# CHRONAXIMETRIC STUDIES IN CATATONIA.

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ONE of the greatest problems facing the psychiatrist of to-day is the ætiology of schizophrenia, particularly that of the catatonic variety. Our ignorance in this respect is reflected by the number of hypotheses which have been put forward by the various schools of thought. It would be a task beyond the scope of this paper to attempt to summarize or criticize these theories, but for the sake of clarity a brief survey may here be made. The ætiological hypotheses fall into three main groups:

## 1. HYPOTHESES PREPONDERATINGLY PSYCHOGENIC.

The most extreme form of these is represented by the Freudian school and its disciples. Mechanisms like regression to the prenatal stage, the pretence to be dead, masochism are all quoted. Jung's analytico-symbolistic psychology affords another variety, as does the psychobiological psychiatry of Adolf Meyer, which attributes the schizophrenic reaction type to a progressive maladjustment to the environment.

## 2. HYPOTHESES PREPONDERATINGLY PHYSIOGENIC.

Numerous organic disturbances have been suggested as directly causative. Thus hepatic hypofunction, tuberculosis, encephalitis, endocrine disease, "toxæmia", vascular disorder, etc., all have their adherents. One of the most radical representatives of this group is Kleist and his school. They hold that some of the extra-pyramidal systems are affected in a constant irreversible manner, the type of the disease being dependent on which particular system is affected. Bourguignon, Claude and Maspes, on the other hand, seek to explain the sudden and rapid changes in the clinical picture and laboratory findings by the occurrence of disorders, such as vascular spasms or locally active toxins affecting some of the subcortical centres. Gjessing, after some epoch-making physiological investigations on catatonia, whilst admitting the importance of constitutional and exogenic factors, brings forward weighty evidence in support of a metabolic disturbance, periodic in nature, and has tentatively coined the term "toxic diencephalosis". Workers like Mott and Reiter, who hold that

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schizophrenia in general is a reaction of the central nervous system to abnormal humoral influences must also be classed with this second category.

### 3. Constitutional Hypotheses.

To this group one must assign authors like Kraepelin or Kretschmer, who frankly ascribe the disease to unknown endogenous factors; likewise the numerous psychiatrists who place the greater part of the onus on heredity.

The present work was contemplated in the hope of contributing something towards this discussion. It has been shown that the chronaxie measurement of the peripheral nervous system is a valuable means of showing changes of the central nervous system which escape other quantitative investigations which are clinically applicable. Thus in Parkinsonism the usual electrical examination according to Dubois Reymond with galvanic and faradic current shows no changes, and yet the organic lesion of the extrapyramidal system brings about a change of the excitability of the peripheral nervous system which is detectable with the chronaxie apparatus. These changes seem fairly constant, as is shown by Bourguignon and others. Normally there is a relationship of 1 to 2 in the chronaxie of agonists and antagonists. In Parkinsonism, on the other hand, this quotient approaches or reaches that of 1 to 1. As the pathogenesis of catatonia, particularly that of the rigid type, has frequently been compared with that of chronic encephalitis, the idea occurred to us of examining a group of catatonics with a view to finding whether comparable changes could be detected.

### Method.

A number of well-marked stuporose catatonics were selected and chronaxie measurements were performed on muscles of the upper and lower limbs. The muscles were so chosen that several fell into each of the three groups proposed by Bourguignon and his pupils.

The method employed was that of Bourguignon, in whose laboratory one of us had the opportunity of assisting in a number of experiments. A chronaximetric apparatus on the condenser principle was constructed at the Cardiff Mental Hospital. The capacity of each of the condensers was kindly tested by Dr. Shaxby, of the Physiology Institute at University College, Cardiff. The circuit and electrodes employed were exactly similar to those recommended by Bourguignon.

Of the readings obtained, only those were used where the value of the rheobase after the determination of the chronaxie was the same (within a 10% experimental error) as before. The great majority of muscles were tested several times, and almost without exception on both sides. In some instances the experiments were repeated on different days, sometimes after a lapse of several weeks.

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## Summaries of Case-Histories.

The following summaries are from 7 cases of well-established catatonic stupor investigated at the Cardiff City Mental Hospital.

Case 1.—Male, æt. 37. Father alcoholic, mother suffered from vascular cerebropathy; one aunt epileptic, two aunts had been in mental hospitals; one brother suffered from dementia præcox and died in Cardiff City Mental Hospital. The patient had previously been in hospital in 1914 for nine months suffering from stupor. He had also been in various mental hospitals in Australia for several years. He was admitted to Cardiff Mental Hospital in 1933. Since admission he had been in a state of profound stupor, was mute, resistive, negativistic, and at times displayed impulsive behaviour with short phases of restlessness. He sat about the ward in fixed attitudes and showed the typical appearance of catatonic stupor. Physically he showed no organic disease.

Case 2.—Male, æt. 28. No history of morbid heredity. Morbid pre-psychotic personality, always quiet and sensitive. Admitted to hospital in September, 1932. Gradually became more solitary, self-absorbed, irritable and detached from reality, eventually merging into a condition of complete stupor. Occasionally displayed catalepsy and "flexibilitas cerea". He rarely spoke, and when he did he was usually echolalic. Physically no organic disease.

Case 3.—Female, æt. 20. Family history, both parents "nervous". Admitted to hospital in September, 1933. Pre-psychotic schizoid personality; from age of 12½ history of occasional outbursts of violence. For four years prior to admission had to be fed. She displayed no spontaneity, and could do nothing for herself. She remained solitary, uninterested and stuporose. Two months later her stupor and negativism became more marked, necessitating tube-feeding daily. She would stand about the ward without moving and exhibited grimacing. Physically no organic disease.

Case 4.—Female, æt. 21. No evidence of psychotic heredity. Admitted July, 1933, after illness of two months, during which she became apathetic and negativistic. Most of the time she was in a profound stuporose condition and offered no response to questions, though on a few occasions she displayed excitement and restlessness. Manneristic behaviour at times marked. During chronaximetric investigations she was in a condition of profound stupor and displayed no mannerisms. Physically no evidence of organic disease except amenorrhœa.

Case 5.—Male, æt. 34. Previous history unknown. Admitted January, 1929. Was then in a semi-stuporose condition, answering questions slowly and in monosyllables. Subsequently became entirely mute and refused to answer questions. He displayed catatonic rigidity, e.g., lay in bed with his head raised from the pillow, frequently negativistic, exhibited "flexibilitas cerea", at times impulsive and violent. Physically no evidence of organic disease except acrocyanosis and œdema of the ankles.

Case 6.—Female, æt. 27. No history of morbid heredity. Pre-psychotic schizoid personality. Admitted to hospital in September, 1929, with history of hallucinations and ideas of reference. She gradually merged into a condition of stupor with stereotypy. In May, 1931, she was discharged "not improved", and re-admitted in August of same year; had gradually deteriorated since then, and during examination was in a state of stupor. Physically no evidence of organic disease except amenorrhœa.

Case 7.—Female, æt. 50. No morbid heredity ascertainable. Pre-psychotic personality; strong-willed, solitary, "nervous". Admitted in November, 1933, with history of three years' mental illness. Gradual deterioration set in with apathy, hypochondriacal ideas and compulsive actions. Had been mute since the autumn of 1932. At the time of examination was completely stuporose and extremely negativistic; no catalepsy. Reacted to painful stimuli by withdrawal;



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TABLE I.—*Showing Chronaxie Values expressed in Microfarads in 7 Cases of Catatonia.*

(R = right; L = left.)

| Muscle. | Normal. | Case No. 1. | 2. | 3. | 4. | 5. | 6. | 7. |
|---|---|---|---|---|---|---|---|---|
| 1. Deltoid (anterior fibres) | ·015–·035 | R ·051 | R ·073 | R ·049 | R ·049 | R ·033 | R ·022 | R ·027 |
| | .. | ·033 | .. | L ·022 | ·051 | ·033 | ·022 | ·022 |
| | .. | ·022 | .. | .. | ·051 | L ·049 | L ·033 | L ·022 |
| | .. | .. | .. | .. | L ·022 | ·049 | ·027 | ·022 |
| | .. | .. | .. | .. | ·033 | .. | .. | .. |
| 2. Biceps brachii | ·015–·035 | R ·022 | R ·022 | R ·027 | R ·051 | R ·022 | R ·022 | R ·049 |
| | .. | L ·022 | ·022 | ·011 | ·062 | ·022 | ·027 | ·051 |
| | .. | .. | ·033 | L ·049 | ·089 | L ·022 | L ·033 | ·022 |
| | .. | .. | L ·011 | ·022 | ·049 | ·022 | ·033 | L ·022 |
| | .. | .. | ·022 | .. | L ·022 | .. | .. | ·022 |
| | .. | .. | ·022 | .. | ·049 | .. | .. | ·022 |
| | .. | .. | ·022 | .. | .. | .. | .. | .. |
| | .. | .. | ·077 | .. | .. | .. | .. | .. |
| 3. Triceps (lateral head): (*a*) Upper point | ·04–·085 | R ·084 | R ·084 | R ·262 | R ·049 | R ·049 | R ·049 | R ·049 |
| | .. | ·027 | L ·184 | L ? | ·051 | ·049 | ·100 | ·033 |
| | .. | L ·027 | ·222 | .. | ·033 | ·033 | ·062 | L ·022 |
| | .. | .. | .. | .. | ·051 | L ·022 | ·077 | ·022 |
| | .. | .. | .. | .. | L ·049 | ·033 | L ·022 | .. |
| | .. | .. | .. | .. | ·111 | .. | ·022 | .. |
| (*b*) Lower point | ·015–·035 | R ·022 | R ·033 | R ·022 | R ·062 | R ·073 | R ·111 | R ·022 |
| | .. | ·027 | ·033 | ·033 | .. | ·033 | ·122 | ·049 |
| | .. | L ·073 | ·051 | L ·027 | L ·062 | .. | .. | .. |
| | .. | ·027 | ·049 | ·033 | ·033 | L ·022 | L ·033 | L ·011 |
| | .. | .. | L ·062 | .. | .. | ·022 | ·038 | ·011 |
| | .. | .. | ·049 | .. | .. | .. | .. | .. |
| | .. | .. | ·033 | .. | .. | .. | .. | .. |
| | .. | .. | ·033 | .. | .. | .. | .. | .. |
| | .. | .. | ·049 | .. | .. | .. | .. | .. |
| | .. | .. | ·022 | .. | .. | .. | .. | .. |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 4. Extensor digitorum communis: | | | | | | | | | | | | | | | | | | |
| (*a*) Upper point | . . | ·04–·085 | . | R ·150 | . | R ·100 | . | R ·078 | . | R ·200 | . | R ·084 | . | R ·200 | . | R ·173 | | |
| | | .. | . | .. | . | ·100 | . | ·062 | . | ·322 | . | ·138 | . | ·211 | . | ·173 | | |
| | | .. | . | L ·160 | . | .. | . | .. | . | ·333 | . | .. | . | ·084 | . | .. | | |
| | | .. | . | .. | . | L ·051 | . | L ·111 | . | ·355 | . | L ·211 | . | ·122 | . | L ·111 | | |
| | | .. | . | .. | . | ·073 | . | ·222 | . | L ·260 | . | ·184 | . | .. | . | ·144 | | |
| | | .. | . | .. | . | .. | . | ·062 | . | ·200 | . | .. | . | L ·260 | . | .. | | |
| | | .. | . | .. | . | .. | . | .. | . | ·327 | . | .. | . | ·184 | . | .. | | |
| | | .. | . | .. | . | .. | . | .. | . | ·244 | . | .. | . | .. | . | .. | | |
| | | .. | . | .. | . | .. | . | .. | . | ·543 | . | .. | . | .. | . | .. | | |
| (*b*) Lower point | . . | ·015–·035 | . | R ·049 | . | R ·295 | . | R ·173 | . | R ·295 | . | R ·111 | . | R ·100 | . | R ·260 | | |
| | | .. | . | ·038 | . | ·311 | . | ·100 | . | ·406 | . | ·084 | . | ·133 | . | ·260 | | |
| | | .. | . | ·049 | . | .. | . | .. | . | .. | . | .. | . | .. | . | .. | | |
| | | .. | . | L ·049 | . | L ·149 | . | L ·149 | . | L ·311 | . | L ·084 | . | L ·276 | . | L ·151 | | |
| | | .. | . | ·084 | . | ·211 | . | .. | . | ·244 | . | ·100 | . | ·238 | . | ·173 | | |
| | | .. | . | .. | . | .. | . | .. | . | ·344 | . | .. | . | .. | . | .. | | |
| 5. Flexor digitorum sublimis | . | ·04–·085 | . | R ·027 | . | R ·049 | . | R ·051 | . | R ·127 | . | R ·027 | . | R ·062 | . | R ·033 | | |
| | | .. | . | ·022 | . | ·100 | . | ·128 | . | ·061 | . | ·027 | . | ·049 | . | ·033 | | |
| | | .. | . | .. | . | .. | . | .. | . | ·111 | . | ·022 | . | .. | . | .. | | |
| | | .. | . | L ·038 | . | L ·011 | . | L ·049 | . | .. | . | .. | . | L ·078 | . | L ·162 | | |
| | | .. | . | ·027 | . | ·033 | . | ·073 | . | L ·051 | . | L ·022 | . | ·078 | . | ·111 | | |
| | | .. | . | .. | . | ·051 | . | .. | . | ·111 | . | ·022 | . | .. | . | ·049 | | |
| | | .. | . | .. | . | ·322 | . | .. | . | ·100 | . | .. | . | .. | . | .. | | |
| | | .. | . | .. | . | ·084 | . | .. | . | .. | . | .. | . | .. | . | .. | | |
| | | .. | . | .. | . | ·238 | . | .. | . | .. | . | .. | . | .. | . | .. | | |
| | | .. | . | .. | . | ·038 | . | .. | . | .. | . | .. | . | .. | . | .. | | |
| 6. Vastus medialis | . . . | ·015–·035 | . | R ·049 | . | R ·023 | . | R ·022 | . | R ·062 | . | R ·038 | . | R ·049 | . | R ·049 | | |
| | | .. | . | .. | . | ·033 | . | ·038 | . | ·078 | . | ·038 | . | ·051 | . | ·049 | | |
| | | .. | . | L ·049 | . | .. | . | .. | . | ·100 | . | .. | . | .. | . | .. | | |
| | | .. | . | .. | . | L ·051 | . | L ·049 | . | ·100 | . | L ·049 | . | L ·051 | . | L ·027 | | |
| | | .. | . | .. | . | ·049 | . | ·049 | . | .. | . | ·084 | . | ·062 | . | ·038 | | |
| | | .. | . | .. | . | ·049 | . | .. | . | L ·038 | . | ·062 | . | .. | . | .. | | |
| | | .. | . | .. | . | ·062 | . | .. | . | ·033 | . | ·049 | . | .. | . | .. | | |
| | | .. | . | .. | . | .. | . | .. | . | ·050 | . | .. | . | .. | . | .. | | |

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TABLE I.—*Showing Chronaxie Values expressed in Microfarads in 7 Cases of Catatonia*—continued.

| Muscle. | Normal. | Case No. 1. | 2. | 3. | 4. | 5. | 6. | 7. |
|---|---|---|---|---|---|---|---|---|
| 7. Vastus lateralis | ·015–·035 | R ·051 | R ·100 | R ·049 | R ·422 | R ·058 | R ·073 | R ·049 |
| | .. | .. | .. | ·111 | ·634 | ·062 | ·049 | ·038 |
| | .. | L ·051 | L ·100 | ·051 | ·049 | .. | ·073 | .. |
| | .. | .. | ·133 | .. | ·062 | L ·049 | .. | L ·073 |
| | .. | .. | ·100 | L ·051 | ·084 | ·049 | L ·049 | ·049 |
| | .. | .. | ·262 | ·151 | .. | .. | ·033 | ·111 |
| | .. | .. | .. | ·138 | L ·149 | .. | .. | .. |
| | .. | .. | .. | ·122 | ·122 | .. | .. | .. |
| | .. | .. | .. | ·111 | ·178 | .. | .. | .. |
| | .. | .. | .. | ·062 | .. | .. | .. | .. |
| | .. | .. | .. | ·084 | .. | .. | .. | .. |
| | .. | .. | .. | ·062 | .. | .. | .. | .. |
| 8. Gastrocnemius: | | | | | | | | |
| (*a*) Medial head | ·100–·175 | R ·045 | R ·233 | R ·130 | R ·111 | R ·073 | R ·289 | .. |
| | .. | 1·125 | .. | ·150 | ·160 | ·078 | ·344 | .. |
| | .. | 1·447 | L ? | ·189 | ·151 | .. | ·373 | .. |
| | .. | .. | .. | .. | .. | L ·078 | .. | .. |
| | .. | L ·029 | .. | L ·051 | L ·149 | ·149 | L ·122 | .. |
| | .. | ·144 | .. | ·100 | ·133 | ·084 | ·144 | .. |
| (*b*) Lateral head | ·100–·175 | R ·073 | R ·260 | R ·049 | R ·162 | R ? | R ·049 | R ·084 |
| | .. | .. | ·211 | ·049 | ·111 | .. | ·049 | ·073 |
| | .. | L ·049 | .. | ·051 | .. | L ·078 | .. | .. |
| | .. | ·062 | L ·151 | ·222 | L ·195 | ·084 | L ·211 | L ·062 |
| | .. | .. | .. | ·151 | ·149 | ·062 | ·084 | .. |
| | .. | .. | .. | .. | .. | ·052 | ·073 | .. |
| | .. | .. | .. | L ·073 | .. | .. | .. | .. |
| | .. | .. | .. | ·100 | .. | .. | .. | .. |
| 9. Peroneus longus | ·04–·085 | R ·049 | R ·162 | R ·084 | R ·222 | R ·062 | R ·311 | R ·100 |
| | .. | ·078 | ·100 | ·051 | ·149 | ·078 | ·322 | ·084 |
| | .. | .. | ·133 | .. | ·162 | .. | .. | .. |
| | .. | L ·100 | .. | L ·100 | .. | L ·184 | L ? | L ·062 |
| | .. | ·049 | L ·151 | ·062 | L ·149 | ·184 | .. | ·062 |
| | .. | .. | ·162 | ·100 | ·127 | ·200 | .. | ·149 |
| | .. | .. | ·049 | ·062 | .. | .. | .. | .. |
| | .. | .. | ·062 | .. | .. | .. | .. | .. |

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TABLE II.—*Showing Mean Chronaxie Values expressed in Microfarads.*

| Muscle. | Normal. | Case No. 1. | 2. | 3. | 4. | 5. | 6. | 7. |
|---|---|---|---|---|---|---|---|---|
| 1. Deltoid (anterior fibres) | ·015–·035 | ·035 | ·073 | ·036 | ·041 | ·041 | ·026 | ·023 |
| 2. Biceps brachii | ·015–·035 | ·022 | ·022 | ·028 | ·054 | ·022 | ·029 | ·032 |
| 3. Triceps (external head): | | | | | | | | |
| (*a*) Upper point | ·04–·085 | ·046 | ·163 | ·262 | ·057 | ·038 | ·055 | ·032 |
| (*b*) Lower point | ·015–·035 | ·037 | ·042 | ·029 | ·052 | ·038 | ·079 | ·023 |
| 4. Extensor digitorum communis: | | | | | | | | |
| (*a*) Upper point | ·04–·085 | ·155 | ·081 | ·107 | ·308 | ·154 | ·177 | ·150 |
| (*b*) Lower point | ·015–·035 | ·054 | ·241 | ·141 | ·333 | ·095 | ·187 | ·210 |
| 5. Flexor digitorum sublimis | ·04–·085 | ·029 | ·103 | ·075 | ·093 | ·024 | ·066 | ·078 |
| 6. Vastus medialis | ·015–·035 | ·049 | ·045 | ·040 | ·066 | ·053 | ·053 | ·041 |
| 7. Vastus lateralis | ·015–·035 | ·051 | ·139 | ·089 | ·212 | ·055 | ·056 | ·065 |
| 8. Gastrocnemius: | | | | | | | | |
| (*a*) Medial head | ·1–·175 | ·718 | ·233 | ·124 | ·140 | ·092 | ·254 | .. |
| (*b*) Lateral head | ·1–·175 | ·061 | ·207 | ·099 | ·154 | ·069 | ·093 | ·073 |
| 9. Peroneus longus | ·04–·085 | ·069 | ·115 | ·076 | ·162 | ·141 | ·316 | ·091 |

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afraid of chronaxie apparatus. Physically some degree of arterio-sclerosis; menorrhagia.

## Results.

The following two tables illustrate the results obtained. Table I records every measurement taken on the various muscles. Table II shows the average of these measurements of the individual muscle. As is usual, the results are recorded in microfarads (mF). To obtain corresponding units in time, each figure must be multiplied by 4 to give the chronaxie value in $\sigma$ (1/1000th of a second).

## Interpretation.

The striking feature of the above results is the fact that abnormal figures were found in every case. Even if one does not adhere strictly to the limits laid down by Bourguignon, and only takes as definitely pathological those values which are less than half Bourguignon's lowest or more than twice his highest limits, there was no muscle of those examined which did not at one time or other show abnormality. This leads us to another important finding, viz., the enormous variability. In normal persons the chronaxie only varies comparatively slightly, but in the catatonics examined chronaxie actually varied in the same muscle. Actual times when variations occurred are not shown in our results, as the recording of the times of examination of each muscle would have made the tables far too cumbersome. It was, however, found that in some instances variations occurred almost from one minute to another, in others the chronaxie remained constant over a period of weeks.

Even allowing for experimental error, there can be no doubt that the figures demonstrating variability and those which are more than double the upper normal limit or less than half the lower normal limit are definitely abnormal. It will be seen from the tables that the majority of abnormal figures were variations upwards rather than downwards. Hence we find a marked tendency for the chronaxie to be lengthened, this lengthening not being constant, but variable. These variations at first sight seem to defy any attempt at interpretation. One fact is obvious: they are decidedly different from the changes met with in Parkinsonism, for it appears impossible to find any determined relationship between agonists and antagonists. Neither could any correlation between the clinical condition of the patient and the chronaxie findings be established. In some of the cases the rigidity increased or decreased, but no relationship could be discovered between these changes and the variations in chronaxie.

## Discussion.

The interpretation of these abnormal chronaxie values is extremely difficult. It seems to us that several points are worthy of discussion.

If it is assumed that an organic constant change in some parts of the central nervous system can be regarded as the cause of catatonia, constant changes of

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chronaxie could of course be explained. Many authors (Kleist, Fränkel, Salmon, Bychowski, Kirschbaum, Küppers, Steck, Lindberg, etc.) have produced evidence to support the theory that anatomical lesions of one or other part of the extrapyramidal system produce the catatonic syndrome. Our findings are so extremely variable, showing as they do so little constancy in the individual case and varying so much from one case to another, that one cannot conclude that the chronaxie findings support the view mentioned above. On the other hand we do not assert that our results disprove it. Further corroborating evidence is required in this connection.

Bourguignon and his pupils have expressed the view that the changes in chronaxie which he found, and which correspond more or less to ours, can be explained by assuming a functional lesion of certain centres. This lesion might be toxic or vascular in origin. It is assumed that some toxins circulating in the blood act over shorter or longer periods on the centres, or that the blood-supply to the latter suffers through arterial spasms, temporary stasis or similar occurrences. As far as we know, however, there is no evidence to support this view seriously, and all that can be said about it is that our chronaxie findings seem compatible with it.

The third explanation, which seemed a possible one to us, is the assumption that the changes of chronaxie are due to the maintenance of certain muscular contractions (increased tone) over long periods. Previous investigations of chronaxie during voluntary muscular contraction have shown no abnormal features, but it must be remembered that such voluntary contractions were necessarily of comparatively short duration, though work and fatigue seemed to change the chronaxie for short periods. When one bears in mind, however, that catatonics frequently maintain the same muscles in a state of contraction (increased tone) over long periods, far longer than normal persons in an experiment, it seems reasonable to suppose that the changes found are secondary to these prolonged contractions or increase of muscular tone. The view is held that the autonomic nervous system is involved in the maintenance of muscular tone. In this connection it might be worth mentioning as a possibility that an increased or changing sympathetic tone can be responsible for changes in chronaxie. It has been shown experimentally that chronaxie can change with alteration of the balance between the cerebro-spinal and the sympathetic systems, although these changes did not reach the degree recorded in our cases.

It would be of interest, in view of the above findings, to ascertain if changes in chronaxie occur in other states where attitudes are maintained over prolonged periods (e.g., melancholia), and we hope to be able to undertake this work at some future date.

## Summary.

The motor chronaxie has been investigated in 7 well-marked cases of catatonia. Marked deviation from the normal was found in every case, these

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changes being variable. Although the results seem compatible with the various ætiological theories of catatonia, a simpler interpretation would be to regard them as secondary changes of excitability due to the prolonged maintenance of catatonic attitudes.

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