at 19 months of age. In each case the parents were healthy, and the mother was a multipara with the history of previous normal births. There was no history of prolonged or difficult labor, but in each case pituitary extract was administered early in labor, and birth was precipitate very shortly after. In each case, efforts at resuscitation were required and convulsions followed later; in two they continue to recur. Heard¹⁶ is convinced that these were cases of cerebral or meningeal hemorrhage and that pituitary extract was the etiologic factor in their production.

Obstetricians generally are coming to a realization that the child has a right to be born alive, and that the license to practice medicine does not also carry with it a license of executioner of the unborn child any more so than of the mother.

A COMPARISON OF STATISTICS OF 1914 AND 1916

1914	1916
Cases, 3,952.	Cases, 1,293.
Ruptured uterus, 8 cases, or 1 rupture in every 494 cases.	Ruptured uterus, 12 cases, or 1 rupture in every 106 cases.
Fetal deaths, 27, or 1 fetal death in every 146 cases.	Fetal deaths, 34, or 1 fetal death in every 38 cases.
	Asphyxia pallida, 1 case in every 32.

Pituitary extract probably has a place in the obstetrician's armamentarium, but for its undoubted bad effects on the child primarily, and on the mother in a lesser degree, it would seem that its field of usefulness is indeed a limited one.¹⁷

16. Heard, A. G.: *Texas State Jour. Med.*, 1916, **12**, 264.
17. In addition to the references already given, the following will be of interest:
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O'Connor, B. J.: *Lancet-Clinic*, 1915, **114**, 302-305.
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Foulkrad, C.: *Therap. Gaz.*, 1916, **40**, 305-307.
Valens, J. A.: *West. Med. News*, 1916, **8**, 128.
Bell, W. B.: *Liverpool Med.-Chir. Jour.*, 1915, **35**, 292-298.
Sheffield, H. B.: *Am. Jour. Obst.*, 1915, **60**, 379.
Trapy: *Monatschr. f. Geburtsh. u. Gynäk.*, 1915, **4**, No. 36.

Production of Camphor.—As set forth in *Commerce Reports*, April 13, 1917, the estimated production of camphor in Japan for the year ending March 31, 1917, was 2,148,197 pounds, an increase of 35,121 pounds as compared with the actual production of 1915-1916. The estimated production in Formosa during this period amounted to 6,619,461 pounds, an increase of 520,821 pounds, as compared with the actual yield of the preceding year. During 1916-1917, the estimated production of camphor oil in Japan was 4,237,852 pounds, an increase of 275,976 pounds as compared with the actual yield of 1915-1916. The estimated production of the oil in Formosa during the period named was 10,332,379 pounds, or 1,249,153 pounds in excess of the actual yield of 1915-1916.

ONE ROAD TO THE CONTROL OF HEART DISEASE

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This note begins with analogy and ends in exhortation. The analogy consists in a fundamental likeness between the demographic aspects of what I will venture to call subclinical infections of the heart, and incipient tuberculosis of the lungs.

No broad scheme for the prophylaxis of tuberculosis was possible until hygienists grasped the fact that tuberculous infection is wellnigh universal. Thereupon the practical procedure became one largely of devising means to prevent a harmless anatomic infection from developing into a dangerous clinical disease. Accordingly, whatever the physical condition may be which the clinician is called on to consider, he has in mind tuberculosis. Particularly in the case of children does he search out in tuberculosis an explanation of morbid symptoms. The result is the control of established tuberculosis.

Just as civilized mankind harbors almost constantly a more or less latent tuberculosis, so infective disease of the heart, provoking no classical symptoms, but potential for catastrophe, is wellnigh invariable at some period of life. The practical conclusion from such a premise is that, as in the case of tuberculosis, the latent anatomic derangement may break down into functional inefficiency in an unfavorable environment, such as added infection or overuse.

The proof of such a proposition can be established only after years of definite research; but sufficient evidence is at hand to justify the clinician in accepting it as a working hypothesis. As has been slowly apprehended in the case of tuberculosis, only on the assumption of the universality of cardiac infection will the clinician give adequate consideration to morbid signs which point to cardiac disease.

From the point of view of the public health officer, Darlington¹ has given statistical evidence that the mortality rate from heart disease is positively increasing throughout the United States, while the general death rate is steadily decreasing.

The fundamental basis on which must rest a belief in my thesis is the incompleteness of the evidence offered by physical signs of the organic condition of the heart. From the point of view of the pathologic anatomist, Warthin² has shown how doubtful is the ultimate cure of any infectious disease and, particularly in the case of syphilis of the heart, that profound organic infection may exist without appreciable clinical signs.

Necropsy records, including careful histologic and cultural examinations of the cardiac tissues, must furnish the crucial evidence as to the dissemination of heart lesions, and none can dispute the urgent duty of the pathologist to provide appropriate records. On the clinician, however, devolves the task of determining the functional capacity of the heart during life, and the practical application of methods to restore power and prevent failure.

We are not here concerned with the obvious cardiac lesions which are causing or threatening broken compensation, but only with what may be called "sneaking heart disease" in which the classical signs of insuffi-

1. Darlington, Thomas: *Tr. Am. Clin. Assn.*, 1908, **24**, 120.

2. Warthin, A. S.: *Tr. Assn. Am. Phys.*, 1910, **25**, 580; *ibid.*, 1914, **29**, 416.

ciency are absent but in which there is a dangerous narrowing of what James Mackenzie calls the field of physiologic response.

It must be granted that the clinical data of physical diagnosis are absurdly insufficient to supply a reliable fund of information. While medical students are taught the words which signify that the pathologic importance of a heart lesion depends on involvement of the heart muscle rather than on changes in its lining membrane, the statement may be boldly ventured that the endocardial murmur is still the sole language through which the heart can speak its limitations to the casual examiner.

It is doubtful if the student keeps clearly in mind that even valve lesions are probably all due to infections conveyed not by the intracardiac flood which bathes the valves but by the capillary streams which spring from the coronary circuit, and that the slightest attack on a valve presupposes a more or less widespread infection of the myocardium. Granting a locus of infection within the heart, the teachings of the new science of immunology must lead us to suspect that at once the vital reactions of cardiac protoplasm become profoundly modified. Even the crude knowledge in our possession today points indubitably to the conclusion that heart tissue so attacked becomes, as it is said, "sensitized" to the particular form of poison with which it is inoculated, and thereafter abnormally receptive of new accessions of the same poison, and susceptible to them. The clinical facts that support this point of view are simply numberless. Fortunately for the preservation of the higher forms of life, protoplasm attacked by a foreign poison, after an initial stage of heightened susceptibility, is apt to acquire a specifically strengthened resistance to the noxa. But this "immunity" is always merely relative, and it were vain in a given case to estimate in advance the ratio of virulence to resistance.

The canny practitioner of medicine wisely takes his generalizations only as inductions from individual experience. Nothing, in my opinion, could be more fruitful for good than for the student of disease to estimate "the field of physiologic response" in the hearts of his patients, according to the views set forth.

EXAMPLES OF LATENT HEART DISEASE

A few concrete clinical examples of a familiar kind may make clearer the source of the impulse that led to this article:

CASE 1.—A man of fine physique, somewhat under 40 years of age, developed articular rheumatism. Nothing wrong had previously been found with his heart, but now there was detected obscurely at the apex a murmur of that peculiar quality which one learns to associate with fresh endocardial lesions. Under some medication and prolonged rest the physical powers seemed to become completely restored and the murmur, changed in quality, had nearly disappeared.

Many months later the patient was accepted for life insurance by a conscientious and competent examiner who, though informed of the history, could detect no cardiac lesion whatever. Since then, a number of times in the course of several years, the patient has applied for treatment of a sore throat, and on each occasion a slight bruit could be detected at the apex, the abnormal sound becoming temporarily a distinct murmur during a recent acute rheumatic attack involving throat and muscles.

My conclusion has been that this active business man carries a chronic infection of the heart which sensitizes the organ toward rheumatic toxins. It is

impossible, of course, to pronounce whether the new endocardial murmur accompanying each attack of sore throat was the mechanical result of an altered circulation or a sign of fresh infective turgidity of the mitral valve. One who watches the quality changes in such a murmur day by day is apt to incline to the latter explanation.

Of the numerous tests to determine the functional efficiency of the heart, nearly all fall within the limbo of "academic" procedure. It is usually doubtful whether a functional inefficiency thus determined shows pathologic impairment or only lack of physical training or weakness of other coordinated organs.

CASE 2.—A man, aged about 25, of powerful physique, who had a perfectly compensated mitral lesion for some years and who was apparently in excellent health and strength, entered a military camp and was set to digging a trench.

A physical examination disclosed too late his unfitness for the task. A prolonged and fatal illness followed, bearing all the features of an infective cardiac disorder.

While the following explanation of this casualty cannot be proved, it harmonizes many facts and suggests a safe working hypothesis: In the foregoing subject, infection and resistance within the heart had been so balanced that functional processes remained normal. When excessive physical strain was put on the anatomically defective organ, its mechanical imperfection led to a "break in compensation," that is, the left ventricle ceased to deliver blood as fast as it was received by the right auricle. At once disturbance of cardiac nutrition was instituted. Immediately the biologic "resistance" of the heart tissues deteriorated, and the infective forces, which had been held in subjection, began to take on strength and to propagate their kind.

A tissue which is the seat of a viable infection is like a seesaw; on one end is seated physiologic resistance, on the other infective virulence; the balance represents the ratio of the two forces. Precisely in the same way we frequently find that a quiescent pulmonary tuberculosis is lighted up into progressive disease by injudicious habits. Indeed, the salutary relation of rest in active tuberculosis is identical, with greatly increased emphasis, in acute heart disease. In tuberculosis, Nature is prone to forgive the careless transgressor; in heart disease, perhaps more rarely.

CASE 3.—A strong youth had a slight indisposition which was evidently rheumatic, but he was not so ill as to refrain from riding several miles on his bicycle to an entertainment. The return journey was made against a strong wind. The boy was obliged to seek his bed on reaching home, and soon developed signs of a general cardiac inflammation that carried him off.

I insist that these cases do not find their complete explanation in mechanical overstrain of the heart, but indicate an intense intoxication, such as the immunologist is prone to demonstrate in his reactions of anaphylaxis. Moderate overstrain of a normal organ is probably an indifferent episode, but the same test applied to an infected organ sets in play chemical reactions which lead to physiologic destruction.

An adequate study of heart disease will disclose another line of contact with pulmonary tuberculosis. In the latter disorder, accumulating clinical experience points more and more frequently to the period of early youth as the time of primary infection. In the same way the student of breaking cardiac compensation can often trace the history through a long period of latent disease to a definite moment in early life when some

Herpes zoster may develop in any phase of paresis,⁹ often coming at the onset before the appearance of the characteristic symptoms. It may develop in the course of the lightning pains of tabes.¹⁰ In tabes and paresis, herpes zoster is incidental to an involvement of the ganglions and roots in the pathologic processes of these diseases,¹¹ although it must not be overlooked that the diseased condition of the nervous system may open the portal for the specific form. It is interesting to note, incidentally, that Knowles¹² mentions the occurrence of herpes zoster together with a maculopapular syphiloderm, and also the occurrence of herpes zoster in a patient with gummas.

Zoster involving the entire length of the upper extremity alone (as in the first case report) appears to be uncommon. Knowles¹² observed four cases of zoster involving the entire length of the upper extremity out of 273 cases of herpes zoster in which the distribution was mentioned.¹³

REPORT OF CASES

CASE 1.—M. K., aged 50, laborer, was an advanced paretic admitted to the Philadelphia Hospital for the Insane, Feb. 10, 1915. Oct. 25, 1915, there was an eruption confined to the right upper extremity as follows: Starting above the axilla the eruption ran along the posterior margin of the deltoid muscle; it occupied an area corresponding to the lower half of the body of the deltoid; below this the outer (lateral) portion of the upper arm, including the outer portion of the flexure of the elbow, and the outer aspect of the forearm, both anteriorly and posteriorly. There were a few vesicles on the dorsum of the hand, slightly beyond the wrist, in line with the metacarpal bone of the index finger; there were a few vesicles toward the palmar aspect of the hand, and along the metacarpal bone of the thumb, extending to the base of the nail. The distribution¹⁴ of the eruption corresponded to the fifth, sixth, seventh and eighth cervical nerve roots (according to the nomenclature of Bärensprung, a cervicobrachial zoster). The vesicles were in groups, on a reddened base; some were filled with hemorrhagic serum. In some places they were broken down with ulceration. The patient had not received arsenic, iodids or any other medication; he had not been injured. There was no evidence of any actual paralysis, or any alteration of the reflexes from the previous condition. The patient did not seem to suffer any pain; this may have been due to his

advanced dementia. The teeth and the tonsils appeared to be normal.

A spinal puncture was done, October 28. The fluid was under much increased pressure (spurred). The Wassermann reaction was positive, culture negative. The Wassermann reaction in the blood, taken the same day, was positive. The patient's temperature was normal throughout the course of the eruption. A white cell count, November 1, revealed: leukocytes, 6,900; neutrophils, 64 per cent.; lymphocytes, 34 per cent.; mononuclears, 1 per cent.; eosinophils, 1 per cent. The patient also had psoriasis, which was present on admission.

The eruption had healed (with a few scars) at the time of the patient's death, which occurred, Nov. 23, 1915.

Owing to an oversight, the spinal cord was not removed at necropsy. Histologic examination¹⁵ of the brain confirmed the diagnosis of paresis. The upper cervical cord showed marked involvement of both the tracts of Goll and Burdach.

CASE 2.—T. M., man, aged 49, vagrant, was admitted to the wards of the Philadelphia Hospital for the Insane, Oct. 26, 1915. The patient was a paretic with some tabetic symptoms. The eye grounds were normal. The Wassermann reaction was positive in both blood and spinal fluid. Several teeth were represented by old decayed roots.

November 5, a redness was noticed on the right side of the forehead. The next day vesicles appeared.

November 6, the border of the eruption started at the hair line laterally and went across to the root of the nose, including the upper lid; the conjunctiva was reddened. There were a few vesicles on the bridge of the nose. The border of the rash then extended up the midline; the upper border was slightly beyond the hair line of the forehead.

November 8, the eruption became infected.

November 9, the patient's temperature was 100 F. It was normal the next day.

November 18, the eruption was practically healed, with rather extensive scarring. The hair line of the eyebrow was involved in a scar with some loss of hair. The patient complained for a time of slight hyperesthesia in the region involved. The sense of pain in this area was considerably diminished; the touch and temperature senses were normal.

The following report on the spinal fluid was submitted by Dr. Kolmer: Clear; total cells, 30 per cubic millimeter, all small lymphocytes; protein reaction positive: Noguchi, +; Pandy, +; Nonne, +; Kaplan, ++; sugar, normal; colloidal gold, 5555544311 (paretic curve); Wassermann reaction (dose 0.8 c.c.), strongly positive; cholesterinized antigen, +++++; alcoholic syphilitic liver, +++++; acetone insoluble lipoids, +++++.

The patient's teeth subsequently became loose. Dementia and physical signs increased. He died, July 23, 1916, in a congestive attack, associated with restlessness and stupor; there were no convulsions or paralyses. Aside from atheroma of the aorta, and chronic myocardial and renal changes, the general organs showed nothing of note. Grossly the brain showed the following lesions of general paralysis: (1) chronic hemorrhagic pachymeningitis; (2) chronic leptomenigitis of the superior and anterior portions of the cerebrum, and (3) granular ependymitis of the floor of the fourth ventricle. There was no apparent cerebral atrophy. Histologic examination confirmed the diagnosis of paresis. The perivascular infiltration was unusually severe in the occipital pole. Gliosis was fairly marked in the column of Goll. The gasserian ganglion and its roots showed fibrosis and also round cell infiltration. The picture was one of chronic and also acute changes, the latter being of a nonseptic type, and probably specific (syphilitic).

CASE 3.—P. H., aged 51, admitted to the wards of the Philadelphia Hospital for the Insane, June 21, 1915. Blindness commenced five years before, and mental symptoms two months before admission. The eyes showed nystagmus; the pupils were equal, slightly irregular and inactive. There was complete optic atrophy of the eye grounds, probably primary. The knee jerks were absent. Romberg's sign was present when the eyelids were closed; there was ataxia in the lower extremities; there had been slight difficulty in holding the urine

15. I am indebted to Dr. Samuel T. Orton for the histologic studies.

9. Hérissé, Paul: *Études cliniques sur les troubles trophique dans la paralysie générale*, Paris thesis, 1903. De Fursac and Rosanoff: *Manual of Psychiatry*.

10. Osler: *Practice of Medicine*.

11. Poincaré and Bonnet (*Ann. méd.-psych.*, 1868 and 1876) first described structural and functional alterations of the sympathetic ganglions in paresis, and Klippel (1892) first described the peculiar microscopic visceral lesions which he attributed to ganglionic changes (Martial Vignaud, *Historique de la paralysie générale*, Paris thesis, 1902). These chronic degenerative changes in the ganglions, which are distinct from the acute changes in paresis causing herpes zoster, may be secondary to involvement of the nervous system central to the ganglions (retrograde degeneration).

12. Knowles, F. C.: *Herpes Zoster, Report of Two Hundred and Eighty-Six Cases, with a Review of the Unusual Features of the Disease*, Pennsylvania Med. Jour., 1911-1912, 15, 615-622.

13. Other cases, to which I have had access, are: Stopin, *Le zona du membre supérieur*, Paris thesis, 1882-1883. The entire length of the upper extremity was involved in six instances out of seventeen cases of herpes zoster of the upper extremity. Gordon, Alfred: *Cervico-Brachial Herpes Zoster*, *Am. Med.*, 1909, N. S., 4, 627-630. Carpenter, G.: *A Case of Herpes Zoster Extending to the Finger Tips*, *Rep. Sc. Study of Diseases of Children*, London, 1905, 5, 3. Pfeiffer (quoted by Blaschko) quotes one case (Footnote 4). In larger series of herpes zoster reported (Hoenicke, Cantrell, Greenough) the exact distribution of the eruption is not mentioned.

14. In determining the level involved, one must take into account the following factors: 1. A given area, though usually supplied by a given root, may instead be supplied by the root above, with corresponding changes in the other roots, or by the root below, with corresponding changes in the other roots. 2. In a very large proportion of cases the eruption does not occupy the whole area of a single nerve root. 3. The shape of the part involved (whether thin, stout, etc.) makes a difference in the appearance of the eruption. 4. There is a certain amount of overlapping of supply in a given segment by the one above and below. In outlining the area of involvement one includes the zone of hyperemia as well as the eruption proper. Cases in which only a single segment is involved are of value in determining the area supplied by a given nerve root. In any one case one cannot be absolutely certain which root is involved unless confirmed by necropsy.

during the last six months; the teeth and the tonsils were normal. The patient had a psychosis, but was not a paretic. Tabes dorsalis was diagnosed; a positive mental diagnosis was not made.¹⁶ Dr. Kolmer submitted the following report on the spinal fluid, June 14, 1916: Clear, 90 cells per cubic millimeter; 92 per cent. small lymphocytes; protein reactions strongly positive: Noguchi, +++; Nonne, Phase I, +++; glucose content, normal. Wassermann reaction (dose 0.8 c.c.), +++++, with the three antigens used above; Lange colloidal gold test: typical paretic curve.¹⁷

June 6, there was redness on the right side of the forehead. June 8, vesicles were well marked. The outer border of the rash extended upward from the outer end of the eyebrow; the inner border was at the midline. The rash included the upper lid, and the nose to the ala; the conjunctiva was reddened, but contained no vesicles. The rash extended backward to slightly beyond the parietal eminence. The blood count was normal. There was a slight rise of temperature a few days later due to secondary infection. June 21, the eruption had well disappeared, except for scabs along the eyebrow.

At present, on the forehead, upper lid and the bridge of the nose, the areas which were formerly the bases of the vesicles show loss of pigment. Neither in these whitish areas nor in the normal appearing skin can any disturbances of sensation be demonstrated.

CONCLUSIONS

Of three patients having an attack of herpes zoster, two were paretics and one a tabetic. The cases of herpes zoster were of the symptomatic variety, that is, due to involvement of the ganglions in the pathologic processes of the disease from which the patients were suffering. The nature of the ganglionic changes is in all probability specific, that is, due to spirochetes or toxins.

Thirty-Fourth and Pine Streets.

16. The patient's mental condition may have been dependent on syphilitic changes or alcoholism. On admission, dementia, though present, was not very apparent; it has since progressed, but is not extreme. There is nothing, however, in the clinical symptoms, which suggests paresis.

17. Solomon, Koefod and Welles state that a typical paretic curve may occur not only in patients who do not have paresis, but also in patients who do not even have syphilis. I have seen two nonparetics with typical paretic curves. In one of these at least, the question of paresis sine paresi does not enter (Solomon, H. C.; Koefod, H. O., and Welles, E. S.: Diagnostic Value of Lange's Gold Sol Test, Boston Med. and Surg. Jour., 1915, 173, 956-960. Solomon, H. C., and Welles, E. S.: Varieties of the Gold Sol Test (Lange) in Several Loci of the Cerebrospinal Fluid System, *ibid.*, 1915, 172, 625-629). A paretic curve is reported in a child suffering from poliomyelitis, with a positive Wassermann in the spinal fluid (Felton, L. D., and Maxcy, K. F.: The Colloidal Gold Reaction of the Cerebrospinal Fluid in Acute Poliomyelitis, *The Journal A. M. A.*, March 10, 1917, p. 752). These observations show that the paretic curve occurs occasionally in conditions other than paresis. All of our consulting psychiatrists have agreed that the patient described above is not a paretic.

Milk Distribution a Public Utility.—A joint commission of Pennsylvania, Maryland and Delaware appointed to study the milk question with reference to price, distribution, etc., headed by Prof. Clyde L. King of the University of Pennsylvania, recommends (*Survey*, February 24), that milk distribution shall be recognized as a public utility. The recommendations extend to both production and distribution, and include suggestions for increased state and municipal supervision, as well as an attempt to harmonize the interests of producers, dealers and consumers. Recognition of milk distribution as a public utility implies the keeping of accounts by dairymen and dealers in a form prescribed by the state, the state examination of accounts, the licensing of all dealers, as at present; the licensing of testers in receiving stations, and the standardization of prices. Seasonal inequality of production has been an obstacle to the maintenance of uniform price to producers, which makes the financial success of the dairy business uncertain. This, it is suggested, can be overcome to some extent by the storage of milk in the form of butter, cheese and condensed milk, and the stimulation of new uses at times of greatest yield. The competition of milk with other food products and the matter of freight rates complicate the milk supply problem.

A CASE OF BROWN-SÉQUARD PARALYSIS*

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Until recently, many workers, following Brown-Séquard, held that impressions of pain and temperature pass upward through the gray matter, while those of touch and sense of position ascend through the dorsal columns. Petré¹, from a collection of reports of a large number of cases, concluded that impressions of pain and temperature pass through the opposite lateral column, and of the muscle sense through the homolateral dorsal column, while two paths are open to touch, one through the uncrossed exogenous fibers of the dorsal column, and another in the opposite lateral column.

Head and Thompson² confirmed these conclusions and showed that the ability to recognize the simultaneous contact of two compass points depends on impulses traveling through the homolateral dorsal column. Head and Holmes³ found that the appreciation of weight and recognition of size and shape are frequently lost on the paralyzed side, and concluded that the impulses which subserve these functions ascend uncrossed through the dorsal columns. Egger⁴ has shown that the sense of vibration of a tuning fork depends on the integrity of the dorsal columns.

Recently Petré⁵, from a review of ninety-four cases of stab wounds of the spinal cord, has concluded that fibers for pain and temperature sense have their course near the lateral column of the cord. For touch two pathways are present: first, in the posterior columns in the long exogenous fibers and, second, in the "crossed lateral columns somewhere along with pain and the temperature sense. The fibers for muscle sense do not run in a crossed tract; they run in two columns, one in the homolateral posterior columns and the other in the lateral cerebellar bundle. They run medially to the fibers for pain and temperature and laterally to the pyramidal tract.

From the notes of forty-five cases of spinal injuries of warfare in which the Brown-Séquard syndrome occurred, Holmes⁶ found that the upper limit of anesthesia to the different forms of sensation varied, and that as a rule the upper border of the contralateral loss did not correspond to the segmental level of the injury. In the middorsal region, the crossing of pain and thermal impulses is complete in one segment. Thermal impressions cross less rapidly than those of pain, and touch requires two segments for decussation. In the upper dorsal segments, pain and temperature cross in two and frequently in three segments. At the fourth cervical segment the decussation of pain requires from five to six segments, and that of thermal impressions from four to five.

Holmes concludes that the fibers which convey impression from the lower spinal roots are probably placed lateral to those which have later reached the contralateral side.

* Read before the Chicago Neurological Society, Dec. 21, 1916.

1. Petré: Skandin. Archiv. f. Physiol., 1902, 13, 9.

2. Head and Thompson: Brain, 1906, 29, 537.

3. Head and Holmes: Brain, 1911, 34, 102.

4. Egger: Rev. neurol., 1902, 10, 549.

5. Petré: Neurol. Centralbl., Jan. 2, 1916.

6. Holmes: Brit. Med. Jour., Dec. 11, 1915, p. 855.