lower than when awake, but this is not always the case, and J. A. Williams, of Aberdeen, has

shown that often, in disturbed sleep, rises occur that may be much higher than are produced by moderate exercise and he thus explains the not uncommon occurrence of sudden death during sleep.

In mild cases of hyperpiesis the patient may go on with his life's work, and it is wonderful how much of the community's best work is done by people who have blood pressures which exclude them from any chance of being accepted for life insurance. All the same, a programme should be arranged which will demand less physical and mental strain. Often one can find on enquiry that some special work causes a special strain on the individual, and this may be cut out without spoiling his general efficiency. In these mild cases gentle exercise, especially of a pleasant nature, will be of benefit, but the patient should be warned to avoid anything that causes him circulatory distress. Short rests in the horizontal posture during the day are valuable, especially after the mid-day meal.

DIET. A general cutting down of the total diet is often advisable. As regards the individual elements of the diet it has long been the custom to lessen the intake of purin-containing foods, such as meats (especially red meats), eggs, fish, and tea and coffee. Lately there has been some doubt thrown upon the effects of a high protein diet and it has been shown that such may be taken for a few days without any rise in the arterial pressure, but this is different from the long continued excess. Nuzum has proved that if rabbits are fed for months on a high protein diet their pressure is higher than the controls, and recently Saile has recorded that the average blood pressure in Trappist, Carmelite and Carthusian monks, who are forbidden the use of meat, fish and eggs, is considerably lower than that of the Dominicans and Franciscans who are allowed meat four or five times a week. Generally speaking I believe that these patients are best on a lacto-vegetarian regime, although an occasional indulgence in meat, fish and eggs will do them no harm. Alcohol does not raise the blood pressure, but should be reduced or barred on general principles. Tobacco has little effect on the pressure and its moderate indulgence by those accustomed to it will do no harm, and is in fact soothing.

The bowels should be gently open, and if they do not act so naturally the regular use of salines in the morning and an occasional mercurial at night are indicated. There is some evidence that a limitation of the intake of sodium chloride is useful and this should certainly be done if there be any oedema.

The limitation of the diet is of special importance when it is suspected that the rise is of a toxic nature.

If a quieter life and regulation of the diet do not relieve the excessive pressure, small doses of iodide of potassium sometimes help, possibly by increasing elimination.

SPECIFIC TREATMENT. An attempt should, of course, always be made to remove the underlying cause of the rise in pressure and this is often possible. Where some nervous strain exists the relief of this may be

all that is required. In toxic cases the careful dieting and free elimination above discussed may rank as specific. The cause, however, is often unremovable, as in most instances of compensatory hyperpiesis, and then all that can be done is to make the sufferer as comfortable as possible under the circumstances. We must remember, moreover, that nature often overcompensates. This is seen when excessive scratching for the removal of some irritant causes damage to the skin; when an excessive cough brings about pulmonary emphysema; when a slight gastritis causes vomiting of an excessive degree, and so on. Nature is often a poor doctor so far as the individual is concerned.

SYMPTOMATIC TREATMENT. In every case of raised blood pressure the regulation of the diet and the manner of living are advisable, and at least cannot do any harm, and many of these people require nothing more and go on year after year in comfort, and in the end may die of something quite unconnected with their hyperpiesis.

If the pressure remains high under such simple regime the question arises of how far it is wise to further interfere with it, and here we may divide all cases of hyperpiesis into two classes, (1) those without symptoms, and (2) those with symptoms.

In the cases without symptoms, where the individual would not be aware of the raised pressure if the doctor had not found it in the course of a general examination, I believe that as a rule we should not actively interfere. The high pressure may be the *optimum* one for them owing to some bodily abnormality, and if we lower it they do not feel so well. A case seen not long ago illustrates this. An old ward patient with hemiplegia had a systolic pressure of 180 mm. The hemiplegia was of long duration and he was quite comfortable except for his somewhat hampered movements. We put him on benzyl benzoate and in a couple of days the pressure was 130 mm., but he felt miserable. The drug was stopped and the pressure soon rose, and with it his feeling of *bien etre*. Another man was admitted with a failing heart and the blood pressure was 100 mm. Under rest and digitalis the heart soon regained power and the pressure rose to 200 mm., and he left hospital feeling comfortable. He needed the high pressure for some compensatory reason. By the way, digitalis does not raise the blood pressure unless it is low due to heart weakness and then it does so very efficiently.

Thus, to repeat, symptomless hyperpiesis that does not lessen under careful dieting and the general regulation of the way of living, as a rule should be left alone, as it is probably the *optimum* pressure for the individual. There is an exception to this which will be mentioned later.

But when high pressure is accompanied by symptoms the case is different. It may be the lesser of two evils to lower the pressure for fear that something worse will happen, and also in order to lessen the distress. The symptoms that are common here are headache, dizziness, irritability, breathlessness on exertion or even while at rest ("cardiac asthma"), passing aphasia or even paresis and angina pectoris. Acute pulmonary oedema may occur at any time. As already mentioned, many

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of these symptoms may be really due to toxæmia rather than to a raised pressure and may be largely reduced or removed by a reduction in diet, free purgation and diuresis. In toxic cases that do not yield to minor measures nothing in my experience equals the effects of a timely blood-letting. Often one sees the wonderful benefit that results from a spontaneous hæmorrhage, and I can recall many cases where this *vis medicatrix naturæ* did more than all our therapy, and therein lies a lesson, and if nature does not act we can do so. In cases of the toxic type I have never seen a venesection do harm, and usually it gives much relief. The pressure usually falls gradually for several days after the removal of blood and it is surprising how slowly it rises again. It may be months before it does so and in some instances, especially in the high pressure that sometimes occurs about the menopause, the fall may be permanent. The little operation is always urgently required where there is pulmonary oedema. In cases where venesection does not affect the high pressure it is probably compensatory and the *optimum* one for the individual.

The vaso-dilators have their place occasionally in emergencies, such as angina pectoris. For rapid effect, amyl nitrite or nitroglycerine are the best, while for more prolonged action sodium nitrite, benzyl benzoate, erythrol tetranitrate and lastly, mannitol hexanitrate are useful. When the pressure is up owing to nervous influences the bromides and luminal are often the best.

Very many causes of hypertension belong to the essential class in that the underlying cause is obscure, and yet, if we can lower the pressure they feel better and no doubt the chances of complications are lessened. Liver extract has been used with much success by some, while others report less favourably. Possibly the differences may be due to the different preparations employed. The drug which in our hands has given the best results here is sodium sulpho-cyanate. It seems that the substance had been tried by Pauli in 1903 on account of its chemical resemblance to the bromides, and he noticed that the blood pressure was lowered in addition to the sedative effect. J. B. Nichols states that he has used it in cases of hypertension for fifteen years with good results. He usually gives five grains thrice daily, and Arthur G. Smith, who was associated with me in the work, and I began with this dose, but soon found that equally good results could be obtained with half this amount. It is interesting to remember that sodium sulpho-cyanate occurs in the body normally as much as 0.1 grm. is excreted daily in the saliva and much more than this in some people, especially if they smoke. We found that the *normal* blood pressure could be somewhat lowered by it and that in cases of hypertension the fall was often great and very long sustained after the drug had been stopped, and the relief of symptoms was decided. The action is equally great in what seem like nervous and toxic cases, and also in the so-called essential ones, but was less evident, or absent, in organic cerebral conditions. Here, probably, the compensatory processes of the body prevent the fall.

As before mentioned, many patients *need* their high pressure, and if it be lowered they feel miserable. An instance of this was one that

Dr. C. L. Taylor brought to my attention, where an elderly man with arteriosclerosis and a blood pressure of 220/120 mm. was given 5 grains of sodium sulpho-cyanate thrice daily for a few doses. Soon the pressure fell to 170/100 mm., but he became mentally deranged and melancholic. The medicine was discontinued and in a week the pressure was up again and with it the mental condition cleared up. When given in moderate doses, such as 2½ grains twice or thrice daily, there are practically no untoward symptoms, although a few have slight gastric disturbance. In very rare instances a skin eruption occurs in the form of dermatitis exfoliativa, but this is so unusual that it need not deter one from the use of the drug.

In conclusion, I would urge that every case of raised blood pressure be carefully investigated, and, if possible, the underlying cause be found and removed. In every case the environment and diet should be carefully regulated. In the nervous cases nerve sedatives are useful. In the toxic ones the toxæmia can often be lessened by purging, diuresis, and in stubborn ones often by the timely removal of blood.

In the many instances where the regulation of the environment and the diet, etc., does not control the condition, and there are no symptoms referable to the pressure, we should be very chary about interfering, although occasionally we can do so cautiously (best, I believe, by the use of sodium sulpho-cyanate), with the object of lessening the strain upon the circulatory apparatus. If upon such careful trial the patient does not feel as well as he did when the pressure was higher such lowering treatment should be discontinued. He evidently requires the increased pressure to compensate for some abnormal condition.

II. LOW BLOOD PRESSURE. Many apparently perfectly healthy people have a pressure well below the average, and in the absence of symptoms a systolic pressure constantly running between 100 and 110 mm. need give rise to no worry and requires no treatment. When the pressure from some cause is below the average for the individual, general tonics and especially strychnine are indicated. When the pressure is for long below what it used to be, in anyone, tuberculosis and Addison's disease should be thought of.

In profound hypopiesis, as it occurs in shock and hæmorrhage, intravenous injections of normal saline or a solution of gum acacia are of value, but when hæmorrhage is the cause, transfusion of blood is often life-saving.

Adrenalin chloride or pituitrin will raise the pressure but the effects are very transient. Ephedrin has proved of special value in these profound falls and the action lasts for hours. In the severe hypopiesis which sometimes complicates spinal anaesthesia, 50 mgs. intravenously may produce a dramatic rise which will enable the operation to be completed with safety.

CANCER

By H. H. PITTS, M.D.

Probably none of the ills which human flesh is heir to has been responsible for more untiring research by innumerable workers in the various scientific branches of medicine in almost every civilized corner of the globe and the expenditure of, one may say, fabulous sums of money, than has cancer. By the same token the fear of cancer in virtually every civilized man and woman over 40 is an ever present one, and the merest suspicion of its presence is enough to cause one to "leave all hope all ye who have this thing," to paraphrase the inscription on the door of Dante's Inferno. Through all the years of effort by legions of painstaking and persevering research workers, including in their ranks many of the most brilliant minds of modern science, we are still wandering in the darkness of ignorance as to the true cause and definite cure of this dread disease.

My time tonight is infinitely too short even to begin to enumerate at any length the various theories advanced, each one of which has its partisans, and so only the high lights of the more recent and more widely accepted ones will be touched upon.

During our college days we had presented to us in the lecture room Cohnheim's and Ribbert's theories and many others, points in each seeming all-sufficient, but weakening in some other aspect—but still these theories have many adherents. The newer theories may be roughly divided into two main groups, (a) Bacterial or parasitic; (b) Cellular.

(A) BACTERIAL OR PARASITIC

Numerous bacteria, parasites and fungi have been heralded as the aetiological factors in cancer, the most prominent supporters of this theory in America being Nuzum, Scott, Glover and McCormack, with Blumenthal in Germany. Nuzum claims to have isolated anaerobic diplococci and streptococci from human and mouse cancer, which, on reinoculation into mice, dogs, but only once in man, will produce cancer. These reproduced tumours, however, are difficult to identify and do not possess all of the histological characteristics of true cancer.

Glover, Scot and McCormack obtained a bacillary organism occasionally showing spore formation, which, inoculated, after special cultivation, into animals, especially the chicken, produces Rous' sarcoma, and in monkeys was said to have produced epithelial tumours of lip, tongue and breast. A toxin was finally produced, an antitoxin and also an agglutinating serum prepared. These experiments have not been satisfactorily confirmed, however.

Blumenthal causes vesiculation on the surface of a carcinomatous growth by focussing the sun's ray through a lens and has found polymorphic organisms in the serum of these vesicles. Inoculation of this serum into animals produces nodules which are, however, more of a connective tissue type, but, with the addition of silica, more typical tumours are

Read before the Osler Society, April, 1930

obtained. He is not entirely certain of the role of this organism, whether specific or merely a carrier for a specific invisible virus.

A (2) COMBINED ACTION OF A FILTRABLE VIRUS PLUS A

CHEMICAL AGENT

Gye and Barnard in England, in 1925, advanced the hypothesis that cancer was caused by a filtrable virus and an element chemical in nature—and from a long series of experiments elaborated the following conclusions:

(1) Every malignant tumour contains an ultra-microscopic virus or group of viruses which may be cultivated, demonstrated and microphotographed, the virus probably being contained within the neoplastic cells.

(2) This virus alone, purified and cleared of all accessory substances, is not capable of producing a tumour by animal inoculation.

(3) When the virus is injected together with a culture of an extract of tumour it produces a malignant tumour. This extract therefore contains a specific factor which renders the virus capable of attacking the normal cell, and transforming it into a cancer cell.

(4) The virus is in no sense specific, since one may produce a tumour in an animal of one species with a virus taken from an animal of another species.

(5) The specific or chemical factor, on the other hand, is strictly peculiar to the species of animal from which it is secured.

(6) Hitherto the specific factor has been found in connective tissue tumours of the sarcoma type. It is particularly abundant and resistant in the Peyton-Rous tumours, while in other tumours it exists in a small quantity and is very labile; in the latter case oxygen-free media must be employed in order to demonstrate it.

Their work has given rise to a great deal of criticism owing to the fact that it has dealt with sarcomatous growths rather than cancer. Professor Gustave Roussy of Paris, at the International Symposium for Cancer Control held at Lake Mohonk, N. Y., in 1926, said that the proof of the inoculability of cancer will be clear only when an epithelial tumour is produced in the same or different species of animal by a medium altogether free from cellular elements, and summarized the present position in regard to the infectious theory of cancer as follows: "It is clear that both early and recent workers have failed to prove the specificity of the organisms they have isolated. The most variable pathogenic agents have been demonstrated, these differing with each worker, and as the tumours produced by inoculation are inflammatory pseudo-tumours, one is led to believe that we are dealing with organisms responsible for secondary infection."

B. CELLULAR THEORIES

Tissue culture, according to the method developed by Alexis Carrel, forms the basis for the bulk of the research along this line, and has very considerably widened the field of investigation of the cancer problem,

especially for exponents of the cellular theory. The cellular theories may be divided into two groups: (1) Cancer is due to a specific principle elaborated by the cell itself; (2) Cancer is caused by abnormality of the cellular glycolysis, these two divisions being the most recent of this school of thought.

1. Cancer is caused by a specific principle elaborated by the cell itself.

Carrel attempted to learn wherein lay the difference between malignant and normal cells of the same type. For his experiments he used the Rous' chicken sarcoma. He was able to isolate two types of cells—fibro-blasts (the fixed cells of connective tissue) and macrophages (the mobile cells of connective tissue or of the blood). He inoculated fibro-blasts in pure culture with the virus of the Rous' sarcoma and found that they resisted it, showing that the aberrant element in sarcoma is not the fibroblast. The macrophage constitutes the malignant element of the Rous sarcoma and also of the spindle cell sarcoma of chemical origin. These cells are fragile, die rapidly, and in dying liberate bodies which Carrel calls "trephones," having the property of producing hyperplasia in neighbouring cells. Thus the malignant element of the Rous sarcoma, known as the virus, is propagated indefinitely.

Carrel claims that the virus is not a definite organism, but is produced only in the presence of cells, and is dependent upon the quantity, activity and even the nature of the cells. In order to produce tumour growth, a number of conditions must be present: (1) A certain strength of the chemical substance; (2) Cells in a given condition; (3) A certain susceptibility of the organism.

However interesting these observations are, and however much they may widen the horizon of the field of cancer causation, certain reservations must be made, for it does not seem fair to apply too dogmatically, results of tissue cultivation *in vitro* to phenomena arising in the body, where such intimate cell interrelationship exists.

2. Abnormality of cellular glycolysis as the cause of cancer. While the cytobiologists, bacteriologists, biophysicists, etc., endeavour to trace the cause of cancer to some factor in their particular field, the biochemist approaches it from a different angle, and Warburg of Germany has tried to show this cause in disordered cell metabolism. Two phenomena are noted in normal cells insofar as their content in carbohydrate, and especially in glucose, is concerned. (A) *Glycolysis*, which splits the glucosa molecule and produces lactic acid. (B) *Respiration*, which on the contrary results in the building up of carbohydrate, one or two molecules of lactic acid disappearing for every molecule of oxygen consumed.

According to Warburg these are the key to the phenomenon of living cell metabolism and are particularly pronounced in tissues of active growth such as the embryo, or in cancer. In the latter, loss of the normal rhythm between respiration and glycolysis occurs affecting cellular respiration. While normally there is a real synchronism between destruction and elaboration of glucose, in cancer it is lost, but the respira-

tory rate is not necessarily affected. As to the cause of this phenomenon Warburg believed that cancer originates in the absence of oxygen, the above being the foundation of his theory. As all normal tissues possess a double metabolic power (glycolysis and respiration) it must be assumed that the constituent cells have gradually become specialized, some in the phenomenon of respiration others in glycolysis. Should anything occur to deprive the tissue of oxygen, be the cause mechanical, inflammatory or otherwise, the only cells which persist are those capable of glycolysis, and the survival of these cells is further favoured by the death of neighbouring cells.

Such is the new conception of cell cancer.

It would appear that the present trend of thought in regard to the cause of cancer is leaning more and more toward an intrinsic disturbance of cell life, the causes of which are probably multiple. Recent work tends to strengthen the proof that cancer is not due to a living agent comparable to those responsible for the infectious diseases, nor does it appear due to a virus; it is therefore not a communicable disease, and this fact should be spread among the public. Cancer then appears to be a special disease whose aetiological factor is still unknown, whose biological and morphological characteristics appear to be distinctly opposite to those of inflammatory phenomena.

Whatever may be the method of development, the inflammatory reaction persists only in the presence of the pathogenic agent which gave it birth. It is thus for example that the nodular or ulcerous lesions of syphilitic, mycotic or dysenteric nature come to a stop and heal, under therapeutic action, which causes the causative spirochaete, fungus or bacillus to disappear. Thus the inflammatory processes stop or even regress as soon as the agent is removed. Cancer on the contrary appears to result from the combined action of known and unknown causes which produce in the cell disturbances of growth or function resulting in quasi-fertility. This fertility which is transmitted to daughter cells constitutes the essential characteristic of cancer cells; it is found in no other morbid process. It matters little whether the occasional or determining agent disappears, be it chemical, physical or living, the new characteristics of the cancer cell will continue to follow the established rhythm. The study of the latent phase of coal tar cancer in animals is a good example of this fact.

Thus the two great morbid phenomena which attack the organism— inflammation and cancer—appear to us today, from the biological point of view, distinctly different one from the other. And perhaps it is because we have mistakenly tried to bring them together that the majority of investigations on the origin of cancer have, up to the present, resulted only in failure.

Maud Slye of Chicago has done voluminous and interesting work on heredity in mouse cancer and maintains that it plays an important role in the causation of cancer, and in her laboratory has been able actually to regulate the percentage of cancer incidence in mice by selective breeding. She states also that apparently two factors are necessary

to produce cancer: (1) an inherited susceptibility to the disease; (2) irritation of the right kind and degree, applied to the cancer-susceptible tissues. For example, in mice that inherit susceptibility to spontaneous breast cancer only, cancer does not occur from irritations other than those applied to the breast tissue. If a mouse is a member of a non-cancerous strain, a similar irritation would produce only scar tissue, which would eventually be wholly or partly absorbed. She cites one notable case of a mouse, a member of a strain carrying both carcinoma and sarcoma, which when struck on the face by a cage door developed both at the site of injury.

We certainly cannot lightly disregard the part that injury or chronic irritation plays in cancer, for almost every day some instance is brought to our attention, where the possibility of this factor being a direct or contributing one in the existing lesion, gives us, at any rate, considerable food for thought. Many examples are quoted, as you will no doubt remember, from your lectures at college, of the marked incidence of cancer at special sites from a definite type of irritation, the betelnut chewers of India (cancer of the cheek), carcinoma of the skin of the abdominal wall in Thibet, where charcoal braziers are carried beneath the cloaks in winter; carcinoma of the oesophagus in males in China. Of course the question of whether or not the irritation is the sole factor must be considered in these instances.

One so often hears the remark that cancer today is more prevalent than say 20 or 30 years ago, and the usual explanation for this is that we are living in an age of high specialization and marked progress, and medical science has participated in this advance so that much more adequate means of diagnosis is at our disposal and command. This, however, is not the whole story, and I believe that cancer is actually rather than relatively more prevalent today, and that the incidence in younger persons is very definite. Paget holds the view that if people lived long enough they would eventually all have cancer, and he believed that the fact that the expected length of life has been prolonged from 40 years in 1850 to 58 at the present time, may account for the increase in both the morbidity and mortality of cancer. Statistics from Holland, to quote one example, give a mortality of 3.4 per 10,000 in 1867 and 11.2 per 10,000 in 1924. One might quote other statistics from various countries *ad nauseam*, all would show definite increases.

I had intended primarily to incorporate in this paper the percentage occurrence of carcinoma in the various sites most frequently involved in the human mechanism, but decided, as I became more and more involved in the maze of theories of causation, that I had much better let well alone, and touch on only the more recent and more widely accepted theories.

In conclusion, I wish to impress upon you further the present chaos that exists regarding the aetiology, prognosis and treatment of cancer, and the broad unexplored field that still lies open to the investigator. Most assuredly, to him or her who is so fortunate as to discover the cause, and a more satisfactory treatment, of cancer, the plaudits of all the world would be little enough to mark the appreciation of suffering humanity for its liberation from this ubiquitous monster.

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APPLICATIONS OF BACTERIOLOGICAL FINDINGS TO DIAGNOSIS

By H. W. HILL, M.D.
Director; V. G. H. Laboratories

What does a positive report mean on a throat (or nose) culture taken for diagnosis of diphtheria? What does a negative report mean? What does a doubtful report mean?

Exactly similar questions may be properly asked regarding the same sorts of report form suspected gonorrhoea pus, whether of genito-urinary origin or from an eye; in examinations of suspected tuberculous sputum, suspected tuberculous urine, suspected tuberculous spinal fluid or other suspected tuberculous material; also, in principle, in the various tests for typhoid fever, undulant fever, etc.

Broadly the same sorts of question apply to all routine bacteriological reports,—and *mutatis mutandis*, to all laboratory reports on any test of material from a patient.

Broadly also, the answers in all cases are of the same order,—a *positive* report means that the *specimen submitted* showed the organism or reaction suspected so far as accepted up-to-date methods *applicable in making rapid reports* is concerned. A *negative* report means that the *specimen submitted*, after a similar thorough examination, did not show the suspected organism or reaction. A *doubtful* report means that a thorough examination failed to yield conclusive evidence of the presence of the organism or reaction sought, but gave indications, not conclusive,

that it might be present.* In such cases obviously another specimen should be submitted. Meantime, the strict truth has been reported; and both the patient and the physician are protected thereby.

Simple, direct, inevitable as the above world-wide procedures in reporting are—the concentrated wisdom of world-wide laboratory experience in its most concise form—the reasons for these forms of report and the applications properly to be made of them in practice are not always as clear to the non-laboratory professional man as to the laboratory professional man; and are therefore here recapitulated.

First. The laboratory obviously examines the *specimen submitted*, not the *patient*. In the *patient* may exist some clinical condition; a proper specimen, properly collected, really representative of the particular discharge involved, should yield (if the *suspected* condition really exists) the given organism or reaction responsible for that condition. Primarily then the laboratory is dependent upon the physician for a *truly representative specimen* of the proper discharge, submitted in proper condition for examination, at the proper stage in the progress of the case. From that point on, but from that point only, is the laboratory responsible. So obvious is this that its mere statement is sufficient to gain instant acceptance.

The laboratory examines the specimen by the best modern accepted methods *that will yield a prompt diagnosis*; because, of course, the physician must have a prompt return, in order to take action during the period when such action will be of some use. Hence the aim of every laboratory is to use those methods which will yield as high a degree of accuracy as possible *within a time limit* which will make the return of practical clinical value.

To illustrate—in diphtheria diagnosis, the actual scientific determination of *the presence* and degrees of *toxicity* and of *virulence* of diphtheria bacilli found may be a matter of days or weeks, concluded perhaps only after the patient is dead or well. Exactly the same might be said of gonococcus or tuberculosis examinations, except that such scientific proof may be still more time consuming. Such scientific accuracy is impossible in practice, because of its really enormous expense; and would be useless practically because of its extreme slowness. Fortunately, morphological methods, 95 to 99% accurate, are available in diphtheria, in gonorrhoea and in tuberculosis, and can be used promptly and to advantage. As contrasted with the 50 to 75% accuracy of purely clinical diagnosis these methods are quite remarkably efficient and constitute a very real advance on older methods. But they are subject to the disabilities of all morphological tests—the variability of the germs sought and the resemblances between them and other non-significant germs; therefore these methods cannot live up to the idealistic halo with which the non-laboratory professional tends to endow them. It is a matter of universal remark that it is the laboratory man who is continually pointing out the disabilities of routine laboratory tests—it is the non-laboratory professional who idealizes them and can hardly be prevented from worshipping them.

* In all tests, positive and doubtful specimens at least should invariably be seen by at least two expert workers, for check purposes.

To illustrate further—a laboratory report of “diphtheria culture positive” means this, *and nothing more*—that the specimen submitted, when grown for 18 hours in the incubator on Loeffler’s serum, yielded organisms giving the *morphology* of diphtheria bacilli. The report does *not* say, or imply, or desire to imply, that these germs are pathogenic, toxic or virulent; nor that, granting them these features, the germs are responsible for any of the patient’s troubles—nor, again, that the patient has any troubles at all, from them or from any other source. All these are matters wholly for the physician to decide from his clinical examination of his patient. All that the laboratory can say is that *the specimen submitted* showed the presence of the organisms reported. How they got into the specimen in the first place, and what they may mean to the patient are *wholly* matters for the physician to decide in each particular case.

True, the laboratories of the world, from long experience and slow, patient investigation of many hundreds of thousands of cases, can offer certain guiding principles to the practitioner which he may use in each case—rules so well and so long established that no practitioner is justified in ignoring them. Some of the more important ones are:

1. The finding of diphtheria bacilli in a specimen by the laboratory, provided all proper technique was followed in collection, etc., of that specimen, shows that the organism was present in the *patient* as well as in the specimen.

2. The findings of clinical *signs* or *lesions* in the patient thus shown to harbor diphtheria bacilli *imply*, but do not *prove*, that the diphtheria bacilli found are responsible for the signs or lesions present. Obviously if the diphtheria bacilli found are non-virulent, they cannot produce lesions of any kind—and if lesions nevertheless be present, they must be due to some other cause. (Virulence can only be determined in the laboratory by special tests, the most rapid of which cannot be ready for three days after obtaining the positive culture.)

Again, even if the diphtheria bacilli be shown to be virulent, this further *implies*, but does not yet *prove*, that they are affecting the patient, since it is true that the patient *may* be immune to diphtheria. If the immune has no lesions, evidently any virulent diphtheria bacilli proved to be present must be harmless to him, and he is a well “carrier.”* But if he has active severe lesions, and yet is immune to diphtheria, the virulent diphtheria bacilli proved to be present must be none the less harmless to him; his lesions must be due to some other infection, Vincent’s, streptococci, etc. He is a “carrier,”* suffering from some intercurrent, non-diphtheria, infection.

Nevertheless, in actual practical everyday work, the examination of throats, etc., for diphtheria bacilli has proved of immense service, despite the occasional drawbacks,—why? Because the total of the sources of error above noted in diagnosing diphtheria by culture are far less than the sources of clinical error—because, therefore, usually (in

* Dangerous to others because of the virulent germs he carries; but not suffering from these germs himself.

about 99% of cases) a "clinical sore throat + diphtheria bacilli" is due to the diphtheria bacilli found.

Negative reports on specimens submitted for diagnosis from clinically suspicious throats are dependent for accuracy first and chiefly on the *representative character* of the specimen obtained; second, on the *thoroughness of the examination* given to the specimen; only for the second of these two items can the laboratory be held responsible. A negative report is universally understood to be inconclusive and in the face of clinical or other reasons for suspicion should always be repeated.

This is particularly true in recovered cases where *release* from isolation is sought. The germs are doubtless reduced in number, may appear on the surface of the throat only intermittently, and therefore are very subject to lack of transfer to the media, or to transfer in very small numbers; hence, they are not difficult to overlook.

To *prove* their absence from the patient is very difficult. Investigations by the writer in Boston, thirty years ago, showed that a *single* negative result in a case for release, if acted upon, resulted in releasing no less than 30% of all the patients while still infective. The demand that *two* consecutive negative cultures from both nose and throat should be obtained before release cut down the number of infective persons released to 2 or 3%. The demand that *three* consecutive negative cultures from both nose and throat be obtained before release cut down the number of infective persons released to one in 1,000, or thereabouts. But note that even with three consecutive negatives, *some* of those released were still infective.

The principles thus painfully established in the now very familiar cultural diagnosis of diphtheria apply in all bacteriological diagnostic work, varying in detail with each disease, i.e., with each specific organism concerned—but yielding the same general story in all.

Above diphtheria is discussed in detail; later articles will deal with gonorrhoea, tuberculosis and typhoid.

SUMMARY

1. Definite clinically suspicious symptoms of diphtheria call for immediate antitoxin, whether a culture be taken or not, and without regard to the returns from any culture taken, whether positive, negative or doubtful. The rule in such cases is—first, antitoxin; then take a culture; and later be guided as to giving more antitoxin by the subsequent developments of the case as well as by the cultural results.

2. Given a sore throat or nose *not* definitely clinically suspicious of diphtheria, and a positive diphtheria report from it, the physician is morally bound to assume that the patient has diphtheria; only conclusive proof that the germs are non-virulent or the patient immune to diphtheria can justify any other conclusion. Since such proof to the contrary can seldom be promptly available, immediate treatment of the case with full therapeutic doses of diphtheria antitoxin is absolutely indicated.

3. Negative results must always be discounted; while positive results are about 99% correct; negative results *for diagnosis* are only about 80% correct; and for release still less correct.

4. Suspicious or doubtful reports mean exactly what they say—that while no final decision has been reached, the patient is not relieved of the possibility of the infection suspected and “safety first” demands treatment at once and another specimen, in the hopes that the latter will prove conclusive. Such reports are not given in release cases, since in such cases, the negative result hoped for is the only one on which action is based. Hence release cultures are reported positive or negative only.

(Occasionally of course, unsatisfactory results, due to overgrowth or no growth or other accidents may be obtained in any bacteriological culture.)

* * *

“DISTINCTIVE TASTES” OF PASTEURIZED MILK AND RAW MILK

At the Board of Trade Health Bureau meeting of Wednesday, January 7th, 1931, twenty samples of milk—ten pasteurized milk, and ten certified raw milk—externally unidentifiable except by a letter of the alphabet attached to each, (corresponding with a written key), were served out to ten members, with the request that each would compare the two bottles handed to him in any way he pleased, but of course using taste as one test, and state whether they were both pasteurized, both raw, or one raw and one pasteurized; and to state, in the latter case, which was which.

The members, about thirty in all, collaborated in groups, so that as a matter of fact 28 returns were made on the 20 bottles.

The returns, despite their paucity, have been considered worth classifying—as follows:

On the 10 pasteurized milks, 16 returns were made. 4 returns were “doubtful”—these four judges would not decide. Of the other 12 returns, 7 stated (correctly) that the milk was pasteurized; 5 stated (incorrectly) that it was raw.

On the 10 certified raw milks, 12 returns were made. One return was “doubtful”—i.e., the judge could not arrive at a decision. Of the other 11 returns, 6 stated (correctly) that the milk was raw; 5 stated (incorrectly) that it was pasteurized.

Thus of 28 returns, 13 were correct, 10 were incorrect, and 5 were non-committal.

This result was compared with the outcome of “deciding” which samples were pasteurized, which raw, *by the flip of a coin*, taking the first 28 consecutive flips.

In order to parallel the above results, the ten pasteurized samples were subjected to the first 16 decisions of the coin, the 10 raw to the remaining 12 decisions. The coin-flips were correct as to the pasteurized

milk in 6 instances out of 16; as to the raw, in 7 instances out of 12. (The respective human decisions were correct in 7 and 6 instances.)

Hence the human judges were correct 13 times in 28 returns—and so were the coin-flips! In other words, in this test, the opinions of interested, educated and intelligent citizens were worth on this subject no more and no less than the tosses of a coin.

It is more than ever difficult now to believe that the "distinctive" tastes of raw and pasteurized milk are distinctive to the "average citizen," as is so often stated.

The following are the actual results of the human judgments obtained:

<i>Pasteurized Milk</i>	<i>Certified Raw Milk</i>
P R	R
R P	R
P	P
P R	R R
R P	P
? ?	P
R	P
?	R
?	P
P P	? R

R = Adjudged Raw P = Adjudged Pasteurized
 ? = Adjudged as Undecidable

* * *

PROGRAMME OF THE 33rd ANNUAL SESSION VANCOUVER MEDICAL ASSOCIATION

Founded 1898

Incorporated 1906

GENERAL MEETINGS will be held on the first Tuesday and CLINICAL MEETINGS on the third Tuesday of the month at 8 p.m. Place of meeting will appear on the Agenda.

- February 3rd—General Meeting:
 Speakers—Dr. F. N. Robertson; "Some Unusual Uses of Common Drugs."
 Dr. Wallace Wilson; "Stenosis of the Mitral Valve."
- February 17th—Clinical Meeting.
- March 3rd—General Meeting:
 THE OSLER LECTURE—
 Dr. R. E. McKechnie; "Reminiscences of Forty Years' Practice."
- March 17th—Clinical Meeting.
- April 7th—General Meeting:
 Speaker—Dr. C. F. Covernton; "Problems of the Primipara."
- April 21st—Clinical Meeting.
- April 28th—Annual Meeting.



A.D. 1853 N° 365.

Deodorizing Cod-Liver Oil.

LETTERS PATENT to Sir James Murray, Knight, Doctor of Medicine, of Dublin, for the Invention of "**IMPROVEMENTS IN DEODORIZING COD LIVER OIL, IN RENDERING IT MORE AGREEABLE AND EASIER TO USE, EITHER BY ITSELF OR MIXED, AND SO AS TO BE CAPABLE OF BEING ADMINISTERED IN LARGER QUANTITIES AND WITH GREATER SUCCESS.**"

Sealed the 13th April 1853, and dated the 11th February 1853.

PROVISIONAL SPECIFICATION left by the said Sir James Murray at the Office of the Commissioners of Patents, with his Petition, on the 11th February 1853.

I, Sir JAMES MURRAY, Knight, Doctor of Medicine, of Dublin, do hereby declare the nature of the said Invention for "**IMPROVEMENTS IN DEODORIZING COD LIVER OIL, IN RENDERING IT MORE AGREEABLE AND EASIER TO USE, EITHER BY ITSELF OR MIXED, AND SO AS TO BE CAPABLE OF BEING ADMINISTERED IN LARGER QUANTITIES, AND WITH GREATER SUCCESS,**" to be as follows:—

- 10 My Invention consists in treating the oil so as to impregnate it most intimately with carbonic acid gas, by subjecting it to great pressure and agitation and passing streams of this gas through it.

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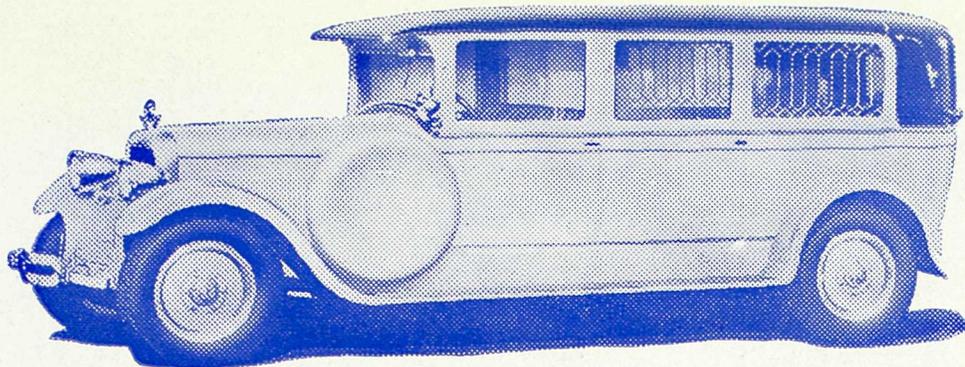
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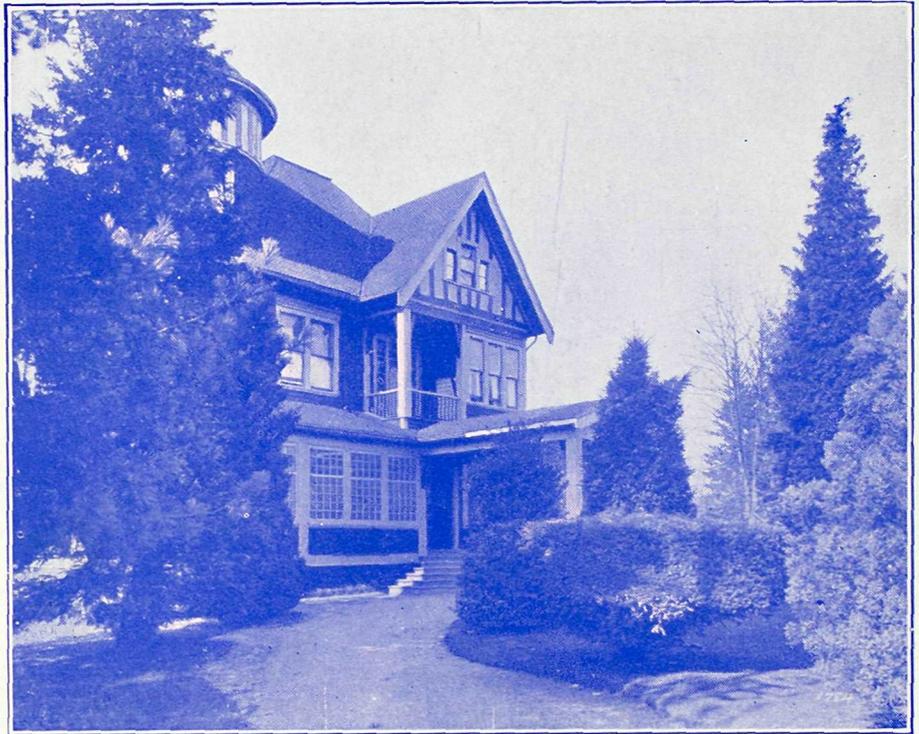
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