

A COMPARISON OF THE MENTAL SYMPTOMS
FOUND IN CASES OF GENERAL PARESIS
WITH AND WITHOUT COARSE
BRAIN ATROPHY¹

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Most promising leads in psychopathology accrue from the well-known neuropathological desire to prove "structural" as many of the so-called "functional" psychopathies as possible. Though the search for truly functional psychopathies—judged by the hard tests of the post-mortem room—has to be very keen, and though the sure and uncomplicated natural experiments which bring to the post mortem room suitable cases for crucial examination are singularly rare, yet the structuralizing neurologist has not yet come at all near to destroying the functionalist hypothesis. The position that mental disease may well be a disease of function involving no more than normal and inevitable physiological changes in the nervous system is still perfectly tenable, perhaps even correct for some cases. For some time now I have been publishing in various medical journals a number of contributions to the study of normal-looking brains in psychopathic subjects. My associates and I have reported on all available material at various Massachusetts hospitals for the insane (Taunton,¹ Worcester,² Westborough,³ Boston⁴) and have made numerous references^{5,6,7} to the largest material (Danvers) which remains as yet unpublished. A large amount of work has had to be done in this search for psychoses that shall be above reproach as to their functionality. As an instance of the intriguing nature of the problem, I may say that out of 153 carefully examined cases at Boston State Hospital, Dr. Canavan and I were able to find but five entirely suited to crucial microscopic examination

¹ Being Contributions of the State Board of Insanity, Number 38 (1915.4). (*Bibliographical Note*.—The previous contribution was S. B. 1. Contributions Number 37 (1915.3) by M. M. Canavan, entitled "A Histological Study of the Optic Nerves in a Random Series of Insane Hospital Cases," JOURNAL OF NERVOUS AND MENTAL DISEASE, March, 1916.)

and that an orienting examination of these cases with the microscope has already led to disquieting suspicions.⁸

One word is due those who take the advanced and (in my opinion) entirely correct ontological view that structure and function are in such very intimate dyadic relation that they form to all intents and purposes a unity. Such a conception I have tried inadequately to develop in previous communications.^{5,6} I trust that the present series of studies will be permitted to rest outside the limits of ontological discussion.

Logically interesting, however, is the progress which can be made by the simple device of cutting an autopsy series or a clinical series in twain on the lines of supposed functionality and structurality. It may be conceded that many cases get pushed to the wrong side of the line, being called structural when they are really (on the present conception) functional, and *vice versa*. But these errors prove themselves in a manner familiar to those employing the statistical method.

The readers of this JOURNAL may recall certain papers on delusions written by Stearns, Tepper, and myself.^{9,10,11} In two of these papers the hypothesis was raised that the various (non-paretic) cases in question were really "functional" in the prevailing sense of cases without neural lesions. In a third paper I resorted to material which had to be regarded as "structural," viz., general paresis; but the conclusions founded thereon depend at least as much on the prevailing mode as did my former conclusions on somatic⁹ and environmental¹⁰ delusions in "normal-looking brain" cases.

How many of the symptoms of general paresis can safely be correlated with the lesions of general paresis as we know them? This question is exceedingly important, dealing as it does with that mental disease about which perhaps we know the most.^{12,13,14} The error in diagnosis is low,^{15,16,17} especially if compared with the error in psychiatric diagnosis at large,^{18,19} and the number of variables in our equations is correspondingly reduced.

In the study just mentioned¹¹ we concluded that the *characteristic* delusions of general paresis (found in 57 per cent. of all cases in a routine series, and in 75 per cent. of all cases showing delusions) are delusions about the patient's personality and that these delusions could be roughly correlated with frontal lobe lesions (non-autopsychic delusions failing to be so correlated). These conclusions were in general harmony with findings in dementia præcox.^{20,21}

For the present purpose I have split a certain series of autopsied parietic cases in twain on the basis of their showing or not showing substantial gross brain lesions. The series was chosen on the basis of personal examination by me at autopsy and of careful registration of all gross lesions found. The descriptions made were very particular and well-nigh final, since they were from the beginning destined to be compared with gross findings in various psychoses at one time commonly regarded as functional (dementia præcox, manic-depressive insanity). Without here considering the medically and therapeutically interesting fact that in this random series 18 brains showed no substantial gross lesions and a bare majority, 20, yielded such lesions, I shall proceed to a brief symptom analysis from a psychopathological point of view, reserving for publication elsewhere²² various medical implications of the work. All cases, both with and without *gross* lesions, possessed the characteristic microscopic lesions developed by the Nissl-Alzheimer school.

Before tabulating the symptoms found in the two "normal-looking" and "abnormal" brain groups or in what might be termed the "mild" and "severe" cases, I must add that we are in no sense dealing with early and late phases of the disease. In fact the mild cases are often the longest cases. There is no question of a progressively severer disease in many cases. The cases progress, it is true, in one sense toward their death, and they do not very often regress. Moreover stationary cases are rarities. But a case lasting five years is not necessarily an anatomically or histologically severer case than one lasting two years.

In explanation of the first two tables, I must premise that (1) The fourth columns contain the number of symptoms (named in the first column) found and catalogued in a series of 17,000 cases clinically analyzed at Danvers State Hospital, only a small portion of which have ever come to autopsy and many of which are still alive. The analysis does not pretend to weigh the importance of the symptoms listed or their dominance in the various cases. The 17,000 list is purely a frequency list. (2) The entries in the *second* column (mild) of Table I represent symptoms in their order of frequency in a series of 18 anatomically "mild" cases of general paresis, whereas in the *third* column (severe) of Table I appear symptoms in their order of frequency in 20 anatomically "severe" cases. (3) The entries in the *second*

column (severe) of Table II represent frequencies in the 20 anatomically "severe" cases and those in the *third* column (mild) the corresponding frequencies in the anatomically "mild" cases.

It occurs to me that some question may well be raised whether anatomical appearances can be safely trusted to gauge severity of processes. Certainly we are aware that in certain cases these appearances can *not* be trusted. But I assume that there can be no doubt that, by and large, the atrophic brain is more deeply affected than the normal-looking brain. At any rate it is a question whether the microscope can be trusted much farther quantitatively at the present time. And in any event the findings both anatomically and symptomatically indicate two groups of cases, whether we choose to regard them as "mild" and "severe" or not.

Without entering the total field of symptomatology in psychiatry, I may perhaps add that I do not necessarily approve the nomenclature of symptoms here adopted and merely record the entries as they stand. The influences of Kraepelin and of Wernicke are plain in the nomenclature, despite the fact that a majority of the facts were collected before the work of either of these masters had come into close contact with practical American psychiatry.

Those symptoms have been included in all columns which occurred in 20 per cent. or more of any of the three series.

TABLE I

SYMPTOMS ARRANGED IN THE ORDER OF THOSE MOST FREQUENT IN THE ANATOMICALLY MILD CASES

	18 Mild	20 Severe	17,000
Amnesia	11	11	3,422
Motor restlessness	10	11	5,428
Disorientation	10	10	2,419
Delusions, allopsychic	9	3	6,844
Dementia	8	9	5,841
Depression	7	9	5,015
Irritability	7	6	2,714
Defective judgment	7	8	2,596
Psychomotor excitement	6	5	6,003
Delusions, autopsychic	6	7	4,897
Destructiveness	6	1	2,362
Resistiveness	6	3	2,051
Insomnia	5	4	4,354
Violence	5	2	3,244
Aphasia	5	9	1,180
Hallucinations, not specified	5	6	885
Convulsions	5	4	413
Hallucinations, visual	4	6	3,186
Sicchasia	4	2	1,597

I have italicized those figures in the 17,000 columns which represent 20 per cent or more of the 17,000.

TABLE II
SYMPTOMS ARRANGED IN THE ORDER OF THOSE MOST FREQUENT IN THE
ANATOMICALLY SEVERE CASES

	20 Severe	18 Mild	17,000
Amnesia	11	11	3,422
Motor restlessness	11	10	5,428
Disorientation	10	10	2,419
Dementia	9	8	5,841
Depression	9	7	5,015
Aphasia	9	5	1,180
Defective judgment	8	7	2,596
Delusions, autopsychic	7	6	4,897
Irritability	6	7	2,714
Hallucinations, not specified	6	5	885
Hallucinations, visual	6	4	3,186
Euphoria	6	3	590
Psychomotor excitement	5	6	6,903
Incoherence	5	3	4,130
Confusion	5	1	2,120
Expansiveness	5	2	386
Insomnia	4	5	4,354
Convulsions	4	5	413
Exaltation	4	2	1,711

If we regard the ten statistically leading symptoms in the 17,000 cases as the most frequent of all psychiatric symptoms, and possibly as the most important (although I do not assert the latter), then it is of interest to inquire how far paresis partici-

TABLE III
SYMPTOMS ARRANGED IN THE ORDER OF THOSE MOST FREQUENT IN
17,000 CASES

	17,000	18 Mild	20 Severe
Psychomotor excitement	6,903	6	5
Delusions, allopsychic	6,844	9	3
Dementia	5,841	8	9
Hallucinations, auditory	5,428	2	1
Motor restlessness	5,428	10	11
Depression	5,015	7	9
Delusions, autopsychic	4,897	6	7
Insomnia	4,354	5	4
Incoherence	4,130	3	5
Amnesia	3,422	11	11
Violence	3,244	5	2
Hallucinations, visual	3,186	4	6
Irritability	2,714	7	6
Defective judgment	2,596	7	8
Disorientation	2,419	10	10
Destructiveness	2,362	6	1
Confusion	2,120	1	5
Resistiveness	2,051	6	3
Delusions, somatic	1,829	0	0

pates in the nature of mental disease at large and how far it is differentiated on this statistical basis.

The following tables bring out the answer :

In a fourth table I have placed the symptoms in order of frequency as they occurred in 17,000 cases of mental disease analyzed at the Danvers Hospital. The first ten of these symptoms occurred in at least 3,400 cases, that is, in 20 per cent. or more of the series, and the remaining nine are added to secure a statistical parallel to the facts in Tables I and II.

TABLE IV

Mental Disease in General	General Paresis	
	Anatomically Mild	Anatomically Severe
1. Psychomotor excitement	9th to 12th	13th to 16th
2. Allopsychic delusions..	4th	Not in first nineteen
3. Dementia	5th	4th to 6th
4. Auditory hallucinations	Not in first nineteen	Not in first nineteen
5. Motor restlessness	2d	2d
6. Depression	6th to 8th	4th to 6th
7. Autopsychic delusions..	9th to 12th	8th
8. Insomnia	13th to 17th	17th to 19th
9. Incoherence	Not in first nineteen	13th to 16th
10. Amnesia	1st	1st
11. Violence	13th to 17th	Not in first nineteen
12. Visual hallucinations ..	18th or 19th	9th to 12th
13. Irritability	6th to 8th	9th to 12th
14. Defective judgment ...	6th to 8th	7th
15. Disorientation	3d	3d
16. Destructiveness	9th to 12th	Not in first nineteen
17. Confusion	Not in first nineteen	13th to 16th
18. Resistiveness	9th to 12th	Not in first nineteen
19. Somatic delusions	Not in first nineteen	Not in first nineteen

Analysis of this table shows that *auditory hallucinations* and *somatic delusions* are the only symptoms which, while appearing amongst the first nineteen symptoms of mental disease in general, fail to appear among the first nineteen symptoms of general paresis in either the mild or the severe group. It will be remembered that the first nineteen symptoms in general paresis were chosen as occurring in at least 20 per cent. of the cases studied, and that but ten symptoms in mental disease at large occur in over 20 per cent. of cases. Hence the failure of *auditory hallucinations* to occur in any considerable number of cases of paresis is made more striking than the absence of *somatic delusions*. The presence of *visual hallucinations*, to be sure at the bottom of the list among mild cases, but in fair proportion among severe cases, is theoretically hard to explain, when taken in conjunction with the paucity of *auditory hallucinations*. Indications in the literature point perhaps to optic nerve lesions as

a possible basis for the *visual hallucinations*, suggesting an almost illusory origin therefor.

The fact that *allopsychic delusions* are so common, at least in the mild cases, seems to show that they are not correlated with *auditory hallucinations* either as cause or effect. It is as if there were not even pseudoreality to the *allopsychic delusions* and as if they did not appear even to the patient as representing centripetal (*e. g.*, hostile) effects. In fact, as will appear below, these *allopsychic delusions* are associated more with refusal of food (hallucinatory tastes ?, comments on indigestion?) than with *auditory hallucinations*. The study of *allopsychic delusions* in the parietic ought therefore to present conceptions of a quite disparate order to those of the victim of dementia præcox, where *auditory hallucinations* are so characteristic (see recent redeterminations of a statistical nature by Stearns²³).

The paucity of *somatic delusions* in both parietic groups is perhaps not surprising and is in line with some previous determinations, including those of Southard and Tepper.¹¹ The peripheral origin of many somatic delusions or at all events their strong peripheral element, as claimed in previous papers,^{9,24} is consistent with this determination. The presence of a fair proportion of *visual hallucinations* remains astounding except on the basis of optic nerve changes mentioned above. Since Canavan²⁵ has shown a high proportion of chronic optic nerve changes in routine autopsied cases of all sorts of mental disease (parietic and non-parietic), it might be argued that *visual hallucinations* should be more common in mental disease at large. In point of fact *visual hallucinations* do seem to stand somewhat higher in order of frequency in mental disease at large than might have been *a priori* supposed. But, why, if *visual hallucinations* are really related (as some assert) with peripheral nerve changes, should not *tactile* and other *haptic hallucinations* occur more frequently in general paresis, in which the peripheral nerves are not infrequently involved? Perhaps such *haptic hallucinations* do occur but fail to reach the medical observer.

The agreement of both parietic groups in placing *amnesia*, *motor restlessness*, and *disorientation* in one, two, three order is of great interest. If we omit the anomalous *allopsychic delusions* from the mild group for the moment, then *dementia* would follow as a fourth common symptom. Further discussion is placed below.

For the purposes of Table IV we extended the list of symptoms from mental disease at large to nineteen for comparison with the nineteen symptoms which we had found to occur in over 20 per cent. of all cases of paresis. As a matter of fact the two lists of nineteen symptoms in paresis are not identical, and the differences are instructive.

The following are symptoms which occur in over 20 per cent. of the mild cases that do not occur in 20 per cent. of the severe cases.

Allopsychic delusions.....	9 in 18	3 in 20	6,844 in 17,000
Sicchasia.....	4 in 18	2 in 20	1,597 in 17,000
Resistiveness.....	6 in 18	3 in 20	2,051 in 17,000
Destructiveness.....	6 in 18	1 in 20	2,362 in 17,000
Violence.....	5 in 18	2 in 20	3,244 in 17,000

I have arranged the list arbitrarily on the basis of a vague conception of the interrelation and possibly the intergrading of some of these symptoms. I believe their mutual relations are plain: *the mild case of paresis*, in more than a fifth of all cases and often in far more than a fifth, *is reacting to his environment* (especially to his personal entourage) *most markedly*. Let us glance at the symptoms which distinguish the anatomically severe from the mild cases, since they fail to occur in 20 per cent. of the latter.

Euphoria.....	6 in 20	3 in 18	590 in 17,000
Expansiveness.....	5 in 20	2 in 18	386 in 17,000
Exaltation.....	4 in 20	2 in 18	2,711 in 17,000
Confusion.....	5 in 20	1 in 18	2,120 in 17,000
Incoherence.....	5 in 20	3 in 18	4,130 in 17,000

Here again, just as perhaps we might separate two symptoms (*allopsychic delusions* and *sicchasia*) from the other three which form a group by themselves among the distinguishing features of the "mild" group, so we may separate *confusion* and *incoherence* from the other three mutually related symptoms, *euphoria*, *expansiveness*, and *exaltation* in the "severe" group.

It was the observation of this contrast which caused me to write out the present paper for this JOURNAL, since I felt there was a general psychopathological interest to the contrast, which must very probably be based on structural differences in disease-process.

I have throughout left the impression that the structural differences in the two groups are largely those of extent. Perhaps

extent, depth, and serial involvement of cortex layers may indeed have something to do with these functional differences. Histological studies of striking instances of these phenomena may well confirm one or other of these conceptions.

Meantime we should also take into account the habitual preference of gross brain lesions in general paresis for the frontal region. With this fact in mind, a somewhat speculative account of the situation might run to this effect: That the *severe cases* with gross brain involvement *tend to leave the parietal regions* relatively intact and subject to operations *unchecked by the great inhibitory frontal areas*. The expansiveness of the parietic would accordingly resemble the hyperphantasia of certain victims of dementia præcox. The latter I have been trying to associate with the mild atrophic lesions of the parietal regions which affect certain cases of dementia præcox.²¹ General paresis very probably often possesses similarly mild lesions of the parietal regions, differing from those of dementia præcox in being exudative rather than merely degenerative. But at a time when these parietal lesions are beginning to develop in paresis, the frontal regions are doubtless often far on the road to coarse atrophy. Inhibitory power the frontal regions no longer possess, certainly over many motor activities, possibly over various conceptual processes. Thus might be explained both the resemblances and the divergences of hyperphantasia (fantastic delusions) and expansiveness (delusions of grandeur).

But now, as has been stated, a large minority of cases of paresis fail to die with coarse brain atrophy. All these cases have exudative lesions of more or less prominence, despite the absence of coarse brain atrophy. Just as the mild lesions of the parietal regions may produce (virtually as irritative symptoms) *expansiveness* and attendant *euphoria* and *exaltation* at the same time as coarse frontal destruction is leading to *confusion*, *incoherence*, and a disintegration of the patient's entire attitude to men and things, so the mild lesions of the frontal region may be leading to the above mentioned *anti-environmental* group of symptoms in the non-atrophic group. Action is not inhibited in its entirety or in its coarser manifestations. The operation of an exudative (and not yet extremely destructive) lesion in this frontal area may act in part to abolish the inhibitions which are very possibly the proper function of this area, but may also act in part to irritate, interrupt, and throw into disorder those inhibitions. The mild microscopic lesions in these non-atrophic cases

may act to bring about not the classical loss of inhibition but a perversion of inhibition, an incoördinate and irregular checking of activities, and of those *inactivities* which proper conduct often requires. On such lines could be explained with some plausibility the *resistiveness*, *destructiveness*, and *violence* which appear to be characteristic of these non-atrophic cases.

As to an explanation of the *delusions of persecution* and *refusal of food*, the situation is perhaps not so clear. The *sicchasia* may sometimes be an example of *resistiveness* and again due to delusions. If the former, then the symptom would best be explained as the result of disorder of inhibition. If the latter, I can only offer the analogy of dementia præcox, in which for some reason or other delusions (except fantastic) are rather closely associated with frontal lobe lesions. The psychopathology of delusions is obscure. I hold the opinion, however, that delusions represent more a disorder of believing than a group of false beliefs, rather more a perversion of volitional process than of intellectual process. On this line of reasoning I find it somewhat easy to reconcile the relation of the mild frontal lesions here found to delusions about the environment. Thus I would align together all five of the distinctive symptoms of the mild group with perversions of inhibition, presumably largely due to frontal lobe lesions even though these are hardly or not at all represented in the gross. In cases with more extensive frontal lobe destruction (coupled often perhaps with the establishment of *mild* lesions elsewhere in the cortex), the perversions of inhibition are replaced by frank losses thereof: the anti-environmental tendencies of the mild cases are replaced by less socially disturbing yet more profound disorder of personality.

SUMMARY AND CONCLUSIONS

The possession of a suitable statistical background (The Danvers Case Symptom Index) has rendered worth while an orienting study in the mental symptomatology of general paresis. A group of 38 general paretics whose brains were specially examined and described by the writer, has been divided into two groups according to whether there was or was not coarse evidence of brain atrophy. The cases without brain atrophy were termed "mild" and those with brain atrophy were termed "severe," although these designations are only approximations to accuracy; the groups are, however in no sense "early" and "prolonged."

Symptomatically the two groups show several surprising concordances and a number of instructive divergences. Thus *amnesia, motor restlessness, disorientation, dementia, and depression* lead both series and in that order (except that *allopsychic delusions* stand fourth in the "mild" series and are far less common in the "severe"). *Are amnesia and dementia therefore in no sense proportionate to brain tissue loss?*

Nineteen symptoms occurred in 20 per cent. or over of the paretic series, viz., the five just mentioned, and nine others (*irritability, defective judgment, psychomotor excitement, autopsychic delusions, insomnia, aphasia, hallucinations* of doubtful or unspecified nature, *convulsions, visual hallucinations*) not always in like proportion in the two series. Five other symptoms occurred in each series, but symptoms quite sundered from one another in general significance.

The "mild" cases showed a group of symptoms which might be termed *contra-environmental*, viz., *allopsychic delusions, sychasia* (refusal of food), *resistiveness, violence, destructiveness*,

The "severe" cases showed a group of symptoms of a quite different order, affecting *personality*, either to a ruin of its mechanisms in *confusion* and *incoherence*, or to the mental quietus involved in *euphoria, exaltation, or expansiveness*.

Some speculations are offered in the text as to the perversion of inhibition or incoördination of inhibition which the largely irritative lesions of the "mild" cases are presumably effecting in the perhaps more seriously affected frontal areas. When these are still more gravely affected, as to the point of atrophy, then the intrapsychic disorder might well become more manifest, *e. g.*, in the distinctive symptoms of the "severe" group just mentioned.

In a series of 17,000 clinical cases (of all sorts of mental disease, alive and dead, recovered and impaired) symptomatologically analyzed, there were but ten symptoms occurring in 20 per cent. or over; These were in order, *psychomotor excitement, allopsychic delusions, dementia, auditory hallucinations, motor restlessness, depression, autopsychic delusions, insomnia, incoherence, amnesia*. Each of these is represented high in general paresis (*i. e.*, in 20 per cent. or over) except that *auditory hallucinations* are infrequent in both "mild" and "severe" cases and *allopsychic delusions* are infrequent in "severe" cases. There may be topographical reasons for the paucity of *auditory hallucinations* in general paresis. The method of production of

allopsychic delusions in general paresis should be studied, since there can be no such alliance of *allopsychic delusions* and *auditory hallucinations* therein as is perhaps the rule in dementia præcox.

If we consider the next *nine* symptoms in order in 17,000 cases of mental disease at large, viz., *violence*, *visual hallucinations*, *irritability*, *defective judgment*, *disorientation*, *destructiveness*, *confusion*, *resistiveness*, and *somatic delusions*, we find only the last, viz., *somatic delusions*, not represented in either group in fair proportion, although (as above stated) *confusion* is poorly represented in the "mild" cases and *violence*, *destructiveness*, and *resistiveness* are poorly represented in the "severe" cases.

Aphasia, *hallucinations* of doubtful or unspecified nature, and *convulsions* appear to be frequent symptoms in general paresis that do not figure at all so largely in mental disease as a whole. Besides these, *sicchasia* of the "mild" group and *euphoria*, *exaltation*, and *expansiveness* of the "severe" group appear to stand out for general paresis against mental disease as a whole.

The most positive results of this orienting study appear to be the unlikelihood of *euphoria* and allied symptoms in the "mild" or non-atrophic cases and the unlikelihood of certain symptoms, here termed *contra-environmental*, in the "severe" or atrophic cases. Perhaps these statistical facts may lay a foundation for a study of the pathogenesis of these symptoms. Meantime the pathogenesis of such symptoms as *amnesia* and *dementia* cannot be said to be nearer a structural resolution, as these symptoms appear to be approximately as common in the "mild" as in the "severe" groups.

REFERENCES

1. McGaffin. A Study of the Forms of Mental Disease in Cases Showing no Gross Lesions in the Brain at Autopsy. Proceedings of the American Medico-Psychological Association, May, 1912.
2. Southard. A Series of Normal-looking Brains in Psychopathic Subjects. American Journal of Insanity, April, 1913.
3. Southard and Canavan. A Series of Normal-looking Brains: Second note (Westboro State Hospital material). JOURNAL OF NERVOUS AND MENTAL DISEASE, December, 1914.
4. Southard and Canavan. A Series of Normal-looking Brains: Third note (Boston State Hospital material), Boston Medical and Surgical Journal, Jan. 28, 1915.
5. Southard. Psychopathology and Neuropathology: The Problems of Teaching and Research Contrasted. Journal of American Medical Association, March, 1912, and American Journal of Psychology, April, 1912.
6. Southard. The Mind Twist and Brain Spot Hypotheses in Psychopathology and Neuropathology. Psychological Bulletin, April, Vol. xi, 1914.
7. Southard. The Association of Various Hyperkinetic Symptoms with Partial Lesions of the Optic Thalamus. JOURNAL OF NERVOUS AND MENTAL DISEASE, October, 1914.

8. Southard and Canavan. Analysis of Five Cases of Quasi Functional Disease of the Mind: Being a Sixth Note on Normal-looking Brains in Psychopathic Subjects. In preparation, to be submitted to *Journal of Medical Research*, 1916.
9. Southard. On the Somatic Sources of Somatic Delusions. *Journal of Abnormal Psychology*, December, 1913.
10. Southard and Stearns. How Far is the Environment Responsible for Delusions? *Journal of Abnormal Psychology*, June-July, 1913.
11. Southard and Tepper. The Possible Correlation Between Delusions and Cortex Lesions in General Paresis. *Journal of Abnormal Psychology*, October-November, 1913.
12. Nissl. Zur Histopathologie der paralytischen Rindenerkrankung. Histologische und Histopathologische Arbeiten über die Grosshirnrinde, Bd. I, 1904.
13. Alzheimer. Histologische Studien zur Differenzialdiagnose der progressiven Paralyse. Histologische und Histopathologische Arbeiten über die Grosshirnrinde, Bd. I, 1904.
14. Kraepelin. General Paresis. (From Ein Lehrbuch für Studierende und Ärzte, III Bd. II Teil, 1913.) Translated by J. W. Moore, Monographs of *JOURNAL OF NERVOUS AND MENTAL DISEASE*.
15. Southard. A Study of Errors in the Diagnosis of General Paresis. *JOURNAL OF NERVOUS AND MENTAL DISEASE*, Vol. 37, No. 1, January, 1910.
16. Orton. An Analysis of Errors in Diagnoses in a Series of 60 Cases of Paresis. *JOURNAL OF NERVOUS AND MENTAL DISEASE*, Vol. 40, 1913.
17. Morse. The Correlations of Cerebrospinal Fluid Examinations with Psychiatric Diagnoses—A Study of 140 Cases. *Boston Medical and Surgical Journal*, Vol. clxx, No. 11, March 12, 1914.
18. Southard. The Margin of Error in the Diagnosis of Mental Disease: Based on a Clinical and Anatomical Review of 250 Cases Examined at the Danvers State Hospital, Massachusetts, 1904-1908. *Boston Medical and Surgical Journal*, August, 1910.
19. Southard and Stearns. The Margin of Error in Psychopathic Hospital Diagnoses. *Boston Medical and Surgical Journal*, December, 1914.
20. Southard and Ayer. Dementia Præcox, Paranoid, Associated with Bronchiectatic Lung Disease and Terminated by Brain Abscesses (*Micrococcus Catarrhalis*). *Boston Medical and Surgical Journal*, December, 1908.
21. Southard. A Study of the Dementia Præcox Group in the Light of Certain Cases Showing Anomalies or Scleroses in Particular Brain-Regions. *Proceedings of the American Medico-Psychological Association*, May, 1910; also *Am. Jour. Insanity*, 1910.
22. Southard. On the Absence of Coarse Brain Lesions in Many Cases of General Paresis (paper to be published in a series of papers read at a conference at Danvers State Hospital, Nov. 19, 1915).
23. Stearns. Occurrence of Hallucinoses in 500 Cases of Mental Disease. *JOURNAL OF NERVOUS AND MENTAL DISEASE*, January, 1915.
24. Southard and Bond. Clinical and Anatomical Analysis of 25 Cases of Mental Disease Arising in the Fifth Decade, with Remarks on the Melancholia Question and Further Observations on the Distribution of Cortical Pigments. *Proceedings of the American Medico-Psychological Association*, June, 1913.
25. Canavan. A Histological Study of the Optic Nerves in a Random Series of Insane Hospital Cases. (*JOURNAL OF NERVOUS AND MENTAL DISEASE*, March, 1916.)