

that there was no marked cyanosis or swelling of the veins of the upper extremity, but as there is diarrhea and vomiting with the loss of a large quantity of fluid, we can understand how there might be this accumulation of blood in the liver and yet not such an accumulation of blood in the upper extremities. One boy had spasmodic croup with severe suffocative attacks. I was asked to intubate the child and on examination no marked obstruction in the larynx was found, but he had this very deep breathing which was mistaken for obstruction. The liver was enormously enlarged, reaching almost to the umbilicus. Another child had cardiac disease. He suddenly had some severe symptoms and developed this deep breathing and also had this swollen liver. I have concluded that this deep breathing and enlarged liver are due to an acute cardiac dilatation which may be incidental to any acute disease, such as acute gastritis, acute gastro-duodenitis or colitis, or severe vomiting forcing the blood into the heart and stretching the right ventricle. So, while I originally considered Parke's syndrome a definite disease, I believe it is merely a group of symptoms occurring in many diseases. The acidosis is due to faulty metabolism in the absence of carbohydrates, and is not the cause of the disease.

DR. ISAAC A. ART, Chicago: I am already on record as having seen a number of these cases, I think a dozen or more. In nearly every one I have had an autopsy performed. In the first case, the child of a physician, the symptoms were pretty much as outlined by Dr. Parke. The child falls suddenly ill, vomiting, rapid respiration, rapid, small pulse, and goes gradually into a coma and last of all develops symptoms of obstruction of the bowel, due probably to extreme atony. They have all shown at autopsy, uniformly, fatty degeneration of all the organs. Dr. Ricketts, who did the autopsy work in the first case, thought that possibly the child might have eaten matches, so profound and extensive was the fatty degeneration. We found not only acetone but leucin and tyrosin in the urine. Dr. Ricketts found that the heart muscle and the intima of the vessel walls showed fatty changes. I think this is a syndrome that is a pathologic entity and depends on some grave toxemia. The acidosis is merely a symptom, and it is of no more importance than the rapid breathing, the rapid pulse, or the mechanical obstruction of the bowels, and it would be wrong to ascribe too much importance to it. I discussed these cases with a pioneer physician of Illinois and he suggested their close resemblance to the disease described as milk-sickness in cattle. Inquiring more closely into the subject one finds that milk-sickness or trembles bears a striking similarity in symptoms to those which Dr. Parke noticed in his patients.

DR. THOMAS D. PARKE, Birmingham, Ala.: There was no coma in any of these cases and there was no mechanical obstruction. There was dysentery and poor digestion and though the bowels did not act during the period of labored breathing, after this stage of labored breathing passed off, the bowels became loose again. I am sure there was no mechanical obstruction in any single case. I am sure not only from the post-mortems that I made but from the patients that recovered. There is a good deal more in the acidosis problem yet to be cleared up and I think you will agree with me if you will study the paper by Dr. Ewing in the December, 1908, issue of the *Journal of Experimental Medicine*. My conception is that the injury done by the toxins of this intestinal trouble cripples the liver-cells and when you get crippling of the liver-cells, the products of intermediate metabolism are carried into the general circulation and produce the air hunger. Dr. Ewing attaches immense importance to this fatty degeneration. He says that these studies of the last few years show the immense importance of fatty degeneration. But we do not know just what work the liver has to do. I have been unable to find any physiologist who could tell me the complete function of the liver. I do not know whether the trouble is in the liver-cells or in the blood that is in the liver, and until we can get thoroughly worked out the full physiology of the liver we shall not know its pathology.

SYPHILITIC DISEASE OF THE ARTERIES OF THE CENTRAL NERVOUS SYSTEM WITH DETAILED REPORT OF A CASE*

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NEW ORLEANS

The importance of arteritis or endarteritis as a lesion of syphilis has become well appreciated, especially since the writings of Huebner, Baungarten, Marchand, Rieder, Nonne, and others. Only recently, however, has sufficient emphasis been placed on the tendency of specific arteritis to appear shortly after primary infection, and on the relative frequency of isolated involvement of the arterial system as compared to the other rather more familiar forms of nervous syphilis implicating the meninges and surrounding structures of the central organ, etc.

As regards the time elapsing between the occurrence of the initial sore and the intervention of symptoms of arterial disease, the tendency of recent writers on this subject, especially Nonne, of Hamburg, is to insist on the very early development of arteritis after the primary infection. The latter author quotes a case in which symptoms of cerebral softening occurred even before the primary sore had healed.

Not only is arteritis now looked on as an early process of syphilis, but it is even held that this lesion in its pure form practically does not occur in very late syphilis, and that nearly all cases occur within three years after the chancre.

Again it is necessary to recognize the frequency of arteritis as one of the grand diseases of the nervous system assignable to syphilis, because both treatment and prognosis are altogether determined by the nature of the processes in the nerve tissues associated with the arterial disease. Only by arriving at a proper conception of the lesions in such instances can the medical attendant properly interpret the phenomena occurring. It is rather probable that disappointment arising out of too sanguine expectations from the application of proper specific treatment, under the mistaken assumption that a gummatous or exudative process is being attacked, in instances of softening and hemorrhage in the central nervous organ, has been the occasion for physicians discarding the correct diagnosis of syphilitic disease, and consequently neglecting the effective agents of treatment subsequently.

It is unnecessary to dwell on the nature of the changes occurring in the arteries, because these are quite well understood. It is important only to recall that the process is practically always a proliferation of the intima with consequent encroachment on the lumen of the vessel and mechanical interference with the passage of the blood-current. And we do not have to wait for complete occlusion of the vessel in the expectation of symptoms, but a very important group of syphilitic nervous diseases is caused by mere diminution of the blood-supply to the tissues, giving symptoms of neurasthenia, transient pareses, and the like, not extending to the gravity of organic disease. In such instances the alterations in the arterial walls, especially in the smaller vessels, further interfere with the interchange of nutritive fluids, thus contributing to the so-called functional disturb-

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ances. As a matter of course, it is very important to interpret such symptoms correctly and early, for proper treatment is then most hopeful.

When actual hemorrhage, softening, or thrombosis occurs, the pathology and symptoms are in no wise peculiar to luetic arteritis, unless we regard the tendency of syphilis to select the branches of the middle cerebral artery as especially great. In my experience with a number of cases, I have found that the involvement of the arteries to the striate body, thalamus and the bulb especially frequent. The interesting lesions seen in the photographs of specimens from the case I am presenting, emphasize this. In this instance the lenticulo-striate group of arteries were almost all completely obliterated, leading to practically complete death of these structures on the right side and pronounced softening on the other.

I deem it somewhat foreign to the purpose of the present paper to more than refer to the fact that syphilis is the remote cause of aneurismal processes very late after the infection, operating in conjunction with agencies tending to raise the blood-pressure, such as atheroma, cardiac and renal diseases. This latter observation, however, serves in this connection to emphasize again the great tendency of syphilis to inflict wide-spread damage on the arterial tree.

I shall finish these few generalizations with reference to a case in which I was fortunate enough to follow the symptoms over a period of twenty months, the case finally coming to necropsy. Although I was not able to get the necropsy done in the best manner possible, and to prepare the specimens completely, I have succeeded in securing photographs and slides of two transverse sections which display the most pronounced lesions in a fairly satisfactory manner.

History.—The patient was a white man, aged 36, single, bartender by occupation, and came under observation March 26, 1908. Aside from the fact that the father was alcoholic, the family history was negative. The patient was exceptionally well as a child and during adolescence. In fact, there was no illness of consequence until he acquired syphilis. Beginning at the age of about 25, he led a very dissolute life, drank alcohol steadily and went on occasional week-end drunks; used tobacco and drank coffee to excess. During September, 1906, he contracted a venereal sore and had well-marked secondaries. Treatment was instituted and kept up in a desultory way for two years after appearance of secondary symptoms. There was seemingly no further manifestation of syphilitic activity until November, 1907, when the patient sustained a stroke of partial hemiplegia of the left side. The hemiplegic stroke was, however, preceded by dizziness and malaise for several weeks; and on treatment the weakness on the left side improved greatly, but never got entirely well. A short time before my observation, he became dull and confused, laughed and cried without cause, and showed other signs of disturbed mentalization.

Examination.—On first physical examination (March 26, 1908), the following observations were made: All deep reflexes were exaggerated, but those of the left arm and leg were most pronouncedly so; ankle clonus partially developed on left, exhausting after a few seconds; the left epigastric and hypogastric reflexes were absent; Babinski sign present on the left, Gordon sign on the right; Romberg test positive. It was impossible to test sensation carefully on account of the mental state, though there was obviously diminution of pain sense on the left side of the body. There was marked weakness of the left arm and leg. With the hands, 38 kilos on right and 12 on left, could be registered on the dynamometer. There was no sign of facial weakness on either side. Tongue deviated slightly to the left; pupils equal, round, active to light and accommodation. There was very slight swelling of the optic disks with obscuration of the edges and what appeared to be

a mild toxic retinitis. The patient articulated awkwardly as if the tongue and lips were stiff on voluntary movement, and the voice was nasal. The soft palate sagged, but was observed to move on forced voluntary effort, not, however, on stimulation. The heart and other important viscera were normal; urine examination being likewise negative. The bowels were obstinately constipated and the tongue had a white coating. Control of the bladder was somewhat impaired, though this could not be determined well on account of the mental state. No hardening could be detected in the peripheral arteries. The mental state was one of universal reduction of all the psychic aptitudes. Patient was unable to tell where he was or anything about his situation. On attempting to answer questions, there was an overplay of the facial muscles of expression and articulation, and the effort would almost invariably end in a fit of crying. Words were enunciated in a clumsy way, amounting to dysarthria. The tendency to weeping obviously did not signify affective depression but the emotional instability seen in cases of gross lesions of the basal structures, especially the optic thalamus and striate bodies. The patient had no care for personal cleanliness. These and numerous other faults of behavior seemingly have their explanation in the mental state of bewilderment and negativism present. Specific treatment was given, but was so poorly tolerated that it had to be used very mildly. The digestive functions seemed to be in abeyance, constipation was obstinate and the general state grew worse.

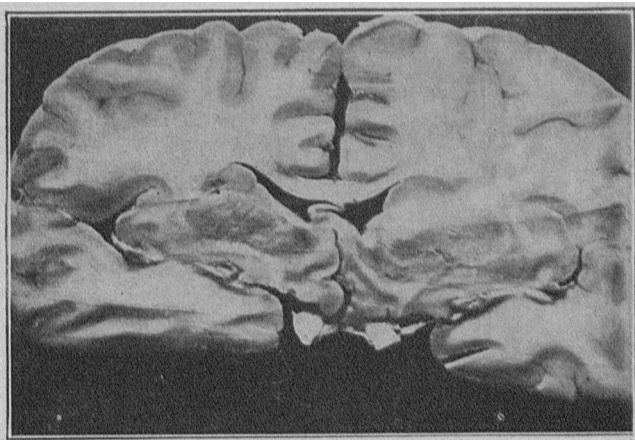


Fig. 1.—Syphilitic endarteritis.

Subsequent Course of Disease.—May 4, 1908: General state somewhat worse than on last observation; patient required careful nursing, drooled saliva and refused food. Emotional instability continued; patient articulated badly; the gait was now somewhat more spastic; condition of the eye fundus remained the same; Babinski sign was present on both sides. Specific medication increased in quantity.

June 9, 1908: The general state was observed to be improved; patient would now articulate his words better, knew that he was in the hospital, took his food better and gained in weight; spasticity in extremities was much diminished; Babinski sign present on both sides; deep reflexes greatly plus; most pronounced on left.

June 16, 1908: Condition quite the same as on previous observation, possibly a bit better. Patient left the hospital.

During the month of October, 1908, patient was seen casually on the street and seemed almost normal. The gait was unimpaired and he asserted that he could use his extremities as well as ever. He felt strong and expressed intention of resuming his occupation. He was readmitted Aug. 15, 1909, having become practically helpless in all four extremities.

October 2, 1909, the following observations were made: Patient's condition was worse than when first seen. He cried almost continually; signs of the old left hemiplegia were again prominent, and the right side was also weak and spastic; the arms were stiff and awkward on voluntary movement; pronounced weakness in both arms and legs, but most pronounced in left; deep reflexes greatly exaggerated on the

left, less so on the right; sustained ankle-clonus on the left; Babinski sign present on both sides. Sensation could not be tested on account of mental state. No athetoid movements or tremors present. The mental state was the same in kind as that seen on first observation, though of greater depth. The patient lay quietly and discharged the functions of his body without concern, requiring to be fed like a child. Articulation was scarcely intelligible, and he had great difficulty in swallowing. Specific treatment was again instituted, but totally disagreed and had to be withdrawn.

Nov. 28, 1909: Patient was bedridden and could scarcely swallow food from involvement of deglutitory muscles. The mental state was one of negativism, due to the profound reduction of all cerebral functions. He still continued to cry when aroused. Babinski sign was present on both sides; sustained ankle-clonus on left. The optic disks remained partially obscured as noted on previous observations. The patient moved his extremities in an awkward, ataxic, spastic manner; was obstinately constipated and abdomen was retracted. Patient was unable to swallow and seemed not to assimilate nourishment given by stomach-tube.

He died December, 1909, from inanition.

Clinical Diagnosis.—Syphilitic endarteritis of the central nervous system, with focus of hemorrhage or softening at first in the right internal capsule; later similar involvement of the left capsule. Probably multiple small foci about the hemi-

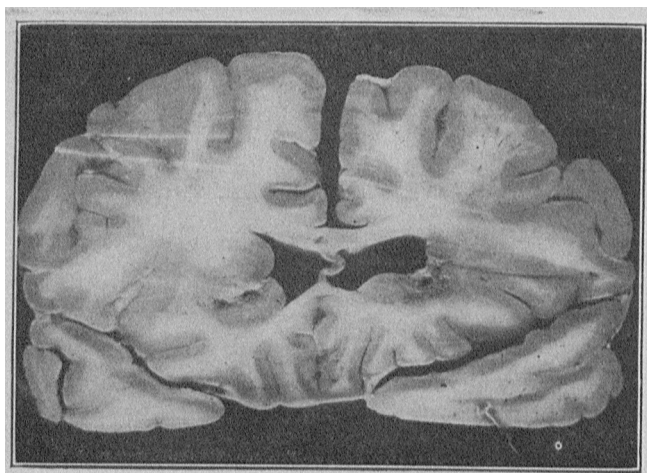


Fig. 2.—Syphilitic endarteritis.

spheres from rupture or obliteration of the smaller terminal vessels.

Post-Mortem Findings.—There were found slight universal thickening of the dura, and wide-spread opacities of the pia with cystic formations in places. The walls of the large vessels at the base were observed to be thickened, especially the middle cerebral and its branches. Areas of softening were seen in the region of the two striate bodies, most marked on the right, where the lesion appeared older and involved the thalamus and practically all structures in the lenticulo-striate region, apparently destroying the majority of the fibers of the capsule. These were the two chief gross lesions, but numerous minute foci of necrobiosis were seen in the cortex and subcortical substance of the hemispheres.

Transverse sections of the spinal cord showed the left half of this organ to be distinctly less in volume than the right. The pyramidal tracts were seen to be greatly atrophied on naked eye inspection, apparently from secondary degeneration alone.

On account of the difficulties under which our observations were made, no Wassermann reaction could be done and no search for spirochetes made; but the clinical history and appearances of the lesions found were so overwhelmingly in favor of syphilis as to leave no reasonable doubt.

I realize the great interest that might have attached to a more detailed observation of the symptoms in the case and the exhibition of microscopic sections of the

brain and cord in further illustration of the interesting pathologic lesions present. Lack of opportunity prevented my doing the former. I hope to complete the pathologic work later.

I have the pleasure to express my thanks to Drs. Daspit, Duval, and Couret, who have so kindly assisted me in securing and preparing the specimens.

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ABSTRACT OF DISCUSSION

DR. WILLIAM W. GRAVES, St. Louis: Dr. Hummel referred to the frequency of early arterial degenerations as a manifestation of syphilis, especially as pointed out by Nonne in recent years. This great clinical fact is one which is not appreciated. Early arterial degeneration after a syphilitic infection, no matter how carefully treated, is the one preeminent clinical fact on which one may oftentimes hinge a provisional diagnosis. Signs of arterial degeneration, occurring in a man before his time, and one being able to exclude metallic and other poisons as the cause, are in the majority of cases due to a former syphilitic infection. I base this conclusion not on that finding alone, but there are other clinical findings demonstrable in cases of syphilis two or more years after the infection, which are almost as constant as the arterial changes.

During the last few years I have given much attention to syphilis of the nervous system and post syphilitic conditions, and I have made careful studies not only of the arterial systems of syphilitics, but also of their nervous systems; and from these studies it may be stated, though I may be considered radical when I do state it, that there are but few patients, however carefully we may treat them, who two years after a syphilitic infection will not show organic changes as demonstrable by a clinical consideration of the nervous system. Indeed during the secondary manifestations of syphilis in some cases which I have had the opportunity of studying, I have found distinct sensory loss, especially to pin pricks. This loss may and probably does remain throughout the life of the individual. A syphilitic may lead a healthful life, but he reacts badly. The individual who has had a syphilitic infection must be regarded as a blighted individual, reacting poorly to stresses and strains. Such individuals when studied, in addition to cardiovascular changes, show in the majority of cases changes in the oculo-pupillary apparatus and in the nervous system.

DR. GEORGE A. MOLEEN, Denver: I have been greatly pleased with the case report that has just been made, and I believe that this condition is too often neglected by reason of its frequent occurrence. The first phase that appeals to me in this paper is the evidence of neglected syphilis which has been the cause of the arterial degeneration. I have seen quite a number of cases of cerebral arterial degeneration in Colorado, and almost invariably the history of the syphilitic infection carries with it an incomplete treatment, or a treatment extending usually not over two or three months. The length of time of specific treatment, I think, is often in direct relation to the occurrence of an arteritis. I have seen in one instance the occurrence of a syphilitic arteritis within three months of the initial sore. In other cases they have gone, as has been the experience of others, as long as fifteen or twenty or more years. The mental confusion in the case just cited, I take it, is an expression of the general toxic effect of the syphilitic virus, which is further evidenced by the neuritis and the retinitis, and these are not commonly, if ever, present in pure arterial lesions. I do not believe that many such cases should occur if the proper mercurial treatment were established and continued, and particularly continued when once established. I think with Osler that it is lamentable that we should find arterial lesions in specific cases like these, which are so amenable to mercurial treatment.

DR. THEODORE DILLER, Pittsburg: There is a good deal of thickening of arteries, a good deal of arteriosclerosis, that is not syphilitic. For instance, I very often find thickening of arteries in boys with dementia præcox, and I believe that there

is a good deal in the inheritance of bad arteries, arteries which degenerate early, and I feel that we ought to be not too hasty in concluding that thickened arteries indicate specific disease, even in young persons.

Dr. E. M. HUMMEL, New Orleans: Fourteen months elapsed between the time of the initial sore and the stroke of hemiplegia which this man suffered, which in the light of subsequent developments was undoubtedly assignable to softening in the right capsule due to obliterating endarteritis. I wish to insist again on the early occurrence of signs of specific endarteritis, dating from the time of the initial sore. Dr. Nonne in his comprehensive treatise, "*Syphilis und Nerven-System*," insists on this repeatedly, and comes to the conclusion that syphilitic processes of this kind always occur practically within three years of the time of the infection. I did not wish to be understood as confusing specific endarteritis with arteritis of other causation, and I quite agree with Dr. Diller that there are numbers of other kinds of endarteritis, but I think, everything considered, there is no doubting the specific character of the arteritic process in this case. There is one point that I should like to mention further in connection with the case, and that is that it is noted in the clinical symptoms that this man underwent great improvement under specific treatment and got practically well. The explanation of that is interesting. It is probable that there was a large exudate around the site of the original softening, and that it was absorbed under specific treatment. One thing further in this connection of and of interest here is that when a person under the age of 40 sustains a stroke of hemiplegia or any other sign of gross focal, brain or spinal cord trouble, coming on suddenly, indicating either hemorrhage or softening, the presumption is very strong that such an accident at so early an age indicates the presence of arterial disease of specific causation and the case ought not to be deprived of a therapeutic trial with antisyphilitic agents.

A NEW METHOD OF VACCINATION

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All physicians will agree that with the vaccination method now in vogue, using the scarifier (*Impf-Lanzette*), we can never measure the extent of the trauma and therefore the resulting lesion. As a rule, with experience, we may strike just about the right thing, but in the case of a restless child an unexpected motion on our part or on the part of the patient makes us go beyond the intentional limits. A further point, which has not been considered sufficiently, is that such a large abraded area is a receptacle of a large quantity of vaccine lymph causing too strong a local reaction. Such and other drawbacks can be prevented by a new method, which I herewith present. This method revolves around the instrument to be used, which is the scarifier devised by Professor von Pirquet for his cutaneous tuberculin test. It is a small chisel with the sharpened edge of which the drilling of the skin is surprisingly painless, hardly perceptible by children.

MODUS OPERANDI

1. Clean the arm with water and soap, and then rub it thoroughly with alcohol.
 2. Apply the vaccine lymph (pulp) on the skin by expressing a capillary tube or by smearing the lymph, in case one prefers the points.
 3. Point the chisel perpendicularly to the surface of the skin and press against it with a few rotary movements or, as a child said, "use it like a screw-driver."
- These few rotary movements suffice to produce both an abrasion of the skin and an implantation of a minimum amount of vaccine virus.

Within a minute a little wheal, a typical, reddish urticarial papule with a central depression, is formed. It is a vasomotor reaction produced by the blunt pressure of the instrument.

I always cease the operation when the superficial layers of the epidermis appear to be removed and a pinkish hue shines through from beneath (vessels of the *pars papillaris corii*). I have never seen a trace of blood oozing from the scarified skin, which is by no means an attribute of a successful vaccination, as some believe.

DEVELOPMENT OF THE PUSTULE

The course of development is as follows: The traumatic papule spoken of fades away in a few hours and the incubation period begins. Nothing is seen or felt until about the end of the first week, when a little elevation is formed around the point of trauma. A very small area of redness may be seen at this time. This papule transforms itself into a vesicle within the second week, and becomes mature at the end of second or the beginning of the third week.

The most striking peculiarity of this form of vaccination is the mild and slow development, so that neither children nor parents are aware of the effects of the vaccination until the pustule is well formed. In the



Vaccine pustules (new method). Strictly circular, of geometric accuracy. Although very close, their individual halo can be distinguished, no confluence having taken place. Surrounding tissue is only slightly involved.

majority of cases, no subjective symptoms worthy of being mentioned are complained of during the first week (itching). During the second and third week, when the pustule is ripening and the area congested, there is seldom felt a pain in the axillary region. I have not yet witnessed an involvement of the whole arm, as the reactive congestion is small and keeps within the nearest boundaries of the pustule; neither does a bacterial infection take place readily. I have not had a single case showing these little accidents, as in previous years, although the vaccinations performed this year surpassed all those of previous years, on account of the smallpox scare in Cleveland. I explain this in the following way:

Scarifying, especially crosswise, means dissemination of little traumatic lesions over an unlimited area. After the inoculation a crop of little pustules will sprout, and a confluence takes place to form a large unbounded pustular surface with an extensive area (halo). The crust later formed cannot completely cover the wound. It is not uniformly thick to resist the exuberant fluid; the scab cracks; matter overflows, sometimes resulting in an unnecessary secondary vaccination, and (what is of more