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Measurement of Rigidity in Parkinson's Disease

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Summary: Clinical assessment of rigidity in parkinsonian patients is largely qualitative. The reliability and validity of the assessments are sometimes in doubt. Several "engineering" methods of quantifying rigidity have been described, but none has been adopted into general clinical practice. A possible reason is that these methods differ in crucial aspects from the clinical exam. We therefore tackled the problem by monitoring the clinical exam itself, using small sensors to measure the forces and displacements applied. Limb impedance (Z) was computed using parameter identification methods and compared to raters' verbalized ratings of rigidity based on a 5-point scale: the Unified Parkinson's Disease Rating System. The qualitative and quantitative estimates of impedance covaried over a fourfold range, depending on the forces imposed and the subject's motor set. Raters differed by up to 1 full point in their mean qualitative ratings and sometimes disagreed on whether levodopa reduced rigidity. This was not due to any significant differences in the overall range of rigidity they evoked, but rather to the way they scored this range [the ratio of mean rating to mean impedance (R/Z) varied between raters and subjects]. On the other hand, the R/Z ratio was reproducible over separate sets of ratings and may therefore serve to convert measured impedance into a standardized rating. Our results indicate that the current clinical exam may be too abbreviated to detect the sometimes quite small reductions in rigidity after levodopa. We conclude that a device that conveniently quantifies the clinical assessment of rigidity is now available and will lead to more standardized protocols for rating rigidity in the near future. Key Words: Parkinson's disease—Clinical assessment—Rigidity.

With the rapidly growing understanding of the function and neuropharmacology of the basal ganglia (1–6), new possibilities for treatment of Parkinson's disease (PD) are becoming available, including microsurgery, electrocoagulation, drug therapy, and foetal tissue implants (7–9). However, testing the efficacy of a new treatment is often problematic and usually relies on qualitative clinical ratings of symptoms such as bradykinesia, tremor, and rigidity (10). There are many other signs and symptoms of PD that are recommended for testing in the Unified Parkinson's Disease Rating System (UPDRS) scale (11–13), but it is the triad described above that forms the basis of the standard neurological examination. The direct measurement of nigrostriatal dopamine depletion should, in theory, offer a quantitative global measure of the severity of PD, but this requires expensive facilities, which rules out its use, for example, in large multicentre clinical drug trials. Further, some symptoms of PD, such as tremor and gait disorders, may not be directly related to nigral dopamine cell death (10). Thus, we have sought to improve the standard clinical assessments of PD by quantifying one of the key diagnostic variables: rigidity.

Quantification of rigidity in PD has a long history. Over the years, the techniques and instrumentation have gradually become more refined, but the correlation to clinical assessments of rigidity has never been entirely convincing, as evidenced by the lack of universal acceptance of any one technique. Although rigidity may not be the most disabling symptom of PD, it forms one of the mainstays of clinical diagnosis. A convenient and reliable way of quantifying it would be useful in evaluating drug treatment and neurosurgical techniques. Agate et al. (14) and Webster (15) were among the first to measure rigidity dynamically, using large electromechanical

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devices to impose movement on the elbow joint of PD patients. Good correlations to clinically assessed rigidity were claimed, but the systems were not adopted clinically. Progressively more sophisticated "engineering" approaches were used to measure rigidity (16–20). Our own experience illustrates some of the problems with these methods. For example, in pilot studies, we found that by using an electromagnetic length servo to impose precisely controlled 5–10° elbow displacements in the frequency range of 1–5 Hz, the impedance computed from the monitored force was quite poorly correlated with the ratings of clinicians subsequently examining the same patients (unpublished observations). Improvement of the coupling of the arm to the muscle puller only made matters worse, likely because it constrained the patient still further, compared with the situation in the clinical setting.

In common with other groups recently (21–25), we thus abandoned the rigorous "engineering" approach to rigidity measurement and instead opted to measure the forces and displacements imposed by clinicians in the course of normal clinical examinations. We developed a new force-sensing cuff through which the rater could grip the patient's wrist and impose movements about the elbow. A compliant transducer was used to monitor displacement. As in other recent systems (21,24,25), the basic concept was to measure manually imposed forces and displacements of the patient's extremity. The new feature of our device was that it allowed the arm to be held and moved normally, without arm rests, splints, or cradles. Parameter identification was used to compute elbow stiffness and viscosity from the force and displacement measurements regardless of the speed or extent of movement imposed on the arm (26,27). In this article we describe some initial results using this system. We compared the computed impedances to concomitant clinical ratings on the UPDRS scale. Impedance varied markedly from moment to moment, as did the clinical ratings. The fluctuating nature of parkinsonian rigidity is common clinical knowledge, but surprisingly it has not been described in quantitative terms before, to our knowledge.

METHODS

Procedure

In all, 14 patients with PD participated in this study with informed consent. The procedures were approved by the University of Alberta Hospitals Ethics Committee. Patients arrived in the morning, having withheld their normal drug treatments since the previous evening. They were then put through a standard clinical assessment for PD, including evaluation of tremor, gait, bradykinesia, and rigidity. Three or four clinical raters independently assessed the rigidity of motion about the patients' elbows, using the 5-point UPDRS rigidity scale. The precise wording of the UPDRS scale was reviewed prior to each session:

0: rigidity absent
1: rigidity slight or detectable only when activated by mirror or other movements
2: mild to moderate rigidity
3: marked rigidity, but full range of motion easily achieved
4: severe rigidity, range of motion achieved with difficulty

The rigidity was then measured quantitatively in the most affected elbow, as described herein. Due to the variability in impedance encountered in early trials, the clinical raters verbalized their clinical ratings every few seconds. These ratings were recorded by the experimenter using a rotary switch that gave a voltage output proportional to the clinical rating. This voltage was sampled along with the force and displacement signals as described. We found at the outset that raters preferred to give in-between ratings such as 1+, 2+, 3−, so we provided nine levels on the rotary switch: 0, 0.5, 1, 1.5, . . ., 3.5, 4. The evaluation procedure was repeated 1 h after the patient took his/her levodopa-containing medication.

Hand-Held Force Measurement Device

To measure the force applied by the rater, we developed a novel hand-held force-sensing device that comprised a balanced pair of force sensors. The sensors were air-filled pads, positioned on the anterior and posterior surfaces of the distal forearm just proximal to the wrist crease. The rater gripped the patient's wrist via these sensors (Fig. 1). The sensor pads were connected by tubes to either side of a pressure-sensitive diaphragm (Validyne DP 45-34). When the wrist was gripped but not moved, equal forces were applied to each pad. The pressure on each side of the diaphragm was similar, and the net force signal therefore remained close to zero. However, when the forearm was moved, there was a difference between the forces on each pad, which was registered by the pressure-sensitive diaphragm.

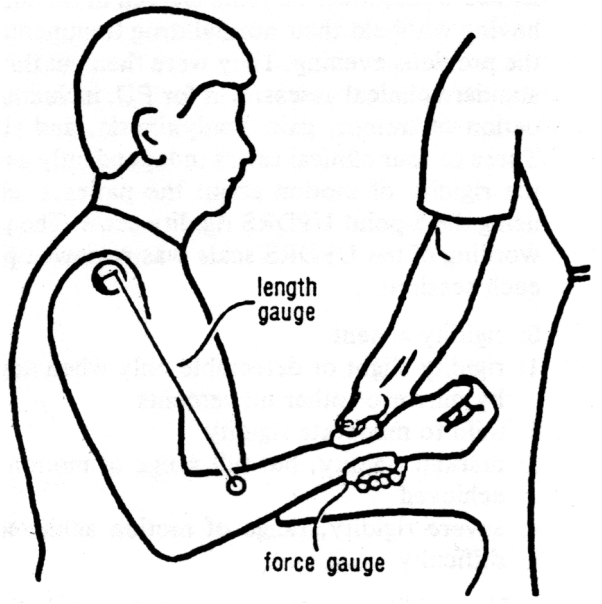

FIG. 1. Transducers used to monitor the force and displacement during a regular clinical evaluation of rigidity. The force sensor was in the form of a cuff, with a pair of air-filled pads through which the rater gripped the subject's wrist. The difference in force between the two pads was monitored, which allowed the resultant flexor or extensor force applied to the forearm to be measured, while the internal forces of the grip were cancelled out. The length transducer consisted of a compliant Silastic rubber band spanning the elbow and deflecting a cantilever force gauge located in the small disc attached to the subject's shoulder.

This difference was proportional to the resultant force applied to the forearm. The experimenter imposed movements in a manner identical to that in a normal clinical exam, except that the movements were made through the force-sensing device. The imposed joint displacement was measured using a compliant displacement gauge attached at one end to the skin over the shoulder and at the other to the skin of the forearm, 5 cm distal to the elbow crease on the lateral side. The gauge consisted of a 300-mm length of Silastic band 1 mm in diameter, attached at the proximal end to a small force gauge, the element of which was a 10 × 3–mm beryllium/copper cantilever instrumented with a pair of Kulite UHP 5000 semiconductor strain gauges. The force gauge registered tension in the Silastic band, which in turn was proportional to its displacement. The gauge therefore registered displacement between the shoulder and forearm attachment points of the sensor. The displacement gauge was linear to within ±2% over the range used and had a frequency response flat to within 3 dB over 0–10 Hz. Although linear displacement was the variable measured, for convenience, calibration was in terms of joint angle.

Joint torque was computed by multiplying the force measured at the wrist by the moment arm to the point of force application. The force and length signals were low-pass-filtered at 30 Hz to avoid aliasing and digitized at 100 Hz for off-line analysis.

Impedance Analysis

The force and displacement measurements were used to compute mechanical impedance Z. A torque–displacement plot was displayed on-line on a storage oscilloscope during the experiment (e.g., Fig. 2, solid lines). The display was not, however,

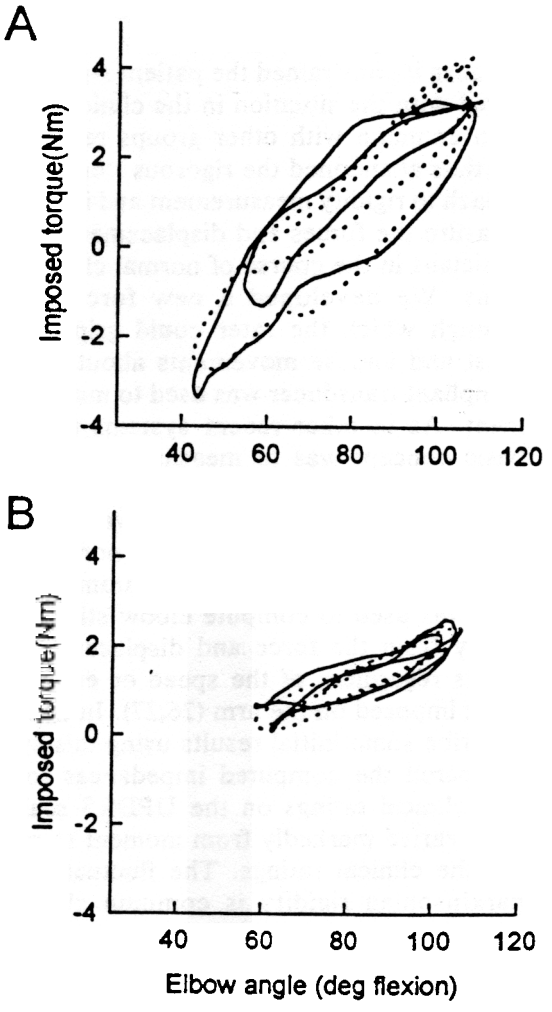

FIG. 2. Torque–displacement plots, each showing two cycles of movement imposed about the elbow in a clinical exam. Solid lines, measured data; dotted lines, torque estimated from displacement using fitted first-order model (see text). A: Slope and width of Lissajous figure were high, indicating that elastic and viscous stiffnesses were high. B: Slope and width of loops were lower, indicating a drop in elastic and viscous stiffnesses. In general, stiffnesses increased at extremes of joint angle (e.g., 40–50° in A).

viewed by the rater or patient. In such plots, an ellipse-shaped Lissajous loop formed with each movement cycle. The width of the loop corresponded to the viscous component of impedance (torque proportional to velocity), and the slope of the loop corresponded to the stiffness component (torque proportional to displacement). The slope and width of superimposed loops gave a rough on-line indication of instantaneous elastic and viscous stiffness, respectively. For example, the elastic stiffness was greater in Fig. 2A than in Fig. 2B. A more accurate method of estimating the components of impedance is to fit a second-order model to the torque and displacement data (26–29). We found that because the movements were of low frequency (<1 Hz) and therefore involved relatively low accelerations, it was unnecessary to estimate the inertial component of the torque response (30). Thus, the relation

$$T = Kx + Bv + C \quad (1)$$

was fitted to the digitized data, where T is the torque, x the displacement, v the velocity (obtained by differentiating x), K the stiffness, B the viscosity, and C any constant offset in torque or length. Standard linear least-squares estimation was used to fit the three parameters K, B, and C to 4 s (400 samples) of data preceding a given time instant (28). This 4-s window was moved along the torque and displacement data, a sample at a time, and the time courses of the elastic and viscous components of impedance were thus obtained. The choice of using data 4 s in the past to compute impedance at a given time was found by trial and error to give the best correlations between the computed and verbalised ratings. The computational method presumably mimics the delays involved in clinically assessing, verbalising, and transferring a rating into the computer.

The net torque sensed by the rater is that due to the elastic stiffness Kx and that due to the viscous stiffness Bv. For sinusoidal inputs of frequency ω, Eq. 1 reduces to the complex function

$$T = (K + jB\omega)\, x + C \quad (2)$$

From this, it is apparent that the viscous stiffness is $B\omega$ (26). Raters typically imposed quasi-sinusoidal movements, so by estimating a mean value of ω and computing $B\omega$, we were able to resolve the elastic and viscous stiffnesses. In the data of Fig. 3, the elastic stiffness was much larger than the viscous

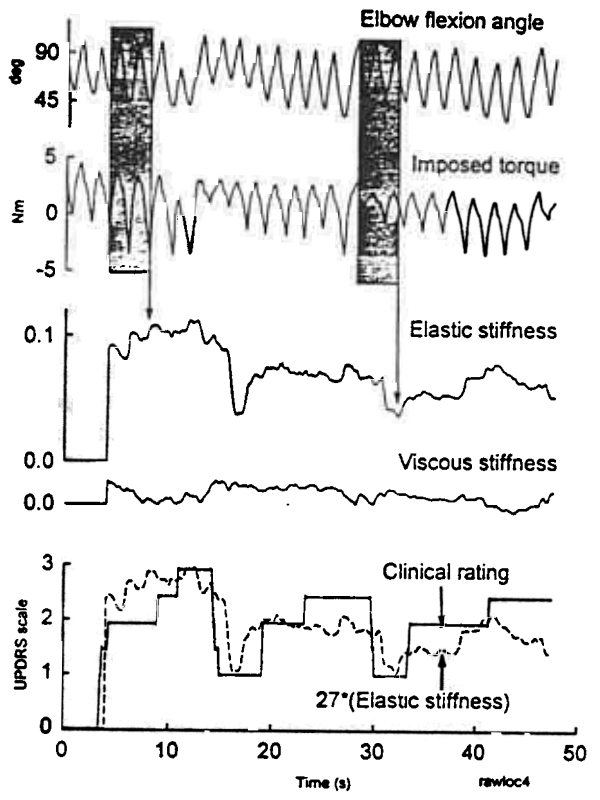

FIG. 3. Illustration of raw data (top), computed elastic and viscous stiffnesses (middle), and a comparison of the computed elastic stiffness with verbalised clinical rigidity rating (bottom). The computed stiffnesses at a given moment were obtained by a least-squares parametric fit of the prior 4 s of data, as represented by the shaded segments. Two examples are highlighted to show a high and low elastic stiffness, respectively, corresponding to large and small excursions in the torque signal. The verbalized clinical rating, which was converted to a voltage and sampled along with the other data, covaried with the computed elastic stiffness. The elastic stiffness plot is reproduced and superimposed as a dashed line, scaled by a factor that corresponds to the slope of the line of best fit between the rating and the stiffness, in this case the factor being 27. UPDRS, Unified Parkinson's Disease Rating System.

stiffness. Some raters moved the arm more rapidly than others, making viscous stiffness more significant. In most cases, the data are therefore presented in terms of the magnitude of the impedance Z (i.e., the vectorial sum of elastic and viscous stiffnesses).

RESULTS

Standard Clinical Assessment

The PD patients studied were at various stages of the disease, ranging from mild to completely wheelchair bound, with a wide range of rigidity. Because

of the way the study evolved, comparable sets of data were obtained in only 7 of the 14 patients we examined. Patients came into the laboratory at 8 or 9 a.m., not having taken their normal medication since the previous day. A brief (~2-min) clinical exam and evaluation of rigidity at wrist and elbow were performed in turn by each of the three or four raters. Rigidity was separately rated in both the mid and full range of motion, because results in the first few patients had alerted us to the likelihood of range dependence. The mean full range ratings at elbow and wrist (1.86 ± 0.96 SD and 1.79 ± 0.86) were significantly larger than the respective midrange ratings (1.01 ± 0.91 and 1.16 ± 0.97; Mann–Whitney rank sum tests, $p < 0.01$). After completion of the premedication clinical assessment and impedance measurements, the patients took their normal morning dose of medication and the assessments were repeated 1 h later. There was a statistically significant decline of 0.49 ± 0.18 after medication across all subjects. However, in individual subjects, the pre- and postmedication difference reached significance in only two of the seven subjects ($p < 0.05$). The rigidity rating of a given patient under identical test conditions varied by up to 2 full points between raters. Furthermore, in nearly all individual cases, raters disagreed on the basic issue of whether rigidity had decreased after drug treatment. The logical and somewhat startling conclusion is that an individual clinician performing a brief clinical exam cannot in general reliably identify a change in rigidity following medication.

Quantification of Rigidity: Impedance Measurement

Immediately after each set of initial clinical assessments, elbow rigidity was again tested, but this time the forces and displacements imposed by the raters were monitored quantitatively by means of the sensors described above. The rater gripped the subject's wrist and moved the forearm in the manner s/he normally employed in a clinical exam. It was apparent from the outset that there were systematic differences in the speed and range favoured by different raters. Whereas one would concentrate on midrange movements, another would deliberately provoke maximal impedance at the extremes of the physiological range. Furthermore, reinforcement manoeuvres, such as patients tapping their knee or squeezing a ball with the contralateral hand, were used to different extents. In the results reported herein, the raters were therefore instructed to test mid and full range of motion separately and to examine the effect of reinforcement in about half of each evaluation period.

As the movements were imposed manually, they were not perfectly sinusoidal (angle and torque traces in Fig. 3 and the pointed ends of the Lissajous figures of Fig. 2). In any case, the interpretation of Lissajous figures is difficult. As discussed in Methods, a more general analytical method is to assume that the muscle can be modelled as a spring damper system and to fit the equation of this system to the displacement and torque data. Provided the model is adequate, no assumptions about the time course of the movement are needed and a good fit results, as exemplified in Fig. 2 (dashed lines). Torque and displacement data were averaged over a moving 4-s window to obtain the time course of elastic and viscous stiffness (Fig. 3; see also Methods).

Continuous Rigidity Assessment

In our initial trials using actuator-imposed sinusoids, there had been a poor correlation between computed impedance and subsequent clinical ratings of rigidity. Indeed, this is what had prompted us to monitor the clinical exam itself. The variability in slopes seen in Fig. 2 and in the modulation of torque excursions in Fig. 3 illustrates that stiffness varied during the clinical exam, changing within seconds. With hindsight, it is common clinical knowledge that rigidity waxes and wanes spontaneously in parkinsonian patients. To compare the quantitative data with the raters' perception of rigidity, we therefore had raters continuously verbalize their qualitative ratings during each exam. Figure 3 (bottom) shows a typical result of such a trial. The time course of these ratings (solid line) closely matched that of the elastic stiffness (dashed line). We fitted a regression line between the ratings and the stiffness to obtain a scaling factor to compare stiffness and rating plots as in Fig. 3 (bottom). The mean error between the two plots was 23% of the mean rating in the example shown. Across all the data, the error between the ratings and the scaled stiffness was 38 ± 20%.

Systematic Differences in Clinical Ratings

We analyzed the data to determine whether there were systematic differences between raters, either in the range of impedance they evoked in the patients or in the clinical ratings they assigned to this range. Figure 4 (top) shows that there was a large range of mean impedance between patients when all

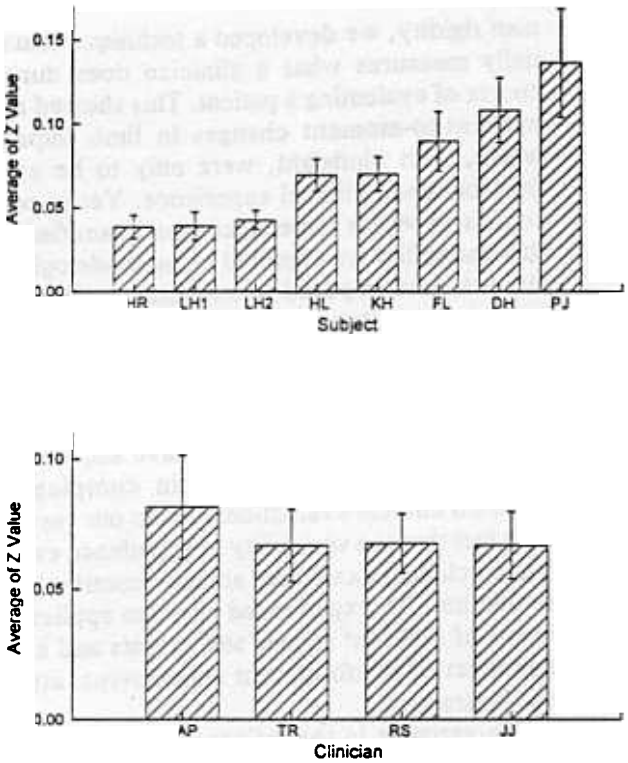

FIG. 4. Top: Mean impedance (*Z:* vectorial sum of elastic and viscous stiffnesses) in different subjects (identified by initials on *x*-axis), averaged across all seven raters and all conditions. There was a fourfold range in impedance between subjects. **Bottom:** Impedance averaged across four subjects and all conditions, raters identified by initials on *x*-axis. There was no statistical difference between raters in the mean and range of stiffnesses they evoked. The rater with the least clinical experience (AP) evoked the largest impedances. Error bars show ±1.96 × SE (i.e., 95% confidence interval).

tests were combined. This was to be expected, given the range of severity of PD in the subject group. On the other hand, the mean impedance evoked by different raters across subjects turned out to be very similar (Fig. 4, bottom). This was interesting in view of the differences between the four raters in their level of experience, the movements they imposed, and the reinforcement techniques they used. TR and JJ are experienced neurologists who routinely evaluate parkinsonian patients in a movement disorders clinic. RS is an experienced physiotherapist who has specialized in evaluating and managing PD for over a decade. AP is a neurophysiologist with biomechanical experience, but limited experience in evaluating patients on the UPDRS scale. To quantify the relationship between the clinical ratings *R* and the impedance *Z*, we computed the slope of the regression line of these two variables and we called this the *R/Z* ratio. The higher the *R/Z* ratio, the greater the qualitative rating of a given "real" impedance. *R/Z* ratios did vary significantly (Student's *t* test, $p < 0.05$), both between subjects (Fig. 5, top) and between raters (Fig. 5, bottom). The between-subject variability indicates that the clinical rating may be influenced by characteristics of the patient other than rigidity (e.g., expectations based on subject's build, clinical profile, etc.). The between-rater variability indicates that the interpretation of the UPDRS scale varies between raters, even when the precise wording has been reviewed shortly beforehand. On the other hand, there was a remarkable consistency in our data regarding the mean *R/Z* ratio across all subjects and clinicians. In Fig. 5, mean *R/Z* was 24.1 ± 8.9. As a separate check on this value, for each subject, we plotted the mean elbow ratings *R* (pre- and postmedication, mid and full range) obtained in the standard clinical assessments de-

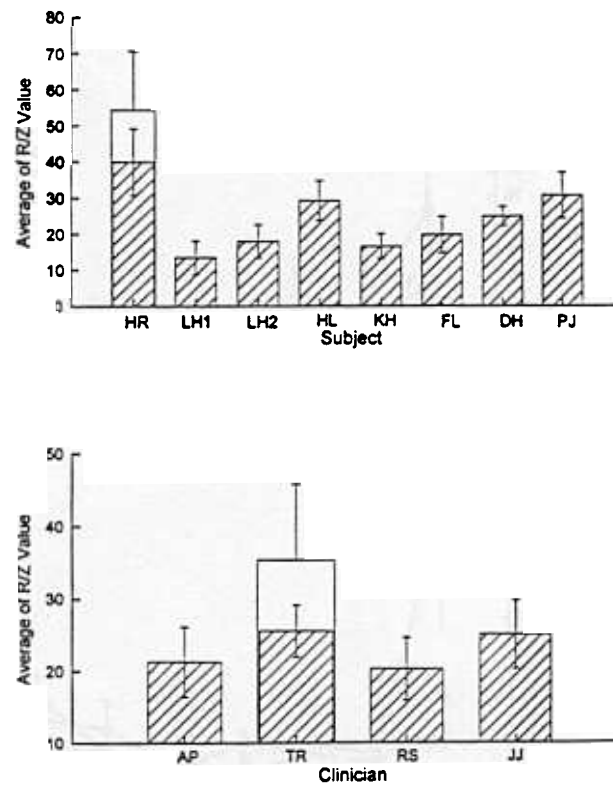

FIG. 5. Ratios of mean verbalized rating to mean computed impedance (*R/Z*). The purpose of these plots was to detect systematic differences in the rating accorded to particular patients (top) or by particular raters (bottom). There were significant differences in both cases (see text for interpretation). In the case of subject HR, one of the raters (TR) was observed to be exerting some force through the web space between his forefinger and thumb. This force was not registered by the force sensor and therefore led to lower *Z* values and higher *R/Z* values (open bars). Error bars show 95% confidence intervals.

scribed earlier against the corresponding mean impedances Z in Fig. 4. The slope of the regression line (i.e., the mean R/Z ratio) was 22.9 ($r^2 = 0.84$). The reproducibility of the R/Z ratio (in our case in the range 23–24) suggests that if it were "calibrated" against the ratings of an internationally recognized group of expert raters, it could be used as a standard conversion factor to transform measured impedances into standardized ratings.

We wondered whether the clinicians' expectation of a lower rigidity after levodopa medication introduced a bias in the qualitative ratings. The data of Fig. 5 (top) were reanalyzed to separate premedication (Fig. 6, top) and postmedication (Fig. 6, bottom) R/Z values. Although a small reduction in postmedication R/Z values is evident, this was not statistically significant in most individual cases.

DISCUSSION

After encountering difficulties with conventional "engineering" approaches to measuring parkinsonian rigidity, we developed a technique that essentially measures what a clinician does during the course of evaluating a patient. This showed up large moment-to-moment changes in limb impedance, which, with hindsight, were only to be expected from common clinical experience. Yet in previous studies in which impedance was quantified, either this variability was ignored or methodological constraints were imposed to minimize it. The tacit assumption was that a given patient has an underlying "true" level of rigidity that is measurable if extraneous sources of variability are minimized. However, the various automated rigidity analyzing techniques that have been tested have succeeded neither in replacing nor even in complementing standard clinical evaluations. From our results, we now feel that the variability in impedance evoked in a good clinical exam may actually contribute to the evaluation. An experienced clinician applies movements of different speeds and extents and explores the effect of reinforcement manoeuvres, attention, and distraction.

The variance in the ratings obtained in the standard clinical assessments led us to the conclusion that in routine examinations a clinician may not be able to judge reliably whether medication has reduced rigidity or not. This is further illustrated in Fig. 7. Both the stiffness curves and the corresponding verbalised rigidity ratings were quite variable. Taking all the curves together, it is clear that postmedication impedance and ratings were indeed slightly reduced, but it is also apparent that a single examination lasting 10–20 s may not suffice to establish this.

We were able to show a close correlation in the time course of impedance and clinically assessed rigidity by recording these two quantities simultaneously. To our knowledge, this has not been attempted with previous methods of impedance estimation. Although the time-varying profiles of impedance and rigidity rating were generally very well correlated and the mean and variance of the impedance evoked by the different raters across patients were similar, there were systematic differences between raters in the qualitative rating of rigidity, as revealed in the R/Z ratios. It could be that clinicians in daily contact with PD patients are more sensitive to the presence of low levels of rigidity than physicians who examine PD patients only occasionally. This would influence their interpretation of the UPDRS scale. Most practising clinicians have only a vague idea of the wording of the UPDRS rigidity

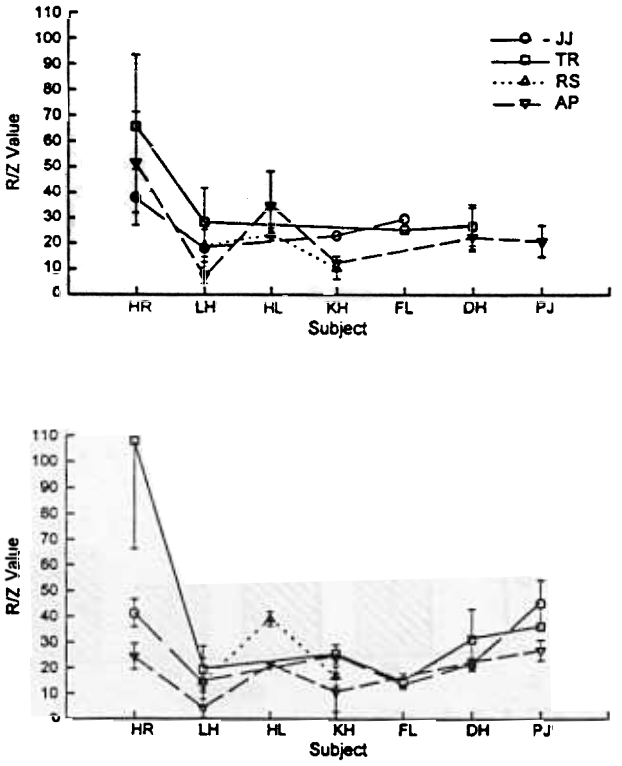

FIG. 6. Same data as in Fig. 5 (top), but with a separation of premedication and postmedication R/Z data. Each rater is indicated by a separate symbol. The purpose in this case was to detect a suspected reduction in R/Z due to the raters' expectation of reduced rigidity after levodopa. The plots indicate that there was indeed a small but statistically insignificant bias of this type. Error bars show 95% confidence intervals.

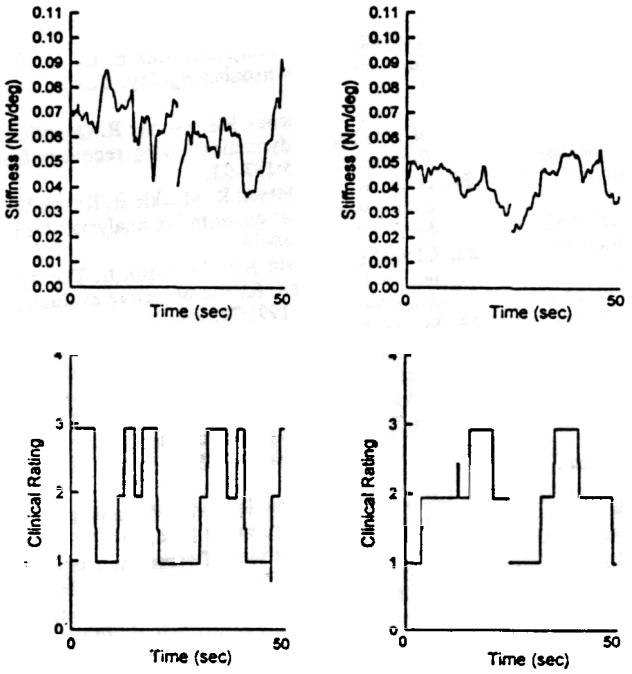

FIG. 7. Time course of computed elastic stiffness (top) and corresponding clinical ratings (bottom) for two pairs of 25-s duration trials, premedication (left) and postmedication (right). These data illustrate how variable stiffness is in a typical clinical exam. Although there is a clear overall reduction in mean postmedication stiffness if the top plots are compared, there are portions of the records in which the difference is unclear. Furthermore, the variability in the verbalised clinical ratings virtually precludes any reliable statement about a postmedication change.

scale. Indeed, some are unsure of whether there are three, four, or five levels in the scale. In retrospect, by providing the raters in our study with written reminders of the UPDRS wording prior to each session, we may have broadened their normal rating range and made them less representative of clinicians at large. In any case, the individual nature of the clinical examination clearly has the potential for creating differences between clinicians and between examinations performed on different days by a given clinician (inter- and intrarater reliability). This is particularly worrisome in multicenter trials with a large number of investigators, each interpreting the UPDRS scale differently.

Limb rigidity is influenced by motor set (31), range and rate of imposed force (19), reinforcement manoeuvres, and random variability, so it is not surprising that the use of electromechanical devices to impose precise movements was not particularly successful in matching clinical ratings. Although the "engineering" approach of minimizing variables offers theoretical advantages over the clinical exam, we would argue that there is little point in pursuing it as a diagnostic tool if, in fact, the narrowing down of test conditions runs contrary to the essence of the clinical exam, which is to evoke a full and representative range of rigidity in a patient. On the other hand, a more standardized protocol of clinical testing, along with a quantification of the test itself, would eliminate some of the intra- and interrater variability we have described, making, for example, multicenter drug trials more reliable. Not all clinicians ask patients to perform reinforcement manoeuvres such as tapping the contralateral knee or clenching the contralateral fist. Some clinicians move the limb back and forth rapidly in the midrange (e.g., at ~1 Hz), whereas others concentrate on the extremes of range of motion, with slow imposed stretches. We suggest that standardized guidelines be identified, which would include a minimal time frame that allows for a statistically reliable estimate of rigidity to be made.

The system we have developed is simple enough for routine use in the clinical setting. Combined with the automated evaluation of other diagnostic variables (10,32), it has the potential to greatly enhance the assessment of PD and other motor disorders, including spasticity. In this study, we chose to use the average impedance measured in a session as the basis for comparing overall clinical ratings and treatment outcomes. Some interrater discrepancies were detected, but the relationship between mean, maximum, and minimum impedance and the clinician's final UPDRS rating needs more clarification. Future studies should also characterize rigidity in relation to range of motion (19), motor set (33), and reflex gain estimated electromyographically (34–37). To be realistic, quantitative evaluation will be adopted only gradually in clinical practice. The qualitative exam will likely prevail for some years to come. It is therefore important at this stage to identify the key factors that influence clinicians in their assignment of rigidity ratings. This should allow a recommended protocol of manual testing to be developed, which may serve to standardize current clinical practice and pave the way for routine quantification in the future.

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