

quately some of the contentions of the dissociationists, while remaining not wholly inconsistent with Kraepelinian ideas.

14. The frontal-paranoid correlation is in line with modern physiological ideas, but it must be granted that the occipital and temporal regions, as elaborating important long-distance impulses, may well play a part also in paranoid states.

15. The cerebellar-katatonic correlation is doubtless in line with some contentions of the Wernicke school, and obvious comments might be made in connection with the proprioceptive functions of the cerebellum (Sherrington).

16. The post-central-superior parietal relations to katatonic symptoms are perhaps theoretically the most novel suggestion from the work, but here again the results are not inconsistent with modern physiology.

17. The topographic study of dementia precox brains, both gross and microscopic, is commended as likely to shed new light on the pathogenesis of certain symptoms, notably paranoidal and katatonic symptoms.

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## IV.

## TEN OBSCURE CASES OF MENTAL DISEASE: A CLINICAL AND ANATOMICAL STUDY.

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In his study of the margin of error in the diagnosis of mental disease, Southard found 10 cases (4%) of a series of 250, specially reported clinically (at Danvers Hospital conferences) and later autopsied, which, unlike the remainder of the series, did not readily fall into accepted clinical groups. As it was thought that a more intensive study of these cases might reveal appropriate diagnoses, the following analysis was undertaken. First, condensed histories and anatomical analyses were drawn up, from which provisional anatomical and clinical diagnoses were made. Secondly, analysis of available microscopic material was made in every case, and provisional diagnoses made therefrom. Thirdly, a comparison of the three provisional — clinical, anatomical and microscopical — diagnoses was made for each case, and on the basis of this comparison a set of probable diagnoses of still greater finality was drawn up.

Owing to the obscure state in which the structural aspect of mental disease still remains, it is obviously impossible from a study of the microscopical brain and gross autopsy findings alone definitely to place a case in a clinical group. A tentative grouping was made of cases with similar morphological findings, and an effort made, first, to place the cases examined in this study in one of these groups, and then more definitely in a clinical group if possible. Four groups were made as follows:

I. Organic group, in which there are prominent blood-vessel changes or well-defined destruction of brain tissue. It includes general paralysis, cerebral syphilis, arteriosclerotic insanity, tumors, trauma, etc.

II. Senile group, in which there is a diffuse destruction of nerve cells without (necessarily) prominent vessel changes, accompanied by either atrophy of convolutions or decrease in brain weight. It may or may not be accompanied by marked visceral disease.

III. A group of which the mental symptoms may be due to toxic and nutritional disturbances. This group includes cases in which the brain shows no changes outside of physiological limits, but where marked visceral disease of such a nature as to produce toxins or disturbances of nutrition is found. It is made up of the infection and exhaustion psychoses, thyrogenous insanity, etc.

IV. Functional group, in which there are no universally accepted findings of note either in the brain or trunk. It includes dementia precox, manic-depressive insanity and the acute intoxication psychoses.

These groups are not entirely satisfactory. The first and second are fairly definite, but the third and fourth are rather loose. It is impossible to say whether a certain combination of lesions would produce sufficient disturbance of nutri-

tion to cause mental symptoms, so all that can be done is to select lesions that must produce some disturbance of nutrition and assume that they were or were not sufficient to cause mental symptoms in the particular case. Unfortunately, this method will not identify the acute toxic and exhaustive psychoses that have no distinct pathology. It also does not exclude the last group, the "functional" diseases, which may be associated with visceral disease. However, the classification was the best that presented itself for the study of a group of mental cases from the morphological side, and the diagnoses are made tentatively, subject to revision when compared with the clinical symptoms.

The cases are presented in the form of condensed clinical summaries and summarized autopsy protocols. The microscopic examinations are by the writer.

CASE I. E. N., Path. No. 850. Male, age seventy years, bookkeeper. Admitted to Danvers State Hospital January, 1883; discharged Jan. 14, 1888. Readmitted Dec. 10, 1903; died Feb. 12, 1904.

One sister insane. Father died of consumption. First admission: Had a common school education. Was cheerful; a moderate drinker; used tobacco to excess. Had been a bookkeeper since 1872. Cause of trouble given as overwork and ill-health. Poor health for one year; for two months in Boston City Hospital. Here he had a cough, vertigo, dyspnea on exertion and pulse of 112. Nervous and excitable.

In hospital was depressed, apprehensive, not violent, had delusions. Took nourishment poorly. Believed he set fire to the city of Portland. Apathetic. Physically on admission was poorly nourished; physical and neurological negative. Diagnosis: Delusional insanity. Discharged on a visit, January, 1907, but was soon brought back by friends.

Second admission, December, 1903. Quiet, feeble, rambled in conversation. Orientation good for place, defective for time. Poor knowledge of current events. No delusions or hallucinations; fair insight. Memory fair for remote events, not so good for recent events; impressibility poor. Emotions normal; at times is confused. Diagnosis: Senile dementia.

Physical examination on admission: Emaciated; arcus senilis. Heart regular, first sound rough, radials thickened. Trace of albumin. Very weak. Tremor and twitching of fingers. Reflexes normal. Pupils equal and sluggish. Sensation dulled.

*Autopsy* by A. M. Barrett: Decubitus, emaciation, miliary tuberculosis of lungs, liver, spleen and umbilicus, with ulceration, chronic adhesive pleuritis, left and right, chronic fibrous myocarditis, milk spot, chronic mitral endocarditis, chronic fibrous peritonitis, arteriosclerosis with calcification of aorta, chronic parenchymatous nephritis, enlargement mesenteric lymph nodes, chronic external adhesive pachymeningitis, arteriosclerosis of small arteries of brain. Brain weight, 1440 gm.

*Cause of death:* Miliary tuberculosis, senile debility.

No. 850. The cortex picture is that of a senile brain. The nerve cells are atrophic and deeply pigmented and are slightly decreased in number. The glial cells are increased in number in the gray matter and contain pigment. A number of satellite cells surround the larger nerve cells. The blood vessels are moderately sclerotic, with an increased amount of pigment in the adventitia. Although no mention of convolucional atrophy was made in the protocol, the diagnosis of

senile dementia is made on the presence of regressive changes in the nerve cells and glia and the cortical arteriosclerosis.

In the protocol, certain visceral lesions are mentioned which must have produced toxic and nutritive disturbances, and which may have had some influence on the mental symptoms. The chronic tuberculosis and chronic nephritis are probably related with the emaciation noted at autopsy. The milk spot, endocarditis, chronic peritonitis and pleuritis may indicate a former acute infection, or even septicemia, which may also have been a factor in causing the mental symptoms.

The first mental trouble, coming on during a period of ill-health and low vitality, is indefinite in nature and can best be explained as due to a disturbance of nutrition acting on a brain with hereditary weaknesses. It seems reasonable to assume that this attack may have been in some way associated with the septicemia mentioned above; at least the mental symptoms cleared up when the physical conditions improved, and for a number of years the patient was probably without mental symptoms. The second mental disturbance was typical of senile dementia and may or may not have had a connection with the first trouble. The toxemia resulting from the tuberculosis could be thought of as helping to produce mental symptoms, since the brain findings are hardly prominent enough to account for great mental disturbance.

CASE II. M. P., Path., No. 896. Female, age seventy-five dressmaker. Admitted to Danvers State Hospital Jan. 1, 1904. Died Aug. 12, 1904.

*Heredity:* Negative. Common school education. Married at the age of twenty-seven; three children, two girls alive, youngest forty-two years old. Husband died thirty-six years ago. Since that time worked at dressmaking. Had articular rheumatism for twenty years. Went to Salem Almshouse twenty years ago. Onset gradual, five years ago. Thought people talked of her, saying she was a murderess and prisoner; calm unless spoken to, when she became excited.

In hospital was quiet, good-natured, talked freely, depressed. Well oriented, fair knowledge of current events. Memory for recent and remote events good. Heard voices talking about her, usually at night, and had delusions of persecution; often cross and irritable. No insight.

*Physical examination* on admission. Poorly developed, emaciated; skin dry, eyes sunken; joints of hands and feet enlarged. Pupils normal, arcus senilis. Hearing and vision defective. Reflexes normal. Dullness of right apex. Arteries knotty. Pulse 100, weak. Second aortic accentuated. No murmurs.

*Autopsy:* Seventy-seven hours post-mortem by A. M. Barrett: Bronchopneumonia of both lungs, acute splenitis, chronic diffuse nephritis, chronic passive congestion of liver, milk spot, old scars at apices, enlargement retroperitoneal lymph nodes, sclerosis of basal vessels, chronic leptomeningitis, increased cerebrospinal fluid, unequal pupils. Brain weight, 1180 gm.

*Cause of death:* Bronchopneumonia.

No. 896. The changes found in the brain are senile in character. The nerve cells show atrophic changes everywhere and are decreased in number to a slight extent. The glial cells are increased focally both in

gray and white matter, and especially so in the subpial region. Many cells contain pigment. The vessels are slightly arteriosclerotic in places. These changes are nowhere prominent, and it is difficult to say whether they are outside the physiological senile limit, especially since no mention of atrophy of convolutions is made in the protocol.

Outside the terminal condition of bronchopneumonia, there is nothing in the gross findings severe enough to have much importance in producing mental symptoms. The only things in the morphology of any importance, then, are the slight senile brain changes, and the diagnosis of a senile condition of a mild degree must be made. The brain weight is low, 1180 gm., and there may have been some atrophy of convolutions that was not recorded.

The diagnosis of senile paranoia was made clinically, and doubtless is correct. Whether the diagnosis can be said to be supported by the morphological findings is an open question. At least there is nothing to disprove it, and the fact that there was little dementia present agrees with the absence of great atrophy of convolutions and the slight changes found in the microscopical picture of the cortex. It must be admitted, however, that these changes are very slight and in another case might not be associated with mental symptoms at all and that the real cause of the mental disturbance is not clearly disclosed in the morphological findings.

**CASE III.** J. M., Path. No. 904. Male, age thirty years, hatmaker. Admitted to Danvers State Hospital Nov. 21, 1903; died Sept. 4, 1904.

*Previous history* not obtained. *Onset* gradual. Used little liquor. Melancholy. Refused to speak; eyes staring and vacant; would not eat or work. Laughed and cried, slept most of time, shunned companions.

In hospital was quiet, stayed in bed, mute. Understood directions pretty well, took nourishment, cheerful, lay in bed quietly for hours. Would not work at one thing for very long. No cerea. Never spoke a word.

*Physical examination* on admission. Well-developed, well nourished. Physical and neurological negative.

*Autopsy* eighteen hours post-mortem by A. M. Barrett. Acute ulcerative ileocolitis, acute valvular endocarditis (aortic mitral), acute lymphadenitis of mesenteric lymph nodes and those of colon, slight hydropericardium, acute proctitis, fatty myocarditis, chronic diffuse nephritis, fatty liver, chronic passive congestion of the liver, chronic leptomeningitis (frontal), granular ependymitis.

Brain weight, 1650 gm.

*Cause of death:* Ulcerative enterocolitis (dysentery).

No. 904. Nissl sections show marked cell changes of a destructive nature, and an actual decrease in the number of nerve cells. None of the cells contain much pigment. The neuroglia is not remarkable. Satellite cells surround the larger nerve cells. The vessels show pigmentation in places, but are not thickened. Marchi sections show myelin degeneration in the white matter.

Outside the terminal condition, the only gross condition of interest is the chronic diffuse nephritis. The nerve cell changes noted are probably due to the terminal condition. Hence no

diagnosis can be made, and the case is placed in the functional group.

Clinically, the diagnosis is obscure because of absence of history and because the patient refused to speak. The mutism without retardation suggests dementia precox, but there were no other signs that would indicate it. Since this diagnosis cannot be either supported or disproved from the morphological picture, it will have to remain in the functional group without being diagnosed more definitely. Manic-depressive insanity is preferred.

**CASE IV.** B. C. S., Path. No. 969. Male, age fifty-eight years, junk dealer. Admitted to Danvers State Hospital Dec. 30, 1904; died April 21, 1905.

Father and mother both lived to old age. No history of insanity given. Born in Massachusetts; was married fifteen and thirty-five years ago; had three children. No illnesses, no alcohol used. Two years ago was operated on at the Massachusetts General Hospital for carcinoma recti and anal fistula. Here was given morphine, which he continued to use after leaving hospital. Had stricture of the urethra and was operated on several months later at the Malden Hospital for pain in the abdomen, and diffuse aneurysm of left common iliac was found. While here had to be restrained with a sheet. No relief from operation. Went to Carney Hospital for another operation, abdominal, which showed nothing; grew worse. Operated on privately for varicocele. Still continued to have much trouble with stomach; vomited and refused to eat. Wife thinks he ate when she was away. Was quiet during the day, but noisy and restless at night. Threatened suicide, but never made an attempt; was destructive.

In hospital lay in bed; seemed to suffer great pain in abdominal regions. Oriented; no speech defects. School knowledge fair. Had no hallucination; memory fairly good; talked of stomach trouble all the time. Complained more when somebody was around. Tidy; restless at night. After some weeks became quieter; also became disorientated. Said he couldn't die.

*Physical examination* on admission: Poorly developed, poorly nourished, cyanosis of legs. Skin dry. Dulness over both scapulae. Heart enlarged to left. Brachials slightly thickened. Neurological examination negative. Hemaglobin, 60%; poikilocytosis.

*Autopsy* ten hours post-mortem by A. M. Barrett: Pulmonary hypostasis, emaciation, pulmonary tuberculosis slight, chronic diffuse nephritis, chronic orchitis, scar of penis, chronic fibrous leptomeningitis (frontal, parietal), chronic external adhesive pachymeningitis, soft edematous brain substance.

Brain weight, 1370 gm.

*Cause of death:* Pulmonary tuberculosis. Marasmus of involutional melancholia.

No. 969. The nerve cells show changes corresponding to the edema changes of Hoch (Dr. Barrett). Cells are not decreased in number. Pigmentation is prominent in all the larger nerve cells. Glia shows regressive changes. Satellitosis is marked in all the cell layers. There is a slight gliosis of the deeper cell layers. Vessels are not changed. Outside the terminal changes, there is not enough upon which to make a diagnosis.

In the autopsy findings there are pulmonary tuberculosis and chronic diffuse nephritis with emaciation, lesions which indicate that toxemia and disturbance of nutrition occurred. With little in the brain to explain the mental condition,

primary importance must be assigned to these changes, so the case is placed in the toxic group.

Clinically, the diagnosis of involutional melancholia was made. From the history one would conclude that disturbances in nutrition played an important part in the causation of the mental symptoms. The various operations, the morphinism and the stomach trouble all indicate such a disturbance, and, since they occurred before any mental symptoms, can be properly regarded as bearing on the mental symptoms. Although these changes were not disclosed at autopsy, they are in line with and confirm the autopsy findings. The brain changes are doubtless due to the disturbance of nutrition, so that this disturbance can perhaps be accepted as the whole cause of the mental trouble.

CASE V. M. G., Path. No. 976. Female, age sixty-five years, housewife. Admitted to Danvers State Hospital May 22, 1905; discharged June 7, 1905.

No insanity in family. Parents lived to old age. Education, common school. Left school to work. At twenty-one married a sailor; husband now a letter-carrier. Had three children, all living, youngest thirty-five years. No serious illnesses. Habits good. Trouble came on six months ago when patient worried much over domestic troubles. Had stomach trouble with vomiting, culminating in extreme weakness, for which patient was sent to Salem Hospital, May 12. Also had prolapse of uterus. After being at hospital for two days her conversation became disconnected and she became restless. Auditory hallucinations appeared and she was given opiates to quiet her. Was very restless and noisy and had to be watched constantly. Imagined she was naked. Mistook strange people for her friends. Transferred to Danvers State Hospital. In hospital confused, restless and resistive on admission. Thought children were outside and tried to get to them. Had auditory hallucinations. Soon became quieter, clearer and more communicative, tidy, lacked spontaneity, confused. Memory fairly good for recent (except most recent) and remote events. Easily became tired and confused. At first disoriented, but later orientation improved. Fair knowledge of current events. Attention poor. Later had insight into hallucinations. Temperature went up, and she had a positive diphtheria culture. Again became confused and disoriented. Given antitoxin, but died.

*Physical examination* on admission: Well developed, poorly nourished, feeble. Skin dry and wrinkled. Arcus senilis, pupils irregular, would not react. Defects of hearing and vision. Reflexes normal. Tremor of tongue and hands. Some inco-ordination. Heart: Systolic murmur. Arteries thick and tortuous. Trace of albumin in urine.

*Autopsy* fourteen hours post-mortem by A. M. Barrett: Pulmonary hypostasis, hemorrhage into adrenal, endometritis, chronic fibrous pleuritis, localized at apex, chronic fibrous pericarditis, chronic fibrous endocarditis, mitral sclerosis, chronic diffuse nephritis, chronic leptomenigitis (slight), diphtheria bacilli in throat.

Brain weight, 1,110 gm.

*Cause of death:* Nephritis.

976. Nerve cells show nothing of note. There are regressive changes in the neuroglia, especially in the gray matter, most marked in the subpial region. A moderate number of satellite cells occur in all cell layers. The only vessel change is some increase in the adventitial pigment.

In the autopsy notes are mentioned chronic diffuse nephritis, chronic fibrous endocarditis and pericarditis. Although they may indicate a disturbance of nutrition, with a septicemia, these lesions are naturally not enough to place the case in the toxic group. The brain weight is very low, but no atrophy of convolutions was mentioned in the protocol. The case is provisionally placed in the functional group.

Clinically the case is one of collapse delirium resulting from the stomach trouble and worry. With the improvement of the physical condition in the hospital came an improvement of the mental symptoms, but when the tonsillitis developed, the mental symptoms became worse again. An erroneous idea of the case is gained from the autopsy findings taken alone. Although the nephritis may have played some part in causing mental symptoms, the principal factor, the stomach trouble, was not such as to be demonstrable at autopsy. The case, therefore, can be shifted to the third group.

CASE VI. D. M., Path. No. 1013. Male, age fifty-seven years, laborer. Admitted to Danvers State Hospital May 18, 1905; died Oct. 24, 1905.

Uncle and cousins are insane in Ireland. Born in Ireland, came to this country when seventeen. Could read and write a little. Worked as a laborer and watchman in a factory. Married at twenty-seven years, had two children who died, one in infancy and one at ten years (fourteen years ago). Wife an invalid for two years. Used tobacco excessively, but never used alcohol until he became sick. Had not worked for twelve years. Became apprehensive and suspicious, thought people were mesmerizing him, had auditory hallucinations, would sit for hours in one position thinking. Gave himself up to the police because voices told him to kill somebody. Became worse the last year; memory good until a year ago when it failed considerably; was restless at night and confused. Once threatened wife. Had two sinking spells, one five years ago and one last winter, each lasting two weeks.

In hospital was confused, restless and noisy. Memory poor for recent and remote events. Disoriented, heard voices. Contradicted himself and fabricated. Sometimes euphoric, sometimes irritable. Tidy; once was in a stuporous condition for two days, was not rigid or resistive, was very weak. Explained it as a hypnotic state. Talks of the powers and God.

*Physical examination* on admission: Extremely well developed and well nourished, feeble. Heart negative. Pulse poor in volume and tension. Arteries not thickened; varicocele. Tremor of hands. Unsteady in Romberg position. Reflexes negative. Pupils equal, sluggish and irregular. Localization of sensation is poor and sensation is dulled.

*Autopsy* four hours post-mortem by L. Hoag: Pyopneumothorax, localized bronchopneumonia going to abscess formation, atelectasis of right lung, emaciation, fibrous pericarditis slight, arteriosclerosis slight, chronic fibrous pleuritis, old scar of left lung, relative insufficiency of tricuspid valve, chronic diffuse nephritis, slight pachymeningitis, chronic sulcal leptomenigitis, subpial edema, gyri narrowed, frontal slightly; atrophy of cerebral vessels.

*Cause of death:* Bronchopneumonia and abscess of lung, involutional psychosis.

No. 1013. Pyramidal cells show atrophic changes with increase of pigment. Neuroglia shows some activity. Considerable gliosis of white matter of one

frontal region. Neuroglia cells contain pigment. Satellitosis involving single cells excessively, but leaving most of them entirely free. No vessel changes of note. Adventitia contains an increased amount of pigment. These changes suggest only a senile condition. The narrowing of the gyri in the frontal region with the cortex changes point to a senile dementia. This is further confirmed by the finding of senile changes elsewhere, such as emaciation, arteriosclerosis and nephritis.

Clinically the case is undoubtedly one of katonian dementia precox, with slight deteriorations until one year before admission. After that time the symptoms suggest senile changes. The case could then be considered as a dementia precox complicated in later life by senile deterioration.

The latter condition is shown in the morphology, but there is nothing to indicate dementia precox. The frontal atrophy and gliosis may have been upon a congenital basis, and, if found early in life, would have led to the correct diagnosis. But, occurring as it did in connection with senile changes elsewhere, there is nothing to distinguish it from the identical condition which is often found in senile dementia. This case shows how easily one may be misled in a study of senile dementia by finding lesions which were present before the onset of the mental trouble. The case is transferred to the functional group.

**CASE VII.** R. Mc., Path. No. 1090. Female, age fifty-five years, no occupation. Admitted to Danvers State Hospital March 23, 1904; died Sept. 6, 1906.

Onset gradual. Patient said she was talking to the Lord. Romances, suspicious, afraid of being poisoned, restless and noisy at night. No further previous history obtained.

In hospital talked much, going into detail. Speech normal, orientation good, consciousness clear. School knowledge rather poor. Denied hallucinations; memory defective, better for recent than for remote events. Loquacious, almost a flight of ideas, had no definite delusions, somewhat suspicious. Untidy. Was fault-finding with nurses and physician, contradicted herself, emotional and irritable. Sometimes was troubled with dyspnea, fast pulse, edema and tendency to suppression of urine. Had bronchitis. In August, in one of these spells, had delusions and perhaps had hallucinations. Attacked other patients. Jan. 24, 1906, developed complete paralysis of right side, of gradual onset. Had a motor aphasia. March 6, 1906, "flaccid" paralysis had improved considerably. July 2, 1906: Convulsions, frothed at the mouth, stuporous for a day. Sept. 4, 1906, had a series of convulsions and died.

*Physical examination on admission:* Well nourished. Eczema of arms and legs, varicose ulcers. Joints enlarged, particularly those of left foot. Bronchitis in both lungs. Heart irregular, second pulmonic reduplicated, also a murmur not definitely located. Arteries thick. Very weak. Hearing and sight impaired. Reflexes normal. Pupils react sluggishly. Urine negative.

*Autopsy* forty-five hours post-mortem by E. E. Southard: Mural thrombosis of left auricle, early bronchopneumonia of lower left lobe, general arteriosclerosis moderate, chronic fibrous endocarditis, mitral sclerosis, fatty changes of myocardium, hypertrophy of heart, emphysema of lungs, chronic diffuse nephritis, infarct of kidney (?), chronic splenitis, cirrhosis of liver, chronic adhesive pleuritis (left), chronic perisplenitis,

diploë absent, cerebral softening of left cerebrum, semi-ovale and right sagittal ramus (cholesterin crystals), slight subpial hemorrhage in left fossa sylvii, chronic external adhesive pachymeningitis, atrophy (aplasia?) of right flocculus of cerebellum, right purulent otitis media. Cultures: *B. coli* and *B. flavus* in heart's blood, *B. coli* and *M. pyogenes aureus* in cerebrospinal fluid, *B. coli communis* and unidentified coccus in thrombus.

*Cause of death:* Cerebral hemorrhage, arteriosclerosis.

1090. Sections of the right frontal region show decrease of nerve cells of focal character and gliosis of the white matter. In other regions the nerve cells are not notable. Spider cells occur both in the gray and white matter and are very numerous in the subpial region. They contain pigment, especially in the gray matter. Satellitosis is marked. Vessels show no changes outside increase in adventitial pigment. Sections of orbital area show numerous compound granule cells. The findings might suggest a chronic intoxication of such a mild grade that no cell destruction resulted, but enough to stimulate the neuroglia to activity.

In the gross brain were found a cyst of softening of the right frontal region, and signs of hemorrhage in left centrum semi-ovale, which, however, was too recent to cause pyramidal degeneration or cell destruction. These lesions doubtless caused mental symptoms, but probably came subsequent to the first mental symptoms.

Other post-mortem findings are mural thrombosis of left auricle, infarct of kidney (?), chronic fibrous endocarditis, chronic splenitis, chronic pleuritis, cirrhosis of the liver and myocardial changes. *B. coli* with other bacteria were found in the thrombus, cerebrospinal fluid and heart's blood. The evidences of old infections in various regions and presence of bacteria in several regions post-mortem indicate that in this case there may have been a septicemia of long standing and mild grade. The evidence of septicemia with disturbance of elimination through the kidneys (chronic nephritis) indicate a chronic toxemia of sufficient severity to cause mental symptoms.

Clinically the case was left unclassified and evidently conforms to no clinical type. But the clinical symptoms are not inconsistent with the anatomical diagnosis of chronic toxic condition. The facts that the symptoms improved slightly after coming into the hospital, that they were very slight for a long time, but again increased during the attacks of heart and kidney trouble, show that the mental symptoms are closely allied to the physical condition. They may have been due more to the nephritis than to the septicemia, which may have developed secondarily as a result of the lowered vitality. In any case, there was a chronic intoxication, and it is reasonable to assume that the mental symptoms were due to this.

**CASE VIII.** L. B., Path. No. 1095. Male, age twenty-eight, shoemaker. Admitted to Danvers State Hospital July 10, 1906; died Sept. 17, 1906.

History of intemperance and insanity. Brothers and sisters are healthy. Had a common school education, worked on a farm for three years, then worked at trade of shoemaker. Was good-natured, quiet and seclusive, used liquors moderately. Had diseases of childhood and probably syphilis four years ago. Two years ago

was operated on, probably for bubos; did not feel well after the operation. For the last three weeks before commitment was restless at night. Two days before admission began to sing, read aloud from the Bible and talked of religion, had crying and laughing spells, and was very active; was never violent or threatening.

On admission was restless, laughed and sang; had to be placed in strong room. Orientation rather poor. Admitted auditory and visual hallucinations of angels singing and God speaking; memory was fair, could give history of life. Was untidy and violent, laughed, sang and talked to God; at times mute and resistive and refused to move. Once was found on the floor, rigid, speechless and refused to move. Took nourishment poorly and grew emaciated. Physically on admission well nourished and well developed. Heart and lungs negative. Arteries slightly hardened. Some incoordination of movements. Reflexes normal. Pupils negative. Sensations normal.

Autopsy three hours post-mortem by E. E. Southard: Acute otitis media (left), distention of bladder, edema of brain substance (encephalitis?), chronic fibrous myocarditis, mitral sclerosis, chronic splenitis, slight sclerosis arch of aorta, chronic orchitis (right), chronic external adhesive pachymeningitis, marked arachnoidal villi, gliosis occipital white matter, heterotopia gray matter in occipital region, more marked than in left.

Note: Culture from heart's blood gave *M. pyogenes* albus; from middle ear, *B. coli communis*; from cerebrospinal fluid an unidentified coccus.

*Cause of death:* Septicemia from infection of hand.

1095. The cortex sections show little. The nerve cells are not notable. The glia is not altered except that in some regions there is a slight gliosis of the infrastellate cell layers. Vessels not altered.

In the autopsy findings there is mentioned a gliosis of occipital white matter and heterotopia of gray matter in the occipital region. These brain findings in the absence of disease indicate a congenital disturbance, and, with the gliosis of the infrastellate cell layer, point to dementia precox. The case is placed in the functional group since there is not sufficient evidence to indicate anything else.

The diagnosis "dementia precox" was made in this case and agrees with the diagnosis from the morphology.

CASE IX. N. P. H., autopsy No. 1204. Male, age fifty-nine, accountant. Admitted to Danvers State Hospital Oct. 14, 1907; died the same day.

*History by son:* No heredity. Left school at fifteen years to work, conductor and auditor for a railroad, got along well. Married and had three healthy children. Had never used alcohol, used tobacco moderately. Always cheerful, had always been dyspeptic. Otherwise no history of illness.

Onset six months ago, when patient complained of loss of sleep and fatigue, but continued to work until three months ago, when he broke down and had to quit work. Often had difficulty in breathing and a doctor diagnosed Bright's disease. About a month ago motor aphasia came on; one week ago became apprehensive and suspicious, afraid wife was trying to poison him; said she had the "evil eye." Suffered from insomnia continuously. Never had any hallucinations, convulsions or dizzy spells. For the last day or two has been violent and tried to cut his throat with a razor. Patient died the day of admission, and no further records of the case were taken.

Autopsy four hours post-mortem by E. E. Southard, J. B. Ayer, Jr., and E. T. F. Richards: Superficial wound of neck (suicidal), slight edema of legs, bilateral mul-

tiple pulmonary thromboses with massive infarctions, thrombosis prostatic venous plexus (source of emboli), aortic sclerosis, ulceration in lumbar region, coronary, renal and pulmonary arteriosclerosis, bilateral hypertrophy of heart, chronic pericarditis (slight), chronic pericarditis (slight), chronic mitral, parietal, aortic endocarditis, chronic obliterative pleuritis (left), chronic splenitis and old infarct of spleen, slight chronic nephritis, chronic appendicitis and peri-appendicitis, chronic peripancreatitis, interstitial pancreatitis, chronic localized external adhesive pachymeningitis, chronic tympanitis (right), calvarium thick.

Brain weight, 1,230 gm.

*Cause of death:* Multiple thrombosis, nephritis.

No. 1204. Weigert preparations of the cord show prominent pyramidal degeneration on both sides; also suggest a cell degeneration in the upper cervical region. In the cortex there is slight focal atrophy of nerve cells in the superficial layers in the frontal region; also some decrease of cells in the deeper layers in the frontal and precentral regions. The Betz cells are greatly decreased in number. The neuroglia is increased in the subpial region and is pigmented in the gray matter. There is a slight satellitosis of the superficial cell layers, and a most intense satellitosis of the infra-stellate cell layer, particularly in the frontal and precentral regions, producing marked atrophy of the nerve cells; vessels not changed.

In the autopsy findings, besides the terminal condition, there are advanced arteriosclerosis, chronic endocarditis and pericarditis, appendicitis, nephritis and splenitis, conditions which indicate a septicemia at one time which may have influenced the mental symptoms in its acute stages, but which could hardly be thought of as producing chronic disturbances. The cortical changes are those of a senile brain, but the diagnosis of senile dementia cannot be made owing to the absence of vessel sclerosis and atrophy of convolutions. The decrease in number of Betz cells and pronounced infrastellate gliosis and degeneration of pyramidal tracts cannot be accounted for, because none of the regions of the brain intermediate were preserved. In the protocol it was mentioned that no changes were grossly visible. There is nothing in the cortex, however, to account for the decrease of nerve cells, so it may be inferred that this was secondary to the pyramidal changes. But whether the pyramidal degeneration was due to hemorrhages too slight for gross recognition, or was a primary affair, cannot be decided.

The clinical diagnosis was difficult because of the short time in the hospital, but was a question between senile dementia and arteriosclerotic insanity. From the morphological findings, arteriosclerosis may be excluded, and the absence of vessel changes and atrophy of convolutions excludes senile dementia, if these be proper criteria. The psychosis might be explained by the factor which produced the pyramidal tract degeneration (could this be determined), but with the data at hand, no diagnosis can be made.

CASE X. O. H., Path. No. 1269. Male, age fifty-three years, carpenter. Admitted to Danvers State Hospital July 29, 1908; died Aug. 31, 1908.

Said to be no insanity in the family. Born in Sweden,



came to the United States at twenty years. Married for ten years, no children, worked at trade of carpenter. No serious illnesses, used tobacco and alcohol moderately. Was considered queer for some time. Onset in February, 1908, when he complained of pain in the epigastrium; stayed in bed. One day climbed to the roof of the house and fell off, breaking some ribs. Was sent to the Boston City Hospital, where he remained four weeks. After leaving hospital acted queerly and was threatening. Thought people were talking about him and that police were looking for him; was noisy and excited. Sent to Danvers State Hospital two weeks later.

Knew date and year, but not the month or sort of place he was in. Education poor. Very weak and was put to bed. Complained of pain in epigastric region. Implored physician not to send him away as he had heard that he was to be taken away and killed. Admitted auditory hallucinations. Was quiet most of the time. Memory cloudy.

*Physical examination on admission:* Fairly nourished, well developed, anemic, bronchial breathing over left apex. Heart negative. Arteries hard, sways in Romberg position. Pupils negative. Reflexes normal. Albumin in urine.

*Autopsy twenty-two hours post-mortem by E. T. F. Richards and M. M. Canavan:* Unequal pupils, emaciation, acute peritonitis, enlarged mesenteric and retroperitoneal lymph nodes, injection of mesentery, purulent bronchitis, acute vegetative endocarditis, acute ulcerative colitis (healing), acute diffuse nephritis, chronic fibrous obliterative pleuritis, necrosis perinephric fat (?), chronic fibrous pericarditis, subacute perisplenitis, atrophy of stomach, fat replacement of pancreas, hypertrophy of prostate (slight), inequality of thyroid lobes, chronic leptomenigitis (sulcal), calvarium thin.

Brain weight, 1,530 gm.

*Cause of death:* Dysentery.

1269. The pyramidal cells show marked changes, but since they are not decreased in number, these may be regarded as terminal. All are prominently pigmented. There is a subpial gliosis and all glial cells contain pigment. The intima of the blood vessels is moderately thickened in places. The adventitial nuclei are increased in number and the amount of adventitial pigment is increased. The spinal cord is negative. These changes indicate a senile condition of a low grade.

In the gross autopsy findings, there is nothing outside the terminal condition which can be regarded as conclusive.

Clinically the case was diagnosed as involutional melancholia, and this agrees with the cortex findings, which indicate senile disturbances of some sort.

At autopsy, emaciation and atrophy of stomach were noted, and for some time before the onset of mental symptoms the patient complained of pains in the epigastrium. It may have been that there was some stomach disturbance that produced the cachexia and that this was a factor in producing the mental disturbance.

#### DISCUSSION.

Summarizing these data, it is found that none of these cases fall in the organic group. One (850) is a senile dementia case with rather slight changes in the brain and distinct visceral lesions; one diagnosed as senile paranoia (896) is accepted

as such, since certain slight senile changes were found in the brain and other findings in the body which might indicate a disturbance of nutrition. Two diagnosed as involutional melancholia (969, 1269) were accepted as such because slight senile changes occurred in the brain, and some toxic disturbances were indicated in each. One diagnosed as collapse delirium (976) was accepted as such from the clinical history, although nothing was found in the morphology to confirm the diagnosis. One case left unclassified clinically (1090) was called toxic from the slight brain findings and the marked lesions found in the trunk viscera. One in which the clinical diagnosis was left open between senile dementia and arteriosclerotic insanity (1204) was placed on the unclassified list since both these conditions could safely be excluded, but no definite diagnosis was made because lesions were found in the nervous system which could not be explained from the material at hand. Two cases (1013, 1095) in which dementia precox was diagnosed were accepted as such because certain abnormal conditions in the brain, probably of congenital origin, were found in each case which were taken as possibly indicating hereditary defects. In one case in which dementia precox was clinically preferred (904), this condition was ruled out through absence of abnormal findings in the brain, and manic-depressive insanity given the preference by a process of exclusion.

In these ten cases the diagnoses made from the morphological findings were changed in two cases (976, 1013) after comparison with the clinical histories. One was due to the fact that the psychosis, a delirium, resulted from a functional stomach disorder that could not be recognized post mortem; the other was a case in which the brain of a dementia precox had undergone certain senile changes. Of the clinical diagnoses, on comparing with the morphological, one was corrected (1204), and two left unclassified were in a measure cleared up (1090, 904). Counting the unclassified as errors, there were three errors in the clinical diagnoses of these ten cases (1204, 1090, 904). Of these, one was due to the poor histories and inability to communicate with the patient; one was probably due to the fact that the patient died the same day that he was admitted; and one error was made because enough importance was not assigned to the disturbance of bodily functions as a causative factor in mental disease. Errors are liable to occur, then, in both the morphological and clinical diagnoses of a case, but when the two are compared it is believed that fairly accurate conclusions may be reached.