

Measurement and Analysis of Single and Multiple Finger Tapping in Normal and Parkinsonian Subjects

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A four-finger tapping sensor and associated software have been developed for the investigation of timing and rhythm performance and mechanisms in normal subjects and their disruption in neurological disorders. The tapping sensor comprises four electronic touch pads and pacing lights. A personal computer (PC) is used to control visual and auditory pacing, record the time and pad of each tap, and carry out several performance analyses including graphing, phase-space plots, calculation of spectra and autocorrelations, filtering and descriptive statistical analysis.

A study was conducted to investigate disruptions of timing and rhythm in subjects with Parkinson's disease (PD). Seven Parkinsonian and ten control subjects undertook paced and unpaced finger tapping tests. The hastening phenomenon—tapping asynchronously at a speed faster than the pacing—was seen with similar occurrence in both normal and PD subjects and appears to be due to perceptual difficulties. No evidence was seen of an increased variability of tapping at particular frequencies, contrary to previous reports. Festinated tapping, in which subjects cycled between acceleration to near-maximum speed and abrupt slowing down, was seen only in PD subjects. As none of these subjects showed significant hastening, it appears that hastening and festination are unrelated phenomena. Inspection of variations of finger tapping intervals gave no evidence for the presence of deterministic chaos in the control of rhythmic tapping. In speed tests performed with and without a weight attached to the finger, it was found that, for reasons which remain unclear, normal subjects increased their maximum tapping speed with the weight attached.

Finger tapping Hastening Festination Parkinson's disease

INTRODUCTION

Nakamura *et al.* [1] and Nagasaki *et al.* [2,3] investigated finger tapping deficits seen in Parkinson's disease (PD). When subjects were asked to tap their index fingers synchronously with a periodic auditory signal, they observed a tendency for the tapping speed to diverge from the pacing speed and speed up to around 5–6 Hz, independent of the input

signal. This was called the *hastening phenomenon*. They concluded that 'hastened tapping represents an intrinsic oscillation in the central nervous system which might be masked in normal subjects but released in patients with Parkinson's disease. In other words there might exist a random oscillation with mean frequency of 5–6 Hz in the central nervous system which is excited and causes the characteristic disturbance of rhythm formation in Parkinson's disease.' They also reported an increase in variability of finger tapping intervals at frequencies around 2.5 and 5 Hz. They considered that hastening was related to this increased variability in that 'the error of response became so large that some patients could no longer maintain the synchronized response and showed a hastened response due to this intrinsic oscillation.'

Hastening appears to have similarities to *festination*, an involuntary tendency to accelerate repetitive movements such as walking and speech. Although

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festination appears to be a frequently observed feature of Parkinson's disease [4], there appears to be little in the research literature on this phenomenon. Narabayashi and Nakumara [5] associated festination with freezing, 'a specific phenomenon characterized by difficulty in starting or continuing repetitive movements such as gait, speech and handwriting. Freezing is also observed *as a result of* festination in these movements.' Festinated gait is the most commonly reported type of festination and was thought to be due to the patient 'falling forward' to walk due to the stooped patient's pursuit of their centre of gravity. However, this assumption may be questioned as it can occur without any anteroflexion of the spine [4].

There is a growing body of evidence which indicates that neurons are complex non-linear devices, capable of generating chaotic behaviour [6–9]. The chaotic behaviour of neurons may manifest itself in periodic activities of the body, such as the heartbeat, which has been found to exhibit chaotic behaviour including 'strange-like attractors' in the phase space plots, $1/f$ spectra, and fractal structures [10]. It is possible that other activities involving the human body clock, such as finger tapping, could also exhibit chaos.

Jones *et al.* [11] have developed a comprehensive PC-based sensory–motor test battery for quantitative assessment of the upper-limb sensory–motor system. The system requires a 386/486 PC, dual high-resolution colour graphic boards and monitors (one for assessor, one for subject), an A/D board, and two input devices—a steering wheel and a floor-mounted joystick. All tests are run and analysed by a program called SMTESTS, which is written in Turbo Pascal, menu-drive, flexible and user-friendly. The sensory–motor tests cover measurement of visuospatial (verbal response only), motor (minimal visual content), and tracking (substantial visual and motor requirements) functions [12,13]. Until recently the tests and input devices focused on proximal arm function with little attention given to the measures of the *distal* arm functions (i.e. wrist and fingers). The only distal test

available was that of grip strength, a gross measure of hand control which gives no indication of finger dexterity, speed and reaction times.

This paper presents a multi-finger tapping sensor and associated software in the SMTESTS system which were developed to help fill some of the needs for measurement of distal function. This is followed by a description of the application of these tests in a study of multi-finger tapping and rhythmic abilities in normal and PD subjects, in which an emphasis has been placed on investigation of the aforementioned phenomena.

APPARATUS

Hardware

An electronic touchpad was constructed with four stainless steel touch plates, arranged in a fan shape to cater for hands of different sizes, and a fifth plate on which the hand rests (Fig. 1). Two 5 mm light-emitting diodes (LED) were mounted at the front of each plate. The green *confirmation/feedback* LED illuminates when the plate is touched. The red *pacing* LED is illuminated, as required, under computer control. An oscillator (1.5 kHz) is attached to the fifth plate which resistively induces a small sinusoidal current ($\approx 2.5 \mu\text{A}$) in the hand. When a finger touches one of the touch plates the induced 1.5 kHz signal on the finger is of sufficient amplitude ($> 1.2 \text{ V}$) to act as a logic high at the input of a high impedance CMOS NOR gate and hence toggle the gate's output. Capacitively induced voltages from surrounding mains sources are also present on the subject's hand and body. Although these are often of sufficient amplitude to be detected by the CMOS gate, they were considered unsuitable for the sensor due to their low frequency (50 Hz) which would introduce a temporal error of up to 20 ms. The output from the sensor is thus a 1.5 kHz square-wave (due to the oscillating input) whenever a finger touches a plate. Each of the four channels of the touch sensor were sampled at 1 kHz by a PCL812 data acquisition board in a 386/486 PC.

Software

Software for the multi-finger tapping sensor was written in Turbo Pascal version 6.0 and integrated into the existing SMTESTS software. Extensive use was made of the existing utilities developed for the system such as the graphing and menu libraries, and file handling procedures.

The result is a user-friendly system with the following features:

- Menu-selectable tests including maximum tapping speed, paced and unpaced tests;
- Pop-up tables (with defaults) for setting test parameters;



FIGURE 1. Multi-finger tapping sensor.

TABLE 1. Performance on tapping speed tests (taps/s)

Test	Median (Range)		Difference	%	<i>p</i>
	Parkinsonian	Normal			
Right index	4.7 (2.3–5.4)	5.2 (3.5–6.1)	0.5	10	~
Right index with weight	3.8 (2.6–5.4)	5.7 (4.1–6.4)	1.9	33	**
Right index repeated	4.8 (2.1–5.6)	5.3 (4.3–6.2)	0.5	9	~
Left index	4.8 (2.4–5.1)	4.8 (4.0–6.0)	0.0	0	
Two finger	1.9 (0.9–5.2)	3.6 (2.3–4.8)	1.7	48	~
Four finger	0.8 (0.4–3.8)	1.1 (0.7–3.4)	0.3	28	
Paced maximum	5.7 (3.1–6.0)	5.7 (4.2–6.8)	0.0	0	
Paced max. with weight	4.9 (3.8–6.4)	6.0 (4.5–7.0)	1.1	18	

~*p* < 0.10, **p* < 0.05, ***p* < 0.01 (1-tailed).

- Automatic storage of all raw data for post-experimental retrieval and analysis at a later stage;
- Several signal processing and analysis tools including artifact removal, filtering, calculation of spectra and autocorrelations, and descriptive statistical analyses;
- Several graphing tools including line graphs, phase-space plots and histograms.

EXPERIMENTAL STUDY

Subjects

The study involved seven Parkinsonian subjects, six males and one female with mean age of 66.5 yr, and ten control subjects, six males and four females with mean age 67.5 yr. The PD subjects were assessed as being within grades I to III on the Hoehn–Yahr severity scale [14], had no 'on-off', no dyskinesia, and had reasonably intact mentation, and remained on their usual anti-Parkinsonian medication throughout the study. All subjects were right handed. One PD subject was deaf and could not carry out the auditory paced tests.

Procedures

Three sets of tapping experiments were performed: maximum speed, paced and unpaced. The speed tests were performed for 10 s (from the first tap) with the right index finger, then with an 84 g weight attached to the finger and again without the weight, then with the left index finger. These were followed by two and four finger speed tests. During the paced tests, 30 visual and auditory pacing stimuli were given via the red LED and a 400 Hz tone, generated by the computer speaker, respectively. Tests were performed at frequencies from 0.8 Hz up to the subject's maximum tapping frequency in increments of 0.2 Hz with the right index finger, the left index finger, the right index finger with a weight attached, and finally the right index finger with visual pacing only.

The unpaced test was performed with the right index finger only. This began with the subjects tapping in time with a sequence of 20 pacing signals at 2.5 Hz, after which the pacing stopped and they continued to tap for 5 min at what they considered was the same rate. The number of missed and double taps, relative

to the number of taps in the test, was calculated as an *index of incoordination*. A missed tap was defined as an interval >1.5 times the mean tap interval and a double tap an interval < 0.75 times the mean tap interval.

The speed, paced, and unpaced tests were repeated with two fingers (alternating index and middle) and four fingers (strumming from little to index fingers).

Statistical comparisons were made using the non-parametric Mann–Whitney and Wilcoxon tests.

To enable the detection of deterministic chaos in the control of finger tapping, a comparison was made of phase-space plots unpaced finger tapping intervals with known chaotic and random signals. The chaotic signals were generated from the logistic equation and the Rossler system. Random intervals were obtained by generating gaussian-distributed random numbers and scaling them to approximately the same amplitude and range as finger tapping intervals. In a phase-space plot each interval in a series is plotted against a previous interval in the same series with the shift between the two intervals being kept constant; a shift of '1' was chosen for this study such that immediately previous intervals were plotted against current intervals. If present, chaotic characteristics show up as a 'near periodic' pattern (i.e. near cyclic but not quite overlapping). Conversely, if the intervals are random there are no patterns in the phase-space plot.

RESULTS

Maximum speed

PD subjects were slower than normal subjects on the single and two finger tests but, with the exception of the weight test, only with marginal significance (Table 1). No difference was found between the right (dominant) and left hands for normal subjects (5.2 vs. 4.8, 7.7%, NS). The maximum speed of the normal subjects was found (by linear regression analysis) to lower with age by an average of 0.21 taps per second per year of age.

When a weight was attached to their index finger the maximum speed of the PD subjects was considerably slower than that of normal subjects. However, whereas PD subjects slowed by 19% (NS) relative

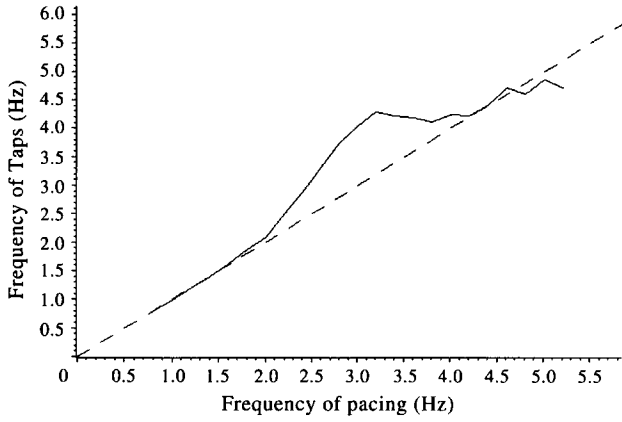


FIGURE 2. An example of hastened finger tapping where a subject suddenly loses synchronism with the visual and auditory pacing.

to their initial single finger speed, normal subjects *increased* their maximum tapping rate by 9% ($p < 0.05$) (Table 1). When repeated without the weight, the speed achieved by both PD and normal subjects was slightly (2%, NS) faster than the initial test in each case, indicating a possible practice effect even on such a simple task. Furthermore, maximum speed achieved after performing many paced tests was considerably higher again in PD and normal subjects (21%, $p < 0.05$ and 19%, $p < 0.01$ respectively). The 'weight' effects observed above were seen again, but to a lesser degree, on comparing maximum speed achieved after performing many paced tests with the weight to that attained without the weight (4.9 vs. 5.7 Hz, NS, for PD subjects; 6.0 vs. 5.7 Hz, NS, for normal subjects).

Hastening

Plots of tapping frequency vs. pacing frequency for paced tests showed that the hastening phenomenon occurred in PD and normal subjects; that is, finger tapping lost synchronism with the pacing signal and sped up, in some cases to near maximum speed (Fig. 2).

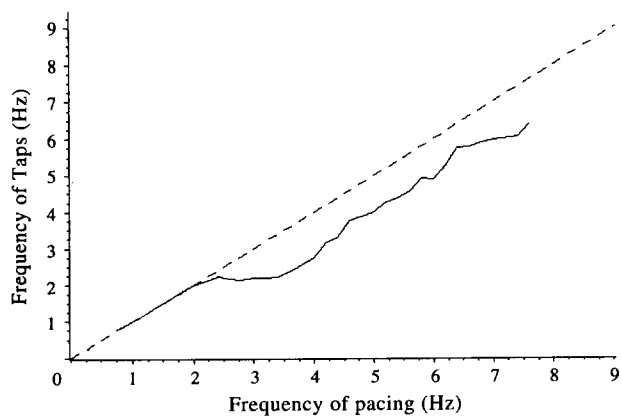


FIGURE 3. An example of finger tapping that slowed down on losing synchronism with the visual (only) pacing.

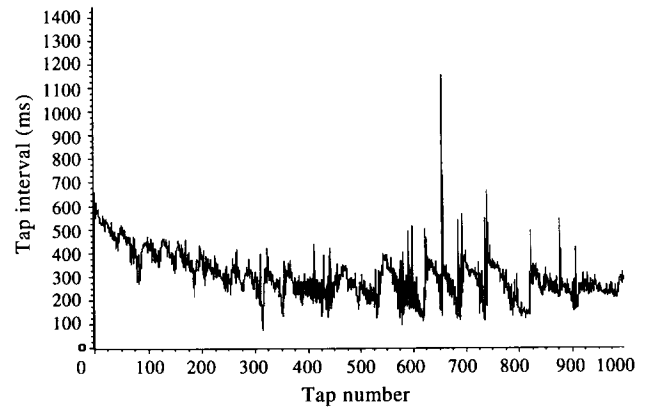


FIGURE 4. An example of festinated (accelerated) tapping during an unpaced test in a subject with Parkinson's disease. Note the cycles of gradual acceleration followed by abrupt slowing down.

Several observations were noted regarding the hastening phenomenon:

- Hastening was not restricted to, nor occurred more frequently in, Parkinson's disease. At least one instance of hastening (out of six series of paced tests) occurred in 5/7 (71%) of PD and 7/10 (70%) of normal subjects.
- There was a strong tendency for hastened tapping to reach a fixed *hastened frequency* irrespective of pacing frequency. A high correlation was found between hastened and maximum frequencies (taken from the highest frequency reached during paced tests rather than the initial speed tests) for both PD subjects ($r = 0.95$) and normal subjects ($r = 0.99$) although the two frequencies were different (4.6 vs. 5.0, $p < 0.01$ for PD subjects; 4.2 vs. 4.4 Hz, $p < 0.005$ for normal subjects). The median frequency for normal subjects was lower than that for PD subjects as the latter could not perform the multi-finger tests (which have lower medians than single finger tests) and were unable to synchronize with the pacing even at the slowest frequencies. In contrast four normal subjects

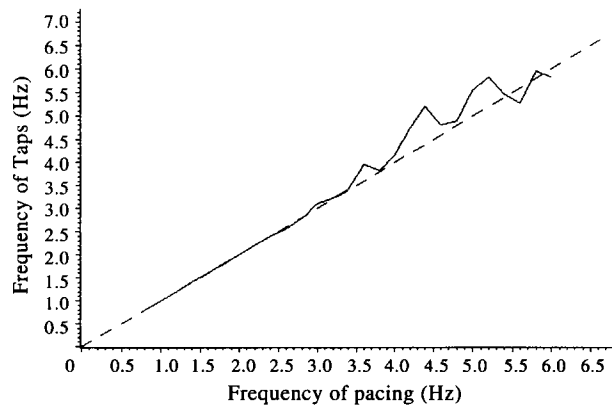


FIGURE 5. Tapping frequency vs. paced frequency for the same PD subject as in Figure 4. Note the lack of hastening despite this subject having exhibited marked festination.

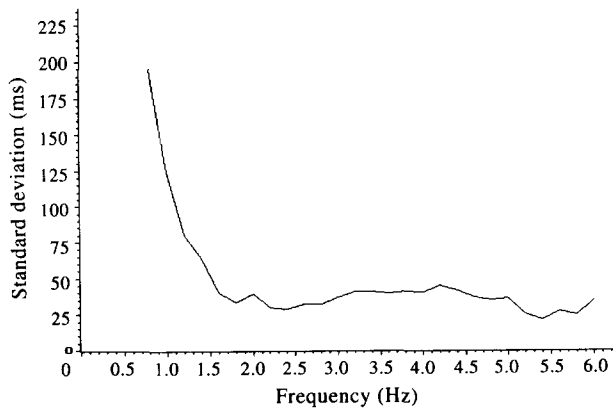


FIGURE 6. An average of 5 standard deviations vs. frequency plots for PD subjects.

showed hastening on two-finger tests and one on the four-finger test.

- The frequency at which PD subjects lost synchronism (the frequency of divergence) was considerably lower than that of normal subjects (2.6 vs. 4.6 Hz, 43%, $p = 0.07$). Some subjects were able to maintain synchronism up to their maximum tapping speed, in which case their maximum speed was used in calculating the average frequency of divergence for the group.
- Visual pacing was more difficult than combined auditory/visual pacing. This was best seen by the lower frequency of divergence with visual pacing (1.0 vs. 2.6 Hz for PD subjects; 1.9 vs. 4.6 Hz for normal subjects). Furthermore, when subjects lost synchronism with the visual pacing, 80% of PD and 67% of normal subjects initially slowed down rather than sped up as was seen in all cases of divergence on auditory/visual pacing (Fig. 3).

Festination

In the single finger unpaced tests, three out of seven PD subjects showed a tendency to accelerate almost uncontrollably towards their maximum speed after initial pacing had ceased. Two of these subjects were able to perceive that they had sped up and were able to

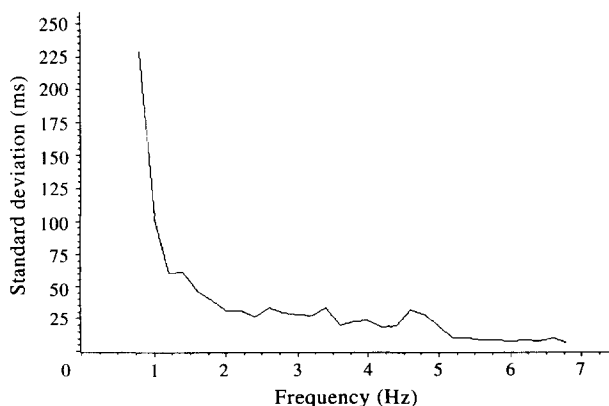


FIGURE 7. An average of 10 standard deviations vs. frequency plots for normal subjects.

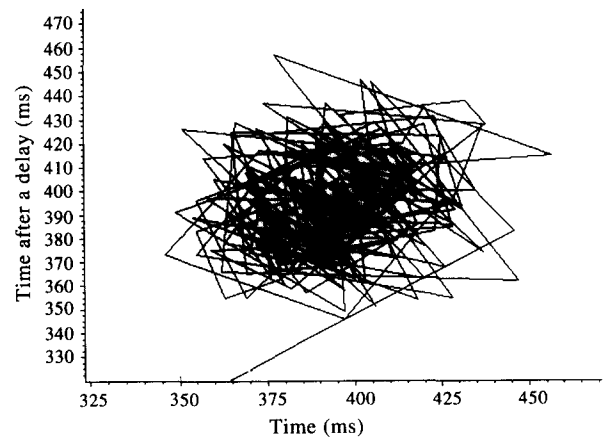


FIGURE 8. A typical phase-space plot of unpaced finger tapping intervals for both PD and normal subjects.

abruptly slow down, although this was soon followed by another burst of uncontrolled acceleration (Fig. 4). The third subject initially showed the same pattern of cyclic acceleration and slowing down but this regressed to staying at maximum speed for the rest of the test. Despite their tendency to festinate none of these three subjects showed any marked hastening during the paced tests (e.g. see Fig. 5).

Tapping variability

Plots of standard deviation vs. frequency of tapping were inspected qualitatively for major increases in tapping variability at and around specific frequencies (i.e. shown by larger than background variations and over more than one consecutive frequency). No definitive increases in tapping variability at specific pacing frequencies were seen in any subjects although several could be considered to have marginal increases. That is, 4/27 (15%) of PD single finger tests (right, left, weight and visual pacing) compared to 8/40 (20%) of normal single finger cases showed equivocal increases in standard deviation. Average standard deviations at each frequency were calculated for five PD subjects (Fig. 6) and ten normal subjects (Fig. 7). The averaging out of much experimental

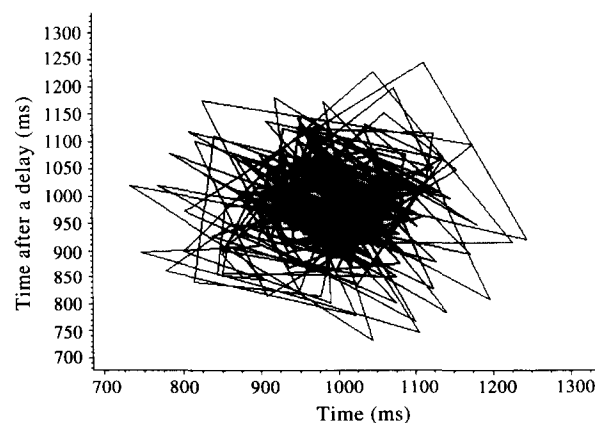


FIGURE 9. Phase-space plot of randomly generated intervals.

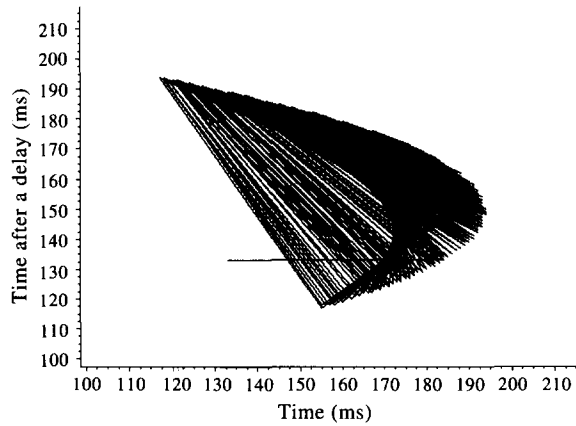


FIGURE 10. Phase-space plot of intervals generated from the logistic map.

fluctuation in individual subject's graphs confirmed a lack of variability peaks at specific frequencies.

Multi-finger coordination

Both groups, but particularly PD subjects, found two- and four-finger paced tests difficult to perform. This was reflected, for example, by 5/7 (71%) PD subjects being unable to perform the four finger tests compared to 1/10 (10%) normal subjects.

For unpaced tests, the index of incoordination was worse in the PD subjects than the normal group for both two fingers (34.5 vs. 2.5%, $p < 0.01$) and four fingers (22.5 vs. 4.5%, $p < 0.05$). There was no difference between the groups in single finger coordination.

Chaos in finger tapping

Phase-space plots of unpaced finger tapping intervals (Fig. 8) can be seen to have very similar characteristics to the phase-space plots of stochastically random intervals (Fig. 9). Similarities between tapping and random intervals were also seen in raw data plots and spectral analysis. Finger tapping intervals showed none of the distinctive near periodic characteristics of deterministic chaos as seen in the

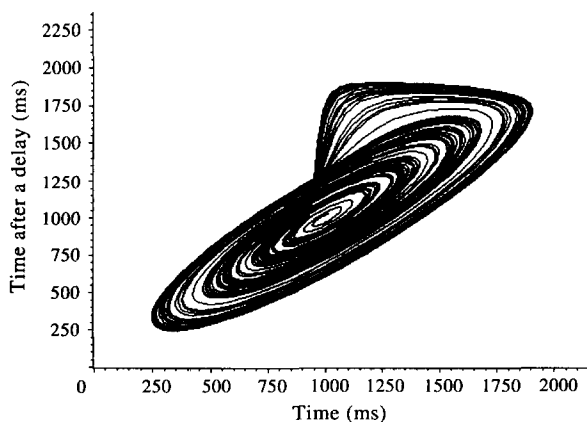


FIGURE 11. Phase-space plot of intervals generated from the Rossler system.

phase-space plots of two well known chaotic systems (Figs 10 and 11). Thus, there was no evidence of, at least, lower dimensional chaos and it would seem that finger tapping intervals are governed by a random process.

DISCUSSION

The weight effect

Perhaps one of the most unexpected findings from this study was the increase in maximum tapping speed seen in normal subjects when an 84 g weight was attached to their tapping finger. The mechanism for this phenomenon is unknown. Although males are stronger than females [15], finger strength does not appear to be a factor as there was no difference in tapping rate between males and females with (4.64 vs. 5.53, 18%, NS) or without the weight attached (5.58 vs. 5.67, 2%, NS).

A possible explanation for faster speed could be an increase in finger stiffness. By modelling the finger as a mass-spring system [16], the natural oscillation frequency of the finger is given by:

$$f_{\text{natural}} = \sqrt{(K_0/m)},$$

where K_0 = spring stiffness, m = mass.

The addition of a small weight to the finger might cause an increase in the finger's stiffness (K_0) due to either increased co-contraction and/or to increased stretch-reflex controlled resistance to movement [17]. If the increase in the muscle stiffness is greater than the increase in mass (m), this would cause an increase in finger natural oscillation frequency. The mass-spring model is, however, a gross simplification of the biomechanics and neuromuscular control of finger movement and, at most, may only give a clue to the mechanism behind the finger weight phenomenon.

Just as the mechanism for weight induced increases in tapping rate in normal subjects is unknown, the basis for the rate decreasing in PD subjects is also uncertain. It can be conjectured that, in addition to a generalized increase in stiffness (i.e. 'lead pipe' rigidity), PD subjects have diminished reflex control of joint and muscle stiffness, thus being unable to compensate for increased finger mass as may occur in normal subjects.

Hastening

Our results on hastening differ from Nakumara *et al.* [1] and Nagasaki *et al.* [2,3] in two fundamental ways. Firstly, they report having observed hastening only in some PD subjects and that 'the other patients and normals could respond synchronously up to 5-7 Hz'. In contrast, our results showed hastening in normal subjects as frequently as in PD subjects. Secondly, Nagasaki *et al.* [2] claimed that the hastened tapping speed was usually independent of a subject's maximum tapping speed, and possibly at an independent

'intrinsic oscillation' frequency. In contrast, our results show a high correlation between hastened and maximum tapping speeds, with hastened tapping occurring at a rate slightly less than maximum.

The hastening phenomenon may well be caused by a perceptual or sensory-motor integrative mechanism, rather than an intrinsic central nervous system oscillation as proposed by Nakumara *et al.* [1] and Nagasaki *et al.* [2,3]. At lower frequencies, subjects could tap synchronously with pacing and were able to constantly monitor and modify tapping speed in order to maintain synchronism. However, as pacing frequency increased, several subjects reported being aware of being out of synchronism but were uncertain whether to speed up or slow down to correct the error. When synchronism could not be achieved they appeared to aim for a rate which was a combination of the perceived pacing speed and that of the previous test. That is, the test was started at this estimated speed, and from then on minimal use appeared to be made of on-going feedback in the form of pacing. As subjects received no indication of performance, initial estimates of speed are likely to have become more inaccurate as testing proceeded due to cumulative error in estimating the speed of previous tests. Consequently, in some subjects, tapping rate rapidly diverged from pacing speed. To verify this cumulative error hypothesis, further experiments would need to be performed with random rather than uniformly increasing pacing frequencies. Under these circumstances tapping frequency might be expected to vary randomly around pacing frequency rather than being consistently hastened.

The hypothesis that hastening may be due to a perceptual or sensory-motor integrative mechanism implies that there is a speed above which subjects cannot process information quickly enough to maintain synchronism with pacing, that is, cannot make use of continuous feedback of performance. When this limit is exceeded, movements become open-loop in nature, unable to be corrected by feedback, and may hasten or fall behind pacing. This limit appears to vary considerably with complexity of task and between subjects, with some people able to maintain synchronism up to a maximum tapping frequency so no limit is detectable.

Other studies have shown a similar closed-loop/open-loop transition. For example, Neilson *et al.* [18] used random tracking tasks with target bandwidths varying from 0.1 to 3.9 Hz to show a progressive loss of synchronism between target and response between 1 and 2 Hz, which is considerably below the neuromuscular limit of voluntary movement (e.g. see maximum tapping rates in this study). Neilson [19] also carried out kinaesthetic tests in which movement of the left elbow was tracked with the right elbow. Again, the maximum frequency at which tracking was coherent was 2 Hz. They concluded that movements

above 2 Hz must be open-loop and therefore learned or programmed. Loss of synchronism occurred at higher frequencies in the tapping task than in either of these studies (medians of 2.6 Hz for PD subjects and 4.6 Hz for normal subjects). The difference may be due to the repetitive, discrete nature of the tapping task, and larger cortical input to finger control. The lower frequencies at which PD subjects lost synchronism with pacing could reflect visual and/or auditory perception deficit or impaired ability to integrate sensory and motor functions.

The frequency at which subjects lost synchronism with visual pacing was much lower than with combined visual and auditory pacing in both groups (1.9 vs. 4.6 Hz for normal subjects; 1.0 vs. 2.6 for PD subjects). Thus it appears more difficult to synchronize tapping with visual than auditory stimuli (based on the assumption that the auditory stimulus is the important factor in combined pacing). This could reflect longer times required to process visual information and is in keeping with visual reaction times being longer than auditory (e.g. 200 vs. 150 ms [20]).

Subjects tended to fall below the pacing frequency with visual and hasten with auditory stimuli, suggesting under and over estimation of pacing rates respectively. It is unclear whether this is related to the longer delays in processing visual information or reflects a more 'urgent' nature in auditory stimuli.

Festination

The basis for festination is uncertain. However, as it occurs during unpaced activities, it may be related to tasks in which subjects rely on their 'internal clocks' to maintain rhythm. Although superficially similar to hastening, festination appears unrelated, with none of the three subjects who exhibited festination showing significant hastening. That is, during paced tests they were able to control their speed over the entire frequency range without accelerating to some maximum as they did in the unpaced tests.

Most normal and PD subjects sped up to some degree during unpaced tests which could indicate that 'internal clocks' (which may control tapping speed) have a tendency to speed up. This has also been observed in other rhythmical activities. Thus, musicians, particularly if novice, tend to slowly increase their tempo throughout a song. Festination in PD subjects could reflect impaired control over this natural tendency. Paradoxically, this appears in conflict with postulated slowing of basal internal clock rate in PD based on underestimation of time intervals [21]. Two explanations for festination are possible. The first is that the intrinsic resting tremor is responsible for festinated tapping. This is supported by festinated tapping occurring at approximately the same frequency as a typical resting tremor (5 Hz) and appearing to be out of voluntary control. The second is

that we may possess two body clocks, namely: (a) a relatively stable real-time clock used in, say, time estimation tasks; and (b) a phase-locked loop clock whose reference frequency may be adjusted to the rate of some pacing signal. If the latter clock is poorly controlled, as may occur in PD, then in the absence of pacing, there may be an involuntary tendency for a rapid increase in tapping (i.e. festination). The real time clock could, at the same time, remain slow. It must be emphasized, however, that the above mechanisms remain speculative and without experimental support.

Although the PD subjects who exhibited festination had difficulty preventing the speeding up, they were able to revert back to their perceived reference speed after a substantial delay. This difficulty may reflect an extreme form of the difficulty PD subjects have in calling up a new motor program [22] or changing motor set [23] to override and correct for involuntary acceleration. An alternative explanation for the sudden decreases in tapping rate seen superimposed on the general trend towards acceleration (e.g. Fig. 4) is 'freezing'. This is considered unlikely, however, because the deceleration was abrupt and in no case sustained for a period long enough to justify this term. It seems more likely that the sudden decelerations were a voluntary attempt on the part of the patients to return to the intended tapping frequency (which may or may not be the same as that of the initiating pacing frequency of 2.5 Hz). For example, in most instances in Figure 4 the duration of the tap interval during these sudden changes approximated to, and was less than the 400 ms interval of the initiating tap pacing signal.

REFERENCES

1. Nakamura R, Nagasaki H, Narabayashi H. Disturbances in rhythm formation in patients with Parkinson's disease: Part I. Characteristics of tapping response to periodic signals. *Percept. Mot. Skills* 1978; **46**: 63–75.
2. Nagasaki H, Nakamura R, Taniguchi R. Disturbances in rhythm formation in patients with Parkinson's disease: Part II. A forced oscillation model. *Percept. Mot. Skills* 1978; **46**: 79–87.
3. Nagasaki H, Nakamura R. Rhythm formation and its disturbances—a study based upon periodic response of a motor output system. *Hum. Ergonomics* 1982; **11**: 127–142.
4. Selby G. Parkinson's Disease. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*, Vol. 6. Amsterdam: North-Holland Publishing; 1968: 173–211.
5. Narabayashi H, Nakamura R. Clinical neurophysiology of freezing in Parkinsonism. In: Delwaide PJ, Agnoli A, eds. *Clinical Neurophysiology in Parkinsonism*. Amsterdam: Elsevier Science Publishers; 1985: 49–57.
6. Musha T, Katsurai K, Teramachi Y. Fluctuations of human tapping intervals. *IEEE Trans. Biomed. Eng.* 1985; **32**: 568–581.
7. Rapp PE, Zimmermann ID, Albano AM, De Guzman GC, Greenbaun MN, Bashore TR. Dynamics of spontaneous neural activity in the simian motor cortex: The dimension of chaotic neurons. *Phys. Lett. A* 1985; **110**: 335–338.
8. Aihara K, Matsumoto G. Chaotic oscillations and bifurcations in squid giant axons. In: Holden AV, ed. *Chaos*. Manchester: Manchester University Press; 1986.
9. Guevera M, Glass L, Shrier A. Phase locking, period doubling bifurcations, and irregular dynamics in periodically stimulated cardiac cells. *Science* 1981; **214**: 1350–1352.
10. Goldberger AL. Fractal mechanisms in the electrophysiology of the heart. *IEEE Eng. Med. Biol.* 1992; **11**: 47–52.
11. Jones RD, Sharman NB, Watson RW, Muir SR. A PC-based battery of tests for quantitative assessment of upper limb sensory-motor function in brain disorders. *Proc. Int. Conf. IEEE. Eng. Med. Biol. Soc. San Diego, USA*, 1993; **14**: 1477–1478.
12. Jones RD, Donaldson IM. Measurement of sensory-motor integrated function in neurological disorders: three computerized tracking tasks. *Med. Biol. Eng. Comput.* 1986; **24**: 536–540.
13. Jones RD, Donaldson IM, Parkin PJ. Impairment and recovery of ipsilateral sensory-motor function following unilateral cerebral infarction. *Brain* 1989; **112**: 113–132.
14. Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. *Neurology* 1967; **17**: 427–442.
15. Jones RD, Williams LRT, Wells JE. Effects of laterality, sex and age on computerised sensory-motor tests. *J. Hum. Mov. Studies* 1986; **12**: 163–182.
16. Schmidt RA. *Motor Control and Learning*. Champaign: Human Kinetics Publishers; 1982.
17. Houk J, Henneman E. Feedback control of movement and posture. In: Mountcastle VB, ed. *Medical Physiology*, Vol. 2, Twelfth Edn. St Louis: Mosby; 1968: 1681–1696.
18. Neilson PD, Neilson MD, O'Dwyer NJ. What limits high speed tracking performance? *Hum. Mov. Sci.* 1993; **12**: 85–109.
19. Neilson PD. Speed of response or bandwidth of voluntary system controlling elbow position in intact man. *Med. Biol. Eng.* 1972; **10**: 450–459.
20. Poulton EC. Human Manual Control. In: Brooks V, ed. *Handbook of Physiology: The Nervous System*, Vol. 2. Maryland: American Physiology Society; 1981: 1358–1389.
21. Pastor MA, Artieda J, Jahanshahi M, Obeso JA. Time estimation and reproduction is abnormal in Parkinson's disease. *Brain* 1992; **115**: 211–255.
22. Marsden CD. The mysterious motor function of the basal ganglia: The Robert Wartenberg Lecture. *Neurology* 1982; **32**: 514–539.
23. Robertson C, Flowers KA. Motor set in Parkinson's disease. *J. Neurol. Neurosurg. Psychiatry* 1990; **53**: 583–592.