Neurology. — Experimental catatonia, produced by auto-intoxication. I.
Experimental catatonia after artificial obstruction of the lumen of
the intestine. By H. DE JONG, D. J. Kok, A. GEESINK and F. J.
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During previous investigations of experimental catatonia in our
laboratory, it became evident, that this syndrome could be produced by
various biological products in different experimental animals 1).

In the following communication we shall devote ourselves to the
question of the effect on the course of experimental catatonia, produced
by removal or functional disturbance of certain organs through disturbance
of metabolism. That such an effect is possible, can be readily imagined
since in this manner, substances are put into circulation, which may have
a toxic effect on the central nervous system. This possiblility is not unlikely,
since it has previously been proved by one of us (D. J.) 1), that experimental
catatonia may be produced by various means in the central nervous system.

If experimental catatonia can be produced in animals by auto-
intoxication, this would support the theory of auto-intoxication in human
catatonia, a theory often mentioned by important authors, such as
KRAEPELIN 2), JELGERSMA 3), SÉRIEUX 4).

In experimental animals we found that generalized catatonia did not
appear after extirpation of: spleen, kidneys, testicles or combinations of
these organs. Injection of extractions of these organs in mice, did not
produce catatonic phenomena.

In contrast we were able to produce experimental catatonia by closure
of the intestine, in 4 of the 12 operated dogs. Artificiel ileus with closure
in 2 places gave these results, as it is shown in the following protocols.

Two illustrative protocols are given here. These experiments are being
undertaken as part of a larger series, in which there will be an extirpation
or functional disturbance of other internal organs.

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"Die experimentelle Katatonie als vielfach vorkommende Reaktionsform des Zentral-
"Annales médico-psychologiques". Nr 2, février 1933.
2) KRAEPELIN: "Lehrbuch der Psychiatrie".
3) JELGERSMA: "Leerboek der Psychiatrie". Leiden.
4) SÉRIEUX: "La démence précoce". Revue de Psychiatrie, juin, 1902.
1. Pronounced catatonia is shown in the following protocol:

Protocol of March 26th, 1934.
The animal is clinically healthy.
Purpose of the operation: artificiel ileus.
Preparation: 24 hours before, castor oil (15 cc). ¾ hours before:
0.25 mg atropine sulf. and 60 mg morphini hydrochl., subcutaneously.
Shaving of the abdomen. Picric acid disinfectant.
Narcosis: chloroform and ether 2:3.
Duration: 15 minutes.
Used: 10 cc of the narcosis-mixture.

Technique:
8.30 A.M. Operation, laparatomy, isolation of an intestinal loop (duo-
denum) by closure at each end. Replacement and closure in
2 stages.

Observation:
9.30 A.M. Pulse 70. Respiration 14. The animal tried to stand.
10.20 Crossing of the forepaws in the lying position was immediate-
11.50 Walked normally, except that movements of the hindlegs
seemed occasionally painful (due to the operative wound).
2. P.M. The animal could be placed in half-sitting, half-lying position
with both forepaws against the rung of a chair. The animal
would let itself be placed on one side on the ground with the
4 paws extended in the air. It would not remain actually on
its back, but immediately turned itself, when free. Crossing
of the 4 paws was immediately corrected.
4. The animal let itself be placed on its back and remained in
this position, with 4 paws extended.
5.50 When the left forepaw was placed on the rung of a chair, it
remained in this position. A forepaw placed behind the neck
was maintained in this position for a long time. Crossing of
the forepaws with the animal in sitting position, became fixed.
There was no evidence of paresis in the extremities; on
whistling, etc, the animal walked well spontaneously, only
somewhat slowly and holding each position several minutes.
9.30 The motility was entirely normal; the animal walked about,
whined somewhat, jumped towards the investigator. There
was no evidence of diminished voluntary motive power. Also
there was no maintaining of the crossed position of the 2
paws or of the position with the paw behind the neck.
10. Pulse 92. normal type.
27. III. 34:
9.30 A.M. The animal was very lively, jumped towards the investigator,
maintained this activity through the whole day.
3.30 P.M. The dog produced a small greenish stool; he ate some rice
and milk hungrily. Motility of the animal was normal. On
March 28th, the animal was found dead in its cage.

Pathological report:
Autopsy: 29. III. 34. 4.15 P.M. (Dr. Pompe).
Rigor was present.
There was some serosanguinous exudate between the skin and the abdominal wall. The abdominal organs were exposed. A localized peritonitis about 1 cm in diameter was found beneath the right kidney. The ligature which closed off the intestinal loop from the duodenum, was intact, the second ligature, 8 cm lower on the intestine had caused perforation.

In the upper part 4 sacculations, in the lower part 5, of which 2 in the colon.

_Cause of death:_ acute peritonitis.

**Conclusion:**

On March 26th, from 5.50 P.M. until 9.30 P.M., the animal showed a _pronounced catalepsy_. Before and after the cataleptic period the motility was entirely normal. The animal died at a time, when the cataleptic condition was not present.

II. _Protocol of August 8th, 1934._

**Technique:**

11. P.M. Midline incision. Exposure of the intestine (ileum), which had a previous operation. This showed only as a narrowing of the loop without obstruction. Three additional ligatures were placed in the intestine.
   1. one single ligature.
   2. one triple ligature.
   3. one double ligature.
   Closure of the peritoneum, muscles and skin.

**Observation:**

2.25 P.M. The animal lay on its bench and whined; it could not stand.
4. With assistance it could stand on its legs. Motility normal.
9. VIII. 34:
9.55 A.M. Crossing of the forepaws was maintained for a very short time, but repetition of the crossing was immediately corrected.
10. VIII. 34:
10. A.M. Crossing of the forepaws was immediately corrected. The dog moved slightly less than a normal animal.
4. P.M. _Crossing of the forelegs was maintained and not corrected. The animal was in addition much slower in its movements._
11. VIII. 34:
4. P.M. _The dog was still cataleptic; crossed position was not corrected. Extending of one paw was maintained._
13. VIII. 34:
10. A.M. _The dog was cataleptic and negativistic, the motility was much slower._

Death after 6 days.

**Pathological report:**

_Section:_ slightly distended intestine, no peritonitis.

**Conclusion:**

Here also very marked cataleptic phenomena were present during a period of 4 days, followed after one day by death.
Conclusion:

Clinical observations suggest the factor of auto-intoxication in catatonia. Therefore a series of experiments have been begun in our laboratory concerning the possibility of the production of catatonia through auto-intoxication in experimental animals.

Some of these experiments with ligatures of the intestine produced pronounced positive results.

We may remember, that in human pathology the hypothesis of entero-genesis in dementia praecox, has been proposed (f.e. by BUSCAINO)\(^1\).

Since experimental catatonia can be produced in many ways, it may not be concluded from the above, that human catatonia results, in every instance from intestinal-intoxication. Therefore, the other biological possibilities of experimental catatonia must be investigated. Such experiments are now in the process of investigation.


Medicine. — On the seasonal longevity of Anopheles maculipennis in Holland with reference to their ability to act as malarial vectors. By A. DE BUCK and N. H. SWELLENGREBEL. (From SWELLENGREBEL’s Laboratory in the Institute of Tropical Hygiene [Director Prof. Dr. W. A. P. SCHÜFFNER] of the Royal Colonial Institute at Amsterdam). (Communicated by Prof. W. A. P. SCHÜFFNER).

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1. Introduction.

JAMES, NICOL and SHUTE\(^1\) have shown that the rate of survival in batches of infected anopheles (i.e. the percentage still alive on the day sporozoites appear in the salivary glands) varies with the month in which they have been infected. The rate of survival is high (about 50\%) from August till November, dropping to about 30\% in December and January, still lower (about 20\%) in February and March, to attain the lowest level (less than 10\%) in April and May. In June and July it starts rising again but not above the level reached in February and March. According to these data the year can be divided into two seasons, one is favourable to malarialtransmission: August to November, the other is not: December