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B. B. LLOYD

The Concept of Regulation in Physiology

M. LINDAUER AND H. MARTIN

Special Sensory Performances in the Orientation of the Honey Bee

Discussions

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THE CONCEPT OF REGULATION IN PHYSIOLOGY

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The rapporteurs of this conference have been asked to set a broad framework for subsequent discussion and not to cover topics in detail and entirety. After a brief discussion of homeostasis I shall therefore concentrate on some quantitative and speculative aspects of respiratory regulation, describing controller and material equations, and briefly discuss frequency of breathing and the effect of high altitude. The problem of physiological set point is then related to the respiratory regulation of pH. Regulation in exercise and voluntary interference with ventilation are then briefly discussed.

Energy, material and information pass through the individual animal at various and varying rates during its existence. Body temperature and posture are typical resultants of a balance between energy input and output, and body weight a typical resultant of a material balance, and are examples of what Cannon [1] called homeostasis, which may be defined as the maintenance of a variable that is subject to throughput within a tolerable and usually fairly narrow range of an average value. Regulation may be defined as the sum total of devices and processes by which homeostasis is achieved.

The information throughput is different from those of energy and matter. Wiener [2, p. 121] has likened the activity and development of a human brain during its life to a single run on a computer: memory and learning, whether neural or immunological, are scarcely examples of homeostasis. And the transmission and multiplication of genetic material by reproduction is largely restrained by the environment, and not subject, except perhaps in civilized man, to homeostatic regulation [see 3 for a different view], which is based on negative feedback, whereas much sexual behaviour, such as mating and parturition, provides examples of positive feedback, which is otherwise rather rare in normal biology, though perhaps not in pathology.

The variables which show homeostasis are legion and the ones most easily and frequently studied are chemical concentrations in the blood. These may be classified into respiratory, ionic, acid-base, nutritional, excretory and hormonal, each of these classes showing more or less overlap with the others; and the list of hundreds of normal values in any reference book of medical chemistry [4] is a list of homeostases, which are also seen in growth and organ size, in the hydrostatic problems of the circulation of the blood, and in the regulation of the frequency, amplitude and wave form of cyclic processes, such as sleeping and

waking and the sexual rhythms, which often profoundly influence supposedly steady levels.

Claude Bernard "Le plus illustre physiologiste de notre époque" (the most illustrious physiologist of his epoch) according to Vulpian [5, **1**, vii], once wrote [5, **1**, 340] that "Tout acte d'un organisme vivant a sa fin dans l'enceinte de cet organisme" (every act of a living organism has its aim within the ramparts of that organism), which may have prompted J. S. Haldane [cited in 1 & 3] to state that "No more pregnant sentence was ever framed by a physiologist" about Bernard's [5, **1**, p. 113] proposition that "La fixité du milieu intérieur est la condition de la vie libre, indépendante". This most famous of physiological generalizations comprises three notions of great importance, the free life, the internal milieu, and the latter's constancy. Physiologists have spent little time debating whether Bernard's 'La vie libre' referred to the mobility of a mammal or to the political freedom of man, but in the last century there has been immense and intense physiological investigation of the identity, composition and regulation of the internal environment, of which Bernard [5, **2**, p. 5] stated 'C'est le sang; non pas à la vérité le sang tout entier, mais la partie fluide du sang, le plasma sanguin, ensemble de tous les liquides interstitiels, source et confluant de tous les échanges élémentaires.' (It is the blood; not indeed the whole blood, but the fluid part of the blood, the plasma, including all the interstitial liquids, the source and confluence of the elementary exchanges.)

This concept of the internal environment is quite complex. Our skin exists in atmospheric air, but the alveolar air in chemical contact with blood in the lungs contains much less oxygen (14 as opposed to 21 %) and far more CO_2 (6 as opposed to 0.03 %) than atmospheric air. We can thus be said to live in an atmosphere containing a partial pressure of oxygen, P_{O_2}, of 100 torr, and a P_{CO_2} of 40 torr, which at once differentiates us from the creatures in the sea, where both these pressures are usually lower.

The arterial blood exchanges gases with the alveolar air, so that for most purposes in the normal man we can accept the equality of the alveolar and arterial partial pressures (P_A and P_a) of oxygen, nitrogen, CO_2 and water, but, just as we really live in alveolar, not atmospheric, air, our tissues live in venous rather than arterial blood, with a P_{O_2} less than 50 and a P_{CO_2} above 40 torr. The immediate environment of cells is the tissue fluid, which tends to be slightly more venous than venous blood, with lower P_{O_2} and higher P_{CO_2}, and the intracellular fluids, in which the cellular components such as mitochondria function, may have P_{O_2}'s of only a few torr, and at the actual points in the cell where oxygen is converted to water its concentration tends to zero. The chemical reduction of oxygen supplies energy to the cell, and the continuous but fluctuating energy demands of the cell depend on an appropriate transport of O_2 from air to the mitochondrion, and of CO_2 from the enzymes of the Krebs cycle to the air. The maintenance of appropriate flows of CO_2 and O_2 is the primary task of ventilation and cir-

culation, and in discussing the concept of regulation in physiology I shall speak mainly about this task. This needs no apology, for not only did Bernard [5, **2**, p. 141] state that 'La respiration est le phénomène le plus caracteristique de la vitalité, c'est-à-dire de l'être en activité vitale', (Respiration is the most characteristic phenomenon of vitality, that is to say of the being in vital activity) but the physiology students being examined in Oxford a fortnight ago were asked to discuss the proposition that 'Respiratory physiologists are the most quantitative neurophysiologists'. Respiration has indeed been much investigated, as its inputs and outputs are relatively easy to measure; it operates over a 30-fold range of activity, and it provides the most characteristically quantitative examples of chemoneural interactions.

We are still in doubt, owing to inadequate experimentation, as to what chemical variables directly affect the ventilation, but it is generally agreed that the H^+ concentration of the blood is effective at the chemoreceptors in the aorta and the fork of the carotid artery, and that there is an effective H^+ concentration inside the cranium. It is also accepted that the partial pressure of oxygen in the arterial blood, $P_{a}o_2$, has a potent effect on the arterial chemoreceptors. The experimentally observed relations between chemical factors and ventilation $\dot{V}$ have been summarized in various equations, of which the following is a recent, though still controversial, example [6].

$$\dot{V} = h\{\psi(\lambda + \log H_a^+/H_{a\theta}^+)/(P_{a}o_2 - \gamma) + \mu + \log H_c^+/H_{c\theta}^+\}. \tag{1}$$

$\dot{V}$ represents the total air breathed out by a subject in unit time (l./min.) and is easily measured by means of a gas meter, while h is partly a size parameter (an elephant will breathe more than a mouse having the same blood chemistry), and partly a measure of overall sensitivity.

The first term inside the curly bracket is meant to represent the activity of the chemoreceptors. These small bodies, situated on the aorta and the carotid artery, fire more nerve impulses to the brain when the pH or Po_2 falls in the arterial blood reaching them. The term shows a linear function of the logarithm of blood hydrogen ion, $(\lambda + \log H_a^+/H_{a\theta}^+)$, multiplying a reciprocal function of oxygen $\psi/(P_{a}o_2 - \gamma)$, and represents the notion, which is still being investigated, that the product of these terms generates the impulse traffic in the chemoreceptor nerves. This traffic adds to two central terms, μ representing a residual nervous effect independent of chemical stimuli, and a final term containing H_c^+, a central hydrogen ion concentration, perhaps in cerebrospinal fluid or brain interstitial fluid. When H^+ falls below the threshold value H_θ^+ in either log term the effect is taken to be zero rather than negative.

We now turn from these informational relationships to the metabolic or material relationships that normally exist between ventilation and the respiratorily important variables. A simple example is the equation relating Pco_2 with ventilation:

$$\dot{V}(P_{A}\,co_2 - P_{I}\,co_2) \simeq j\dot{V}co_2. \tag{2}$$

This states that the product of ventilation and the difference between alveolar and inspired P_{CO_2} is approximately proportional to the metabolic production of CO_2, so that if $\dot{V}$ is doubled at constant $\dot{V}_{CO_2}$, $P_{ACO_2} - P_{ICO_2}$ is halved, and so on, a relationship we term the metabolic hyperbola. This means that if $\dot{V}$ changes for any reason, such as a change in one of the terms in the right-hand side of equation (1) there will usually be a change in P_{ACO_2}, which is itself a main determinant of H_a^+ and H_c^+ and hence of $\dot{V}$. The form of the equations shows that when $\dot{V}$ rises, P_{ACO_2} approaches P_{ICO_2} and hence H_a^+ and H_c^+ tend to fall, causing a depression of ventilation. This negative feedback is an example of the myriad of negative feedback arrangements in physiology. A similar negative feedback applies to oxygen, for which we may write

$$\dot{V}(P_{I\,O_2} - P_{A\,O_2}) \simeq j\dot{V}_{O_2}. \tag{3}$$

When $\dot{V}$ rises, $P_{IO_2} - P_{AO_2}$ falls, so that, at constant P_{IO_2}, P_{AO_2} rises. This reduces $1/(P_{aO_2} - \gamma)$ in equation (1), so that the stimulus is reduced and $\dot{V}$ tends to fall. In short, knowing P_{IO_2}, P_{ICO_2} and $\dot{V}_{O_2}$ ($\dot{V}_{CO_2}$ is usually close to it in value) and the basal characteristics of the blood and c.s.f. of the subject, we should be able from equations (1), (2) and (3) to predict the steady-state values of $\dot{V}$, H_a^+, H_c^+ and P_{aO_2}. The non-steady state is more complicated, and a masterly treatment of the relationships corresponding with equations (2) and (3) in the non-steady state for lungs, blood, tissues, brain and c.s.f. is to be found in the paper of Grodins, Buell and Bart [7].

Their paper, like our discussion so far, treats ventilation as a steady process, represented by a steady current of air passing over the lung surface, but breathing, as Yamamoto and Raub [8] amusingly point out in an intriguing theoretical paper, is of course rhythmic, involving V_T, the tidal volume or amplitude, and a frequency f, such that

$$\dot{V} = fV_T. \tag{4}$$

Hey et al. [9] have shown that over a wide range of ventilations with a variety of respiratory stimuli (increased body temperature is an exception, causing m to increase) there is a linear relation between $\dot{V}$ and V_T of the form

$$\dot{V} = m(V_T - k). \tag{5}$$

The parameter m has the dimensions of a frequency, min^{-1}, and it is highly correlated with the sensitivity-size parameter h of equation (1), which has the dimensions $l.min^{-1}$. This high correlation implies some important overlap between chemical and neural phenomena, and raises the question of the origin of the rhythm of ventilation and of its optimal frequency. The latter is related to work of breathing, to the volume and resistance of the respiratory tubes that do not exchange gases with blood, and with the notion of length-tension appropriateness

[cf. 10, 11]. Mathematical ideas of optimality are of growing importance in biology [12].

Although equations such as (1), (2) and (3) provide a basis for respiratory regulation, they are far from adequate as explanations of every important respiratory phenomenon. Altitude and exercise provide the most striking examples of physiological respiratory alteration, and both provide interesting examples of regulatory peculiarities.

At altitude P_{IO_2} and hence P_{AO_2} are reduced, and equations (1), (2) and (3) permit a prediction of $\dot{V}$ in the steady-state which is reasonably borne out by experience. The increase in $\dot{V}$ without an increase in P_{ICO_2} will by equation (2) lower P_{ACO_2}, and this will cause $H_a{}^+$ and $H_c{}^+$ to fall below their threshold values $H_{a\theta}^+$ and $H_{c\theta}^+$, so that the log terms are effectively zero. Whereas an abnormal alkalinity of the blood is only slowly rectified by renal excretion of HCO_3^-, an abnormally alkaline c.s.f. is rapidly rectified by a secretory lowering of HCO_3^- so that the pH of c.s.f. is soon restored to its usual figure of about 7.32, at which $H_c{}^+$ is sufficiently high to promote ventilation and hence a raised P_{AO_2}. This extremely interesting phenomenon, by which c.s.f. pH is maintained and P_{AO_2} considerably augmented at altitude, was hinted at by Kellogg [13] and confirmed by Mitchell, Severinghaus and their collaborators [14], when they heroically analysed their own c.s.f.'s at an altitude of 3800 m.

A well-known effect of altitude is the increase in red cells per unit volume of blood. This is yet another example of negative feedback, the hypoxia calling up a response in the blood-forming system which tends to negative the effect of hypoxia. There are, however, two disadvantages of polycythaemia, the obvious one being the rise in blood viscosity, which can overload the heart and circulation. Secondly, and this is less obvious, because haemoglobin is the main blood buffer polycythaemia depresses the slope of the blood log H^+, log P_{CO_2} line and thus tends to cause a reduction in ventilation which could lead to further hypoxia and hence more haemoglobin, a positive feedback to which there is no defence except blood-letting or a return to lower altitudes. This is a speculative contribution to the aetiology of the undesirably high polycythaemia of chronic mountain sickness, but brings out the point that where an element in a normally effective homeostatic mechanism develops positive feedback with an open-loop gain greater than 1, disease and perhaps death ensue. This question is discussed in Milhorn's [15, pp. 102, 358] comprehensive text, in which control theory is systematically expounded in a physiological context, at much greater length than in the useful shorter book by Bayliss [16].

The phenomenon by which c.s.f. pH tends to be jealously regulated near 7.32 without a steady-state error, by a combination of ventilation, secretion, brain bloodflow and metabolism, raises the whole question of set-points in physiological regulation. Why is the blood pH 7.4, the c.s.f. pH 7.318? On the former, Priban and Fincham [17] have made the ingenious and stimulating suggestion that the

normal pH of blood is set at 7.4 because this is the pH at which haemoglobin is most effective as a physiological acid-base regulator. Haemoglobin is a buffer system which accepts protons and lessens pH change as CO_2 diffuses into blood from the tissues, but this simple physicochemical buffering is further supplemented by the Bohr-Haldane effect, by which oxyhaemoglobin is a stronger acid than reduced haemoglobin, and oxygen is driven off haemoglobin by an increase of CO_2 or H^+ concentration. Priban regards this effect as being most marked at a pH of about 7.4, and suggests that this property of haemoglobin acts on the ventilation to bring blood pH back to the figure at which haemoglobin is most effective, using the device known to control engineers as hill-climbing to provide a mechanism in detail.

This suggestion is open to serious criticism as a full explanation of respiratory regulation, but it contains the important idea that the set-point to which a regulatory system returns may be embodied in the properties, physicochemical or otherwise, of a large molecule evolved in more primitive evolutionary conditions. Once evolved the large molecules can tend to improve their biological effectiveness by promoting the selection of an internal environment which enhances the biological value of their physicochemical peculiarities. Priban's idea is also close to the notion that an enzyme could during the course of evolution bring its intra- or extracellular environmental pH to the value at which it is most effective as a catalyst.

A further suggestion as to the respiratory regulation of blood pH comes from the work of Rahn [18], who has shown in cold- and warmblooded animals that over a range of body temperatures the pH to which the blood is normally brought by regulation varies with temperature in parallel with the pH of neutral water, so that the pH of blood is about 0.6 above that of physicochemical neutrality. Albery [19] has argued that this may be related to the change with temperature of the pK of some biologically important buffer system.

Yet another aspect of blood pH regulation is the possibility that it is set to give a minimum effective total of $[H^+]+[OH^-]$, both H^+ and OH^- being powerful hydrolytic catalysts for biologically important polymers such as proteins, esters and polysaccharides. If we can argue that because H^+ is about twice as mobile as OH^-, and that the ideal compromise pH is reached when $[H^+]$ is 0.5 $[OH^-]$, we should expect at 37° a biological pH of 6.95, 6.8 being the pH of neutral water. This seems rather far from that of blood, but is probably close to intracellular pH's [20].

The point emerging at this stage of our discussion is that the pH's of blood, interstitial fluid and cells undoubtedly show homeostasis and are subject to regulation, but that the set point to which they regulate is not some standard pH tucked away in a little compartment to which reference is continually made, or a pointer reading as in a chemical engineering plant, but may be embodied in the physicochemical or biochemical properties of key macromolecules. The

links between these properties and the regulatory devices have yet to be worked out, and until this is done in detail this notion remains speculative.

In discussing the pH regulation of the blood it may be convenient to think of factors tending to disturb it and of those tending to correct it. A low-protein vegetarian diet tends to cause alkalinity, and a high-protein diet acidity by the oxidative production of sulphuric acid: these are relatively slight effects dealt with in the long run by the kidney. The ingestion of any sort of food leads to the secretion of acid in the stomach, and Dodds [21] showed that the resulting alkalinity of the blood is dealt with by a depression of ventilation, a consequential retention of metabolic CO_2, and a partial restoration of pH to the norm. Haldane [22] showed that 10 or 15 g of ammonium chloride could be ingested daily without apparent ill effect and that it gave an effect very similar to that of the administration of hydrochloric acid. This causes acidity of the blood, an increase in ventilation, a consequential lowering of the CO_2 concentration of the blood and hence a partial restoration of the pH to normal.

As long as the daily ingestion of 10 g of ammonium chloride continues, the blood pH remains at about 7.3, this giving the steady-state error of 0.1 pH needed to drive the kidney to dispose of the 10 g of ammonium chloride into an acid urine: similarly the oral ingestion of sodium bicarbonate can lead to a steady-state blood pH of 7.45, this being the pH at which the kidney will dispose of the bicarbonate into an alkaline urine. This dietarily imposed range of 0.15 pH is quite large, and implies that the kidney operates as a proportional controller with a steady-state error, that is as if the rate of excretion of acid is a direct function of the acidity of the blood, and there is no evidence, from the persistence of abnormal pH's when the diet is abnormal, that integral control operates to bring the final steady-state error to zero. The kidney seems also to lack derivative control responding to the rate of change of blood $[H^+]$, but this is not a defect, for the most precipitate change in blood pH comes during and after the most violent exercise, such as the running of 400 m. in 45 seconds. Energy for this comes partly from oxidation and partly from the conversion of some 40 g of glycogen to lactic acid in the muscles. The concentration of lactic acid in the blood may rise from 10 to 150 mg per 100 ml, and, if there were no ventilatory adjustments, the pH would fall to 6 or less. As we all know from personal experience there is during or immediately after violent exercise a rise in ventilation (partly predictable from equation (1)) which drives off CO_2 and moderates the fall in pH so that the lowest value reached is about 7, and then during recovery there is a steady and rapid return to normal as lactic acid is removed from the blood, mainly by the liver. If the kidney responded immediately to the low pH of exercise, we should be in grave difficulties. Lactic acid, a valuable source of calories under aerobic conditions, would be lost in the urine, and the metabolic cost of violent exercise would rise ten-fold, a situation of no advantage to primitive man and of benefit only to the obese of the twentieth century (if they could be persuaded to make use of it).

This short-term and very effective ventilatory regulation of blood pH depends on the volatility of the main acid product of metabolism, CO_2. By hyperventilation we can lose roughly one molecule of CO_2 for each molecule of lactic acid added, though once again, as with the longer-term kidney, there will be a steady-state error, showing the the ventilation-blood-H^+ system seems to work as a proportional controller without integral control. The importance of the blood H^+-CO_2-ventilation system is its rapidity of action, which is second only to that of the first line of defence, the physicochemical buffers of the body fluids, of which haemoglobin and bicarbonate are the most important.

When $P_{A}co_2$ is raised by inhalation of CO_2, the blood supply to the brain generally rises through the agency of a roughly proportional control mechanism by which the diameter of the brain arterioles is a direct function of $P_{A}co_2$, so that $P_{c.s.f.}co_2$ rises less than $P_{A}co_2$. This means that the stimulus to raise c.s.f. [HCO_3^-] by secretion is smaller than it would otherwise be. If this regulation of brain bloodflow did not exist, raising blood CO_2 would entail a secretory rise in $HCO_{c.s.f.}$, a change which could lead to serious underventilation if the extra CO_2 were suddenly removed. The raising of brain bloodflow in response to high $P_{A}co_2$ is thus a valuable short-term regulation by a rapidly reversible process, similar in function to the respiratory as opposed to renal adjustment of blood pH during the lactacidaemia caused by violent exercise. It should be noted, however, that the final regulation of c.s.f. pH to 7.32 in a wide variety of conditions [14: but see 23] may imply integral control without a steady-state error.

Exercise is the most normal and least understood stimulus to ventilation. It can raise $\dot{V}co_2$ and $\dot{V}o_2$ of equations (2) and (3) 25-fold above their resting values, and this must cause an increase in either or both of $\dot{V}$ and $(P_{A}co_2 - P_{I}co_2)$. It is obviously possible in principle to calculate $\dot{V}$ in exercise from equations (1), (2) and (3) and the appropriate subsidiary equations relating H^+ to Po_2 and Pco_2, but it is found in practice that $\dot{V}$ is nearly always greater than the prediction, and indeed greater than the value predicted when values actually measured in exercise are substituted in the controller equation. It is therefore suggested that exercise increases μ, possibly by impulses coming from active limbs, and we [24] have found in man, following in the wake of M. Dejours [see e.g. 25] that the increase in $\dot{V}$ at the beginning of exercise is independent of the pre-existing Po_2 and Pco_2 in the chemical background, which argues strongly that this initial exercise effect is an increase in μ. We [26] are currently investigating short-term and steady-state aspects of the hypoxia parameters (ψ and γ) in exercise, which now appears to evoke a change in the form of equation (1). The hyperventilation seen in exercise over and above the prediction of equation (1) is obviously of great interest, and has been attributed to feed forward, showing up as an increase in μ. The absence of changes (steady-state error) in $P_{a}co_2$ and $P_{a}o_2$ in moderate exercise points also to integral control.

Professor Dejours has always grasped the most interesting nettles, seldom if

ever being stung, of respiratory physiology, and I see from the programme of the June 1967 meeting of French-speaking physiologists that he is now working on respiratory aspects of speech. Speech is the most human of motor activities, using one of the most primitive of quasi-autonomic activities, respiration, for its purposes: and it has for some time been known [27] that during speech the respiratory muscles are used primarily not for maintaining the homeostasis of blood pH or brain P_{O_2}, but for keeping a constant pressure of gas against the vocal cords. This capacity for over-ruling the usual chemoneural respiratory rhythms is of immense biological importance for drinking, swimming, talking, spitting, yawning, grimacing, laughing, sobbing, cleaning one's teeth, snorting, singing, diving, sneezing, sniffing, coughing, blowing, sucking, eating, drinking and for rejecting some hostile atmospheres and inhaling others, and it is interesting to speculate as to where the will to hold one's breath, presumably cortical in origin, conflicts with the chemical drive which ultimately forces one to breathe. Measurements during and after breath-holding provide, albeit at a low level, objective and reproducible data on the will and its pharmacology [28, 29], and it is of interest that Dr Bhattacharyya has found in our laboratory that at the breath-holding breaking point the movement of the chest is affected by the blood gases, but the effects, though fairly consistent for an individual, differ widely between subjects.

By definition, but by nothing else, the gaps in this presentation cannot be its most salient features. By largely confining it to respiratory regulation we have been able to go into some detail, though the treatment of the only controller equation we have discussed has been brief and dogmatic. The unsolved problems in regulatory physiology are the details, largely requiring physiological experimentation, of these control equations, the conceptual problems of the non-steady-state, in which Grodins has given an admirable lead, and the search for the physical counterpart of the set point to which many homeostatic systems return, with or without steady-state error.

The problem of ventilation in exercise remains partially unsolved, and the central nervous links between the chemical input and the rhythmic neuromuscular output remain largely unexplored. There is no doubt that the physiology of regulation has already benefited from the limited application of the techniques and ideas of mathematics, physics, engineering and physical chemistry that has so far been made. During the next twenty years a systematic extension of these applications to the scores of regulations which are now merely verbally described will transform this branch of physiology into a rigorous quantitative discipline. Regulation was made for man and his free life, not man for regulation. May this neo-Bernardism bring many more physically trained theorists into biology!

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SPECIAL SENSORY PERFORMANCES IN THE ORIENTATION OF THE HONEY BEE

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Adaptation, regulation and homeostasis, these fundamental performances of the living organism, are only possible when the latter can gain information about conditions and changes in its environment. Every animal, including human beings, can however register only a diminutive fraction from the *objective* environment through its "interpreters"—the sense organs. In order to understand the behaviour of a living organism it is important to recognize the *subjective* part of its environment.

Three examples, drawn from the orientation behaviour of the bee, may us reveal a section of a world, inaccessible to our sensory system.

1. The suncompass-orientation

Bees use the sun as a compass. It is important to realize, that they use the sun not only for their private orientation but mainly as a point of reference in their mutual communication: When a forager bee in the darkness of the hive indicates the location of a food source by the tail-wagging dance [1] the angle between sun and goal is transposed into the field of gravity. Astonishingly enough—our bees communicate by dances even if the sun is hidden behind clouds and if they can see only a patch of blue sky. That is possible because the light that comes from the blue sky is polarized showing definite relations to the sun's position with both the direction of vibration and the intensity of polarization. V. Frisch discovered this twenty years ago and today we know that there exist true analyzers for polarized light in the bee's eye. The fine structure of the rhabdomeres gives the prerequisites for the dichroic absorption of polarized light [2, 3, 4, 5].

Another question is whether the bees are familiar with the whole pattern of polarization on the blue sky and its regular arrangement around the sun. If you keep young unexperienced bees in a closed room under artificial light and let them fly out for the first time after three weeks than they are unable to use the sun as a compass. It takes at least 5 days, or 500 foraging flights until they learn to use the moving sun and the pattern of polarization on the blue sky as a compass. Another experiment demonstrates that this suncompass is not innate but must be learnt by the bees: I brought a bee colony from Ceylon to Poona (India) and from Ceylon to Munich. The bees behaved in the northern

hemisphere as if they would be still under the Ceylonese sky e.g. they calculated the sun movement anticlockwise instead of clockwise; it took 40 days until they got familiar with the orientational cues on the new sky [6, 7].

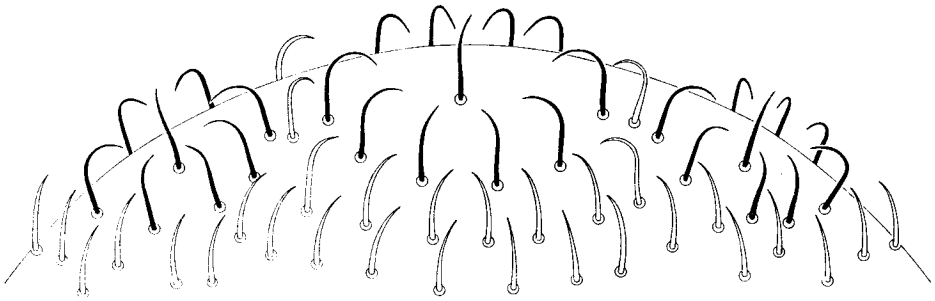
A prerequisite for suncompass-orientation is an exact time memory. The bees have to know the azimuth of the sun every minute of the day for calculating the changing angles between the moving sun and the fixed position of the food source. Beside the findings of Beling [8], Wahl [9] ,and Renner [10, 11] the following experiment may surprise every one how precisely the time sense works in suncompass-orientation: In specific situations [6, 12] you can induce "marathon-dances"; in this case the bees continue dancing for many hours without leaving the hive. Therefore they cannot control in the meantime the changing angle between sun and goal; time memory and knowledge of the sun movement exclusively have to save this critical dilemma. Even at night with artificially induced dances the bees show where on the sky the sun could be found; and —the human observer hardly can believe it—they don't calculate the sun's movement at an average speed: they take into account the seasonal variations of the change of azimuth of the sun [13].

2. Problems of orientation during building activity

A masterpiece of the regulated communal work in the beehive is the building of the honeycomb, a performance of orientation marvelled at since long time not only by biologists but mainly by mathematicians, and physicists, in front of all by REAUMUR. In this situation the mechanical senses are alone responsible. The honeycomb must be built in the darkness of the hive; without the guidance of optical clues the builders have to measure the overall dimensions of the space within the cells and have to control the angles between the comb-cell walls as well as their inclination towards the foundation. Of the different control mechanisms in the building activity we know only two:

1.) The bristle fields in the neck of the bees are used as gravity receptors to orient the cells in the gravity field. If you glue these sensory bristles with paraffin in all bees of a colony not a single comb cell can be built (even during 4 weeks); but if you remove the paraffin, the bees start with construction of combs immediately.

2.) A group of highly specialized pressure receptors on the antennal tip controls the thickness of the cell wall, which is strictly 72 μ in worker cells and 95 μ in drone cells (fig. 1a/b). These receptors work on the following principle: after the foundations of the wall are laid down the builders move along one side of the wall, continually making pressing movements with the rounded edges of their mandibles. The cell walls get indented about 5 μ deep under controlled pressure of about 4 dyn—just as when one draws two fingers over a tightly stretched curtain. The tips of the antennae—by bipolar scanning movements—register the change dynamically during the indenting and return movements. At the same time they perceive through the sensory hairs on their tips any counter



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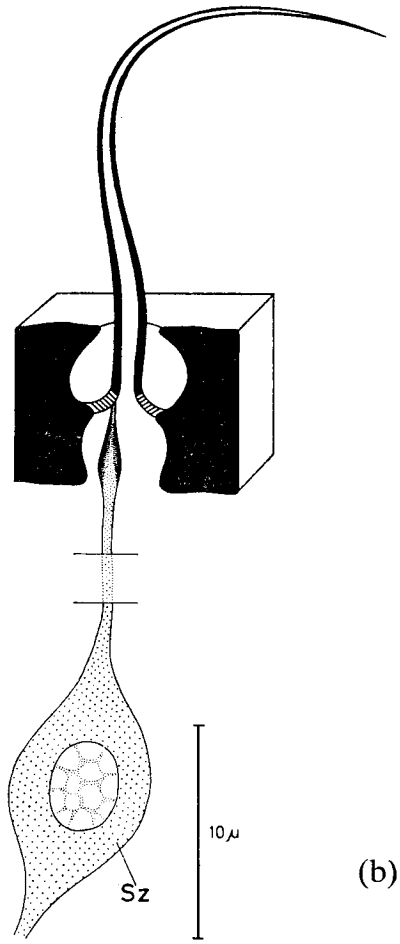


(b)

Fig. 1. Three groups of specialized sensory hairs on the tip of a bees' antenna; in the center of each is a straight bristle surrounded by 7–10 hooked bristles in a circle which measure the counterpressure of the wax wall, when it is dented by the mandibles. Note in fig. 1b that the chitinous membrane on the basis of the bristle allows a downward movement of the hair for less than 5 μ. This is exactly the range in which the builders indent the cell walls.

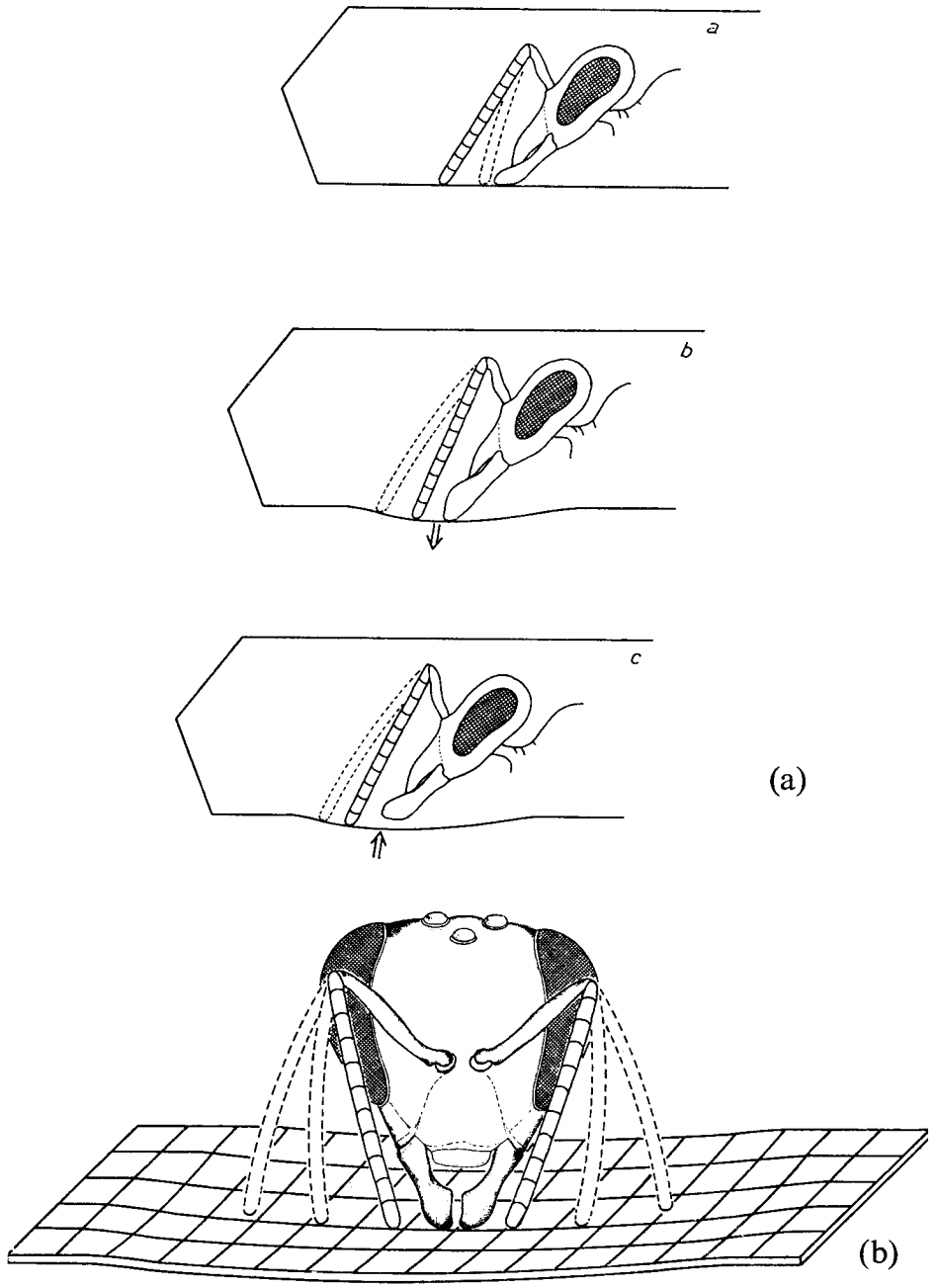
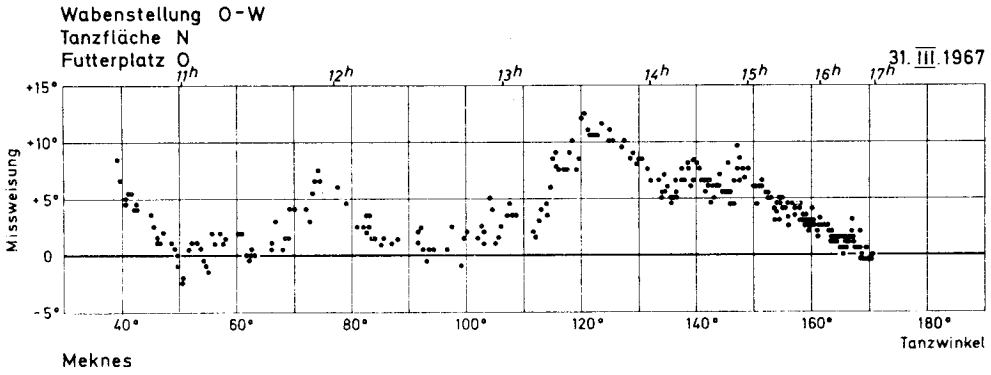


Fig. 2. (a): Pressing movements of the mandibles (a, b), the elastic aperiodic returning movement of the wall (c) and the simultaneous control of the counterpressure by the tips of the antennae provide the information to measure the thickness of the wall.
(b): The tips of the antennae measure three parameters relevant to the thickness of the comb-wall: counterpressure, change in shape and its speed.

pressure (fig. 2a/b). This control mechanism—in order to be effective—has two prerequisites:

1.) One has to use always the same raw material; in fact, this is the case: the building material is intrinsic wax (palmitic acid ester and myricil-alcohole) which is transformed into an amorphic stage by kneading them together with the secretion of the mandibular glands.

2.) The temperature must be kept exactly at 35° C because in this temperature range only the wax wall—after mandibular pressure—shows "aperiodic displace-



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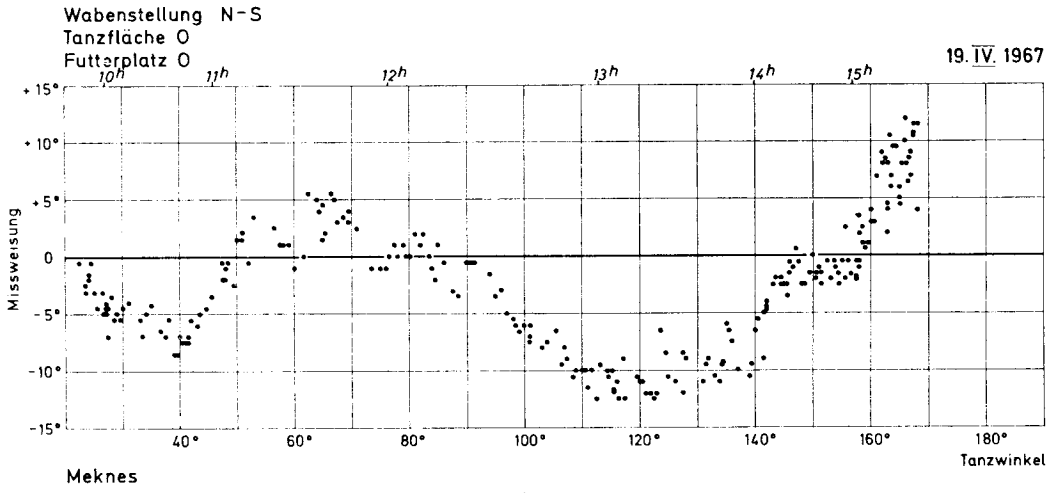


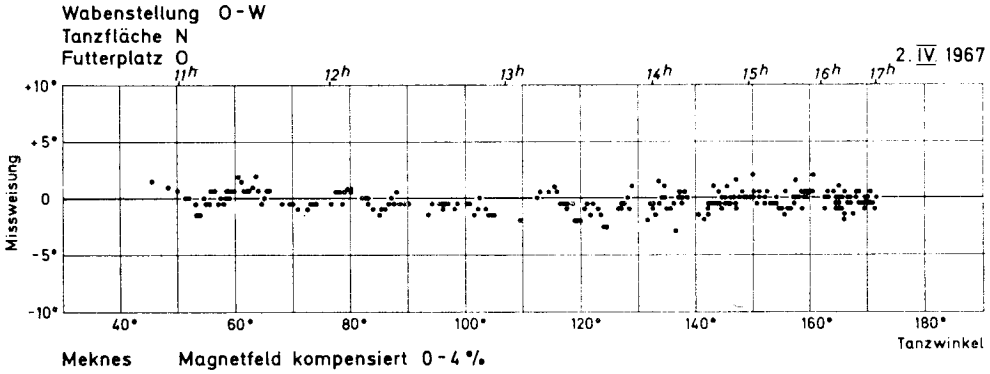
(b)

Fig. 3. Bees visiting a feeding table 400 m eastwards of the hive announce by their dances on the vertical comb the angle between sun and goal (transposed into the gravity field). The abscissa gives the changing angle between goal and the advancing sun throughout the day. All spots on the 0-line correspond to this angles performed by the dancing bees indicating the direction to the goal without mistake. All spots above or below the 0-line are deviations. The diurnal curve of these deviations depends on the position of the dance floor in the earth magnetic field. (In fig. 3a the dance floor faced northwards, in fig. 3b eastwards.)

ment". In fact since long time it is known that the temperature in the building cluster is kept day and night exactly at 35° C [14].

3. Orientation in the gravity field influenced by the earth's magnetic field

In the last ten years studying of orientation of dancing bees we encountered much trouble when the bees apparently made small genuine "mistakes". But there was a system in these errors: As I have mentioned already the optical angle between sun and goal is transposed into the gravity field; the wagging line of the dancer keeping the same angle with the perpendicular. When one measures, for a whole day long, this dance angle on the vertical comb, than—as the angle between the advancing sun and the goal changes the dance angle deviates in a characteristic course up to 25° from the expected normal (fig. 3a). The recruits in spite of this



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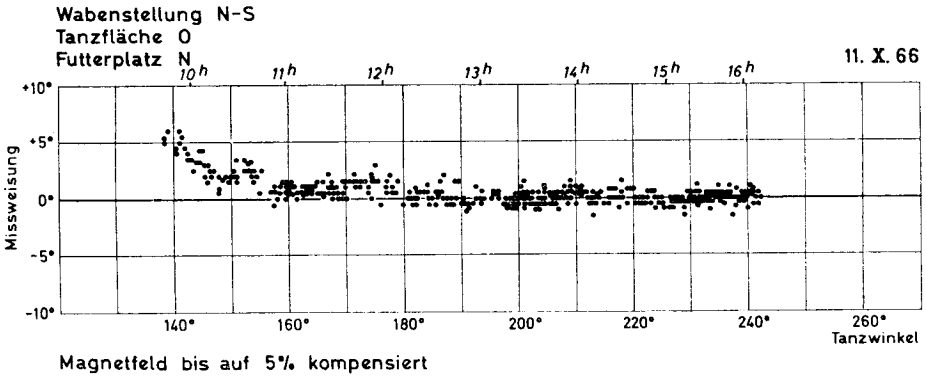

(b)

Fig. 4. The experimental situations in fig. 4a and 4b are identical to those of fig. 3a and 3b respectively. In both cases the dance floor was put in the center of a Helmholtz-drum and the earth's magnetic field was compensated up to 4 percent. The bees dance almost without deviation —they transpose the angle between goal and sun exactly into the gravity field throughout the day.

indicated deviation search for the right direction without misorientation. The communication system therefore in the "bee language" is correct, it demands however the right interpretation. To our astonishment we found the last year, that this diurnal „deviation" curve ran a quite different course when the vertical "dance floor" was put at different position in the earth magnetic field (fig. 3a/b). In the same way this deviation curve alters when the bees from Frankfurt are taken to Marocco where the earth's magnetic field has a different intensity and inclination. (Frankfurt: total intensity 0,43 Oersted; inclination 64°. Marocco: total intensity 0,41 Oersted; inclination 49°.) The deviations disappear almost completely when the hive, with its dancing bees, is put under a Helmholtz drum and the magnetic field is compensated (fig. 4a/b). The results will be discussed in detail elsewhere [15].

From this it seems that the bees are receptive to the fields of the earth magnetic force. We do not yet know the receptive mechanism for this nor its exact biological meaning. We hope the bees will give us some chance to enter in this unknown field step by step.

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DISCUSSIONS

H. C. LONGUET-HIGGINS: Perhaps one could be told how big the measurements of the vertical squares are. The deviations shown are deviations between the angle of the dance and the correct angle for locating the flowers?

M. LINDAUER: It is just at the 0 line.

H. C. LONGUET-HIGGINS: Your interpretation, then, is that the bees make a composite function of the earth's magnetic field and the polarization of the light from the sky?

Dr. MENDELSSOHN: Just one short question. It looks immediately of course, since the acting mechanism is a polarization of the light and is affected by magnetic field, that there should exist somewhere in the mechanism of the bee a rotation of polarization by a magnetic field. Has it been tried to see what happens if you do not just compensate the magnetic field but put a rather strong magnetic field in another direction, I mean, can you force the bee that way.

M. LINDAUER: It has been done. But I mentioned that when they go from Francfort to Meknes, the total intensity is different. Then the curve is different.

F. HALBERG: La figure 1 est intéressante d'un point de vue historique. Elle a été publiée à Leipzig, en 1840, par Gauss et Weber dans "Resultate aus den Beobachtungen des Magnetischen Vereins im Jahre 1839". Cette figure représente des courbes de déclinaison magnétique. Gauss et Weber ont utilisé des informations recueillies en différents points du globe, et, en particulier, à Milan, à Berlin et à Uppsala.

Malgré le déplacement géographique Milan–Berlin–Uppsala, les courbes sont presque identiques entre elles. Vous voudrez bien remarquer que ces graphiques sont publiés sans analyses complémentaires, par exemple, sans faire appel à la méthode des moindres carrés. Ce qui est intéressant est que cette publication a été sanctionnée par Karl F. Gauss, le "roi des mathématiques" et père de la méthode des moindres carrés, bien que les résultats n'aient pas été soumis à une critique statistique. En effet, la figure ne comporte pas d'étude de la variation statistique des valeurs représentées. Une telle étude était réellement superflue même pour ne discuter que de "l'harmonie des courbes", c'est à dire, sans vouloir essayer de quantifier les composantes du phénomène.

Les aspects statistiques du géomagnétisme ont été discutés d'une façon élégante