forehead-nose continuum, whereby the bridge of the nose is obliterated, by not showing any indentation, narrowing or other indications for it (Figure 1, NGr- and NGl-types).

For delineating a new syndrome, the present data would be greatly enhanced if additional information were available on the Ss's acumen in recognizing actual individual faces. They themselves would most likely be unaware of any disability in this respect, as are most prosopagnostic adults, who orient themselves by features other than the most involved upper portion of the face. Tests on face recognition have grave limitations too, as MEADOWS[17] emphasizes, moreso as clinically prosopagnostic patients often do well on such tests.

A review[18] of the neurological literature on brain-lesioned prosopagnostic patients shows some reports in which there also existed reading disturbances – at least in the beginning (JOSSMANN[20], HOFF and PÖTZL[21], FAUST[22], BODAMER[16], HEIDENHAIM[23]), while others did not report about such association (HÉCAEN et al.[18], WILBRAND[24], DONINI[25], FAUST[26]). In addition ENGERTH[19] discusses the case of a brain-lesioned prosopagnostic with 'reading disturbances'. Their specific pattern, though not commented upon, supports the here postulated analogy between the global manner of approaching the face pattern (especially its upper part 'eye and nose') and letters, syllables or words (frequently 'turning letters up or down', distorting words by reading and writing them 'in a reversed manner'). Another patient of ENGERTH[19] with cerebral contusion had a temporary defect in reading and drew a neolithic-like face configuration, though again not commented upon. (Supporting KLEIN's[5] hypothesis, this patient had also finger agnosia, acalculia and right-left distortion and drew a 'paw-like' hand.)

Thus, these brain-lesioned cases[19] also suggest that a *'de-differentiation'*[5] *in experiencing* hand and face reveals specific features, that are congruent with an early and more primitive utilization and cognition, respectively, of both these essential body parts.

Identification of a new clinical subgroup or new syndrome – aside from its heuristic value – opens the way for an early diagnosis with practical implications.

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Experimental Coupling of Crab (*Carcinus maenas*) Second Maxilla Neural Motor to an Alternating Current

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Summary. Movements of the crab gill bailer were entrained to an alternating current applied to the thoracic ganglion. There was little distortion of the muscle recruitment cycle, both absolute and relative coordination were observed, and the phase of the driven system in the driving cycle was a function of the difference between free running and driving frequencies.

The suboesophageal ganglion of decapod crustaceans generates a rhythmic pattern of motor activity driving the muscles of the 2nd maxilla in a restricted number of recruitment sequences to produce either forward or reversed beating of the gill bailer[2]. Loose bilateral coupling of the bailers in a preferred phase relationship has been observed[3]. Experimental coupling of the 2nd maxilla neural motor to an external signal could be used to locate the neural oscillator, and, if the coupling were tight, to set its period so that signal averaging techniques could be used for analysing small electrical events recorded in the neuropile.

The possibility of achieving an experimental coupling was investigated using *Carcinus maenas*. The animals were dissected to expose the suboesophageal ganglion and the muscles of the 2nd maxilla.

If the recruitment sequence is driven by quasi-sinusoidal variations in the membrane potential of a single oscillator neurone[4], a slowly alternating current might be a suitable entraining signal. This sort of signal was chosen for these experiments.

The entraining signal was applied to the dorsal surface of the ganglion through a suction electrode, orifice diameter 0.3 mm. Alternating current was drawn from a low impedance source and taken to the suction electrode through a 50 Ω series resistance. A silver chloride reference electrode encircled the suction electrode near its tip. The voltage developed across the electrodes and 50 Ω resistance was monitored using a chart recorder.

Entrainment was observed to signals of 0.3–11.0 V peak-to-peak; a signal of about 5.0 V peak-to-peak was generally used in these experiments. It is probable that with the concentric electrode system the electric field strength attenuated rapidly with distance from the suction orifice.

Electromyograms from 2 bailer muscles were also recorded, so it was always possible to determine whether the bailers were being driven in the forward or reverse modes. The results here are derived from forward mode recordings only.

A short section of a recording is shown in Figure 1. The muscle recruitment sequence became entrained to the driving signal within one cycle, although in other experiments the stable phase relationship was reached only

[1] I am grateful to Professor K. SIMKISS for providing facilities in the Department of Zoology, Reading University. Some of the equipment was provided by University of Otago Research Committee grant No. 37–097.
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after several driving cycles. It is evident that the driving signal did not seriously distort the muscle recruitment sequence observed while the bailer was free running.

Absolute entrainment as illustrated by the traces in Figure 1 occurs only over restricted ranges of driving frequencies in the neighbourhood of the free running frequency and simple multiples or submultiples of it. The preparations used in these experiments were spontaneously active only for about 30 min after dissection and the free running frequency changed during that time, generally increasing terminally. It was therefore more appropriate to determine the range of driving/free running frequency ratios over which a preparation could be entrained, than to determine the absolute frequency range of entrainment which would have varied during the lifetime of a preparation.

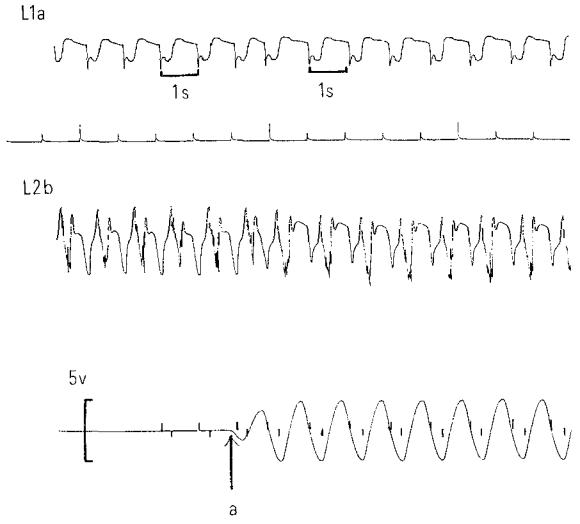

Fig. 1. Pen recorder trace of a typical entraining session. Driving signal switched on at a. Top: e.m.g., muscle L1a (nomenclature after YOUNG[2]). Middle: e.m.g., muscle L2b. Bottom: entraining signal voltage on which have been superimposed marks indicating the start of the e.m.g's. Upward strokes, L1a, downward strokes L2b. Time scale: sec.

Spontaneously active preparations were subjected to the driving signal for sessions of 10–20 cycles. The preparation was allowed to free-run between sessions, and the mean of the free running frequencies before and after each session was taken as an estimate of the 'free running' frequency of the neural motor during a session. Free running frequencies in the order of 1.0–2.0 Hz were commonly observed, and driving frequencies in the range of 0.5–5.0 Hz were used. A number of frequencies within this range were presented in a random order during each experiment.

Results from one such experiment are presented in Figure 2. The driven frequency/free running frequency ratio is shown on the ordinate. Most of the points do not lie on the line where the ordinate value is unity, i.e. where the driven frequency (the bailer beat frequency during a session) equals the mean free running frequency. On the abscissa is shown the driving frequency/free running frequency ratio. Points in the range of 0.84–1.16 on the abscissa lie exaclty on the line for which the driving and driven frequencies are equal. Some other points lie on the line for which the driven frequency is half the driving frequency.

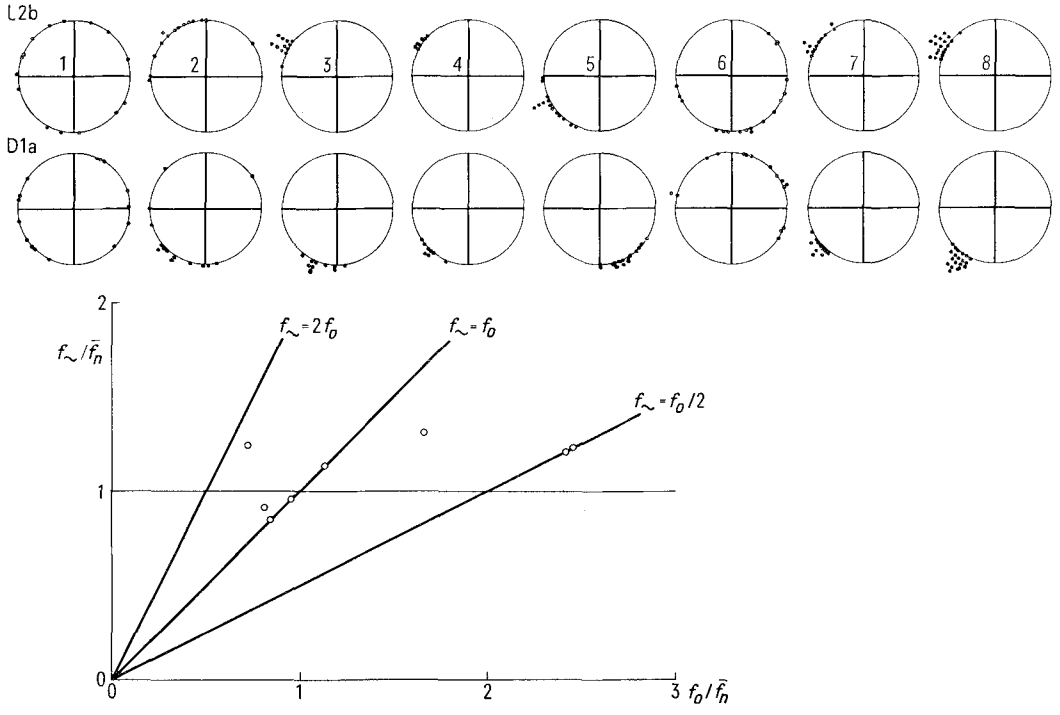

Fig. 2. Coupling over a range of driving and free running frequencies. Graph: ordinate, driven frequency/mean free running frequency; abscissa, driving frequency/mean free running frequency.
Circular histograms: Muscle recruitment phase in driving signal cycle. Top, muscle L2b; bottom, muscle D1a. Positive-going zero voltage crossing at 3 o'clock, histograms read anticlockwise. In histograms 7 and 8 one histogram cycle is equal to two driving signal cycles, the muscle being entrained to alternate signal cycles.
Abbreviations: $f\sim$, bailer beat frequency during an entraining session (= driven frequency); $\bar{f}_n$, mean of the bailer free-running frequencies before and after a session; f_0, entraining signal frequency (driving frequency).

Each point on the graph is a result from a single session. Above the graph are pairs of circular phase histograms which are arranged in the same order as points on the graph. That is, the first pair of histograms shows the phase of the driving cycle at which muscles L2b and D1a were recruited in each cycle during the session from which the first point on the graph was derived. Histograms 3, 4, 5, 7 and 8, relating to sessions in which the driven frequency was equal to the driving frequency or was half the driving frequency, show that the muscle recruitment cycle became phase locked to the driving current during these sessions. During entrainment the normal L2b–D1a phase angle of about 80° was maintained.

During sessions 1, 2 and 5, absolute coordination of the sort described for the other sessions was not observed, but the neural motor exhibited a tendency to maintain a preferred phase relationship with the driving signal, slipping rapidly through regions of unfavourable phase relationship in the manner described by VON HOLST[5] as relative coordination.

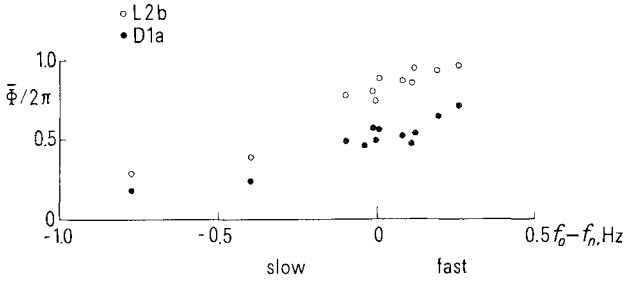

Fig. 3. Regression of coupling phase angle on frequency difference. Ordinate: phase of muscle recruitment in driving signal cycle (positive going zero voltage crossing = zero phase). Abscissa: difference between driving frequency (f_o) and mean free-running frequency (f_n). Abbreviation: ∅, mean recruitment phase angle during an entraining session (radians). Other abbreviations as in Figure 2.

In Figure 3 results from a different set of experiments are presented. The phase in the driving cycle at which muscles were recruited is shown on the ordinate. Each point is obtained from a single session during which forward beating was absolutely entrained to the driving signal for a minimum of 10 cycles, and represents the mean recruitment phase angle during that session[6]. The difference between the driving frequency and the mean free running frequency for each session is plotted along the abscissa. The neural motor evidently becomes advanced in the driving cycle when it is forced to run slowly, and retarded when it is speeded up.

Four features of these results indicate that the driving signal entrains the neural motor rather than that it drives the muscles independently of the neural motor. These are, 1. the maintenance of intermuscular phase angles during entrainment; 2. the restricted range of driving frequencies over which absolute coupling is observed, 3. the relative coordination observed outside the absolute coupling frequency bands, and 4. the phase retardation observed when the system is made to run fast[7].

It is not possible to decide whether the neural oscillator is entrained directly to the applied current, or whether that current modulates activity in a set of neurones to which the neural oscillator can become entrained. Nevertheless now that experimental entrainment has been demonstrated, it should be possible using a small electrode to map the regions of the ganglion having the greatest sensitivity to the driving signal. In this way one could hope to locate the neural oscillator, control its period, and even obtain an indication of its size and structure.

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The Oxygen-Linked Hydrogen Ion Binding (the Haldane Coefficient) of Bovine Hemoglobin

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Summary. The Haldane coefficient (the amount of the oxygen-linked hydrogen ion binding of hemoglobin)was determined in bovine erythrolysate (Hb concentration = 13.5 m*M*) by means of the differential titration method with varying PCO_2 from 0 to 74 mm Hg and pH from 6.0 to 8.5 at 37 °C. The maximum value of the coefficient was found to be 0.49 m*M* per m*M* Hb at PCO_2 = 0 and pH 7.20. With increasing of PCO_2, the coefficient became smaller in all ranges of pH studied. The coefficient under the conditions of pH 7.20 and PCO_2 = 45 mm Hg that are normally prevailing in the interior of bovine erythrocytes was 0.31.

At a physiological pH hemoglobin releases protons as O_2 binds (the alkaline Bohr effect). The magnitude of this effect is represented by the Haldane coefficient expressed as $-\delta Hb\text{–}H^+/\delta Hb\text{–}O_2$. The Haldane coefficient of human hemoglobin has been reported to be 0.47 under the conditions of pH 7.20, PCO_2 = 34 mm Hg, DPG/Hb_4 = 0.84 and Hb concentration (on a monomer basis) = 12 m*M*[2]. DPG stands for 2,3-diphosphoglycerate. With oxygenation of blood, an increase in the negative charges of the hemoglobin due to the alkaline Bohr effect leads to a decrease in the pH of the interior of erythrocytes at a given plasma pH, the magnitude being proportional to the Haldane coefficient[3]. Recently, we had an opportunity to measure the erythrocyte pH for bovine blood[4]. It was 7.24 on oxygenation of the blood and 7.252 on deoxygenation at plasma pH of 7.4, the difference between the two values being not significant. This finding for the bovine blood made us wonder whether the Haldane coefficient of bovine hemoglobin is very small at physiological pH and PCO_2. Results of studies on the Haldane coefficient for bovine hemoglobin and blood are not

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