

# Evidence for an Error Deadzone in Compensatory Tracking

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**ABSTRACT.** Humans and monkeys show intermittent arm movements while tracking moving targets. This intermittency has been explained by postulating either a *psychological refractory period* after each movement and/or an *error deadzone*, an area surrounding the target within which movements are not initiated. We present a technique to detect and quantify the size of this deadzone, using a compensatory tracking paradigm that distinguishes it from a psychological refractory period.

An artificial deadzone of variable size was added around a visual target displayed on a computer screen. While the subject was within this area, he received visual feedback that showed him to be directly on target. The presence of this artificial deadzone could affect tracking performance only if it exceeded the size of his intrinsic deadzone. Therefore, the size of artificial deadzone at which performance began to be affected revealed the size of the intrinsic deadzone.

Measured at the subjects' eye, the deadzone was found to vary between 0.06 and 0.38°, depending on the tracking task and viewing conditions; on the screen, this range was 1.3 mm to 3.3 mm. It increased with increasing speed of the target, with increasing viewing distance, and when the amplitude of the movement required was reduced. However, the deadzone size was not significantly correlated with the subjects' level of performance. We conclude that an intrinsic deadzone exists during compensatory tracking, and we suggest that its size is set by a cognitive process not simply related to the difficulty of the tracking task.

**Key words:** motor psychophysics, on-line control, visuomotor tracking

In compensatory tracking, in which the main cue is tracking error, the human arm moves in a series of discrete positional corrections. In 1947, Craik suggested that human tracking performance is like that of a "sampled servo-controller" (p. 56), and discussed the advantages of such intermittently sampled control systems. He concluded that an intermittent process was a fundamental component of the limb control system, which could be suppressed or replaced by smooth pursuit after sufficient practice and when the targets were predictable.

There are three possible mechanisms underlying the intermittency seen in compensatory limb tracking. The first, an internal clock that simply times out series of movements (Bekey, 1962; Lemay & Westcott, 1962), is easily ruled out. The rate of movements is not constant from moment to moment but depends on, among other things, the size of the movements made and on the delay in visual feedback of the subject's movements (Pew, Duffendack, & Fensch, 1967; Smith & Sussman, 1970; Miall, Weir, & Stein, 1985). The second mechanism is that a psychological refractory period (a period after the start of one movement during which a subsequent movement can not be initiated) may interpose a delay between each corrective response (Smith, 1967; Vince, 1947). After making a corrective movement, the subject is briefly refractory to further errors. This mechanism has been postulated for both limb and eye movement control, and considerable evidence suggests that it operates to limit the rate of ocular saccades (Westheimer, 1954). The third proposal is an error deadzone that inhibits small movements. The term *deadzone* is used widely in engineering to describe an input range to which the system does not respond. In other words, there may be a threshold above which the positional error must rise before a corrective movement is initiated. Craik (1947) argued that this is unlikely because the threshold of human visual acuity is much smaller than the observed error before each movement. Furthermore, he argued that an error deadzone would mean that the rate of corrective movements depends on the speed and amplitude of the target's motion. Craik therefore favored the psychological refractory period mechanism. The difficulty in deciding between the last two hypotheses has been to separate out the effects of a refractory period

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from a possible error deadzone. Therefore, we have developed a manual tracking paradigm by which we can identify the error deadzone, regardless of whether a refractory period also exists, by measuring tracking error in the presence of an external error threshold on visual feedback.

We define the size of the subject's error deadzone in the horizontal axis as the distance on either side of a visual target within which the subject makes no corrective movements. In our visually guided tracking paradigm (Figure 1a), we were able to set up an artificial deadzone (ADZ) surrounding the target: If the subject held his manipulandum within this ADZ, he was then given visual feedback that showed him to be exactly on target (Figure 1b). At all other times, his visual feedback was an accurate monitor of manipulandum position.

Provided that the ADZ is smaller than his intrinsic deadzone, the subject's tracking ability should remain unaltered, because, by definition, he would not normally correct for errors smaller than his own deadzone. Once the ADZ is made greater than the real deadzone, however, there will be a region outside the real deadzone but inside the artificial one in which movements normally would have been initiated but now will be suppressed. This will lead to an increase in error between target and real manipulandum position (manipulandum error). The magnitude of the intrinsic deadzone therefore can be estimated by plotting the subject's average manipulandum error against the size of the ADZ. While the ADZ is smaller than the real deadzone, performance should be independent of ADZ size; hence, the initial part of the plot should be horizontal. Once the ADZ is greater than the intrinsic deadzone, the error should rise because of the impaired performance. The point at which the error starts to rise will give the magnitude of the intrinsic deadzone.

Our aim, therefore, was to determine whether an error deadzone exists in visually guided compensatory tracking, and if so, to see which factors of the task affects its size. In addition, to test whether either motor or visual acuity limit the subjects' performance, we measured the deadzone size at different viewing distances and with different movement ranges.

### Method

The experiments reported here were carried out on 2 normal male right-handed human subjects. Because we are interested in using tracking as an experimental paradigm to assess the role of the cerebellum (and therefore the effects of cerebellar lesions), we carried out similar experiments on 1 normal adult male rhesus monkey. All 3 subjects were experienced in tracking the target waveforms used in this study.

### Human Paradigm

The subject sat 50 cm in front of a monochrome computer monitor on which a target was displayed as a small rectangle ( $4 \times 4$  pixels; 1.3 mm wide  $\times$  3.2 mm high).

The horizontal excursion of the target was 500 pixels, which corresponded to a distance of 16.5 cm on the screen.

The subject held a light-weight, low-friction manipulandum in his preferred hand (Figure 1a). The forearm was supported, and only horizontal wrist flexion and extension was allowed ( $65^\circ$  to cover the target amplitude). The angular position of the manipulandum was digitally sampled at 60 Hz with 12-bit resolution and was displayed on the screen as a small monitor spot ( $2 \times 2$  pixels;  $0.67 \times 1.6$  mm). Because the target was only 2 pixels wider than the monitor spot, the subject could, in theory, position his monitor spot exactly at the center of the target.

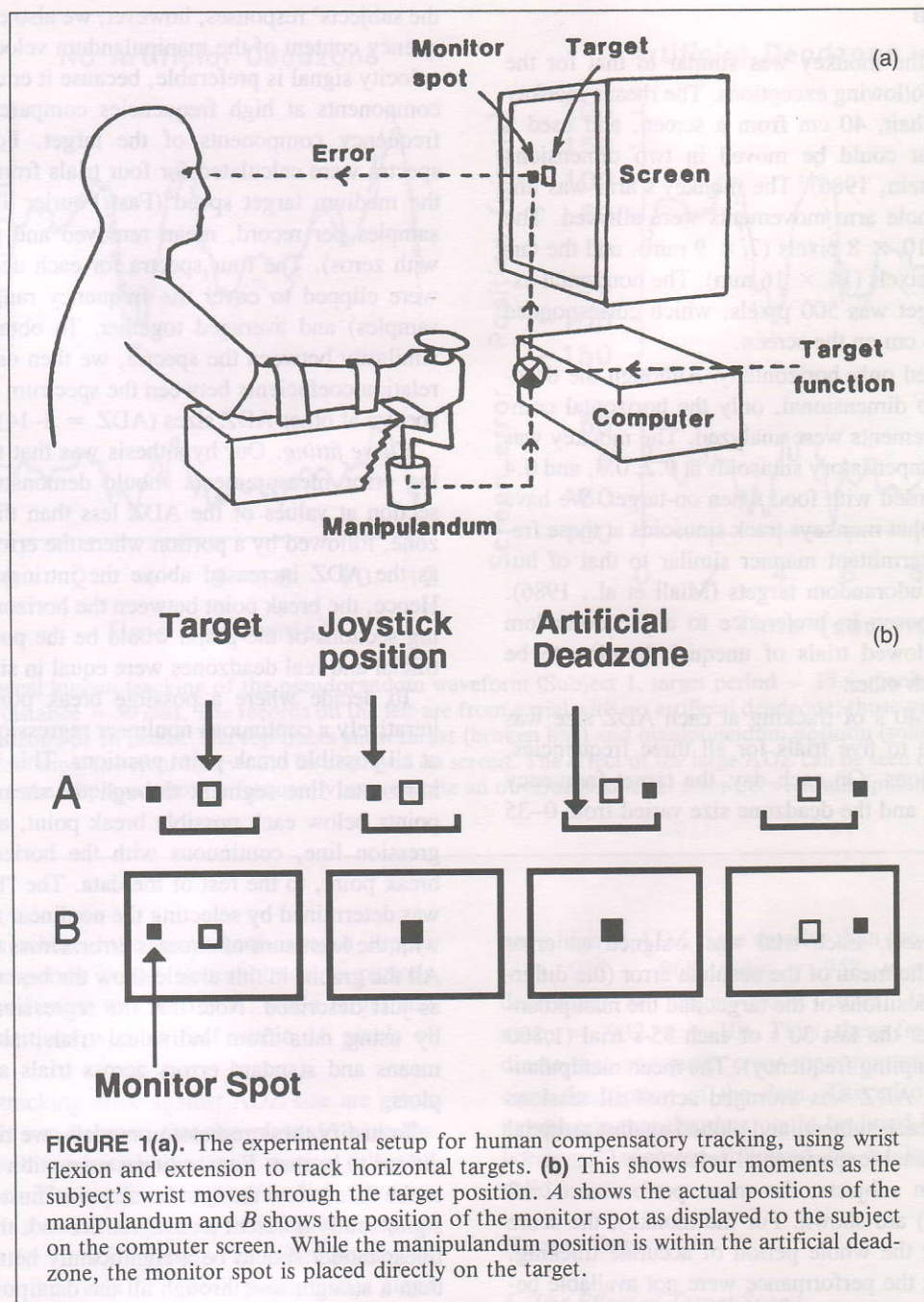
Because our hypothesis was that the deadzone is a threshold on positional tracking error, we needed to give the subject an error-correction task. Hence, the target waveform was pseudorandom, and a compensatory display was used. In a compensatory task, the monitor spot is offset from the central, stationary target by the test waveform; the subject's task is then to compensate for this displacement and return the monitor spot to the screen center. Thus, the display indicates to the subject only the error in angle of his wrist. In a pursuit task, additional cues about the target motion are available (Weir, Stein, & Miall, 1989).

The pseudorandom target function was generated by adding four nonharmonic sinusoids of equal amplitude (Poulton, 1974). Human tracking is most obviously intermittent in these circumstances; if the target is easily predicted, for example, a single sinusoid, the responses rapidly become smooth (Weir et al., 1989). A further advantage of using a compensatory task is that subjects maintain visual fixation on the stationary target during compensatory tracking (Weir et al., 1989), whereas in normal pursuit tracking the eyes fix the continuously moving target. Hence, by using the compensatory task, we could remove the effects of eye movements from the paradigm.

The artificial deadzone (ADZ) was a zone on either side of the target, whose width was measured in screen pixels (1 pixel subtended  $0.0378^\circ$  at the subjects' eyes from a viewing distance of 50 cm). If the manipulandum position was such that the monitor spot fell within this zone, then the monitor spot was displayed exactly on target (Figure 1b). Once the difference between actual wrist angle and target position exceeded the ADZ, the monitor spot jumped out of the deadzone and once again accurately reflected the positional error of the manipulandum. Therefore, the visual feedback only told the subject his manipulandum error if he was outside the artificial deadzone. Note, however, that tracking error was measured as the difference between target position and actual manipulandum position, which was recorded continuously by the computer in each trial.

Three series of experiments were undertaken:

1. The subjects were tested with pseudorandom waveforms at three different speeds. The normal period of the waveform was 15 s (with component sinusoids of 0.13, 0.2, 0.33, and 0.47 Hz). The faster speed was obtained by reducing the waveform period to 10 s (component sinusoid



**FIGURE 1(a).** The experimental setup for human compensatory tracking, using wrist flexion and extension to track horizontal targets. **(b)** This shows four moments as the subject's wrist moves through the target position. *A* shows the actual positions of the manipulandum and *B* shows the position of the monitor spot as displayed to the subject on the computer screen. While the manipulandum position is within the artificial deadzone, the monitor spot is placed directly on the target.

frequencies increased by 50%), and the slower speed by increasing it to 30 s (component sinusoid frequencies decreased by 50%).

2. To test for a relationship between visual acuity and the size of the deadzone, we used the 15-s period pseudorandom waveform to test one subject at viewing distances of 150 and 250 cm. The motor task was unaltered in that the required wrist deviation remained  $65^\circ$ . Thus, this experiment tested whether the observed deadzone was related to the visual aspects of the task.

3. To test for a relationship between motor acuity and the deadzone size, we also tested the same subject with reduced wrist movements. Again, the 15-s pseudorandom waveform

was used, and the viewing distance remained at 50 cm. The gain of the manipulandum signal was changed to 150% and 200% of the initial setting so that the angle through which the wrist had to be moved was reduced to 66% or 50% of the initial range of  $65^\circ$ .

Each daily session consisted of 20 trials with randomized artificial deadzone size. Each trial consisted of 35 s of tracking. To avoid starting errors, we excluded the first 5 s of each trial from the analysis. Within each session only the size of the artificial deadzone was changed between trials; each deadzone size was tested eight times per subject for Experiment (1), and four times for Experiments (2) and (3).

## Monkey Paradigm

The set-up for the monkey was similar to that for the humans, with the following exceptions. The rhesus monkey sat in a primate chair, 40 cm from a screen, and used a manipulandum that could be moved in two dimensions (Miall, Weir, & Stein, 1986). The monkey's arm was unsupported, and whole arm movements were allowed. The monitor spot was  $10 \times 8$  pixels ( $7 \times 9$  mm), and the target was  $20 \times 15$  pixels ( $14 \times 16$  mm). The horizontal excursion of the target was 500 pixels, which corresponded to a distance of 36 cm on the screen.

The target moved only horizontally. Although the deadzone was also two dimensional, only the horizontal component of the movements were analyzed. The monkey was trained to track compensatory sinusoids at 0.2, 0.3, and 0.4 Hz, and was rewarded with food when on-target. We have previously shown that monkeys track sinusoids at these frequencies in an intermittent manner similar to that of humans tracking pseudorandom targets (Miall et al., 1986). Sinusoids were chosen in preference to a pseudorandom waveform; this allowed trials of unequal duration to be compared with each other.

A total of 80–240 s of tracking at each ADZ size was collected over two to five trials for all three frequencies, in nine daily sessions. On each day, the target frequency was kept constant, and the deadzone size varied from 0–35 pixels.

## Data Analysis

**Error measurement.** Each trial was assigned an error score, which was the mean of the absolute error (the difference between the positions of the target and the manipulandum) averaged over the last 30 s of each 35-s trial (1,800 points at 60-Hz sampling frequency). The mean manipulandum error at each ADZ was averaged across all sessions (eight or four) for each subject, and plotted against artificial deadzone size for each experimental setup (see Figures 3–5). For the human subjects, the mean performance  $\pm 2$  standard error (SE) are shown. For the monkey, the score was averaged over the whole period of accurate tracking. Standard errors on the performance were not available because the data for each deadzone was recorded over two to five trials of unequal duration.

We chose to plot tracking error as a measure of performance rather than to try to quantify the degree of intermittency, because subjects show a continuum between clearly intermittent responses and moments of virtually smooth pursuit. Therefore, although it is possible to detect and analyze individual intermittent corrections (Miall et al., 1986), we have not found a clear statistic that describes the contribution of intermittent corrections to the whole response. Others have used measures of signal power (Pew et al., 1967) or total path length (Beppu, Nagaoka, & Tanaka, 1987), neither of which would be appropriate in this situation.

**Fourier analysis.** Because our measure of the mean tracking error may not be sensitive to small changes in shape of

the subjects' responses, however, we also examined the frequency content of the manipulandum velocity records. The velocity signal is preferable, because it emphasizes spectral components at high frequencies compared with the low-frequency components of the target. Fourier amplitude spectra were calculated for four trials from each subject at the medium target speed (Fast Fourier Transform, 2,100 samples per record, mean removed and padded to 4,096 with zeros). The four spectra for each deadzone size then were clipped to cover the frequency range 0–6 Hz (411 samples) and averaged together. To obtain a measure of similarity between the spectra, we then calculated the correlation coefficients between the spectrum at zero ADZ and spectra at other ADZ sizes ( $ADZ = 1-14$ ).

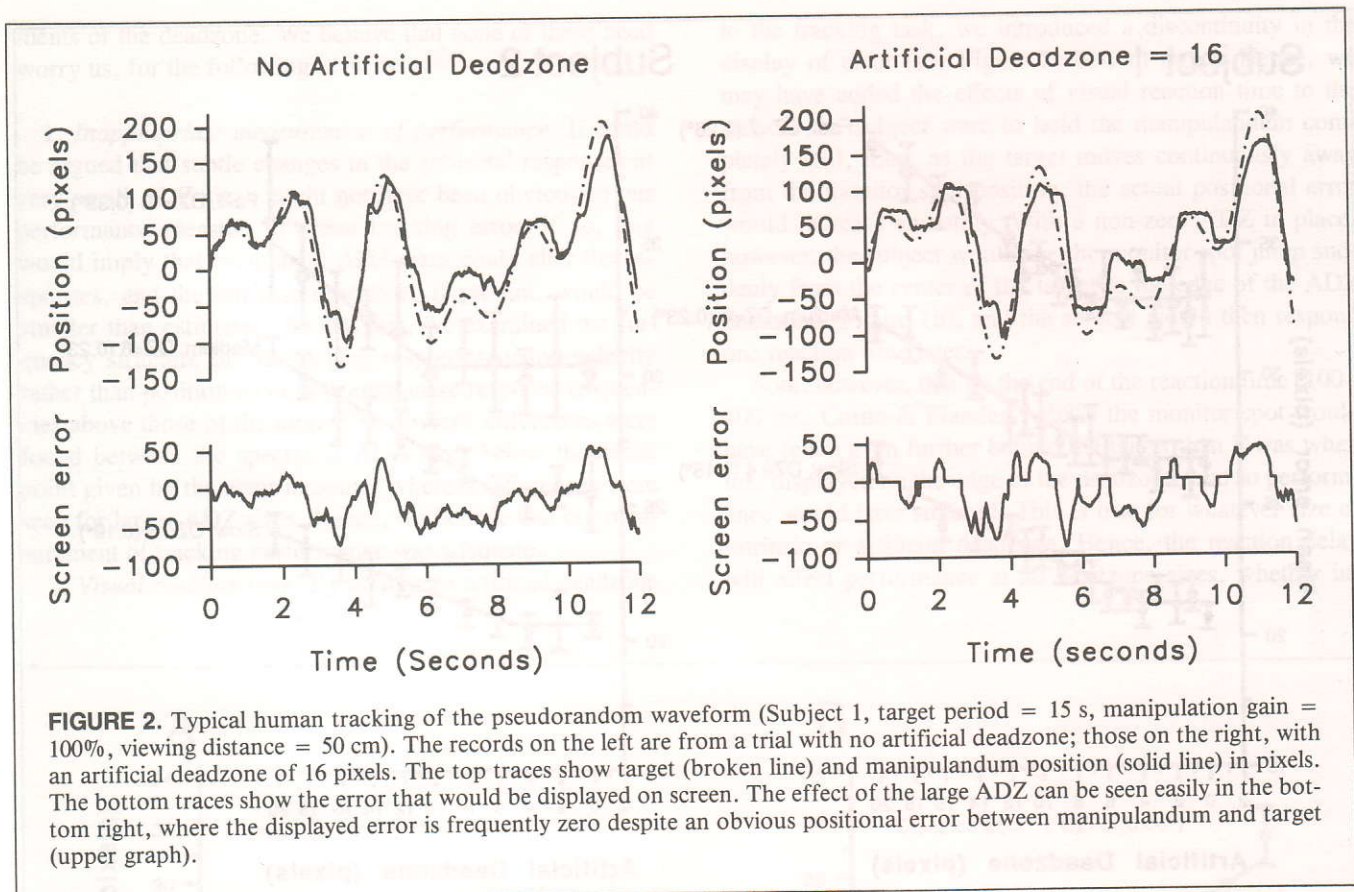
**Curve fitting.** Our hypothesis was that the plotted tracking error measurements should demonstrate a horizontal section at values of the ADZ less than the intrinsic deadzone, followed by a portion where the error would increase as the ADZ increased above the intrinsic deadzone size. Hence, the break point between the horizontal and the sloping sections of the graph would be the point where the artificial and real deadzones were equal in size.

To decide where a possible break point lay, we fitted iteratively a continuous nonlinear regression line to the data at all possible break-point positions. This involved fitting a horizontal line segment through the mean of all the data points below each possible break point, and a straight regression line, continuous with the horizontal line at the break point, to the rest of the data. The "best" break point was determined by selecting the nonlinear regression model with the least sum-of-squares error across the whole graph. All the graphs in this article show the best nonlinear model, as just described. Note that the regression was calculated by using data from individual trials, although only the means and standard errors across trials are shown in the plots.

To justify these regression models, we required two conditions to be met. First, a linear regression line applied only to the data below the best break point must have a slope not significantly different from zero. Second, the complete nonlinear model should be a significantly better fit to the data than a straight line through all the data points (using the *F* test), given that the nonlinear regression has one extra degree of freedom. The break point, provided that these two conditions were met, was taken to be a measure of the intrinsic deadzone for that experiment.

## Results

A characteristic tracking trace is shown for Subject 1 in Figure 2. The left-hand graphs show tracking with no artificial deadzone. The top traces show the target (broken line) and manipulandum position (solid line) over a 12-s period. The bottom traces show the error in the compensatory task as shown on the screen. The subject can be seen to track the smoothly moving target intermittently. The right-hand graphs show the same task but with an artificial deadzone of 16 pixels. The tracking is now more intermittent, and



**FIGURE 2.** Typical human tracking of the pseudorandom waveform (Subject 1, target period = 15 s, manipulation gain = 100%, viewing distance = 50 cm). The records on the left are from a trial with no artificial deadzone; those on the right, with an artificial deadzone of 16 pixels. The top traces show target (broken line) and manipulandum position (solid line) in pixels. The bottom traces show the error that would be displayed on screen. The effect of the large ADZ can be seen easily in the bottom right, where the displayed error is frequently zero despite an obvious positional error between manipulandum and target (upper graph).

there are periods where little movement is seen (upper right). This corresponds in the bottom trace to periods where the displayed error was zero (i.e., when target and manipulandum positions were in fact within 16 pixels of each other).

Graphs of the tracking error against ADZ size are given in Figures 3–5. It can be seen that the plots do have the predicted form, with an initial horizontal section followed by a rising section. The nonlinear regression line fits were significantly better than a straight line fit for all the experiments carried out ( $p < .05$ ,  $F$  test). The initial portion of each graph, below the “best fit” break point, had a slope that was not significantly different from zero ( $p > .1$ ) in all but one graph, when modeled with linear regression. The one exception (Figure 5a) had a significant but negative slope.

Similar results were found when the data were examined in the frequency domain. At all ADZ sizes below the intrinsic deadzone size, as calculated from the error plots, the average frequency spectra looked grossly similar, with a broad band of components between 0.5 and 2 Hz (for example, Pew et al., 1967). At the largest ADZ sizes, there was a shift in this broad band to slightly lower frequencies, as might be expected from the reduced response rate of the subjects (Figure 2, upper right). The similarity of the spectra was quantified by calculating a correlation between the zero-ADZ spectrum and all other spectra. The correlations

were high at ADZ sizes smaller than the intrinsic deadzone ( $r = .945 - .968$ , mean = .958,  $n = 9$ ) and gradually decreased at larger ADZ sizes ( $r = .896 - .949$ , mean = .932,  $n = 10$ ). Thus, these frequency spectra indicate that our mean error measurement provided a sufficient description of the data. This also suggests that our hypothesis that the data could be fitted by a model with a horizontal segment followed by a sloped segment was valid.

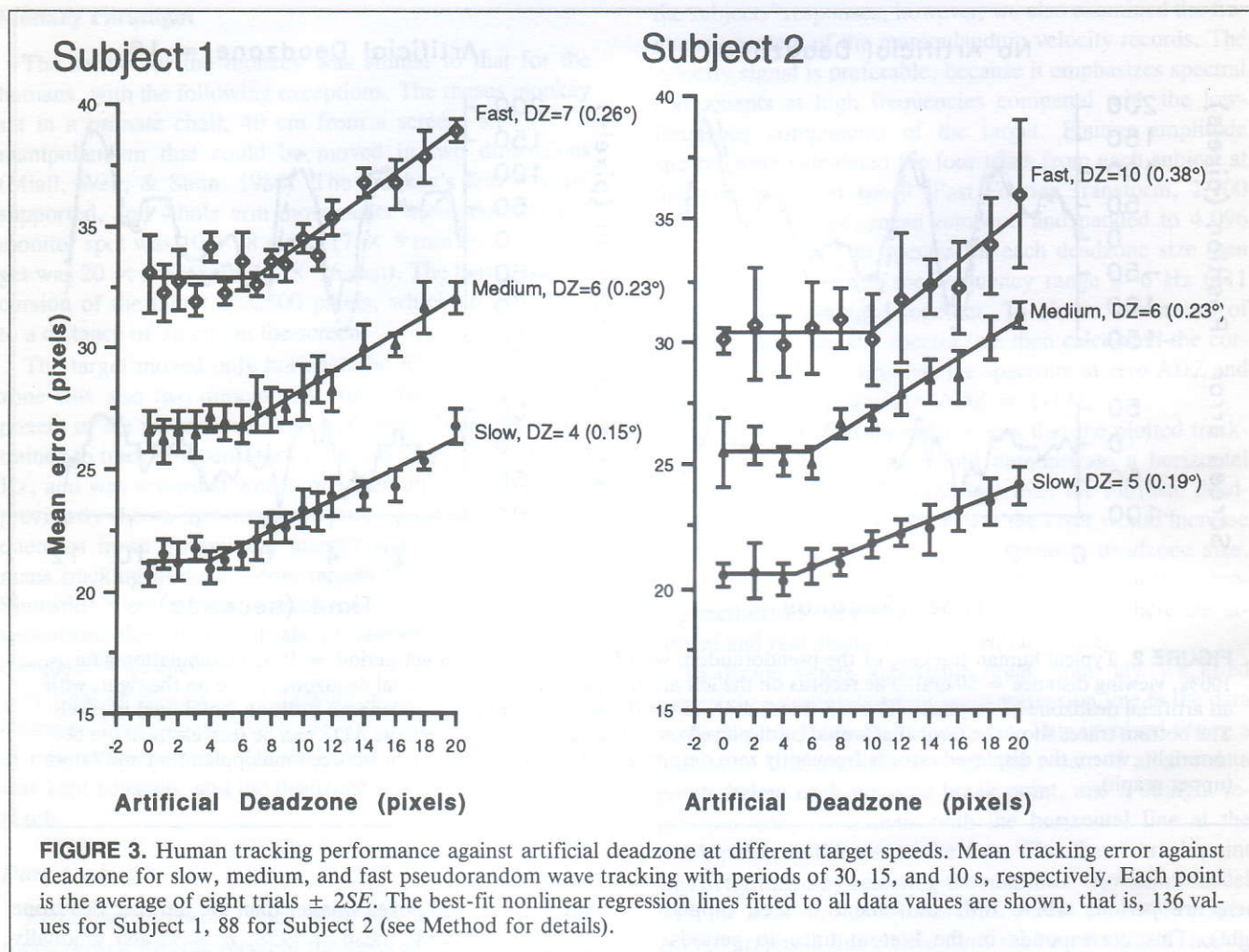
## Human Data

### 1. The Effect of Target Speed

Figure 3 shows the error against artificial deadzone plots for three different target speeds for the 2 human subjects. As expected, errors increased with increasing target speed for both subjects. Both subjects showed an increased variability in tracking error for the fastest target. For Subject 1, the break point was at 4, 6, and 7 pixels (0.15, 0.23, and 0.26° at the eye) for target periods of 30, 15, and 10 s, respectively. For Subject 2, the values were 5, 6, and 10 pixels (0.19, 0.23, and 0.38° at the eye) for the same targets.

### 2. The Effect of Viewing Distance

The results for increased viewing distances of 150 and 250 cm are shown in Figures 4a and 4b. Equivalent data for



the normal viewing distance of 50 cm were given in Figure 2, Subject 1, middle plot. The break point was measured to be 6, 8, and 9 pixels (0.23, 0.10, and 0.07° at the eye) for viewing distances of 50, 150, and 250 cm, respectively.

### 3. The Effect of Manipulandum Gain

The results for trials with reduced movement amplitude (increased manipulandum gain) are given in Figures 4c and 4d; again the equivalent data is that of Figure 2, Subject 1, middle plot. The break point was 6, 9, and 8 pixels (0.23, 0.34, and 0.30° at the eye) for movement gains of 100%, 150%, and 200%, respectively.

### Monkey Data

The data for the monkey tracking sinusoids at three frequencies are shown in Figure 5. The break point was measured at 9, 8, and 11 pixels (0.93, 0.83, and 1.13° at the monkey's eye) for target frequencies of 0.2, 0.3, and 0.4 Hz, respectively.

### Discussion

In these experiments, we have attempted to measure the size of the error threshold or deadzone during manual track-

ing. Our hypothesis was that the addition of an artificial deadzone (ADZ) larger than the subject's intrinsic deadzone should increase tracking error. Hence, a characteristic plot of tracking error against ADZ width should be obtained. This was found to be the case (Figures 3–5), and the fit of nonlinear regression models to the data was in all cases statistically significantly better than that of a linear regression model ( $p < .05$ ). As described in the introduction, the break point of these curves gives an estimate of the size of the intrinsic deadzone. Hence, these results support the proposal that a positional deadzone exists in compensatory manual tracking.

We chose to use a compensatory tracking paradigm in order to restrict the task to error correction and to prevent possible contamination of our results by eye movements. Intermittency is also seen in pursuit tracking, however, and it may be that this intermittency also is related to an error deadzone. For example, pursuit of a pseudorandom target shows clear intermittent responses. It is an open question whether the smooth tracking of predictable targets eliminates the deadzone, or avoids its effect by utilizing feedforward control.

There are four possible contaminants of our measure-

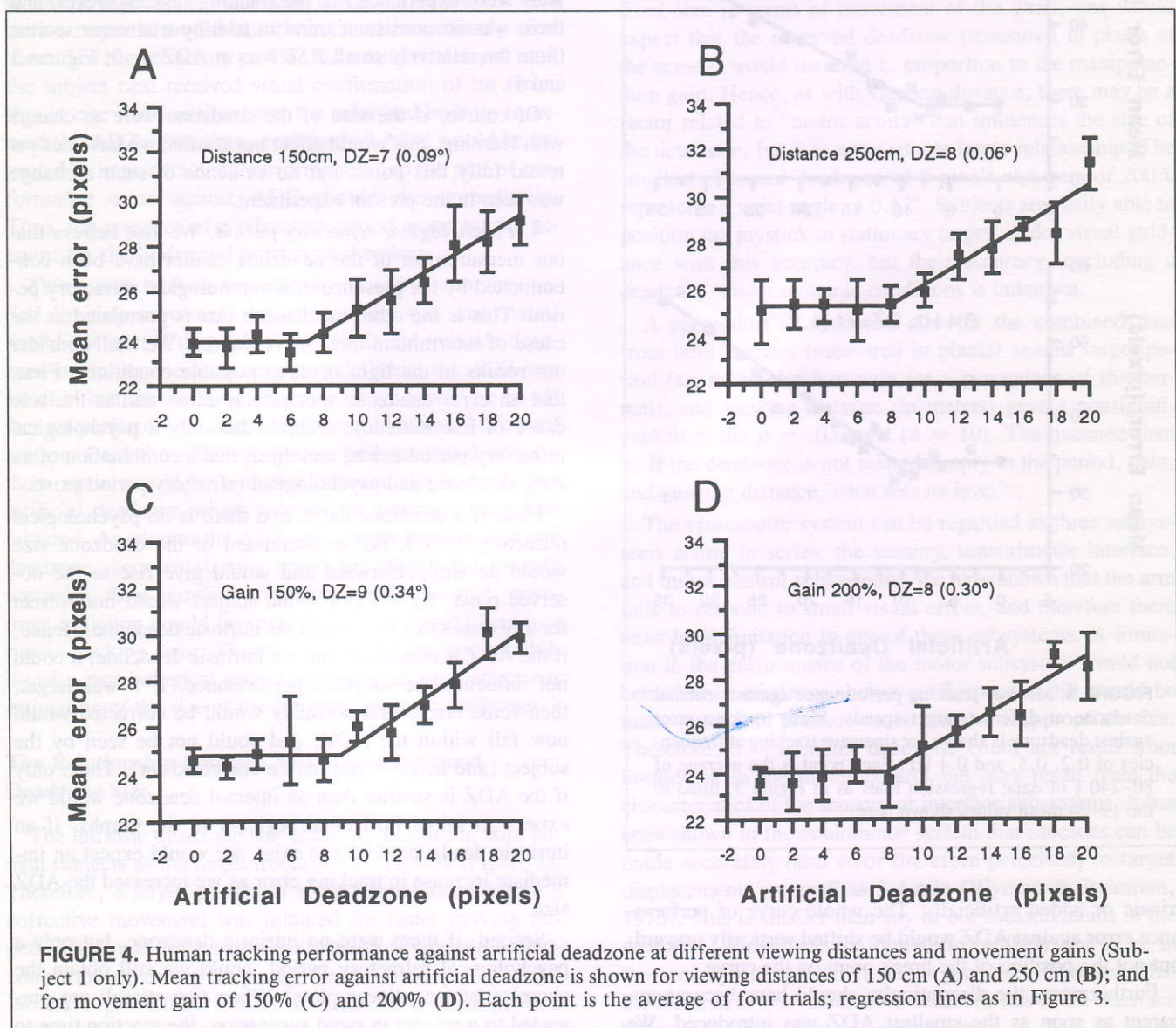
ments of the deadzone. We believe that none of these need worry us, for the following reasons:

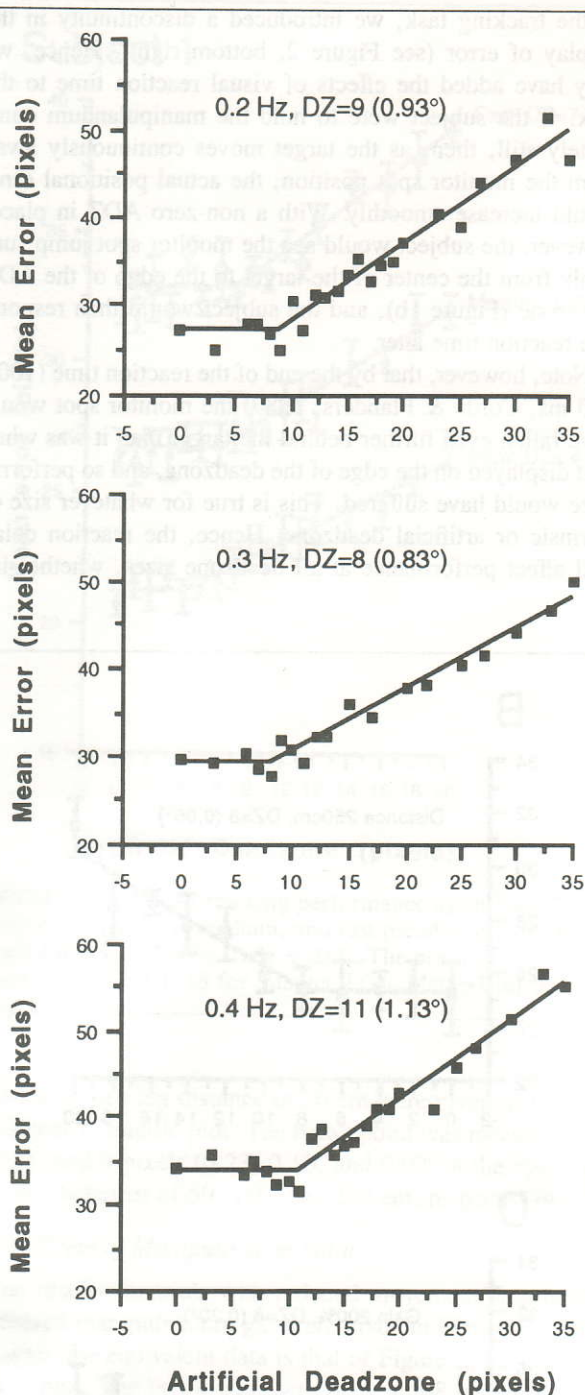
1. *Inappropriate measurement of performance.* It could be argued that subtle changes in the subjects' responses at very small ADZ sizes might not have been obvious in our performance measure of mean tracking error. If so, this would imply that even small ADZ sizes could alter the responses, and the intrinsic deadzone, if present, would be smaller than estimated. To test this, we examined the frequency structure of the tracking responses, using velocity rather than position records to emphasize response frequencies above those of the target. No obvious differences were found between the spectra at ADZ sizes below the break point given by the error measure, whereas differences were seen for larger ADZ sizes. Hence, we believe that our measurement of tracking performance was adequate.

2. *Visual reaction time.* By adding an artificial deadzone

to the tracking task, we introduced a discontinuity in the display of error (see Figure 2, bottom right). Hence, we may have added the effects of visual reaction time to the task. If the subject were to hold the manipulandum completely still, then, as the target moves continuously away from the monitor spot position, the actual positional error would increase smoothly. With a non-zero ADZ in place, however, the subject would see the monitor spot jump suddenly from the center of the target to the edge of the ADZ deadzone (Figure 1b), and the subject would then respond one reaction time later.

Note, however, that by the end of the reaction time (100–400 ms, Cordo & Flanders, 1989) the monitor spot would have fallen even further behind the target than it was when first displayed on the edge of the deadzone, and so performance would have suffered. This is true for whatever size of intrinsic or artificial deadzone. Hence, the reaction delay will affect performance at all deadzone sizes, whether in-





**FIGURE 5.** Monkey tracking performance against artificial deadzone at different target speeds. Mean tracking error against deadzone is shown for sinuswave tracking at frequencies of 0.2, 0.3, and 0.4 Hz. Each point is the average of 80–240 s of data; regression lines as in Figure 3, fitted to the 19–24 mean values shown here.

trinsic or added artificially. The whole curve of performance error against ADZ would be shifted vertically upward, but not the position of the break point on the curve.

Furthermore, the discontinuity should have become apparent as soon as the smallest ADZ was introduced. We

therefore might expect a step increase in performance errors between tracking with no deadzone ( $ADZ = 0$ ) and  $ADZ = 1$ . This is not seen (Figures 3–5). Therefore, we believe that the effects of the subjects' reaction time did not affect our measurement of the size of their intrinsic deadzone.

**3. Learning.** Our identification of the best break-point position in the curves depends on the relative difference in performance between trials for each subject, rather than the absolute value of performance error. Thus, the effect of the subject's learning more about the task should not affect the results. As just argued for the effects of reaction time, the effects of learning would only shift the whole curve vertically (downward) and should not affect our measurement of the break point in the curve.

An indication of learning would be that the performance measured with no ADZ should improve over the course of the experiments. This would be obvious as a trial-by-trial reduction in the performance error at  $ADZ = 0$ . The subjects were experienced at the tracking task, however, and there was no consistent trend in trial-by-trial error scores; (note the relatively small 2 SE bars at  $ADZ = 0$ , Figures 3 and 4).

Of course, if the size of the deadzone were to change with learning, this would affect our results; we have not yet tested fully this point, but no evidence of such a change was seen in the present experiment.

**4. Psychological refractory period.** We also believe that our measurement of the deadzone cannot have been contaminated by the presence of a psychological refractory period. This is the other mechanism that is postulated as the cause of intermittent tracking responses. We shall consider our results in the light of three possible conditions: First, that an error deadzone mechanism exists and is the sole cause of intermittency; second, that only a psychological refractory period exists; and third, that a combination of an error deadzone and psychological refractory period exists.

First, if a deadzone exists and there is no psychological refractory period, our measurement of the deadzone size would be straightforward and would give rise to the observed plots. By definition, the subject would not correct for any errors that fall within his intrinsic deadzone. Hence, if the ADZ was smaller than the intrinsic deadzone, it could not influence the subject's performance. If it was larger, then some errors that normally would be corrected would now fall within the ADZ, and could not be seen by the subject (and hence could not be corrected for). Thus, only if the ADZ is smaller than an internal deadzone would we expect an initial horizontal segment to the graphs. If an intrinsic deadzone does not exist, we would expect an immediate increase in tracking error as we increased the ADZ size.

Second, if there were no intrinsic deadzone, but only a psychological refractory period, could we still obtain the characteristic nonlinear plots? When two stimuli are presented to a subject in rapid succession, the reaction time to

the second stimulus is increased. The increase in reaction time has been referred to as the psychological refractory period (Smith, 1967). The psychological refractory period can be defined, for a tracking task, as a period after the start of one movement during which a subsequent movement cannot be initiated. In this case, each corrective movement should start one psychological refractory period after the previous movement. Only if positional error was actually zero at the end of the refractory period would the subject fail to make a subsequent correction, whereas failure to correct for any nonzero errors of course implies the existence of an error deadzone. We have shown that the size of errors measured at the start of each corrective movement has a roughly normal distribution (Miall, Weir, & Stein, *in press*). This means that movements are initiated from a broad, continuous range of starting errors, and so, if the refractory period exists, then most movements indeed should be initiated one refractory period after the previous one. If an arbitrarily small ADZ is now introduced, some errors would be expected to fall within this zone; hence, the corrective response would not be initiated immediately after the end of the refractory period, but would be delayed until the subject next received visual confirmation of his error, that is, one reaction time after the error had built up to exceed the ADZ. Therefore, even a small ADZ would be expected to impair tracking performance, and the plot of performance error against ADZ should rise immediately. Thus, the presence of a refractory period alone cannot account for the horizontal portion of the plots.

Third, with both an error deadzone and a refractory period limiting movement initiation, corrective movements would not start if they fell either within the intrinsic deadzone or during the refractory period. While the ADZ was smaller than the intrinsic deadzone, there would be no effect on performance, and the plot would have a horizontal section. Once the ADZ was greater than the real deadzone, there again would be a region between the intrinsic and artificial deadzone where movements normally would be initiated. As soon as the ADZ was greater than the intrinsic deadzone, because starting errors are distributed roughly normally, there would be occasions when normal movement initiation would be suppressed. This would lead to a rise in tracking error. Therefore we argue that the possibility of a psychological refractory period did not affect our estimation of the size of the subjects' intrinsic deadzones.

### The Relationship Between Task Parameters and Deadzone Size

The intrinsic deadzone was found to increase in both human subjects as the target frequencies increased (Figure 3). Therefore, a larger positional error was tolerated before a corrective movement was initiated for faster moving targets. There was not such a clear increase in size of the deadzone with increasing frequency for the monkey (Figure 5). We have previously found, however, that monkeys best track sinusoids of frequencies of about 0.3 Hz; whereas fre-

quencies of less than 0.1 Hz or greater than 0.5 Hz are followed poorly (Miall et al., 1986). This may explain why the deadzone was found to be smaller at 0.3 Hz than at 0.2 Hz in this experiment.

The intrinsic deadzone also increased as viewing distance increased (Figure 4). The increase from 6 pixels at 50 cm, to 7 pixels at 150 cm, and 8 pixels at 250 cm was less than could be explained simply on the basis of viewing angle, however. Furthermore, the smallest observed deadzone (of 8 pixels at 250 cm, or  $0.07^\circ$  at the subjects' eye) was still greater than this subjects' acuity at distinguishing separate points on the screen, whether stationary or moving at the velocity of the target used in this experiment. Hence, the size of the deadzone cannot be simply related to visual acuity.

As the manipulandum gain increased, deadzone size increased somewhat (from 6, to 9, to 8 pixels at gains of 100, 150, and 200%; Figure 4). Note that as gain increased, finer wrist movements were required. If the deadzone were of fixed size in terms of movement of the wrist, one would expect that the observed deadzone (measured in pixels at the screen) would increase in proportion to the manipulandum gain. Hence, as with viewing distance, there may be a factor related to "motor acuity" that influences the size of the deadzone, but it is not a simple linear relationship. The smallest observed deadzone of 8 pixels at a gain of 200% represents a wrist angle of  $0.52^\circ$ . Subjects are easily able to position the joystick to stationary targets under visual guidance with this accuracy, but their accuracy (excluding a deadzone) under dynamic conditions is unknown.

A regression of deadzone size for the combined data from both humans (measured in pixels) against target period (s), manipulandum gain (as a percentage of the normal), and viewing distance (in meters) gave a nonsignificant fit at the  $p = .05$  level ( $n = 10$ ). The question then is: If the deadzone is not related simply to the period, gain, and viewing distance, what sets its level?

The visuomotor system can be regarded as three subsystems acting in series: the sensory, sensorimotor interface, and motor control subsystems. We have shown that the arm fails to respond to small visual errors, and therefore there must be a limitation in one of these subsystems. A limitation in the performance of the motor subsystem would not behave as a deadzone, however. Responses still would be made but would produce movements of inappropriate size. Therefore, a visuomotor deadzone could not result from limitations in the motor control but may result from the characteristics of the sensory or interface subsystems. It has been shown in the oculomotor system that saccades can be made accurately (and error therefore perceived) to target displacements as small as 3.4 min (Wyman & Steinman, 1973). This is smaller than any of our measurements of the deadzone. Therefore, the limitation is likely not to be in the sensory subsystem. It seems probable, therefore, that the location of the visuomotor deadzone lies in the interface between sensory input and motor output.

## The Relationship Between Deadzone Size and Tracking Performance

The plots in Figures 3, 4, and 5 show that the errors measured at ADZ = 0 were not significantly better than at other ADZ values below the best-fit break points. A baseline error for each experiment therefore was taken to be the mean of all the points below the best-fit break point; this corresponds to the horizontal segment of the regression model plotted in each figure. The baseline error was seen to vary with target speed in the human subjects and in the rhesus monkey, and also varied with viewing distance and manipulandum gain for the one human subject tested.

A regression of baseline error (BE) for the combined human data against target period (P), manipulandum gain (G), and viewing distance (D) gave the following:

$BE = 39.6 - 0.48P - 4.03G - 1.65D$ , with a significant correlation of  $r = .916$  ( $p \leq .01$ ,  $n = 10$ ).

We therefore attempted to regress our measurements of deadzone size on the baseline errors for all human data; this correlation was not significant ( $r = .53$ ,  $p > .1$ ,  $n = 10$ ). Hence, although the subjects' performance varied from trial to trial, presumably as the task difficulty varied, this did not correlate with the measured size of the error deadzone. It may be that the size of the deadzone is set by the subject on the basis of his subjective assessment of the task difficulty. But, there appears to be no simple relationship between a small deadzone and good tracking performance. In fact, a large deadzone may be a "good" strategy for tracking: Subject 2 had lower tracking errors but a larger intrinsic deadzone than Subject 1. (see Figure 3). This corresponds closely to Craik's (1947) original suggestion, taken up by others (Neilson, O'Dwyer, & Neilson, 1988), that sampling of the tracking error, in this case by an error deadzone, confers the advantages of sampled feedback in an uncertain task.

In summary, therefore, we believe our results demonstrate that an error deadzone must be present in compensatory manual tracking. We cannot state that it is the only cause of intermittency, although our measurement of its size is independent of psychological refractory period or reaction time. We are confident that these results could not be achieved by a mechanism that does not include an error deadzone. We have also shown that the size of the deadzone varies with a number of factors but is not simply related to the difficulty of the tracking task. Thus, the determination of deadzone size must incorporate both the visual and the motor aspects of the task: Its size is not constant when measured in either visual or movement coordinates alone, but is a mixture of the two.

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## REFERENCES

- Bekey, G. A. (1962). The human operator as a sampled-data system. *I.E.E.E. Transactions Human Factors in Electronics*, HFE3, 43-51.
- Beppu, H., Nagaoka, M., & Tanaka, R. (1987). Analysis of cerebellar motor disorders by visually guided elbow tracking movements. *Brain*, 110, 1-18.
- Cordo, P. J., & Flanders, M. (1989). Sensory control of target acquisition. *Trends in Neurosciences*, 12, 110-117.
- Craik, K. J. W. (1947). Theory of the human operator in control systems. I. The operator as an engineering system. *British Journal of Psychology*, 38, 56-61.
- Lemay, L. P., & Westcott, J. H. (1962, September). *The simulation of human operator tracking using an intermittent model*. Symposium conducted at the meeting of the International Congress of Human Factors in Electronics, Long Beach, CA.
- Miall, R. C., Weir, D. J., & Stein, J. F. (1985). Visuo-motor tracking with delayed visual feedback. *Neuroscience*, 16, 511-520.
- Miall, R. C., Weir, D. J., & Stein, J. F. (1986). Manual tracking of visual targets by trained monkeys. *Behavioral Brain Research*, 20, 185-201.
- Miall, R. C., Weir, D. J., & Stein, J. F. (in press). Intermittency in human manual tracking tasks. *Journal of Motor Behavior*.
- Neilson, P. D., O'Dwyer, N. J., & Neilson, M. D. (1988). Internal models and intermittency: A theoretical account of human tracking behaviour. *Biological Cybernetics*, 58, 101-112.
- Pew, R. W., Duffendack, J. C., & Fensch, L. K. (1967). Sine wave tracking revisited. *I.E.E.E. Transactions Human Factors in Electronics*, HFE8, 130-4.
- Poulton, E. C. (1974). *Tracking skills and manual control*. London: Academic Press.
- Smith, M. C. (1967). Theories of the psychological refractory period. *Psychological Bulletin*, 67, 202-213.
- Smith, K. U., & Sussman, H. M. (1970). Delayed feedback in steering during learning and transfer of learning. *Journal of Applied Psychology*, 54, 334-342.
- Vince, M. A. (1947). The intermittency of control movements and the psychological refractory period. *British Journal of Psychology*, 38, 149-157.
- Weir, D. J., Stein, J. F., & Miall, R. C. (1989). Cues and control strategies in visually guided tracking. *Journal of Motor Behavior*, 21, 185-204.
- Westheimer, G. (1954). Eye movement responses to a horizontally moving visual stimulus. *Archives of Ophthalmology*, 52, 932-941.
- Wyman, D., & Steinman, R. M. (1973). Small step tracking: Implications for the oculomotor "dead zone." *Vision Research*, 13, 2165-2172.

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