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ON THE STRUCTURE OF THE TEMPORAL SENSORY SYSTEM

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Evidence is reviewed that favours the hypothesis that temporal performance depends on a temporal sensory system based on a biological source of temporal information. A model for the pacemaker, initially proposed by Treisman (1963) and elaborated by Treisman, Faulkner, Naish and Brogan (1990), predicts that if regular stimulus pulses (such as auditory clicks) are presented at suitable rates during intervals whose durations are estimated by a subject, the pulses may perturb the frequency at which the subject's temporal oscillator runs and so perturb time estimation, resulting in a characteristic pattern of interference. Such interference patterns have been found for time estimation when subjects were exposed to auditory clicks, or visual flicker, and for the timing of movement. Records of the electroencephalogram were taken while subjects estimated time durations accompanied by trains of auditory clicks. Analyses provide evidence for the presence in the EEG of click-sensitive oscillations. Implications of this finding for the organization of an underlying temporal sensory system are discussed.

We can orient in space and measure distance by the use of visual and auditory cues. But how do we orient in time and measure durations? Clearly, we sometimes use temporal cues, such as the position of the sun, and the belief that all time experience is so based has been popular. However, spatial perception also employs a dedicated sensory system, the vestibular system. Analogously, I believe that we also possess a temporal sensory system that allows us to perceive and operate in time. This system derives its information from mechanisms, temporal oscillators, contained wholly in the nervous system. Below I briefly review work we have done in the attempt to put this hypothesis on a firmer basis.

Various approaches have attempted to establish a physiological basis for time perception. Hoagland (1933) proposed an internal chemical clock whose rate was affected by body temperature, and François (1927) and Hoagland (1933) showed that time is felt to pass more rapidly if body temperature is raised. But there is a problem with this, as the variations in temporal judgment associated with temperature change are greater than change in the speed of a chemical reaction can explain.

Further evidence for a physiological basis is provided by the effects of hormones and neurotransmitters, drugs, and brain lesions. Other approaches seek to find evidence of internal timing processes in motor performance and the reproduction of rhythmic sequences, or attempt to identify privileged time intervals or temporal quanta (Stroud, 1955; Kristofferson, 1984). (For references and a fuller review see Treisman et al., 1990.)

Various observations presented anomalies. For example, not only may rise in temperature speed the clock, as shown by changes in time estimation, so also may exposure of lightly dressed subjects to low temperatures (Lockhart, 1967). To understand such effects, a theory of temporal processing is needed. Treisman (1963) put forward an information-processing model in which temporal information was provided by a pacemaker that emitted regular pulses. The model contained a short-term memory and other devices that allowed the temporal information to be used to perform tasks such as time estimation. To account for the similar effects of low and high temperatures, monotony, and other observations, the model allowed the pacemaker to vary in its degree of arousal or activation. If this rose its speed increased; and both stressful low or high temperatures might increase its arousal.

A more direct approach would be to identify a rhythm in the EEG that could be shown to behave like the theoretical pacemaker output. A common speculation, that the alpha rhythm represents the output of a temporal clock (Hoagland, 1933) has been investigated several times with inconclusive results. Treisman (1984) re-examined this question by recording the EEG while subjects repeatedly produced a four second duration. If the clock slows down over a period, time productions should get longer, and if the alpha rhythm represents the clock it should slow down in the same proportion. But it was found that time productions and alpha frequency could vary equally well in the same or opposite directions, which seemed to exclude the hypothesis.

Various observations present difficulties for a unitary pacemaker. For example, a concert may seem longer if listeners are bored than if they enjoy the music, but their ability to assess its tempo is unaffected. This suggests that differing timing processes underlie the two judgments. We may also ask what features temporal pacemakers involved in controlling motor performance would need to have. Treisman et al. (1990) argued for a model in which multiple pacemakers are embedded at different levels of a motor control hierarchy. A motor program would prescribe the general parameters of movement, and

individual pacemakers would determine the exact timing. But this model imposes conflicting demands on the pacemakers. To coordinate two limbs engaged in a cooperative action (e.g. catching a ball with both hands) the pacemaker controlling each limb should run at the same stable frequency. But if pacemakers always ran at the same unvarying rate the system would be inflexible. To allow continuous modifications of pace (e.g. increasing one's speed while running), it would be most efficient for the governing motor program to be unchanged, i.e. to prescribe the same timing parameters to the pacemakers, but for the rates of the latter to be capable of being varied as required.

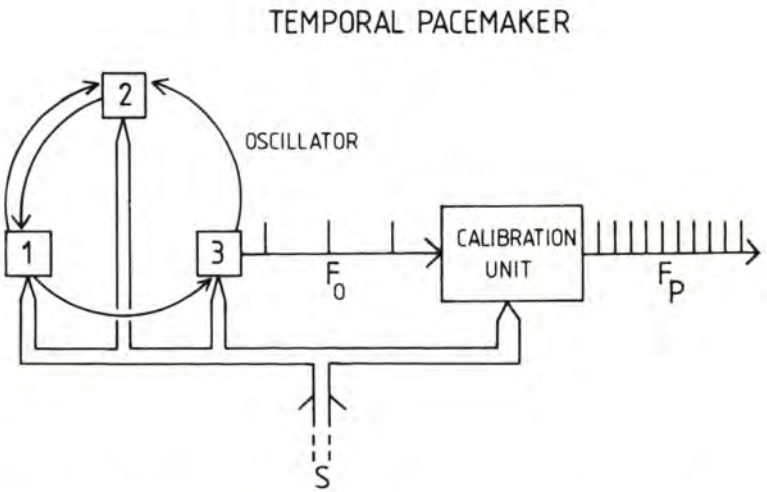

Figure 1. The calibrated temporal pacemaker model. Inter-connected units form a non-linear self-exciting temporal oscillator (TO) which connects to a calibration unit (CU). S: an external stimulus input. F_O: the output frequency from the TO. F_P: the output frequency from the CU. (Reprinted from Treisman et al., 1990.)

These considerations led to a "calibrated pacemaker" model in which the pacemaker has two components: a Temporal Oscillator (TO) that emits pulses at an oscillator frequency, F_O, whose characteristic value is $F_{c,O}$, and a gain control or Calibration Unit (CU) that multiplies F_O by a calibration factor C_f to determine the frequency of the final pacemaker output F_P. If high stability is required F_O might be employed directly. In other tasks, such as temporal judgments, the final output

F_P would be employed by the information-processing mechanisms. If intense sensory stimuli increase the arousal of the CU, or messages from higher neural levels raise it, Cf might be increased. Thus this model provides both a stable reference frequency (from the TO), and can exhibit controlled flexibility (F_P).

We assume the TO is a non-linear oscillator with resemblances to those that may underlie circadian rhythmicities (Treisman, Cook, Naish and MacCrone, 1994). Such oscillators have the property that an external periodic force whose frequency lies in a limited range about the natural frequency of the oscillator, ω, may excite phase-locked oscillations. Also, if the two frequencies are different, but n periods of the applied frequency correspond to m periods of the spontaneous oscillation, where n and m are relatively prime integers, entrainment may occur over a limited range about this frequency. An applied frequency ω_f related to the oscillator frequency by $m\omega_f = n\omega$, will be referred to as a simply related frequency. Thus entrainment might occur near simply related applied frequencies given by $\omega_f = (n/m)\omega$. As the effects are strongest for low values of m and n, they might be observed, for example, near the applied frequencies $(1/4)\omega$, $(1/3)\omega$, $(1/2)\omega$, $(2/3)\omega$, $(3/4)\omega$, ω, $(3/2)\omega$, 2ω, and 3ω, if we set limits of 3 on n and 4 on m. Frequencies just below a given simple frequency, e.g. $\omega_f = (3/4)\omega$, would slow the pacemaker so that the number of pulses counted during a presented interval, say T_e = 1.0 sec, would be reduced, and the duration would be estimated low. Frequencies just above $\omega_f = (3/4)\omega$ would speed the pacemaker so that a high estimate of T_e would be given. Thus as the applied frequency is increased from just below to just above $(3/4)\omega$, and if T_e is estimated for each frequency, we would see a dip in the estimates succeeded by a peak as the applied frequency increases. We call a pattern of dips and peaks spaced in this way an interference pattern.

So the model could be tested by applying forcing frequencies and examining for such effects of entrainment on temporal performance. As TOs are probably protected from too ready entrainment to external frequencies, an effect is most likely if we employ strong and sudden pulse trains such as loud auditory clicks.

We can now examine some interesting questions. Can an interference pattern be shown in temporal estimation? If so, could it be used to estimate $F_{c,O}$? Can such patterns be shown for motor performance? Can physiological correlates of the oscillator frequency be demonstrated?

In the first experiment ("Expt 1") of Treisman et al. (1990) subjects estimated the durations of intervals (T_e) presented as an asterisk on a VDU screen simultaneously (within the accuracy allowed by the VDU scan rate) with a regular click train. Click rates from 2.5 to 27.5 Hz, at 0.5 Hz intervals, were employed. On each trial a click rate (from a set of 11 for the session) and a reference duration (from a set of two, "short" (S) and "long" (L) - L was always 250 msec longer than S) were randomly selected. The duration actually presented (T_e) was the nearest duration larger than the reference duration capable of coinciding with a click train at the selected rate. For example, for a 500 msec reference duration, T_e might be 571 msec accompanied by $N_a = 7$ clicks at 10.5 Hz. The mean S and L values of T_e for click rates from 2.5 to 7.5 Hz were 827 and 1067 ms, and for the higher click rates 536 and 784 ms. Thus T_e (and N_a, the accompanying number of auditory clicks) varied at different click rates.

We expect the estimate E to be a positive function of T_e, and it may also increase directly with N_a if each click increases the arousal of the CU. To separate these effects from the non-linear effects of click rate on the temporal oscillator that constitute the interference pattern, the regression of E onto T_e and N_a was found: $E = c_t T_e + c_a N_a + c$ (where c_t, c_a and c are constants), and the residuals about this regression were examined.

The residuals showed an interference pattern, consisting of dip-peak perturbations in E, with considerable similarity between different subjects. Results of a replication with a single subject are shown in Figure 2. The mean residuals are plotted against auditory click rate, separately for the first and second halves of the sessions for S (S1 and S2) and L intervals (L1 and L2). The positions of the main dips that were found in Expt 1 are indicated by vertical arrows.

The interference pattern replicates the results of Expt 1 and is very consistent from the first to the second half of each session, the main dips being identically positioned or displaced by 0.5 Hz. The S and L data are similar except that at low click rates the major S dip is at 4.5 Hz, while the main L dips occur at 3.5 and 5.5 Hz. Dips represent a slowing of the clock, giving a lower estimate of a fixed interval, peaks the reverse.

Does the spacing of the major dips match the spacing of the simply related frequencies expected for a non-linear oscillator of frequency ω [an example of which was given above: $(1/4)\omega$, $(1/3)\omega$, $(1/2)\omega$, $(2/3)\omega$, etc]? If so, the value of ω giving this pattern would be an estimate of

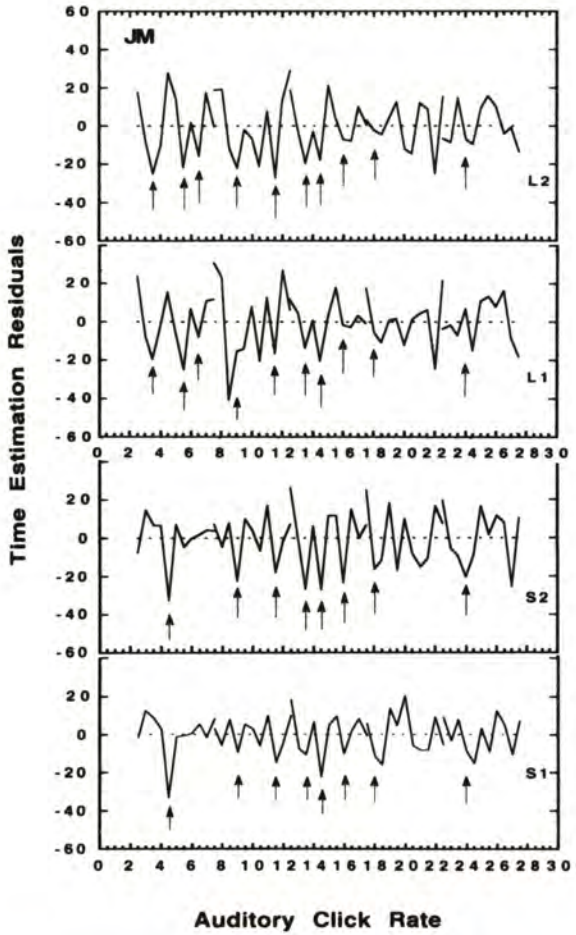

Figure 2. Time estimation results for subject JM (Treisman et al., 1990). Residuals plotted against click rate. Upper two panels: L durations; lower panels: S durations. S1 (L1) label the first halves of the S (L) sessions, and S2 (L2) the second halves. The arrows show dips obtained in Expt 1.

$F_{c,O}$. To examine this, a set of eight major dips (from the Expt 1 data) was taken to define the experimental interference pattern. A search procedure was then applied: for each candidate frequency from 1.0 to 100.0 Hz (at 0.1 Hz intervals) all the corresponding simply related frequencies (with limits of 3 on n and 5 on m) were generated. For a small range of candidate frequencies centered on 24.75 Hz only, 7 of

the experimental dips could be matched to a simple frequency so generated. (The probability of a random set of dips giving such a match was p = 0.013.) If the limit on m was extended to 10 an estimate of 49.5 Hz was obtained (Treisman et al., 1990). Larger limits on m gave no further estimates. A second experiment gave the estimate $F_{c,O}$ = 37.3 Hz. These three values correspond quite closely to 2, 3 and 4 times 12.4 Hz. Further studies showed that related interference patterns can be obtained using visual flicker (Treisman and Brogan, 1992), or substituting choice reaction time for time estimation (Treisman, Faulkner and Naish, 1992). The latter gave an interference pattern related to that for time estimation and from which $F_{c,O}$ = 49.8 Hz could be estimated.

These studies have answered some of our questions. We have found related interference patterns in time estimation and motor timing, and we have estimated $F_{c,O}$. It remains to ask: Can a physiological basis be demonstrated for these effects? Can we identify the output of the hypothesized TO as a component in the EEG?

The observations above suggest a way to examine this question. If the TO output is present as a component oscillation in the EEG, its frequency should decrease if clicks are presented at rates at which they reduce time estimates. However, even if such a component exists, how could we observe it? It would be like trying to determine whether, in a large orchestra playing a chord, one violinist has shifted the note he is producing.

Even if we could observe an effect, what pattern of changes would we expect to see? Our arguments suggest three models. (1) The first and simplest model would propose that there is a single TO producing a single oscillation in the EEG. (2) However, our model for the control of movement required multiple pacemakers, which suggests that the EEG may contain a set of click-sensitive TO outputs, corresponding to the different pacemakers. These would have similar frequencies, distributed, perhaps normally, about a mean value. We will call this the Single Distribution model of TO organization. (3) But even this may not be enough to account for our performance. Since we estimate intervals of very different orders of duration, we might require TO distributions centered at different frequencies. To facilitate coordinating and translating between them, their frequencies should be related, e.g. they might be centered at equal steps in the spectrum, like a series of harmonics - the Harmonically Related Multiple TO Distribution model.

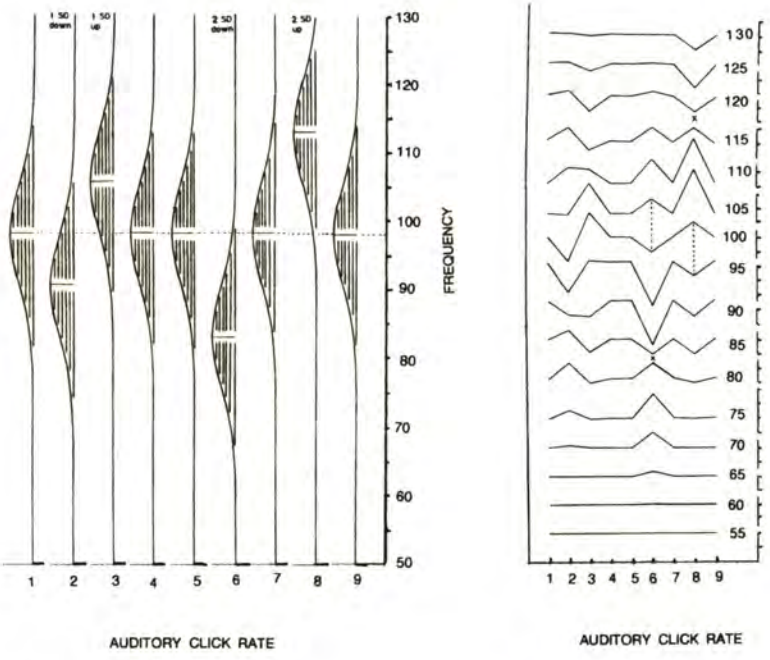

Figure 3. EEG_{sim}: Simulation of the effects of arousal pulses on a single click-sensitive temporal oscillator distribution. Left: a model of the EEG spectrum is plotted against frequency (on the ordinate). The plot is repeated for a series of click rates. Right: centroid frequencies of 5 Hz bands (labeled with the highest frequency in the band) plotted against click rate. Two 'diamonds' are marked by dotted lines and two 'crosses' by an 'x'.

To explore a method of analyzing the EEG that might allow us to demonstrate the presence of TO components in it, the Single Distribution model was simulated for arbitrary parameters, and the simulation output analysed as illustrated in Figure 3. On the left the power spectrum of the simulated EEG, "EEG_{sim}", is plotted (for "Auditory Click Rate 1" on the abscissa, and "Frequency" on the ordinate) in its resting state. What we see is a rectangular density function (plotted vertically, against Frequency, over the range of arbitrary values 50 to 130 Hz) representing non-click-sensitive frequencies in the spectrum of EEG_{sim}. Superimposed on this is a normal distribution of click-sensitive frequencies whose mean lies at 98 Hz (indicated by the horizontal

dashed line) and with SD = 7.5 Hz. This plot is repeated for "Click Rates" 2 - 9, some of which (4, 5, 7, 9) have no effect on the click-sensitive TO distribution. Rate 2 slows the TO outputs, shifting their frequencies one SD downward. Rate 3 shifts the distribution one SD up. Rates 6 and 8 cause 2 SD shifts.

The analysis on the right demonstrates these effects. For each click rate, the power spectrum is divided into 5 Hz bands, e.g. 1 - 5, 6 - 10. (Each band is labelled with its upper limit; e.g. the band from 51 to 55 Hz is labelled "55"). The centroid frequency (*CF*) or weighted mean of each band is plotted. For "Click Rate 1", which has no effect, the *CF* value in each band is the resting value. At "Click Rate 2" the downward shift changes the *CF* in bands from which power is removed or to which it is added. Thus in Band 100 (96 - 100 Hz), after the distribution's downward shift power densities are relatively greater in the lower part of the band (they were previously symmetrical) so that the weighted mean moves down, producing a local dip. In Band 110 power density is lost mainly from the lower part of the band so that *CF* rises, giving a local peak. In each band the *CF* values for the different click rates are joined to give a *CF* curve. (The unit on the right ordinate is 0.05 Hz.)

At click rate 6 the oscillations are slowed down 2 SD units (the mean frequency is reduced to 83 Hz), and at rate 8 accelerated. At "6" Band 100 (the resting mean is 98 Hz) shows a dip and Band 105 a peak. The dip and peak are joined by a dotted line to illustrate that together they form a "diamond". Dips are also seen in the bands below Band 100, down to Band 85 (which contains the new mean 83 Hz). Band 80 then shows a peak, so that 85 and 80 together form a "cross" (marked with an "x"). Thus a "diamond above a cross" configuration represents a downward shift of a distribution. At click rate 8, a "cross above a diamond" represents an upward shift. Similar but smaller effects are seen at rates 2 and 3. The diamond at "2" spans three bands, from 100 to 110.

An experiment was run on three subjects in which the EEG was recorded while the subjects estimated time intervals presented concurrently with clicks at rates varying from 2.5 to 17.9 Hz. The first 500 ms of the EEG during each presentation was subjected to power spectrum analysis (Treisman et al., 1994). The mean power spectrum for each click rate was divided into 5 Hz bands and the *CF*s plotted. Some results for subject PN are shown in Figure 4. Similar data were obtained from the other subjects.

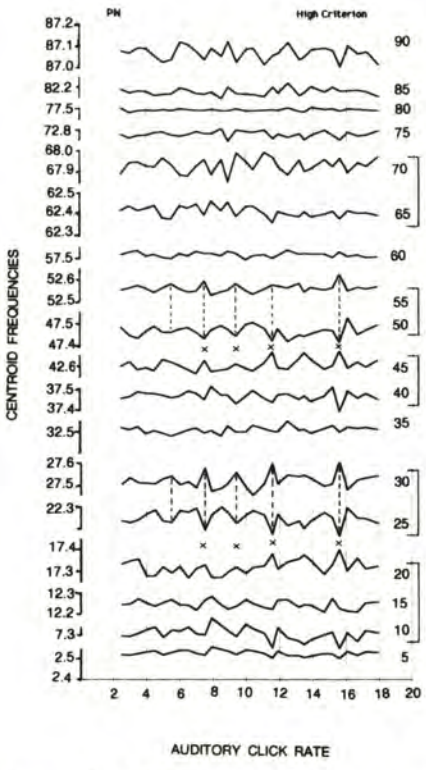

Figure 4. EEG data (subject PN). The centroid frequencies of successive 5 Hz bands up to Band 90 (the parameter is the upper limit of the band) plotted against auditory click rate. Diamonds between Bands 25 and 30, and 50 and 55 are indicated by vertical dashed lines. Crosses between Bands 20 and 25, and 45 and 50 are marked with an 'x'. Band Pairs are bracketed on the right.

The data show that auditory clicks can affect components of the EEG. The effects are patterned, not a general increase in noise. The patterns shown by some of the *CF* curves are similar to each other.

The time estimation results gave 24.75 Hz as one estimate of the $F_{c,O}$. This would lead us to expect that the *CF* for Band 25 (21 - 25 Hz) should show evidence of click-sensitivity: we should see dips and peaks as click rate varies, in a pattern with some resemblance to the time estimation interference patterns. Band 25 does show dips (marked by vertical dashed lines) at 5.5, 7.5, 9.4, 11.6 and 15.6 Hz. While this curve, taken alone, could be attributed to noise, the band above shows peaks at the identical click rates, an unlikely coincidence. The two bands between them present us with a series of "dia-

monds". Furthermore, at 7.5, 9.4, 11.6 and 15.6 Hz there are also "crosses" between Bands 20 and 25. This is not the pattern a single TO would produce. It is consistent with a distribution of click-sensitive TOs having similar $F_{c.O}$ values.

There is more to be seen in the figure. The earlier data also gave estimates of 37.3 and 49.5 Hz for $F_{c.O}$. These would predict interference patterns in Band 40, or Band 50. In fact the *CF* curves for both these bands show a pattern of dips similar to Band 25. Further, Bands 45 and 55 are similar to Band 30.

A pair of bands symmetrically bracketing diamonds will be referred to as a "Band Pair". The square brackets on the right of Figure 4 pick out five similar Band Pairs (one brackets three bands). Such recurrent patterns would be very unlikely to occur by chance. Band 25 is similar to Bands 10, 40, 50 and 65; its mean correlation with them is 0.836; with Bands 20, 30, 45, 55 and 70 it is -0.848; in each case $p < .0001$. While these calculations are post hoc, examining the same bands gives similar results for the other subjects.

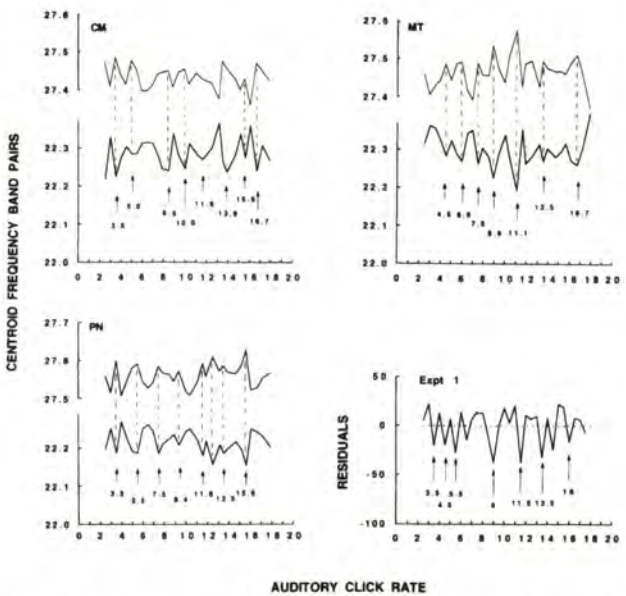

Figure 5. Bands 25 and 30 are shown for each subject. Dashed vertical lines indicate the locations of diamonds; where peaks and dips differ by one point, dotted lines are used. The mean time estimation interference pattern from Expt 1 of Treisman et al. (1990) is also shown.

For each subject a sequence of Band Pairs recurred in the spectrum, at almost identical positions, at mean intervals of 12.8 Hz. Thus similar patterns of click-sensitive effects occur in bands spaced at regular intervals in the spectrum, as the Harmonically Related Multiple TO Distribution model would require. Although subjects were consistent within themselves, there was more variation in the location of diamonds in the Band Pairs between subjects. Figure 5 shows Bands 25 and 30 for each subject, with diamonds between them indicated by dashed lines (dotted lines where peak and dip differ by 0.5 Hz). The mean plot of the residuals from Treisman et al.'s (1990) Experiment 1 (6 subjects) is also shown. Similarities between the *CF* interference patterns and time estimation and motor response patterns are discussed by Treisman et al. (1994).

To conclude, studies have been described that support the hypothesis that a temporal sensory system underlies time perception and the temporal control of action. The EEG results suggest that click-sensitive oscillators occur in the brain and form one component of the temporal system and that they are organized as distributions of TOs that recur at equal frequency intervals, of the order of 12 - 13 Hz. These parallel TOs may allow time measures recorded or produced at one frequency to be related to or coordinated with measures taken at another frequency (for example, in relating the production of bars in music to the durations of notes).

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