THE

CLINICAL EXAMINATION

OF THE

NERVOUS SYSTEM

BY MONRAD-KROHN

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PREFACE
TO THE TWELFTH EDITION

MORE than half a century has elapsed since the author as a young neurologist started his work on " The Clinical Examination of the Nervous System," his goal being originally to write out a plan for his own clinical work. The result of this he published in 1914 in Norwegian with the following philosophical words of *Claude Bernard's* as a guiding motto: " *Recueillir les faits et ne s'astreindre à les interpréter qu'ensuite, est la condition indispensable pour arriver à la verité.*" As this small book met with some success the first English edition followed in 1921.

Clinical Neurology was at that time rather uneven and clinical examination consisted in many clinics mostly in a haphazard hunt for various " pathognomonic signs " with a view to fixing a diagnostic label on the case, often only to await confirmation or correction at the post-mortem autopsy. (According to one of the leading textbooks of those days there would be 176 diagnostic labels to choose between). The therapeutic consequences could under such conditions only be deficient. On the whole the clinical neurologist's work in many places strongly resembled that of a stamp collector's with the interest chiefly confined to labelling and classification, with an undue interest in rare and weird cases, which are of minor importance unless they throw light on general problems.

The author's guiding idea was to ensure a *complete* routine examination and at the same time to try to replace the diagnostic *labels* of " the stamp collecting era of neurology " by a methodical diagnostic *orientation*, consisting of the following three links:

1. a complete survey of the *functional state* comprising all the loss and alterations of functions to be found,
2. based on this functional state, an anatomical *focal diagnosis* and finally
3. based on the historia morbi and ancillary tests, an *etiological diagnosis*, each of these links having its particular therapeutic purpose:

    1. the functional state as a necessary guide for *re-education and rehabilitation*,
    2. the focal diagnosis as an equally necessary guide for *eventual surgical interference*,
    3. the etiological diagnosis as a guide for " *therapia causalis* " where such is available.

This approach to the cases, always aiming at a *diagnostic orientation in three stages* (*each of which also serving therapeutic issues*) still remains a central point in our clinical teaching. To deal with the special diagnoses of the various neurological disorders is, however, outside the scope of this book.

From the introduction to the first edition the following passages may suitably here be rendered:

" In neurology more than in any other branch of medicine the diagnosis is built up of a number of details found by clinical examination. It is the main object of the examination, then, to furnish *all* the details, to elicit *all* the functional disturbances that the case presents, and in order to do this the clinical examination has to be both systematic and complete.

" The neurological examination, without at least an honest attempt to investigate the patient's mental condition, must be regarded as incomplete. It is fully realized that the correct estimation of a patient's mental state often necessitates continued observation during many days and weeks. What this book proposes to give is only a rough outline of the first mental examination, which in the majority

of cases will give the examiner a rough orientation as regards the patient's mental state.

" On the other hand, it cannot be emphasized too strongly that the psychiatric examination is equally incomplete unless it is accompanied by a complete neurological examination; and the author therefore ventures to hope that this book may be of some use to the medical officer of the mental hospital who feels the need of supplementing his mental examination by a *complete* neurological examination.

" Although this is not intended to be a textbook on the diagnosis of nervous diseases, some diagnostic considerations have been introduced, mainly in order to illustrate the significance of various tests.

" It must be emphasized that proficiency in the neurological examination can only be attained through practical work; this book is therefore intended for use in close connection with the clinic.

" The author recommends his students the following procedure: First, complete the whole systematic examination; give the examination your whole attention without speculating about the diagnosis till the examination has been completed. Then write out a tabulated list of your findings; it is most convenient to arrange them in two columns corresponding to the two sides:

Right	Left.

" Next, try to arrive at a *focal diagnosis* based on your anatomical and physiological knowledge. Finally, consider the *nature* of the lesion, aided by your knowledge of general pathology. Here the various anamnestic data—particularly the mode of onset and the development of the illness—should guide you.

" Only in this way can the student be trained to arrive at a diagnosis in cases which are not typical and which he does not find described in his textbook. Nothing is more injurious to the student's training than the usual way of proceeding, in which the student first reads a textbook, and then, without any logical reasoning, tries to remember what type of disease the case ' reminds ' him of, with the final conclusion that the case ' looks like ' this, that, or the other."

The enormous development steadily going on has necessitated a steady increase in size of the book but the guiding idea and the plan have always been the same and it still remains a book " from the clinic for the clinic."

The development of all the ancillary technical methods is still going on in a very gratifying way and we have tried to keep abreast of this development. Yet a note of warning has again to be sounded. The development of technical methods involves a danger to the budding neurologist: he may lose sight of the patient as an individual, who should first and foremost be studied by means of direct clinical observation and in the second place by technical methods so excellent and valuable as *ancillary* aids.

As the direct clinical observation may be a very lengthy procedure it is understandable that many an unripe youngster in this machine-ridden era of hustle and bustle finds some difficulty in settling down to patient observation and clinical examination of the living individual, hoping that the modern technical methods will offer short cuts to diagnosis. In the face of this tendency it cannot be emphasized strongly enough that the direct clinical observation and meticulous clinical examination of the patient still constitute the main pillars of neurological clinical science, representing, as they do, " the sacred facts "—to quote C. P. Scott (of *The Manchester Guardian*).

Scott's dictum: " Comments are free, facts are sacred ", applies equally well to science as to journalism. Correct observations recorded in phenomenological terms are unchangeable " sacred facts ", not to be disputed. Interpretations and comments are both open to be disputed and changed.

The findings should as much as possible be recorded in *purely phenomenological* terms. This is the reason why, following Sherrington, we keep the expression *rigidity* for *all* conditions where a limb presents resistance to passive movements.*

The clinical examination of the nervous system is—*and should be*—a time consuming proceeding.—This particularly

* "Spasticity" we consider a term of doubtful value as it calls up multiple associations—some of them misleading. We therefore consider the purely phenomenological term "moulding rigidity" preferable to "spasticity."

applies to the difficult examination of aphasia and allied processes of psychosomatic transitions—where we still stick to the author's plan of 1917 (*Journal of Mental Science*) as the basic examination.

A great number of additions and alterations have been made, trying at the same time to avoid any great enlargement of the book.

The chapter on the cerebro-spinal fluid has been revised. In the chapter on pneumoencephalography several additions have been made. In the chapter on electro-myography a short description is given of the measurement of conduction velocity of the nerves.

Our thanks are due to Doctors Arne Engeset and Arne Lundervold for interested and instructive help and also to Doctor Gunnar Rövig and the late Doctor Öystein Farbrot and Professors Tormod Hauge and Kristian Kristiansen for contributions given to the three previous editions and used also in the present one. The examination of the VIII cranial nerve has been remodelled and we are indebted to Mr. Gordon Flottorp, Audiophysicist of the Rikshospitalet, for his valuable aid in respect to this.

We also beg to thank the publishers, Messrs. H. K. Lewis and Co. Ltd., for their unfailing help and assistance.

Professor Sigvald Refsum, who has succeeded Professor Monrad-Krohn in the professorial chair and the leadership of the Neurological University Clinic in Oslo, is responsible for the major part of the revision for this edition, assisted by some notes of Professor Monrad-Krohn.

We hope the present edition will meet with the same benevolent approval with which the previous editions were received.

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SIGVALD REFSUM.

Oslo, *September* 1963.

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THE CLINICAL EXAMINATION OF THE NERVOUS SYSTEM

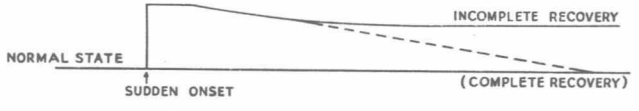
ANAMNESIS.

THIS must be elicited without any suggestion; the patient must be allowed to relate his history in his own words. First and foremost it is of importance to obtain a definite idea of how the illness has developed. *Was the onset sudden or gradual?* If sudden, can the patient give the exact date or even the exact hour when it started? Has he been getting steadily worse—or better—since the onset? Have there been remissions? Exacerbations? Diurnal alterations? (often very marked in myasthenia gravis).

A few paradigmata of some typical "curves of development" (decursus morbi) may be instructive.

(1) *Decursus Morbi of Vascular Cerebral Catastrophes.*

When the disturbances are established suddenly in their full strength in order to give way to a gradual improvement



which is either complete or, more commonly, incomplete, we have the typical curve of development of a vascular catastrophe, which may consist in Hæmorrhage, Embolus, Thrombosis or angiospastic Occlusion (*cf.* pp. 340-341).

Of these vascular catastrophes, thromboses and hæmorrhages are the commonest. (Of 1,000 cases collected by Dalsgaard-Nielsen in 1955, thrombosis was responsible in 500, hæmorrhage in 432 and embolism in 68 cases.) With the introduction of anticoagulation therapy the differential diagnosis between hæmorrhage and thrombosis has become more important than before as such treatment is contraindicated in the initial stages of hæmorrhage. (Later on cerebral hæmorrhage is probably also followed by a local tendency to thrombosis.) The differential diagnosis may be very difficult. It has to be based on: (1) the clinical background (RR, renal function), (2) the onset (more sudden and violent in hæmorrhage; more gradual

and gentle in thrombosis), (3) the relation between disturbance of consciousness (absent or slight in thrombosis—severe, often coma in hæmorrhage) and the loss of function.

A possible source of embolic material (valvular heart disease, particularly mitral stenosis, auricular fibrillation, coronary thrombosis, thrombotic conditions in the lungs) will be in favour of embolism.

Traumatic disturbances obviously have the same sudden onset (when retrograde amnesia is present and there is no visible injury the traumatic origin may sometimes be overlooked).

(2) *Decursus Morbi of Tumours and of Progressive Degenerations in the Nervous System.*

Here the curve of development is represented by a steady crescendo suggestive of the steady growth of a tumour or

the continuous progression of chronic degenerative processes (*e.g.*, progressive spinal atrophy, progressive bulbar paralysis, myopathy, etc.).

Some tumours (*e.g.*, some of the gliomata) are predisposed to hæmorrhage. When such a tumour has grown in a silent area* symptomless for some time, a hæmorrhage may cause a sudden clinical onset, and the curve of development may have the same beginning as in (1).

(3) *Decursus Morbi of Cerebral Arterio-sclerosis.*

A staircase-like crescendo curve is characteristic of cerebral arterio-sclerosis, miniature vascular catastrophes (clinically characterized by attacks of vertigo, transient

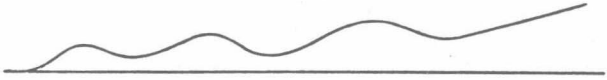
paralyses or aphasia, etc.) succeeding one another, adding their effects to one another in such a way as to produce an increasing cerebral invalidity, the final stages of which

* For a complete neurological examination, aided by the modern supplementary methods of air encephalography, angiography and electroencephalography, there are hardly any "silent areas" any longer. In one sense, however, there will always be silent areas, viz., when the lesion does not cause any so pronounced symptoms that the patient is prompted to seek a physician, who brings him under special neurological observation.

may vary, but commonly correspond more or less to the clinical picture of "pseudo-bulbar paralysis" (better called "central bulbar paralysis"—Collier).

(4) *Decursus Morbi of Disseminated Sclerosis.*

An undulating curve of crescendo development (remissions alternating with exacerbations) is typical for dis-

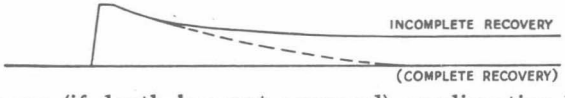


seminated sclerosis, except in the last stages of this very common disease, which generally begins with an undulating crescendo curve and ends in a steady crescendo without noticeable remissions.

Often the apparent onset is preceded by some transient disturbance (diplopia, retrobulbar neuritis, precipitate micturition), occurring months or years before.

(5) *Decursus Morbi of Acute Poliomyelitis.*

In acute poliomyelitis we meet with yet another curve of development. The onset is as a rule not quite sudden, but development is generally rapid, and the full amount of disturbance is usually reached in the course of a few days,



whereupon (if death has not occurred) amelioration begins, leading to an incomplete or, very rarely, to a complete recovery.

Often (in about 40 per cent. of all cases) an uncharacteristic febrile state of a few days' duration (by Ritchie Russell* called "minor illness") precedes the onset of the actual illness (the "major illness"—Ritchie Russell*), with an intervening symptomless and deceptive interval of a few days' duration.

The somewhat schematic description of these few curves of development may help the student to see how important it is to obtain a complete description of the onset and development of the illness.

Above all try always to obtain precise information of the initial symptom or symptoms, whether motor or sensory.

* *Cf.* W. Ritchie Russell: "Poliomyelitis" (Edward Arnold and Co., 1956).

Thus initial difficulty in mounting stairs and getting up from a chair is often a sign of failure of the proximal muscles of the lower limbs. Initial pains of fixed localization (not necessarily extended to the whole segmental area) may be the first root pains so important in extramedullary spinal tumours.*

To complete the history, some leading questions should be asked particularly about the following symptoms:

Headache (constant or intermittent, diurnal or postural variations; diffuse or localized; hemicrania).

Vomiting, with or without subjective feeling of nausea.

Vertigo.—Is it more pronounced in the dark than in light? "wash-basin symptom"? tendency to fall to one side? any subjective sensation of rotation?

Disturbances of Vision.—First and foremost *diplopia* (frequent in "epidemic" encephalitis, disseminated sclerosis, tubercular and syphilitic meningitis, intracranial tumours).

Pains and Paresthesia (initial root pains in extra-medullary tumours; "cushion sensation," "girdle sensation," in tabes, *e.g.*).

Disturbances of Consciousness (loss of consciousness, automatism, petit mal, dreamy states, "déjà vu" feelings, depersonalization).

Convulsive Fits (or Seizures) (tonic or clonic, general or localized, followed by localized paresis; Jacksonian fits, attacks of opisthotonos).†

Both in regard to disturbances of consciousness and convulsive fits one must ascertain if the patient has any recollection of what has happened. If not, *total amnesia* is said to exist for the attacks—a characteristic point in epilepsy.‡ Also it should be ascertained if the patient has any kind of previous warning (aura). An exact description of this should be obtained.

* Cp. my article on Diagnostic Errors in Neurology (*Acta Medica Scandinavica*, 1952—Supplementum CCLXVI, (266), p. 733.)

† Whilst formerly opisthotonos was considered a typical hysterical manifestation, it can no longer be regarded as such. It may be a symptom of meningeal irritation; it may also be a symptom of a lesion of the corpora quadrigemina. (Quadrigeminal syndrome: paralysis of upward movement of *both* eyes, bilateral deafness whilst vestibular reactions are unimpaired, tendency to fall backwards, attacks of general extensor spasm of all four limbs, and opisthotonos.)

‡ In all such cases *electro-encephalography, hyperventilation*, and possibly the *hydration tests* should be carried out (*cf.* Appendix).

It should also be ascertained if such paroxysmal events are related to or brought on by any specific circumstances such as, *e.g.*, the menstrual epoch, long fasting interval since last meal (hypoglycæmia), the wearing of a too tight collar (carotid sinus reflex), strong (particularly flickering) light.

When consciousness has been lost in trauma capitis, it should be ascertained if there be *retrograde amnesia* (*i.e.*, loss of memory comprising also a period *prior* to the accident, of a duration from seconds to hours). This is frequent in cerebral concussion. In cerebral compression (due to hæmorrhage from injured vessels) loss of consciousness comes on more gradually *after* the accident.

Bladder Trouble.—Precipitate micturition* (frequent in disseminated sclerosis); incontinence; retention. In case of incontinence it should be ascertained whether the bladder empties itself involuntarily at certain intervals—automatism of bladder mechanism (as in infants);—or a steady, dribbling incontinence obtains, "ischuria paradoxa," an incontinence by overflow.

Constipation.—There is no doubt that many neurotic complaints are accentuated by constipation.

Disorders of Sleep: Insomnia—Hypersomnia (as detailed an account as possible).—In insomnia, is it the process of going to sleep that is impeded by pains, by persevering thoughts or by anxiety? Or does the patient wake up with any peculiar sensation? How many hours does the patient sleep during a night and a day? Dreams?

In women the *menstruation* should always be inquired into (amenorrhœa? irregularity? menstrual exacerbations?).

The *patient's previous life* (previous illnesses) should then be inquired into.

Inquiries about *syphilitic infection* should always be made, if not directly, then indirectly—abortions, still-births, rash, etc. Intemperance with regard to *tobacco* and *alcohol* should be noted, and the quantity consumed should be stated as accurately as possible.† Also *the patient's diet* should be

* By patients often erroneously recorded as "inflammation of the bladder."

† It is wise always to *ask about tobacco first.* Few people have any objection to admitting that they smoke a lot, whilst most people dislike admitting that they drink a lot. Asking for exact and detailed information about tobacco consumption paves the way for putting corresponding questions about alcohol consumption.

inquired into, particularly with regard to *possible vitamin deficiency*.

Has the patient been exposed to any *episode of anoxia*? (*e.g.* drowning with prolonged resuscitation, difficult and prolonged birth in breach presentation, CO-intoxication).*

Traumata will as a rule be mentioned by the patient in his own narration, but may also have to be inquired for (sometimes from others, *cf.* retrograde amnesia in cerebral concussion, which may sometimes occur without any visible injury).

In cases of fractures it should be ascertained if the trauma was an adequate one or not ("spontaneous fractures").

Finally, the different data regarding *the patient's family* should be obtained.

It is important to obtain a complete record of the patient's occupation and an equally complete picture of his occupational and family environment and of his mode of living. Make the patient give you a full account of his daily routine from when he wakes up in the morning till he goes to sleep at night.

As regards work it should always be remembered that *work as a fatigue-producing factor* is a much more comprehensive item than the *occupational or professional work sensu strictiori*. Worries, conflicts and difficult decisions constitute a considerable amount of hard mental work as a fatigue-producing factor, though of no outward utility.†

Whilst the *focal diagnosis* (where is the lesion situated?) is chiefly based on the actual findings by the clinical examination—*the "etiological" diagnosis* (of what nature is the lesion—inflammatory, neoplastic, traumatic? etc.) rests to a larger extent on the anamnesic data and the more or less typical curves of development, as outlined above.

* Cp. the author's article "The Clinical Aspect of Cerebral Anoxia," *Acta Psychiatrica et Neurologica Scandinavica*, 1956, Vol. XXXI, Fasc. 1.

† It is of little use to let an overworked clerk or factory worker stay away from the office or the factory if his chief occupation at home consists in quarrelling with other members of his family, which in itself may be the chief cause of the patient's neurotic state representing, as it does, an added burden of useless, but nevertheless fatigue-producing, "work."