

mal. Endarteritis obliterans of anterior and middle cerebral arteries, but no complete occlusion nor thrombosis was found. No aneurysms nor hemorrhage. No gross macroscopic lesions of brain. Of the other organs of interest the kidneys showed a condition of subacute parenchymatous nephritis.

I believe that we have sufficient proof of syphilis in the keratitis, iritis, adenopathy and clinical course of the disease, although the post-mortem findings are not conclusive, being limited in the brain mostly to the arteries. I will not deny that there may also have been a uremic element in the case. I will offer no explanation for the presence of leucin in the urine, occurring as it does most commonly in acute yellow atrophy of the liver, phosphorus poisoning, leukemia and various infectious diseases.

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DISCUSSION.

DR. MOYER—It needs no emphasis at this time to point out the immense importance of syphilis as a causative factor in disease of the brain and spinal cord. There is, however, a trifling misconception growing out of the mere use of terms. I see the doctor has labeled his paper, cerebral syphilis with report of cases, and he referred to the fact that Gowers had no chapter in his work on this subject. He would probably not find one upon syphilis of the spinal cord, and yet most of our German confrères have a chapter devoted to these subjects. It is not, however, that the English and American neurologists and the practitioner of general medicine do not recognize the importance of syphilis in the etiology of these disorders. The term cerebral syphilis and syphilis of the spinal cord, as used by our German confrères, is incorrect, as it puts into the nomenclature a new pathologic entity, like chorea, tabes, etc., a something which is caused by syphilis. We do not think it is possible to distinguish clinically the cases of tabes and paretic dementia having their origin in syphilis from those caused by other conditions. There is no doubt that most of the cases occurring in the meninges along the base of the brain, involving one or more of the cranial nerves, or a cerebral peduncle, or perhaps a single branch of the third nerve, or those which occur with a comparatively abrupt onset of symptoms, those cases are almost without exception syphilitic, but I do not think this justifies the term cerebral syphilis; I should say rather they were such and such conditions, affecting certain portions of the brain as the case might be, which were caused by syphilis. There was one case described in the paper which to my mind might justify the term cerebral syphilis. It was the one in which a diffuse endarteritis was found, but no local lesions. I believe that makes a clinical type that perhaps ought to have a chapter in works upon practice labeled cerebral syphilis. I have seen several such cases in which there was a rather abrupt onset with scarcely any local paralysis, early interference with speech and sluggishness of the pupils; some exaltation of the mental functions, rapidly passing on into mild delirium, stupor and death. At the post-mortem, the findings are not different from those described by the doctor; but, as I say, this discussion really relates to terms and not to facts. The great importance of the doctor's paper is in directing our attention very pointedly to the influence of syphilis in these affections, and particularly in the cases that he describes, those presenting irregular manifestations and those with a comparatively abrupt onset, but this has been recognized for the last twenty years. Within the last eight or ten years, there has been an increasing importance attached to syphilis in the slow progressive degenerations, the ordinary cases of paretic dementia and tabes, and we must now admit, although

still denied by a few, that syphilis causes 80 or 90 per cent., or even more of these cases. There is, too, a striking difference in regard to treatment in this class of cases. Those with an abrupt onset are usually quite amenable to treatment; those in which the symptoms come on slowly and insidiously are most rebellious to treatment, no matter how energetically pursued, and I think the reason is found in the fact that the changes in these other conditions are really secondary. Take, for instance, an endarteritis that leads to a rupture of an artery or thrombosis. The secondary conditions are not amenable to anti-syphilitic treatment. The same applies to cases of insidious onset, in which specific treatment does so little and yet it is undoubtedly true that they are of syphilitic origin. The reason of it is that this slow, progressive syphilitic endarteritis has set up the secondary changes, sclerotic in character, and when those changes are once set up, it makes no difference what the cause is, the treatment is practically *nil*, no matter how energetically pursued. But coming to the practical point; the evidence is now so strong that in all these cases it is the duty of the practitioner, even in the absence of a history of syphilis and even where it is denied, to give an energetic anti-syphilitic treatment. I prefer the mixed treatment, but give the iodids in relatively very large doses.

DR. WM. HESSERT—I think that Dr. Moyer and I agree, only that we express our views in different ways. I meant to say that the subject of cerebral involvement by syphilis has been described as an entity under a special heading of late by German authors; especially Oppenheim in his last work gives a classic description of cerebral syphilis, meningitis and inflammation of the cerebral cortex. I admit certainly that the subject of cerebral syphilis is as old as the subject of syphilis itself, and thousands of years ago we may say that mankind was affected with the cerebral involvement just as they are now; but the point is that it was not recognized as of late years.

PNEUMONIA; THE NON-ALCOHOLIC TREATMENT.

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Contrary to all that has been written showing the pernicious effect of alcohol on the system in health and disease, and contrary to the admonitions of some of the greatest lights of the medical profession, past and present (a few of whom I may mention, viz., Sir Astley Cooper, Prof. Willard Parker, Dr. Benjamin Ward Richardson, Dr. Wm. B. Carpenter, Dr. N. S. Davis and numerous others equally as well known), as to its deleterious effects in disease, and notwithstanding the fact that the mortality rate from pneumonia is higher than fifty years ago, still we persist in using alcohol in pneumonia crouposa.

You can scarcely pick up a text-book or journal for reference as to the treatment of pneumonia, without being confronted with the fact that you must stimulate your patient with alcohol.

We boast of the great advancements made in surgery and medicine, and contrast our knowledge of to-day with that of fifty years ago. The contrast is certainly very great; our knowledge of materia medica and therapeutics is certainly very extensive; in diagnosis we have become very proficient, and we differentiate one disease from another with that degree of certainty that if some of the lights of the medical profession who lived fifty, or even twenty years ago, were to come to life, they would be amazed at the advancement made in diagnosis alone.

In treatment of disease their surprise might not be so great, because of the fact that we do not differ materially from the various ideas entertained in the past.

In pathology we have made the greatest advancements, and inasmuch as this article has to do only with pneumonia crouposa, we shall dwell on it more or less from a pathologic standpoint in order to show

why alcohol is contraindicated, and why it should not be used in pneumonia.

Pneumonia crouposa is distinctively an inflammatory disease, and as in all cases of inflammation occurring in any part of the body, so in the first stage of pneumonia we find a hyperemia or increased vascularity; engorgement.

In the second stage we have exudation, red hepatization, in which the alveoli and bronchioles become filled with a sero-plastic exudate, and that portion of the lung affected becomes partially solidified, and in it the blood current is apparently stopped.

The third stage is the one in which we find fatty degeneration of the exudate, and an extrusion of the same. It is called the stage of resolution.

Space will not permit, nor need we make a more elaborate description of the various minute pathologic changes that take place in the affected lung in order to prove the theory I advance. It will suffice to say, for all practical purposes, that we have an inflamed condition of lung to deal with, a congested, engorged condition of the blood vessels of the part affected, and later on a state of solidification in which all blood is excluded.

Now then, inasmuch as the foregoing statements are beyond dispute, having been accepted by all, we shall endeavor to prove from the above facts why alcohol is contraindicated in this disease.

In the first stage we find on microscopic examination the blood vessels to be distended with blood; that is to say, the volume of blood has been enormously increased and the capillary network surrounding the alveoli is greatly enlarged and all adjacent portions of bronchioles are similarly engorged; thus we have a hyperemia marking this stage of the inflammatory process, lasting from twenty-four to thirty-six hours.

We will now see what affect alcohol has on the system and compare it with the above.

According to the latest views, the physiologic action of alcohol on the arterial system is that it causes the heart to beat faster but not stronger. The increased heart beat is attributable to the dilatation of the arterioles; that is, there is less resistance to overcome and the heart therefore beats faster. It also, through its action on the vasomotor nerves, paralyzes the minute capillary vessels, and fills them with blood. In other words, it produces an hyperemic condition substantially the same as we find in the first stage of pneumonia.

Now then, if alcohol produces in health this condition of affairs, why is it not contraindicated in an already congested lung? Does it not assist in complicating a difficulty we are endeavoring to overcome?

Again, it is a well established fact that alcohol in the system diminishes oxidation, and inasmuch as the oxygen needed in the blood is received from the pulmonary air cells by the hemoglobin and serum of the blood, and in them conveyed to the systemic capillaries, where it comes in contact with and exerts its influence on every cell and structure of the body, and furthermore the feeble affinity it manifests for oxygen is offset by its strong affinity for water, albumin and hemoglobin. Therefore if alcohol is introduced in the circulation, instead of uniting with the oxygen, it manifests its strong affinity for the hemoglobin and sero-albumin, and consequently interferes with the reception of oxygen from the pulmonary air cells. In this manner the presence of alcohol prevents the hemoglobin from being converted into oxy-hemoglobin in

the pulmonary arterioles, and in the same ratio diminishes the amount of oxygen conveyed to the systemic capillaries, and in the same ratio the nerve sensibility and metabolic changes diminish.

Alcohol, as can be demonstrated, is a narcotic poison in large doses, and kills through suffocation by its paralyzing influence on the respiratory nerve-centers, and in small but continued doses it produces the same result by the structural changes which it produces in the organs and tissues of the body. By its action on the blood cells it checks oxidation by limiting their power of eliminating carbonic dioxid and of absorbing oxygen.

Therefore, if, as is above shown, alcohol prevents the elimination of carbonic dioxid and the reception of oxygen by the blood cells, how can we expect by introducing it into the circulation, that it is going to afford any relief to an already congested lung which is endeavoring to rid itself of an overabundance of carbonic dioxid, and to obtain more oxygen?

During the stages of red and gray hepatization up to the point when resolution takes place, we find the area of lung involved almost solid. The venous circulation falls abnormally and the right heart weakens from over-work, and as a result of this condition the patient has a flushed, anxious and slightly dusky countenance. We have substantially asphyxia, and in many cases the patient is in a semi-comatose condition. This condition is the result of carbonic dioxid poisoning and a lack of oxygen. The asphyxia is not necessarily due or dependent upon the amount of lung tissue involved, nor to any change in the heart muscle, but rather a toxemia which undoubtedly weakens the heart.

It is also an established fact that all natural molecular or metabolic changes, nutritive, secretory and disintegrating, taking place in the living tissues, are absolutely dependent on the presence and movement of blood containing its natural proportion of oxygen. Consequently it must follow that when this free movement of blood is interfered with, as it is in the solidified portion of the lung (as we find it in the second stage of pneumonia), the metabolic molecular changes taking place in the lung are interrupted, and as a result the blood is loaded with toxins. It is very evident, therefore, that we should direct our treatment so as to assist the system in ridding itself of these toxins.

Can we, by administering alcohol, accomplish this? I say unhesitatingly, no. I say so because of the fact that it is contraindicated, for the following reasons: The effect of alcohol in the system is to paralyze the minute capillary vessels and fill them with blood, thereby substantially producing a stasis. The removal of toxins, which have been produced by the diseased condition of the lung, is interfered with, and we have as a result a systemic poisoning.

How often have we observed, during the second stage of pneumonia, when a patient is apparently dying from asphyxia, the subnormal temperature, pulse hardly to be felt, cold perspiration, respiration labored and stertorous, a cyanotic appearance of the face and a more or less comatose condition? How often do we attribute all of the above symptoms to the venous stasis and carbonic dioxid poisoning, resulting from the diseased portion of the lung, to which it is true they may be due? But as a rule what have we been doing for the patient during this time? Filling him up with alcohol in order that we may, as

we have been taught, tide him over this condition by stimulating him.

The question now arises: How much of the above condition can we attribute to the disease and how much to alcohol? We shall answer the question in the following manner: The imbibition of small doses of alcohol produces through its action on the cerebro-spinal system a relaxation of all terminal capillaries, and there is no rise in temperature. In large doses or a very frequent repetition of the small dose, there is a partial paralysis of the terminal capillaries, a lowering of temperature, loss of muscular power, lack of coördination and finally more or less delirium, anesthesia and collapse. In larger doses it is a true poison.

Now then, inasmuch as the physiologic efficacy of alcohol is considered to be one ounce well diluted, taken in divided doses during twenty-four hours, any considerable quantity taken above this amount may be considered toxic, and being a true poison note the effect it produces on the system. In complete alcoholic poisoning, the temperature is subnormal, pulse hardly to be felt, cold perspiration, respiration labored and stertorous, pupils do not react to light and the lips are blue.

Observe the similarity of the symptoms of alcoholic poisoning and those of carbonic dioxid poisoning of the second stage of pneumonia, then ask yourself if you are justified in giving alcohol in any form in this condition. No more dangerous, pernicious treatment has ever been adopted or advised than to give, in this critical stage (or any other) of pneumonia, alcohol in any form or quantity. You are no more justified in administering alcohol in pneumonia than you would be in giving it in a case of alcoholic poisoning.

Dr. Henry Hartshorn stated in a paper read before the College of Physicians of Philadelphia, 1888, that pneumonia was much more fatal then than when the practice of venesection prevailed; and that the increased mortality could not be explained by change in type or change in constitution, and that it must be due to change in treatment; *ergo*, the modern treatment must be wrong. Then he goes on to prove, by statistics taken in Great Britain and Europe, and also this country, that the mortality from pneumonia was relatively twice as great then, than it was fifty years previously. It would be inferred from Dr. Hartshorn's article (as he says) that the reason for this greatly increased mortality, was we had dropped venesection and taken up a new line of treatment, and that, *ergo*, this new line of treatment was wrong.

Dr. Hartshorn is not alone in thinking that the modern method of treating pneumonia is wrong. Many of the older members of the profession will tell you that when they began the practice of medicine forty or fifty years ago, when it was customary to give a dose of calomel and bleed for almost everything, they did not have such a high death rate as we do to-day.

Dr. N. S. Davis, at the AMERICAN MEDICAL ASSOCIATION meeting in 1888, during the discussion on pneumonia, stated that it was a curious fact that the mortality was greater then than it was forty-five years before. He referred to the statements of a Canadian physician, "who stated that during a practice of thirty years he had adopted three plans of treatment. During the first decade the treatment consisted of blood-letting; the second decade constituted the transition from the blood-letting to the expectant plan; and the third the stimulating method. He found in

footing up his records that the mortality was least during the first decade and greatest during the last."

Although I must agree with Dr. Hartshorn and those older members of the profession as to the high mortality rate, still I can not attribute the higher mortality in this age to the lack of venesection. Some recent writers advocate venesection in special cases. I have never seen a case where I felt justified in venesection. I never could see any reason in blood-letting, particularly in pneumonia. I have noticed that those who do advocate blood-letting in pneumonia do so only in the early stages. The object, apparently, in venesection early in the disease is to relieve the over-loaded venous circulation, thereby assisting the right heart.

There are those of us who have never done any blood-letting, who have had sufficient experience in the treatment of pneumonia to warrant us in saying we do not think it should ever be resorted to. We have seen cases get well without being bled, that had they been attended by one who advocated blood-letting, and bled, the result would undoubtedly be attributed to the blood-letting.

The question of venesection has been so thoroughly discussed in years past, that we of this generation have considered it settled, and relegated to the past, but ever so often it seems we have to be reminded of the fact that it still has its advocates. We have such authors as Ziemssen, Hare, Austin Flint, Bartholow and others of equal note, to be cited as against venesection in pneumonia; consequently we say, do not bleed in pneumonia.

I attribute the favorable results in pneumonia of fifty years ago chiefly to two things: First to the almost universal use of calomel, and second, to the very limited use of alcohol. Calomel was used generally from the first to the last stage of the disease. The effect of this administration as a rule was to cause the bowels to move freely, oftentimes causing watery discharges; thus the physicians of those days were unconsciously using a remedy sanctioned by the leading lights of the profession of to-day; particularly by those who believe that pneumonia is a germ disease. Binz and others claim that calomel partly becomes converted into corrosive sublimate in the digestive tract, there unfolding its powerful antiseptic action. This disinfection and its coexisting purgative action, cleans the bowels to a very great extent of whatever germs may be present.

Whether we view the use of calomel from the standpoint of its being a germicide or not does not matter, but we do know that acting as a purgative it increases intestinal secretion, and has an indirect effect on the circulation by lowering the pressure of blood in the blood vessels, thus substantially producing what may be called a physiologic blood-letting, without the venesection and without the shock. It is not necessary that we should use calomel; any active cathartic or purgative would do as well; but the free movement of the bowels at the onset is indispensable, in fact is one of the cardinal principles in the treatment of pneumonia.

It is generally understood that if no complications set in, pneumonia (crouposa) is a very regular and typical disease. It is self-limited, and inasmuch as it is such, our chief aim should be to not interfere too zealously in natural processes, but by judicious handling bring about a favorable termination. Generally speaking the less medication the better.

Fothergill used to say that about every new remedy had to be used in pneumonia. The chief remedy in this disease is plenty of air. Liebenmister (Ziemsens's cyclopaedia) says: "A patient with a high temperature can not take cold." Do not be afraid of too much air; good fresh air will be found beneficial in any case; but be careful to avoid drafts. See that your patient has plenty of nourishment, preferably in liquid form.

Do not give alcohol in any form during any stage of the disease. I firmly believe that the use of alcohol has been the chief cause of the high mortality in recent years. Let the fever alone. The danger is not from the fever but rather the heart, and inasmuch as all of the recent antipyretics act as heart depressants, we should be very cautious in their use in pneumonia. Stimulate your heart if necessary with digitalis, using a good reliable fluid extract; strychnia or nitro-glycerin. Oxygen should always be thought of when there is a tendency toward cyanosis. I have seen such excellent results follow its administration that I unhesitatingly say: Use oxygen in cyanosis and use it freely. I have never taken very kindly to cold packs or compresses and have resorted to their use only in a few cases. Still in those cases where I have used the cold compresses I was pleased with the result. Poulitices are disagreeable things at best, and only tend to worry and fatigue the patient.

I use the chlorid of ammonia in preference to the carbonate, and use it up to the crisis and during convalescence.

OBSERVATIONS ON GUMMA OF THE HYPOPHYSIS; AND PRIMARY CARCINOMA OF THE URETER.

Read before the Chicago Pathological Society, April 13, 1896.

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GUMMA OF THE HYPOPHYSIS.

In hereditary syphilis, the hypophysis may be enlarged and indurated, due to connective tissue proliferation.¹ In acquired syphilis, gumma of the hypophysis has been described by Troisier,² Weigert,³ Barbacci,⁴ Birch-Hirschfeld⁵ and Sokoloff.⁶ In these cases there can not be much doubt concerning the truth of the diagnosis. In Troisier's case the origin of the gumma is referred to the capsule of the hypophysis. Boyce and Beadles⁷ detail a case of granulomatous infiltration of the hypophysis which they regard as tuberculous, but without having shown tubercle bacilli to be present. The same uncertainty of diagnosis is attached to Wagner's⁸ case of "tubercle of the pituitary," which he observed in a girl aged 13 years, without tuberculosis elsewhere.

The following case of gumma of the hypophysis consequently merits report on account of the rarity of the lesion:

Woman, age 45, died on the same day as she entered the hospital without being able to give any information concerning her history. The post-mortem was made twenty-four hours after death.

The anatomic diagnosis reads: Chronic interstitial nephritis; chronic perihepatitis and perisplenitis; syphilitic cirrhosis of the liver with gummata; gumma of the hypophysis; thick skull; chronic interstitial myocarditis. Bacteriologic examination showed the heart's blood, the lung, the liver, the kidney, and the spleen to be sterile. Only the liver and the hypophysis need to be described at this time.

The liver weighs 1,570 grams. It is adherent to the diaphragm, especially along the falciform ligament. The adhesions are firm and fibrous. The surface of the organ is irregular and in the vicinity of the falciform ligament are puckered cicatricial depressions in the bottom of which the tissue is firm and whitish in color. The consistence is firm. On the cut surface there is observed a marked but irregularly arranged increase in fibrous tissue, but this is also best marked about the insertion of the falciform ligament; here the substance of the organ contains whitish nodules or areas that are inclosed in puckered capsules of fibrous tissue from which trabeculae radiate in all directions. Larger, homogeneous districts are also present.

The hypophysis is about twice the usual normal size; it is firm, rather homogeneous and grayish-red on the cut surface; the walls of the sella turcica are rough. The hypophysis weighs 1.8 grams.

The skull, which is plagiocephalic, is unusually thick, measuring, at the line of the incision to remove the calvaria, from 8 to 10 mm. in thickness, its bone being very dense, the diploë almost entirely absent, or replaced by compact tissue.

Microscopic examination of the hypophysis shows the entire organ the seat of a diffuse round-cell infiltration, throughout which are scattered numerous multinucleated giant cells. Only in a few places are indistinct remnants of follicles present. There are present a few irregular areas of necrosis in which the substance is homogeneous or finely granular. Blood vessels are sparse and their walls are often diffusely infiltrated. There are no miliary tubercles at the periphery of the mass. Under high power the nuclei of the cells are seen to be oval, spindle-shaped and stained but lightly; or smaller, round and deeply colored. Irregular shaped nuclei are also present. The giant cells show the protoplasm to be red (eosin), finely granular or homogeneous, the nuclei being heaped up mostly at the periphery. Large, distinctly epithelioid cells are not present. The ground substance is homogeneous or freely granular with but slight filtration. In the distinctly necrotic districts are nuclear fragments of all shapes and sizes. Eight slides were examined carefully and repeatedly for tubercle bacilli (carbol-fuchsin) but with negative results.

The liver shows marked thickening in Glisson's capsule with areas of diffuse cell infiltration in which are giant cells and encapsulated necrotic districts. This is most marked near the surface of the organ. There are no tubercles in the sections. Tubercle bacilli were not found.

From this examination it is believed that the diagnosis of gumma of the hypophysis is justified upon the following grounds:

1. The absence of typical tubercles and of tubercle bacilli, the structure being that of a degenerating granuloma.

2. The presence of a distinctly syphilitic process in the liver.

¹ Lancereaux, *Traité historique et pratique de la syphilis*, 2d ed., Paris, 1873, p. 288.

² Bull. de la Soc. Anat., 1874, T. xlix, p. 25.

³ Virch. Arch., 1875, Bd. 65, p. 223.

⁴ Centralbl. für Allg. Path. and Path. Anat., 1892, iii, p. 301.

⁵ Path. Anat., 1894, Bd. i, p. 281.

⁶ Virch. Arch., 1896, Bd. 143, Heft 2.

⁷ Journal of Path. and Bact., Vol. i, No. 3, p. 359.

⁸ Arch. f. Heilkunde, Bd. ii, 1892.