How can one account for obsessive preoccupation with ideas that provoke anxiety (one would expect avoidance of them) and other aspects of this neurosis purely on the basis of Wolpe’s variety of learning theory? Are the cortical symptoms in neurones functional or physical, analogous to ‘concussion’, often of reversible nature, as a result of drug administration or psychotherapy involving alteration of neural circuits and subcortical processes? I have attempted to point out that the medical practitioner—not merely the psychiatrist, but all colleagues, including the paediatrician and obstetrician—should become more, rather than less, intensely interested in observation of emotional factors in his patients; not merely from the point of view of humanitarianism but as a scientist. The psychologist can help us experimentally, but the man in the clinical field should be in the front line. It will yet be an art to use this science skilfully.

SUMMARY

The use of psychotherapy as a scientific procedure in the treatment of the psychoneuroses and psychosomatic disorders is described. The history of the development of the scientific study of disease is traced. The progress from belief and intuition towards scientific theory tempered by scepticism and the demand for evidence is described. The formulation of various analytic and other psychological theories by clinicians as a result of clinical experience is stressed. The apparent similarity of results claimed by various schools of thought necessitates further study. The behaviourist (conditioning) school have cast doubt on the clinical method, considering more readily testable autonomic and muscular reactions in the psychological laboratory the sole method of studying psychotherapy scientifically, and consider the use of statistics essential. The value of animal experimentation is discussed. Laboratory experiments in this field, involving the production of experimental neurones in man, are described. A combination of the clinical method practised by the doctor and the experiments in the psychological laboratory is considered desirable, the psychologist assisting the clinician as the biochemist/physiologist assists the clinician in physical medicine.

The deficiencies in conditioning theory as sole explanation for complex human neurones are discussed. The final section of the paper deals with the factors which the theories have in common and attempts to stress the import of a scientific attitude towards psychological factors by the medical practitioner.

REFERENCES


PROBABLE FACTOR XI DEFICIENCY IN BANTU SUBJECTS*

M. G. A. FORREST, M.A., M.B., B.CH., M.C. PATH. AND A. C. B. WICKS, M.B., B.CH., Harari Central Hospital, Salisbury, Rhodesia

During the 4-year period June 1963 - May 1967, 108 patients were referred to the clinical laboratory for investigation of suspected haemorrhagic disorders. In 60 patients no abnormality was found; 23 were suffering from haemophilia (factor VIII deficiency), 1 from Christmas disease (factor IX deficiency) and 4 from thrombocytopenia. In 3 the bleeding was secondary to uraemia, and 11 cases were not sufficiently investigated for a firm diagnosis to be made. The remaining 9 patients, belonging to 2 families, appear to be examples of contact factor (factors XI and XII) deficiencies. Factors XI and XII are involved in an early stage of thromboplastin generation. Factor XII is activated by contact with a foreign surface and then reacts with factor XI, yielding a product which takes part in the next phase of this complex process. It seems probable that factor XI rather than factor XII is at fault, since factor XII deficiency is not usually associated with abnormal bleeding. The majority of published cases of factor XI deficiency have been in persons of Jewish descent, and, as far as we are aware, the disorder has not been reported in Bantu subjects.

CASE HISTORIES

Case 1. Bantu male, aged about 2 years, first admitted in May 1963 suffering from uncontrolled bleeding following a fall on the head. There have been 6 subsequent admissions due to haemorrhage caused by minor injuries, including one episode of intracranial bleeding.

Case 2. Bantu male aged about 12 years; first cousin to cases 1 and 4. He was originally admitted in March

*Date received: 30 October 1967.
1965 with haematemesis and melaena and gave a past history of bleeding into joints. There have been 5 subsequent admissions due to haemorrhagic episodes, in which haemarthroses have been conspicuous.

**Case 3.** Bantu male aged about 10 years; apparently not related to the other patients described. He was admitted in August 1965 with a haematoma of the right buttock, and had noticed that he bled more than other children from minor injuries. This patient has not been seen again.

**Case 4.** Bantu male infant, younger brother of case 1. He was first admitted in June 1966 with signs of meningitis and a right hemiparesis. Lumbar puncture yielded purulent cerebrospinal fluid which was sterile on bacteriological culture. There was persistent bleeding from the lumbar-puncture site and transfusion was required. The meningitis responded to treatment and the child was discharged. He was readmitted in coma 6 weeks later. The cerebrospinal fluid was now uniformly blood-stained and xanthochromic, with a protein content of 2,100 mg./100 ml. A provisional diagnosis of cerebral haemorrhage was made and death occurred a few hours after admission. No postmortem examination was performed.

This patient had a twin brother who was admitted in April 1967 with excessive bleeding after biting his tongue. No coagulation studies have as yet been carried out on this child and he is not further considered in this paper.

**LABORATORY INVESTIGATIONS**

Relevant laboratory findings are summarized in Tables I and II. It is clear, both from the clinical histories and from their prolonged whole-blood coagulation times, that these patients have a significant abnormality of the coagulation mechanism. The fact that the one-stage prothrombin time is normal, whereas the prothrombin consumption test is abnormal, indicates that the defect is to be found early in the coagulation process, i.e. in the stage of thromboplastin generation. If haemophilia (factor VIII deficiency) were the diagnosis, the thromboplastin generation test would yield an abnormal result when absorbed plasma from the patient was used in the generation mixture. If these were cases of Christmas disease (factor IX deficiency), thromboplastin generation would be abnormal when the patient's serum was used in the test.

Reference to Table II shows that in case 1 the thromboplastin generation test was completely normal. In case 2 an abnormal result was obtained on one occasion when normal absorbed plasma was used together with serum from the patient (he was initially thought to have Christmas disease), but subsequently no defect could be detected.

### Table 1. Results of Coagulation Studies*

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole-blood coagulation time</td>
<td>21 min.</td>
<td>&gt;30 min.</td>
<td>29 min.</td>
<td>22-5 min.</td>
<td>Method of Lee &amp; White. Normal range in this laboratory 5-9 min.</td>
</tr>
<tr>
<td>One-stage prothrombin time</td>
<td>18 sec. (control 17 sec.)</td>
<td>13-5 sec. (control 12 sec.)</td>
<td>15 sec. (control 13 sec.)</td>
<td>14 sec. (control 13 sec.)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>14 sec. (control 13 sec.)</td>
<td>16 sec. (control 13 sec.)</td>
<td>13 sec. (control 13 sec.)</td>
<td>13 sec. (control 13 sec.)</td>
<td></td>
</tr>
<tr>
<td>Prothrombin consumption test</td>
<td>15 sec.</td>
<td>Abnormal (substrate clotting time not recorded)</td>
<td>17 sec.</td>
<td>16 sec.</td>
<td>Warner-Chilcott reagents used according to manufacturer's instructions. Normal plasma gives a substrate clotting time greater than 25 sec.</td>
</tr>
<tr>
<td>(substrate clotting times are given)</td>
<td>15 sec.</td>
<td></td>
<td>15 sec.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelet count</td>
<td>560,000</td>
<td>360,000</td>
<td>195,000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Where more than 1 entry appear, the test was performed on more than one occasion.

### Table II. Results of the Thromboplastin Generation Test*

<table>
<thead>
<tr>
<th>Case</th>
<th>Source of absorbed plasma</th>
<th>Source of serum</th>
<th>Result of test</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Patient</td>
<td>Normal</td>
<td>Normal</td>
<td>Identical results were obtained on a second occasion</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>Patient</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Patient</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Patient</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>Patient</td>
<td>Abnormal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Patient</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Patient</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Patient</td>
<td>Normal</td>
<td>Abnormal</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Patient</td>
<td>Normal</td>
<td>Normal</td>
<td>Identical results were obtained on a second occasion</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>Patient</td>
<td>Abnormal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Patient</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Patient</td>
<td>Normal</td>
<td>Abnormal</td>
<td></td>
</tr>
</tbody>
</table>

*Where more than 1 entry appear, it signifies that the test yielded different results on different occasions. All tests were performed with Warner-Chilcott reagents used according to the manufacturer's instructions.*
The haemoglobinopathies are a group of haematological diseases which are characterized by abnormalities in haemoglobin structure and synthesis. This concept arose following the demonstration by Pauling et al. in 1949 that the haemoglobin found in sickle-cell anaemia had a different electrophoretic mobility from normal adult haemoglobin. Ingram, in 1956, using a delicate new process which he described as 'finger printing', showed that this was due to a specific chemical difference between the globins of normal adult haemoglobin (Hb.A) and sickle-cell haemoglobin (Hb.S), found to reside in the beta-chain. Since that time a large number of haemoglobin variants have been described, with amino-acid substitutions detected in the alpha-, beta-, gamma- or delta-chains.

These diseases are genetically determined. Following