AGRAPHIA AND MICROGRAPHIA: CLINICAL
MANIFESTATIONS OF MOTOR PROGRAMMING AND
PERFORMANCE DISORDERS *

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Rigorous studies involving cases of acquired language and memory disorders have contributed
substantive information regarding higher cortical functions. Acquired disorders of handwriting,
however, have remained a largely untapped resource which could be of great value to students of
motor control. In this study, we demonstrate how two such disorders provide specific information
regarding the motor control of handwriting. In a case of acquired agraphia due to a cerebrovascu-
lar accident there was a problem in programming the proper letter strokes and achieving the
correct spatial location of strokes. In contrast, Parkinsonian patients with micrographia produced
the appropriate letter strokes but could not maintain adequate force to preserve letter size. It
appears that such information will help to dissect the handwriting skill into a number of
functionally and anatomically separable subcomponents.

Of the two forms of visible language, reading disturbances have cer-
tainly received the bulk of attention, disorders of handwriting having
been relatively neglected by neurologists and neuropsychologists. The
analysis of handwriting errors in normals has provided the basis for a

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model of the motor control of handwriting which consists of several discrete subcomponents (Ellis 1979, 1982). Acquired disorders of handwriting are another important source of information which has been grossly underutilized.

In this report we investigate two writing disorders which affect different components of motor control. In a patient with agraphia due to a brain lesion, there was an apraxia limited to handwriting which resulted in a breakdown in the formation of some letter strokes. This syndrome was associated with a neocortical lesion. In contrast, in patients with micrographia secondary to Parkinson’s disease there was a disturbance in the force and timing of writing movements which seemed to be independent of higher order processing. The pathologic substrate in this disease is primarily subcortical. In studying these and other acquired disorders of handwriting one hopes to further delineate the functional subcomponents involved in the motor control of handwriting and define their neuroanatomical correlates.

1. Acquired agraphias

Acquired agraphia refers to a decline in writing performance that is not accounted for by a primary sensory or motor dysfunction (e.g., blindness or paralysis). Agraphias are most often associated with lesions of the cerebral cortex, but no “writing centers” analogous to Wernicke’s or Broca’s area which are critical for speech have been clearly identified. A fundamental distinction should be made between linguistic and non-linguistic agraphias. Linguistically based agraphias are common and usually accompany oral language disturbances. Linguistic agraphias are essentially disorders of written spelling which are characterized by letter omissions, substitutions, additions, and reversals, while letter morphology is preserved (Marcie and Hecaen 1979). In keeping with the theme of this volume, this form of agraphia will not be discussed further but has been reviewed elsewhere (Leischner 1969; Marcie and Hecaen 1979; Margolin and Binder in press; Margolin submitted).

Non-linguistic forms of agraphia include apraxic and spatial agraphia. Apraxic agraphias are characterized by faulty letter formation with intact spelling. Like other forms of apraxia, the term refers to a disorder of motor execution not accounted for by a disturbance in elemental sensory or motor processes and may include a breakdown in the morphology and sequencing of movements.
Some instances of agraphia appear to be due to perceptual disturbance. Patients with visual perceptual dysfunction, usually due to non-dominant hemisphere lesions, may display a particular combination of writing changes which has been termed spatial agraphia (Hecaen et al. 1956). Changes include slanting of the line of writing off the horizontal, widening of the left hand margin and letter repetitions (Hecaen et al. 1956; Lebrun 1976).

While lesions of the dominant parietal lobe are often associated with disturbances of letter formation, there are usually spelling errors as well (Marcie and Hecaen 1979). Apraxic agraphia without linguistic disturbance is thus quite rare, and is usually associated with very discrete lesions or atypical cerebral dominance. It is these cases, however, which are the greatest potential source of information about the motor control of handwriting. We had the opportunity to study a patient who developed a profound disturbance of letter formation with preserved spelling which permitted a detailed analysis of his errors in letter formation.

1.1. Case report

A 52 year old man, R.B., with mixed handedness (left handed for writing) suffered a right hemisphere stroke the day following abdominal surgery. Neurological deficits included left sided paralysis and sensory loss, blindness in the left visual field (complete homonymous hemianopsia) and a typical non-dominant hemisphere syndrome of visuospatial and visuoconstructual deficits. A computerized brain scan revealed an extensive right hemisphere stroke involving mainly the frontal and parietal lobes. Details of the neurologic exam and neuropsychological testing are reported elsewhere (Margolin and Binder in press). Notably, auditory comprehension, verbal output, reading, letter identification and oral spelling were intact. In contrast, there was a profound graphic disturbance. During the first week after the stroke, he could produce only an amorphous scrawl when asked to write or draw with his non-paralyzed right hand. This was particularly striking since strength, coordination, and sensation were intact on the right side. He performed a variety of transitive (e.g., comb your hair, hammer a nail), and intransitive (e.g., wave goodbye, salute) gestures to verbal command without error and could imitate finger movements presented in random or fixed sequences. It was thus evident that his dysgraphia was
not merely the reflection of a general inability to carry out complex movements with his non-preferred hand. In contrast to his success with other gestures, he could not mime the formation of letters in the air.

Ten months after the stroke, his handwriting and drawing were still markedly impaired, the visual perceptual deficits were unchanged, and he remained completely paralyzed in the left upper extremity.

1.2. Description of graphic performance

The writing samples displayed here were obtained between one and ten months following the stroke. All samples were produced with the right hand since his left hand was completely paralyzed. No clear improvement in handwriting was observed during this 10 month interval.

Within one month following the stroke one could observe a clear

Fig. 1. One month following stroke: A = signature; B = printed name; C = copied name (model above copy).
distinction between cursive and printed writing (fig. 1). There also was
some segmentation between “letters” but none of the letters were
legible. Copying (fig. 5) was clearly superior to his other writing (figs.
1–3) and he could trace letters or figures accurately. There was no
difference between graphic performance with eyes opened or closed.
Letters were formed with normal speed [1] and R.B. was usually aware
when he made an error. Letter malformations were inconsistent as
demonstrated in three immediately repeated attempts to produce the
lower case printed letter a (fig. 2).

When instructed to write the alphabet in cursive (fig. 3) or print (fig.
4), write numbers, or take dictation he did so rapidly and without

[1] No specific instructions were given the subject regarding speed or accuracy. He usually
performed spontaneous writing and copying at about the same speed.
Fig. 3. Cursive alphabet: 1st box = letter f; 2nd box = letter i; 3rd box = letter q.

Fig. 4. Printed alphabet: 1st box = Letter J; 2nd box = letter l.
Fig. 5. Copied alphabet (models above copies).
further cueing. The letters, however, were very poorly formed and many would have been illegible if viewed individually.

In addition to the overall dysmorphic quality of his handwriting, he often produced incorrect letter elements or strokes. In forming the lower-case $f$, for example (fig. 3), the upper loop is repeated once in lieu of the appropriate descending loop. A repetition of an appropriate stroke is seen in the cursive $q$. Here too, the descending stroke is omitted but the terminal stroke is appropriate. In the cursive $l$ there is simply a single repetition of the proper upstroke and downstroke. The upper case $G$ in fig. 5 provides a particularly clear example of stroke substitution. He proceeds correctly up to the formation of the short horizontal segment. Instead of proceeding horizontally, however, he descended vertically thus forming a loop. The loop was then repeated and finally the horizontal component was inserted in proper sequence but in an anomalous spatial location.

Only one letter substitution, $B$ for $P$, was documented (fig. 5). This was not clearly a linguistic error, however, in that it involved the

Fig. 6. Top left = drawing of circle and top right = drawing of square, no models provided; bottom = copy of square, design, and house (models below copies).
production of a single anomalous stroke and may thus have been an apraxic or perceptual error. Occasionally letters were relatively well formed but rotated as seen in copying the letter S (fig. 5). A combination of 180° rotation and rotation plus the addition of an extraneous stroke were seen in the J and L respectively (fig. 4).

Both in spontaneous writing and copying (e.g., E, M, fig. 5) strokes were not put in the proper spatial location. In other cases the orientation between strokes was faulty (e.g., K, T, V, fig. 5). A similar difficulty was seen in spontaneous or copied drawings (fig. 6).

1.3. Analysis of graphic performance

It is clear from the preceding case description that R.B. was suffering from a disturbance of motor control which was specific for graphic tasks and was not explained by a lack of coordination. The following error analysis demonstrates that this was a multi-component agraphia, consisting of apraxic and spatial components.

The model of handwriting control developed by Ellis (1979, 1982) provides a framework for developing the concept of writing apraxia. In this model there are three main representations for a letter; the grapheme, allograph, and graph. The grapheme is an abstract specification of a particular letter which can be expressed in a variety of forms including writing, typing, and oral spelling. Next, the allograph assigns a particular shape to the letter, thereby specifying the case and form. The graph refers to the actual production of the letter in handwriting. The successful production of a graph is dependent upon a graphic motor pattern which specifies the appropriate sequence of strokes and their relative size. Finally, these specifications must be translated into a particular pattern of neuromuscular activity.

Within this schema, a disturbance at the grapheme stage would produce errors in letter form without errors in letter selection and would thus fit the generally accepted definition of apraxia. R.B. demonstrated no clear errors at the grapheme stage such as errors in letter selection, nor at the allographic stage such as mixing upper with lower case or cursive with print. There were however numerous errors involving a single stroke which is consistent with a breakdown at the level of the graphic motor pattern. However, these data do not permit a discrimination between damage to the graphic motor pattern itself vs. faulty input to or output from the graphic motor pattern.
These single stroke errors which occur in normals as well (Ellis 1979), suggest that even after writing becomes overlearned and automatic, the stroke serves as a fundamental unit of letter formation with each letter programmed as a sequence of one or more strokes. The fundamental role of the stroke in letter programming is consistent with data from other types of motor tasks. Restle (1970) has studied serial pattern learning in normals where subjects are required to press buttons in a particular sequence. Normals tend to err by overextending two types of patterns, sequential patterns or runs (e.g., 1, 2, 3, 4, 7 → 1, 2, 3, 4, 5), and repetitive patterns or trills (e.g., 5, 4, 5, 4, 3 → 5, 4, 5, 4, 5). These patterns appear to represent a universal organizing tendency in planning movement sequences. The appearance of errors involving overextension of repetitive letter stroke sequences, such as the upstroke-downstroke-upstroke-downstroke sequence in the printed letter M, would thus support the role of the stroke as a fundamental unit in the motor programming of letters. Indeed, such errors were seen in R.B. and other agraphic patients (Gordinier 1899) and also occur in normals (Ellis 1979).

In addition to the apraxic disturbance, some of R.B.'s graphic disturbance was probably related to his visual perceptual deficits. Several lines of evidence indicate that the translation of a graphic motor pattern into a graph can be influenced by changes in sensory feedback. Patients with visual perceptual deficits frequently slant the line of writing off the horizontal, show widening of the left hand margin, and add additional strokes to some letters, particularly M and N (Hecaen et al. 1956; Lebrun 1976). When normals write while attending to delayed visual feedback of their own writing, handwriting velocity and legibility diminish, and there is an increase in letter repetitions and letter malformations. The high incidence of letter repetition errors suggests that visual feedback is important in updating the graphic motor pattern so to which graphs have already been executed (Van Bergeijk and David 1959; Smith et al. 1960; Kalmus et al. 1960; Lebrun 1976).

In R.B.'s case it is quite likely that his perceptual deficits contributed to his writing disorder. His visual perceptual difficulties included a severe breakdown in the ability to judge the proper orientation of lines (Benton et al. 1978), which may well have contributed to the difficulty he had in achieving the proper location and orientation of lines in writing and drawing. The widening of the left hand margin seen in
R.B.'s writing is probably related to his left visual field loss and neglect. Some of R.B.'s letter formation errors, such as letter stroke repetition and the generalcrudeness of letters are consistent with the changes produced by altering visual feedback in normals. However, there are several important differences between R.B.'s performance and that of the delayed feedback subjects which makes it improbable that all of his errors were due to his visual perceptual deficits. Some of R.B.'s errors involved the insertion of an anomalous stroke which resulted in the formation of a non-letter (as described earlier in copying the letter G). It is unclear from the reported data whether these errors are ever seen in the experiments with normals. In addition, in the delayed feedback paradigm, handwriting was only disturbed when the subjects continuously attended to the visual feedback display. In contrast, R.B.'s performance was not altered when he closed his eyes. The most cogent discrepancy is found in copying and tracing tasks. In the delayed feedback studies, copying and tracing were also markedly disrupted. R.B.'s writing was clearly improved by providing a model to copy, and his tracing performance was facile. The ability to copy or trace requires visual motor integration which is at least as great as that required for handwriting. Therefore, the relative preservation of copying, and the intact tracing performance in the face of markedly disturbed handwriting strongly argues for a distinct apraxic component to R.B.'s agraphia rather than a purely perceptual etiology.

1.4. Discussion

This analysis of a case of acquired agraphia is intended to illustrate the manner in which this group of patients may contribute to our understanding of the motor control of handwriting. Unfortunately, there are several factors which have limited this approach. Apraxic agraphia is usually due to a dominant hemisphere lesion; thus it is usually associated with language disturbance including written spelling errors. Frequently, the writing is too agraphic to be legible, the patient producing only an illegible scrawl. However, even when the degree of agraphia could have permitted a detailed error analysis, this has not been obtained. Existing reports of apraxic agraphia therefore do not provide much information about handwriting control from an information processing standpoint, although they can be useful in determining the anatomical localization of handwriting control. Overall, the reported
cases of apraxic agraphia indicate that handwriting can be disrupted without affecting other motor skills. A detailed review of these cases can be found elsewhere (Margolin and Binder in press; Margolin submitted).

2. Handwriting disorders in Parkinsonism

Parkinson's disease is predominantly a motor disorder, the symptoms relevant to this discussion being a paucity and slowing of voluntary movements. These patients usually have an increased reaction time and movement time on motor tasks but, in contrast to apraxics, the form and sequence of their movements are normal (Pirozzolo et al. 1982). The symptoms are believed to be due to a decrease in the neurotransmitter dopamine which innervates a group of motor nuclei known as the basal ganglia. The basal ganglia constitute an important part of the subcortical or extrapyramidal motor system.

Parkinsonism affects all voluntary movements, but handwriting appears to be particularly vulnerable, in that it is frequently the first manifestation of this disease (McLennan et al. 1972). There is no disturbance of higher order handwriting programming such as increased spelling or stroke errors. Instead, handwriting difficulties include slowing, loss of accuracy, and diminution of letter size, the last characteristic accounting for the use of the term micrographia. Micrographia may include progressive diminution of letter size as the patient continues to write in addition to overall smallness. The incidence of micrographia in Parkinsonism has been estimated at 10–15% (McLennan et al. 1972) but this was based on changes apparent to the naked eye. If handwriting is indeed sensitive to disturbances of the extrapyramidal motor system than a quantitative analysis of writing could serve as a useful tool in evaluating diseases which affect this system and provide insights into the dynamics behind these handwriting changes.

According to the force impulse model of handwriting (Wing 1978), changes in letter height can be produced by changes in applied force (amplitude or duration) or timing (interpulse interval). Within this model, micrographia could be caused by a primary change in force (which would keep movement time relatively constant) or a decrease in interpulse intervals (which would result in a decreased movement time). The analysis used in this study involved the measurement of the height
and movement duration of individual letter strokes, thus providing the necessary information to determine the role of force vs. timing changes with respect to micrographia.

In designing this experiment attention was paid to two common methodological problems encountered in research with Parkinsonian patients. Selection of an appropriate control group is problematic in that Parkinsonian patients often have other deficits in addition to the motor disturbance, such as dementia (Pirozzolo et al. 1982). The second problem is related to the medication which most Parkinsonian patients are taking, since, in most research the level of anti-Parkinsonian medication is not systematically controlled. In the present study, and in other research in our lab (Posner et al. submitted) these two problems were avoided by using the patients as their own controls and testing them during two different states of medication.

2.1. Method

Five patients, between the ages of 60 and 68 (M = 63.6), with idiopathic Parkinson's disease of varying severity were studied. Patients were not found to be demented on neurologic examination or suffering from any other neurologic disorders. Only one patient was severely micrographic, and at least two patients were not micrographic to the naked eye. One subject had no observable tremor, two had mild tremors and two had moderate tremors. All were taking anti-Parkinsonian medication. There were four males and one female, and all were right handed. A Talos graphic writing tablet (31.75 × 31.75 cm) equipped with a moveable stylus was interfaced with a digital computer (LSI-11/02). Movements were recorded at the rate of 8.3 msec per coordinate pair at an accuracy of 0.25 mm.

Patients adjusted the angle of the tablet to attain a comfortable writing position. They were allowed to watch their handwriting without any manipulation of visual feedback. On completion of a line of writing a visual display of the computer-sampled trace was provided. Subjects performed handwriting tasks involving two letters, lower case cursive e and l, and two contexts, repetitive single letter or repetitive alternating letters. Thus, there were three writing tasks: repetitive e, repetitive l, and repetitive el. Ten trials of each of the three tasks were obtained in counterbalanced fashion under two conditions. In the first condition patients were relatively undermedicated (UM). This was accomplished
by having them skip at least one dose of their medication. In the second condition they were relatively well-medicated (WM), testing usually beginning 45 to 60 minutes after their last dose of medication. Both conditions were verified by the patient's subjective report and examination by a neurologist (D.I.M.). Examination findings were quantified on an ordinal rating scale, with a maximum score of 44 representing the most severe Parkinsonian signs. The mean score in the UM condition was 3.6 points higher than in the WM condition.

For purposes of analysis, the data was retrieved from the computer and the vertical component of writing was displayed as a function of time. The height and duration of the downstroke of the e and I was then computed. An example of the display used is presented in fig. 7. If the curve at a maximum of a letter was flat over more than one time unit the last point was taken. If it was flat at a minimum the first point was taken. In this way the narrowest boundaries of the downstroke were consistently identified.

2.2. Results

Both elements of micrographia; an overall decrease in letter height (vertical extent of downstroke), and a progressive decrease in letter

Fig. 7. Top left = display of handwriting sample as vertical (0.22 cm/division) and horizontal (0.82 cm/division) components. Bottom left = display of vertical component only (0.22 cm/division) as a function of time (1 sec/division).
Right = magnified display of first 40% of sample shown on left. (Top plot: y axis = 0.13 cm/division, x axis = 0.31 cm/division. Bottom plot: y axis = 0.13 cm/division, x axis = 0.41 sec/division).
height within a line of writing, were documented. The mean downstroke size (averaged across letters and repetitions) was 7% smaller in the UM than the WM condition, and in the former condition, stroke size decreased 6.3% from position one to position ten. Associated with the decrease in letter size in the UM condition was a 26% increase in movement time per letter stroke as compared to the WM condition. This combination of a decrease in letter height and increase in movement time is reflected in a drop in velocity in the UM condition of 19%.

The means for height, movement duration, and velocity are presented in table 1, and a sample of these handwriting changes can be seen in fig. 8.

A two-way repeated measure ANOVA was performed on each of the letter/context combinations (i.e., e, I, e( e/), and l(e/l) with drug state (2 levels) × position number in sequence (2 levels) as factors. The effect of these factors on letter height, movement time and velocity is summarized in table 1.

Table 1

<table>
<thead>
<tr>
<th></th>
<th>UM</th>
<th>WM</th>
<th>Med</th>
<th>Pos</th>
<th>Med × Pos</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>e_1</td>
<td>e_{10}</td>
<td>e_1</td>
<td>e_{10}</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>0.38</td>
<td>0.36</td>
<td>0.39</td>
<td>0.39</td>
<td>*</td>
</tr>
<tr>
<td>Duration (ms)</td>
<td>274</td>
<td>241</td>
<td>249</td>
<td>224</td>
<td>*</td>
</tr>
<tr>
<td>Velocity (cm/sec)</td>
<td>1.58</td>
<td>1.50</td>
<td>1.95</td>
<td>1.86</td>
<td>*</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>l_1</td>
<td>l_{10}</td>
<td>l_1</td>
<td>l_{10}</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>0.73</td>
<td>0.69</td>
<td>0.76</td>
<td>0.76</td>
<td>*</td>
</tr>
<tr>
<td>Duration (ms)</td>
<td>299</td>
<td>340</td>
<td>232</td>
<td>224</td>
<td>*</td>
</tr>
<tr>
<td>Velocity (cm/sec)</td>
<td>2.93</td>
<td>2.61</td>
<td>3.31</td>
<td>3.55</td>
<td>*</td>
</tr>
</tbody>
</table>

Note: An asterisk indicates a significant F value.
In order to assess the effect of context on velocity a three-way repeated measures ANOVA was performed for the letters e and l using drug state (2 levels) × position number in sequence (2 levels) × context (2 levels) as factors. The main effects of position \( [F(1,31) = 15.42, p < 0.01] \), drug \( [F(1,31) = 6.04, p < 0.05] \) and context \( [F(1,31) = 10.29, p < 0.01] \) were significant for the letter e. Only the main effect of
drug was significant for the letter \( l \) \( [F(1,31) = 24.01, p < 0.01] \). There was a significant interaction term of position \( \times \) context for the letter \( e \) \( [F(1,31) = 4.36, p < 0.05] \).

Inspection of the data reveals that \( e \) in the repetitive \( e \) context was written with higher velocity than in the \( el \) context, and that the decrease in velocity from position one to ten was greater for the \( e \) in the \( el \) context. This is consistent with data from normals which indicates that the context in which letters are written influences the writing velocity (Greer and Green 1982).

2.3. Discussion

This quantitative analysis of handwriting in Parkinsonian patients is able to detect changes in letter height and movement time per letter which are related to the level of anti-Parkinsonian medication and the amount of preceding writing. Velocity of letter formation is the most sensitive measure of these changes since it incorporates both the drop in letter height and the increase in movement duration. This appears to be a relatively sensitive analysis since patients had only missed a single dose of medication, the average clinical difference between the UM and WM conditions was relatively small, and at least two patients were not micrographic to the naked eye.

In preliminary studies, we have collected data on two Parkinsonian patients with predominantly unilateral Parkinsonism (two females, age 44 and 69). In these patients, the comparison was between the more-Parkinsonian and the less-Parkinsonian hand. In both cases the dominant right hand was also the Parkinsonian hand. In both patients the more-Parkinsonian hand wrote smaller and with an increased movement time, and these deficits became more pronounced with continued writing. Thus, the right vs. left hand differences paralleled the UM vs. WM differences emphasizing that these two paradigms can provide complementary data in research with Parkinsonian patients.

As mentioned earlier, the primary process underlying micrographia could have been a diminution in effective force or a decrease in interpulse interval. Since micrographia was found to be associated with an increase in movement time, it is clear that a diminution in effective force is the primary underlying process. If a decrease in the interpulse interval were the primary process, then the movement time would have decreased. The observed increase in movement time is presumably an
attempt to compensate for the underlying force diminution. The increase in movement time is not adequate to compensate for the loss of force, however, and there is a decrease in letter size. As writing continues the force diminishes progressively, and the micrographia becomes more marked. This explanation of micrographia is consistent with the clinical observation that Parkinsonian patients have difficulty initiating and maintaining adequate force to execute movements (Marsden 1982).

Our data do not allow us to determine whether the decrease in effective force was due to diminished force impulse amplitudes or decreased duration of force impulses. Data from EMG studies, however, indicate that both of these factors contribute to the effective force diminution in Parkinsonian patients (Petajan and Jarcho 1975; Hallett and Khoshbin 1980).

The dynamics underlying the letter height changes in these Parkinsonian patients is distinctly different from the strategy displayed by normals when intentionally changing overall size of writing. When writing a small $e$ or $l$, normals have a shorter movement time than when writing a large $e$ or $l$, but velocity is relatively constant. The combination of decreased velocity and increased movement time when writing smaller, which was seen in the Parkinsonian patients, is never encountered in normals (Wing 1980). This discrepancy between Parkinsonian patients and normals support the interpretation that micrographia represents a disturbance in motor control rather than an intentional process.

Both the Hollerbach (1981) and Vredenbregt and Koster (1971) studies of handwriting are compatible with the finding that micrographia is due to force diminution. In the Hollerbach model, letter formation is independent upon the action of two separate oscillators. Amplitude modulation in a vertical oscillator produces letter height changes and a horizontal oscillator controls letter width. Letter shape is determined by the coupling of the oscillators. Diminution of force amplitude in the vertical direction would produce the height changes seen in the micrographic patients. With the methodology utilized in this study, it is also possible to quantify changes in the width of letters formed by Parkinsonian patients which are apparent by gross inspection (figs. 8 and 9). It would then be possible to determine the correlation between changes in the vertical and horizontal components of letters.
Fig. 9. A: Repetitive e interrupted by tremor [star] (top plot: y axis = 0.11 cm/division, x axis = 0.28 cm/division, bottom plot: y axis = 0.11 cm/division, x axis = 1.24 sec/division).
B: Magnification of tremor and the two preceding letters. The beginning of tremor is noted in the two e(s) preceding the fully developed tremor (Top plot: y axis = 0.11 cm/division, x axis = 0.09 cm/division. Bottom plot: y axis = 0.11 cm/division, x axis = 0.415 sec/division).

Vredenbregt and Koster (1971) have experimented with a mechanical handwriting simulator which utilized two pairs of orthogonally mounted motors to produce letter shapes. This simulation was instrumental in the development of a model of handwriting which postulates that letters are formed by discrete bursts of muscle activity in appropriate muscles (Wing 1978). This model is compatible with our data in that it predicts inadequate muscle force would result in alteration of letter size and shape.

Micrographia is not necessarily associated with tremor (McLennan et al. 1972), but in one of the patients we studied, tremor was clearly contributing to the deterioration in handwriting. Fig. 9 illustrates a line of e(s) written by this patient while undermedicated. The patient's handwriting was interrupted by his tremor after the eighth e, and a 5 hertz oscillation is recorded at this point. Prior to this point there was no definite tremor observable. A magnified view of this portion of the record (fig. 9b), however, reveals the beginning of tremor in two e(s) before it was evident to the naked eye. The tremor oscillations are superimposed on the e without altering the basic upstroke and downstroke, but the vertical oscillations do add to the overall movement time.

This method of analyzing handwriting appears to be a sensitive indicator of tremor. The development of tremor during a movement,
seen in the preceding example, documents the clinical observation that while Parkinsonian rest tremor may initially be attenuated by movement it often returns during the movement.

The alterations in handwriting seen in these Parkinsonian patients appear to be due primarily to changes in force with secondary changes in timing, and in some subjects, to the effect of tremor. With respect to the model of handwriting control presented earlier, these changes represent problems executing an intact graphic motor plan. This is consistent with a current view that the basal ganglia are not involved in the highest levels of motor planning (Marsden 1982).

3. Overview

In this paper we have demonstrated the manner in which acquired disorders of handwriting can contribute to our understanding of the functional and anatomical correlates of the motor control of handwriting.

In a case of acquired agraphia due to a neocortical lesion there were marked errors in letter formation. An analysis of these errors revealed that there were two separable deficits underlying the agraphia; a deficit at the level of the graphic motor pattern (apraxic agraphia), and alteration in sensory feedback due to perceptual deficits.

In contrast, Parkinsonian patients with micrographia were able to access an intact graphic motor pattern but could not generate the proper neuromuscular activity to produce a letter of adequate size. The subcortical structures which are involved in this disease are apparently instrumental in modulating the magnitude of movements [2].

Data from acquired handwriting disorders can be synthesized with knowledge pertaining to the motor control of handwriting gained from experiments with normals. In this way an understanding of the discrete subcomponents of motor control and their anatomical substrates is emerging.

[2] Ideally one would like to have compared the two types of patients on the same tasks. Unfortunately, the computerized handwriting analysis was not available when R.B. was under study.
References


