Clinical Implications of a Neuropsychological Approach to Aphasia

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ABSTRACT
This paper deals with the clinical implications arising out of a study designed to evaluate Luria's approach to the cerebral organisation of higher mental functions such as language. The research took the form of four in-depth case studies of aphasic patients and involved a comparison of neuropsychological predictions as to the site-of-lesion with radiological findings (cranial computerised tomographic scanning). Correspondence was felt to be good in most instances, indicating that Luria's theory provides a valid framework within which to relate clinical symptomatology and focal brain damage. The fact that Luria places clinical practice on a firm theoretical foundation is seen as being advantageous and is discussed in relation to assessment and therapy.

OPSOMMING
In hierdie studie word Luria se benadering tot die organisasie van hoër-verstandelike funksies soos taal, evalueer. Die kliniese implikasies van die studie kom veral onder die soeklig. Die navorsing het uit vier gevalle studies van afatiese patiente bestaan. 'n Vergelyking is tussen die neuropsigologiese simptome as voorspelling van die lokus van die letsel, en radiologiese bewysings, getref. Die ooreenkoms tussen die neuropsychologiese voorspelling en die lokus van die letsel was in meeste gevalle goed. Dit dui aan dat Luria se teorie 'n geldige raamwerk verskaf waarbinne kliniese simptomatologie en fokale breinskade gekoppel kan word. Die kliniese praktyk wat Luria op so 'n vaste teoretiese grondslag bou, word met betrekking tot diagnose en behandeling bespreek.

The speech clinician involved in the assessment and remediation of aphasia is confronted by a bewildering array of approaches to this complex disorder. This paper focuses on some clinical implications arising out of a study designed to evaluate, within the context of speech pathology, the neuropsychological approach developed by Luria, who has been described as "this century's leading Soviet figure in aphasiology". According to Hatfield, Luria is one of the few leading aphasiologists who has combined clinical practice with theory. This is seen as a major advantage, but it does imply that an understanding of the general neuropsychological principles upon which Luria based his work is essential for the clinician who wishes to use this approach. A brief resumé of theoretical aspects relevant to this paper follows.

Luria viewed his work on the organisation of higher mental functions in the brain as incorporating the positive aspects of two opposed approaches to the subject. He felt that neither the mechanistic narrow view, given its major impetus by the work of Broca, nor the integral or noetic view, supported by authorities such as Goldstein, successfully accounted for the cerebral organisation of higher mental function. Both views in Luria's opinion contributed to misunderstandings of this complex subject.

In a re-examination of concepts such as function, localisation and symptom, Luria retained and integrated parts of both the extreme views mentioned above, while successfully avoiding the less desirable aspects, namely, the idea that complex mental processes can be located in a single focal area of the brain on the one hand, and that such activities involve the whole brain in an undifferentiated fashion on the other.

According to Luria, one should conceptualise the higher mental functions as functional systems with an extremely complex composition. The performance of any such mental activity necessarily involves the co-operation of many different parts of the brain.

Luria divides the brain into three main functional units. These units and their further subdivision into hierarchically arranged cortical zones, are described in detail in his works and are represented schematically in Figure 1. Speech and language are thus viewed as complex functional systems to which many different cortical areas contribute (See Table 1). Luria refers to these cortical areas as "links" within the functional system. Damage to any one of these areas affects its ability to function optimally and it thus becomes a weak or broken link within the chain of the functional system. Although any damaged link

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will affect the functional system, the effects will vary depending upon the particular link involved. The interference with the normal function of a specified cortical area results in a primary or fundamental problem. The secondary or systemic effect of this primary problem is the breakdown in the functional systems to which the area normally contributes.

Two points should be noted at this stage. First, what appears to be the same symptom complex, may actually be related to one of several primary problems. For example, the common aphasic symptom of word-finding difficulty may be caused by a breakdown in phonemic hearing, problems with simultaneous synthesis, or difficulties at the articulatory level. Second, because the function of a specific cortical area may be a link in several functional systems, a focal lesion might result in a group of symptoms which are seemingly unrelated. However, appropriate neuropsychological analysis reveals that the symptom complex consists of "externally heterogeneous but, in fact, internally interconnected symptoms". For example, a lesion of the overlapping tertiary zone in Unit II results in a specific primary problem, namely, difficulty with simultaneous synthesis. However secondary or systemic effects include a problem with logical-grammatical relationships as well as acalculia. Rather than attempting to localise complex functional systems such as receptive or expressive language, an attempt should be made to identify the various focal areas of the brain which make a specific contribution to the system as a whole. The clinician who is able to localise the lesion and who understands how the resultant primary problem may affect the functional system under consideration, is then in a position to plan appropriate treatment. Thus within Luria's framework, a classificatory label attached to an aphasic patient is not merely descriptive but is rather an indication of the primary problem underlying the presenting symptom cluster (see Table 2).

Luria himself recognised the need to evaluate his work in terms of some objective criterion and it was with this in mind that the present research evolved. The study was designed to assess Luria's views on the manner in which symptomatology relates to brain damage. More specifically, the aim was to compare the site-of-lesion in aphasic patients as ascertained by Luria's neuropsychological tests, with the results of radiological findings based on cranial computerised tomography (C.T.) scanning. The study itself will be described very briefly and will be followed by a discussion of clinical implications.

METHOD
This research took the form of four in-depth case-studies.

SUBJECTS
All subjects were diagnosed as aphasic by a speech therapist and a neurologist. Etiology was required to be a CVA resulting in a focal lesion that could be observed on a cranial CT scan. The upper age-limit was set at 55 years and hearing was required to be within normal limits. A minimum period of three months had to have elapsed since the onset of the CVA to allow for spontaneous recovery in accordance with the study by Demeurisse, Demol, Derouck, deBeuckelaer, Coekaerts and Capon. See Table 3 for a description of the subjects.

PROCEDURE
Subjects were scanned (cranial CT scanning) in order to establish the presence of a focal lesion. As this was a blind

### Table 1

| UNIT I | Maintains the optimal level of cortical tone necessary for the functioning of the systems of speech and language |
|--------|----------------------------------------------------------------------------------------------------------|
| UNIT II | Zone 1 Receives auditory impulses | Zone 2 Receives visual impulses | Zone 2 Zone 2 Recognition and processing of speech sounds or phonemes |
| Auditory analyser | | Visual analyser | Visual auditory functions |
| | Tactile-kinesthetic analyser | | Tactile-kinesthetic auditory functions |
| UNIT III | Zone 1 Outlet channel for movement | Zone 2 Planning of movement | Zone 3 Integration of successive input |
| | | | Zone 3 Regulation and verification of complex behaviour |

**Figure 1** Schematic view of Luria's division of the brain into units and zones.

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Table 2  Luria's classification of aphasia as related to primary problems and areas of cortical damage

| Type of aphasia          | Primary problem                                      | Cortical area                                     |
|-------------------------|------------------------------------------------------|--------------------------------------------------|
| Efferent motor          | Skilled sequential movements                         | Pre-central                                      |
| Afferent motor          | Motor kinesthetic afferentation                       | Post-central                                     |
| Dynamic                 | Speech drive                                         | Frontal lobes                                    |
| Sensory                 | Phonemic hearing                                     | Superior, posterior temporal lobe                |
| Acoustico-mnestic       | Audio-verbal memory                                  | Middle temporal lobe                             |
| Semantic                | Simultaneous synthesis affecting quasi-spatial operations | Posterior inferotemporal lobe (tertiary, overlapping zone) |

Table 3  Description of subjects

| Subjects          | O      | P      | V     | D     |
|-------------------|--------|--------|-------|-------|
| Age at time of testing | 49 years | 50 years | 44 years | 23 years |
| Sex               | male   | male   | female | male |
| Time elapsed since onset | 9 months | 5 months | 1½ years | almost 2 years |
| Immediate effects of the CVA including progress in first 3 weeks | Could not speak for first 3 days. Recovered slowly | Could not speak for first 3 days. Recovered slowly. Paralysis of right upper limb — recovered completely | Could not speak. Dense right hemiplegia. | Coma for 2½ weeks. Right-sided hemiplegia |
| Speech therapy    | Immediately after stroke for 4 months | | Immediately after stroke. Discontinued after 1 year | Immediately after stroke until the present time |
| Pre-morbid conditions | Heart condition (3 previous heart attacks). High blood pressure. Diabetes diagnosed at age 35 years | | | |
| Post-morbid complications in addition to aphasia | Athetoid movements of right hand. Hemianopia | | Severe hemiplegia | Residual hemiplegia and hemiparesis. Jacksonian epilepsy |
| Pre-morbid handedness | Right | Right | Right | Left |
| Drugs taken at the time of testing | Aldomet, Isoptin, Syndol | Persantion, Librium | Epinutin | Epinutin, Persantion, Aspin, Tegretol |
| Home Language      | Afrikaans | English | Afrikaans | English |
| Pre-morbid occupation | Lawyer | Sheet-metal worker | Secretary | University student |
| Present occupation  | Same | Housewife | | Student issues clerk |
| Pre-morbid educational level | University degree | Standard five | Matriculation, Secretarial diploma | University student |

RESULTS

The results of the study are summarised in Table 4 which compares neuropsychological predictions as to the site-of-lesion with radiological findings (cranial CT scans). Bearing in mind normal individual variation in the cerebral organisation of higher mental functions, as well as subject characteristics encountered in this study (for example, one left-handed subject and one with a premorbidly low level of intellectual function), the correspondence between the neuropsychological and radiological findings is felt to be good. However, mention must be made of instances of non-agreement. In certain cases.

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### Table 4 Summary of results comparing neuropsychological and radiological findings for all four subjects

| Site-of-lesion | SUBJECT O | SUBJECT P | SUBJECT V | SUBJECT D |
|---------------|-----------|-----------|-----------|-----------|
| HEMISPHERE    |           |           |           |           |
| RIGHT         | 1         | 0         | 1         | 1         |
| LEFT          |           | 1         |           |           |
| CORTICAL AREAS|           |           |           |           |
| Sensorimotor  |           |           |           |           |
| Tertiary parietal |       |           |           |           |
| Prefrontal    |           |           |           |           |
| Superior, posterior temporal | 1 | 1 | 0 | 0 |
| Middle temporal |           |           |           |           |
| Occipital     |           |           |           |           |

**KEY**
- Indication of damage
- Contra-indication of damage

Radiological Confirmation of Neuropsychological Findings

| 1 | Confirmed |
|---|-----------|
| 0 | Not confirmed |

There was no evidence of structural damage to support clinical symptomatology. In this instance, it is possible that there is damage that has not been picked up on the scans. It should be borne in mind that the scanning techniques used here provide a static representation of extant neuropathology. It is our belief that neuroradiological procedures which can detect physiological or functional change such as blood-flow, for example, might correlate better with neuropsychological dysfunction, than the static structural damage observed on the scans used in this study.

It is felt to be more important to account for the cases in which there is a definite indication of damage on the scan but no evidence of the clinical symptoms one would expect from such damage. In both subjects V and D, for example, there was little or no clinical manifestation of observed damage to the superior posterior temporal lobe (Wernicke's area). In subject D who was premorbidly left-handed, there is a strong possibility of bilateral representation in the premorbid cerebral organisation of language, lending support to work done by Hardyck. However, it is more difficult to account for the same situation arising in Subject V, who is a right-hander. The fact that damage to Wernicke's area did not result in the expected symptomatology, despite good correspondence in other areas, leads one to consider the possibility of a premorbid bilateral representation of language in this subject.

Despite the above instances of non-agreement, the fact that the principles of Luria's neuropsychological analysis were useful in arriving at the site-of-lesion in most cases, indicates that his theory of functional localisation, in which the concept of the primary problem plays such a central role, is a valid means of relating observed symptomatology to focal brain damage.

**DISCUSSION**

This result has clinical implications for the speech clinician dealing with aphasia as Luria's views on diagnosis and therapy are logically related to his theory. First, as regards classification, Luria's approach differs from many traditional approaches.

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While accurate classification is stressed, this does not, as mentioned previously, merely involve the attaching of descriptive labels. Rather, classification gives an indication of the real (primary) problem or problems underlying the often confusing picture presented by the patient. As can be seen from Table 5, although the types of aphasia represented range from the highly specific, such as afferent motor aphasia, to those of a broader nature, such as dynamic and semantic aphasia, they all relate to underlying brain damage in the same fashion, namely, focal brain damage results in specific primary problems which are manifested in the symptoms we observe.

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One of the advantages of Luria’s approach is that it enables one to be specific. For example, the commonly used term ‘global aphasia’ indicates that there are gross expressive and receptive language defects related to an extensive lesion probably involving both anterior and posterior parts of the brain. It might be far more useful clinically to have more detail about the exact extent of the lesion and the primary problems which need to be tackled. In other words, global aphasia should be viewed as a complex combination of several primary problems rather than as an undifferentiated whole. Similarly it would be more useful theoretically and clinically to classify complex problems.

### Table 5 Classification of subjects according to the primary problem and types of aphasia

| Subject | Primary Problem | Type of aphasia in terms of Luria’s classification |
|---------|----------------|--------------------------------------------------|
| O       | Visual agnosia for letters, Spatial and quasi-spatial difficulties, Mild audio-verbal memory difficulties | Semantic aphasic, Mild acoisctico-mnestic aphasia |
| P       | Some quasi-spatial problems | Mild semantic aphasia |
| V       | Efferent and afferent organization of movement and speech, Quasi-spatial difficulties, Audio-verbal memory, Regulation of movements and actions | Efferent motor aphasia, Semantic aphasia, Acousticomo-nestic aphasia, Possibility of dynamic aphasia |
| D       | Efferent and afferent organization of movement and speech, Quasi-spatial difficulties | Efferent aphasia, Afferent aphasia, Semantic aphasia |

### Table 6 Primary problems and effects relevant for therapy (Subject V)

| Primary problem | Effects on speech and language | Other major effects of relevance to the speech clinician |
|-----------------|--------------------------------|-------------------------------------------------------|
| Efferent and afferent organization of movement and speech | Severe articulation problems, Absence of narrative speech, Quasi-spatial functions, for example, difficulty in understanding logical-grammatical speech, Difficulty with series of verbal stimuli, Possibility of problems with internal speech | Hemiplegia, Problems of motivation, attention, perseveration, attention to irrelevant stimuli, inhibition of actions, etc. |

Note: Some difficulties, such as naming and comprehension in some instances cannot be related to one specific primary problem in this subject. These difficulties may be due to a combination of some or all of the above primary problems.

### Table 7 Primary problems and effects relevant for therapy (Subject O)

| Primary problem | Effects on speech and language | Other major effects of relevance to the speech clinician |
|-----------------|--------------------------------|-------------------------------------------------------|
| Efferent and afferent organization of movement and speech | Word-finding difficulty, Some difficulty with confrontation naming, Mild problems with logical-grammatical structures, Severe alexia, Difficulty with the retention of series of auditory material in the absence of semantic cues | Hemianopia |

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cases of aphasia, such as Broca's, in terms of the primary problems involved (see Table 5) instead of grouping them together in a broad symptomatological category often inaccurately related to an isolated focal area of the cortex.

Second, in order to plan therapy appropriately, it is necessary to relate the primary problem to speech and language. Other effects of the primary problem while not directly related to speech and language, must also be taken into account. The following charts (see Tables 6 and 7) have been drawn up as an illustration of one way in which the speech clinician can relate the primary problem to the overall symptom complex.

Subjects V and O have been chosen as examples because they represent different levels of severity and complexity. In the former case (V), brain-damage was extensive and the clinical picture highly complex, making it difficult to identify the underlying primary problems. In the latter case (O), the primary problems were relatively easy to identify.

These therapy charts need to be individually tailored for each patient. Being able to isolate the primary problem does not mean that the entire symptom profile can be predicted, although symptoms should be able to be explained in terms of the primary problem. The neuropsychological examination, in addition to giving information about the site-of-lesion, enables the clinician to be fairly specific about the functional effects of this damage in the individual being examined. For example in subject V, there was evidence of both efferent and afferent motor aphasia. However, the latter predominated and would therefore require more attention initially. It was observed, that if this subject was able to circumvent kinaesthesia by compensating with her strong visual abilities or a combination of audio-visual stimulation for example, she was able to articulate reasonably well. This fact should be used in constructing an appropriately graded therapy programme. In addition, the major effects of prefrontal damage in the case of Subject V would definitely affect therapy adversely and would make for a far less favourable prognosis. Examples of the symptoms being referred to are problems of motivation, attention to irrelevant stimuli, and inhibition of actions, among other things.

The clinician must therefore be aware of which problems are of major importance and require most attention. It must be understood that ‘important’ in this case refers to the fact that the particular problem is having a major effect on the patient’s ability to communicate. For example, in the case of Subject O (see Table 7), the mild audio-verbal difficulties noted did not affect communication significantly.

CONCLUSION

The implications of the findings of the study reported here have been discussed in relation to classification and therapy. Although these have been discussed separately, it is hoped that the logical and cohesive nature of Luria’s neuropsychological theory is evident in the way in which these topics relate to each other.

The fact that Luria places clinical practice on a firm theoretical foundation is seen as being advantageous to the speech clinician, who, with her theoretical background and access to clinical material, has in turn a significant contribution to make to the neuropsychological study of aphasia. Further research in the field would be usefully augmented by clinical studies evaluating, in particular, Luria’s approach to assessment and remediation in aphasia. Feedback from such studies could for example be used to remodel aspects of Luria’s classification of aphasia which do not seem to tie in with clinical reality.

On a more practical level, Luria’s work should be made available in a format especially designed for use by the speech clinician involved in the field of aphasia.

Although a considerable investment of time and energy is required of the speech clinician interested in applying Luria’s approach, the effort is felt to be worthwhile “for practical-minded field speech pathologists, who are willing and flexible enough to try to understand the underlying philosophy”.8

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