Orientation by weighted randomness

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Abstract

This paper deals with the problem of how motion control can be achieved in very simple systems under a minimal amount of preconditions. The system's design therefore is as minimal as possible, reflecting the basal reflex-arc as observed in biological systems. The model for the movement of the agent is a multiplicatively modified random walk and thus does not represent a diffusion process of Langevin type. The mobile agent shows a reliable and fast homing behavior towards a defined area and finally stays in some defined neighborhood of this area. Moreover, obstacle avoidance is shown to be an immediate result of the system's properties.

Keywords: bio-mimetic system's architecture, stochastic motion control, homing behavior, obstacle avoidance

1 Introduction

Motion, including spatial movement and growth, is regarded as a universal feature of biological systems. Although motion and its control may be realized by very different mechanisms in different organisms, the guess is that there

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exist only very few principles of motion control which are fundamental to all these different levels, from uni-cellular organisms such as protozoa or bacteria, up to multi-organismic organisms like invertebrates or higher animals. Due to the complexity of even simple biological systems, a common approach has been to regard their motion as a random process and to consider motion control by "trial and error" (see Marken & Powers¹ and Murray²). The approach proposed in the following is based on a diffusion-like process (random walk), but differs form Langevin-models in a fundamental way. The basic random walk is extended by a multiplicative term, which corresponds to some (deterministic) internal mechanism of the system, rather than to an externally superimposed force term. As such the dynamics of the system is due to some "weighted" random walk (Reimann & Mansour³). The corresponding weight is shown to direct the motion towards an externally defined target and gives rise to obstacle avoidance as a simple consequence.

The model to be proposed in this paper is strongly inspired by biological systems: The fundamental functional architecture underlying our approach was deduced from observations of the ontogenetical as well as the phylogenetical development of nervous systems (Reimann⁴). A well-known example is the basal reflex as can be observed in vertebrates for example. This architecture appears to be fundamental for the persistence of biological systems and therefore is proposed to be analogously realized on very different levels of biological organization.

The key assumption involved is that this agent possesses "internal states" (essential variables in the terminology of $Ashby^5$), that have to be kept within certain given bounds during existence (for the relevance of homeostasis for physiological processes see Cannon⁶, Cannon⁷ or Adolph⁸). It is shown that this homeostasis conditions directs the motion of the agent in a signaling field. Both, simple homing behavior as well as obstacle avoidance, are shown be immediate results of the internal homeostasis condition of the agent. This result suggests that motion, spatial movement as well as growth, may be regarded as results of compensations of exogenously induced perturbations of the intra-systemical homeostasis condition.

2 Motivation of the model

Our paradigmatic example for the design of an agent is the basal reflex-arc as observed in recent animals, see Figure 1. The same functional architecture can be observed to be analogously realized in cells like nematocysts and thus is not restricted to neural systems. Accordingly, we design an Agent as a system which consists of three functional components: a sensory pole S, an effectory pole E, and an intermediary component I. (We call an open entity *intermediary*, if its state can be determined by a strictly internal measurement and if it is functionally interposed between the sensor and the effector.) Concerning the reflex-arc, the sensor of the agent corresponds to the set of sensible ganglia cells,

its effector is the set of moto-neurons, while the intermediary component can be identified with the set of interneurons.

— Figures 1 and 2 to be inserted about here —

These functional components are coupled in a particular way: Along the SIG-NALLING PATHWAY $\mathbf{F} : \mathbf{S} \to \mathbf{E}$, sensory activity may induce effectory activity directly, so that this pathway mediates the re-action of the AGENT to external signals. Parallel to this pathway, there exists a second one $\mathbf{M} : \mathbf{S} \to \mathbf{I} \to \mathbf{E}$, which causes the self-modulatory dynamics of the system. Both pathway differ concerning their respective function: While sensory activation may directly induce effectory action along \mathbf{F} , no effectory activity is induced by \mathbf{M} . This difference is most clearly seen by considering the effect of neural activity in *nematocysts*: Without any neural influence, the effector of a nematocyst, the *cinocil*, becomes activated, i.e. hurled out, if the mechano-sensor is activated by a sufficiently strong physical stimulus, while the effect of neural activity neither is to activate the sensor, nor to activate the effector. In fact, the effect of neural activity is to increase the threshold of the mechano-sensor. In other words, the action of the pathway \mathbf{F} is to alter the probability for the sensor to become activated. In this sense, this pathway may be called TRANSFER-MODULATING.

As a technical example, one may think about a simple **mobile robot**, which can move in a minimal environment. In order to enable the agent to move freely in its environment, we assume that its motor **E** has an appropriate number of degrees of freedom. According to classical mechanics, its spatial state if given by its spatial coordinates $Q \in \mathbf{R}^d$, and its momentum $P \in \mathbf{R}^d$, where *d* denotes the dimension of the environment. The action of its motor is to change its spatial state due to

$$\mathbf{F}: \begin{pmatrix} Q \\ P \end{pmatrix} \mapsto \begin{pmatrix} Q' \\ P' \end{pmatrix} := \begin{pmatrix} Q+P \\ \mathbf{D}(\alpha) P \end{pmatrix}, \tag{1}$$

where $\mathbf{D}(\alpha)$ denotes a rotation of the momentum around some randomly chosen angle $\alpha \in (0, 2\pi]$. The corresponding dynamics is simply a random walk in \mathbf{R}^d . As our key assumption, we assume that the AGENT has an internal component \mathbf{I} , whose states are called the *internal states* of the AGENT. The role of internal state or *essential variables* was already mentioned by R.W. Ashby⁵. A simple model for the adaptive regulation of cells by modulation of sensitivity was analyzed in Reimann⁹. More general considerations of the biological background can be found in Reimann⁴.

The internal states are supposed to be affected by the signals, being induced at the sensor of the AGENT according to some function g, so that to each position $Q \in \mathbf{R}^d$ corresponds an internal state $x = g(Q) \in X$. Let Q' denote the agent's next spatial position due to the dynamics defined above. Then the internal state x' corresponding to this new position is a function of the coordinates (Q, P). As such the dynamics of the AGENT's internal state is closely related to its spatial movement.

As a key assumption about the internal state set, we assume that there exists a particular subset $Y \subset X$ of internal states, which is called the "homeostatic range" of the AGENT. The definition of this set is part of the definition of the system and refers to basic properties of the AGENT. For example: The metabolism of cells is phosphat-oriented, i.e. due to the breakdown of chemical energyequivalents, nucleotide-triphosphats, there always exists a certain amount of free anorganic phosphat in the cell. This poses intracellular constraints on the ionic content of the cell in that if, for example the amount of Calcium is on a too high level, Calcium-Phosphat $(CaPO_4)$ would be formed, which in turn would damage the cell lethally. Therefore the strong need exists to control the amount of Calcium on a sufficiently low level, at least for longer times. Accordingly, the condition for intracellular Calcium is due to basic physico-chemical properties of the cellular components and its organization. Biological research has elucidated a variety of systemic variables which are homeostatic, for example see Kennedy¹⁰, Lubmann¹¹, Westbrook¹², Cheek¹³, Dawson¹⁴. Accordingly, homeostasis, i.e. to keep the AGENT's internal state close to its homeostatic range during its time development, appears to be closely related to the persistence of the system (for the relevance of homeostasis to physiological processes see Cannon⁶, Cannon⁷ or Adolph⁸). Since biological systems are thermodynamically open, the homeostasis of their internal states is permanently perturbed so that the maintenance of homeostasis is a result of regulatory processes, which permanently re-constituted the "internal milieu" of the system. As such, homeostasis is due to the action of self-regulatory mechanisms compensating long-lasting perturbations of the internal states and therefore is a systemic property.

As the distance measure, define the distance between the internal state $x \in X$ and the homeostatic range $Y \subset X$, i.e. d(x, Y) = 0 if and only if $x \in Y$, i.e. if the internal state is homeostatic. A direction P is regarded as "GOOD", if the internal state related to the new position Q' is closer to the homeostatic range than that related to the former position Q. This "weight" is formally defined as:

$$c(Q, P) := \begin{cases} +1 & \text{iff } d(x, Y) \ge d(x', Y) \\ -1 & \text{else,} \end{cases}$$
(2)

Suppose that the actual position of the AGENT is (Q, P). Then define the forward cone

$$K_{+}(P) := \{ p \in \mathbf{R}^{d} : a \le \langle p, P \rangle \le P^{2} \},$$
(3)

where $\langle \cdot, \cdot \rangle$ denotes the ordinary scalar product on \mathbf{R}^d and $0 \le a \le P^2$. Analogously, K_- is defined by the property $a \le -\langle p, P \rangle \le P^2$. *a* is related to the opening angle of the cone: $\gamma_a = \arccos(\frac{a}{P^2})$. Obviously, if a = 0, then $\gamma_a = 0$, so that $p \parallel P$, while if $a = P^2$, then $\gamma_a = \frac{\pi}{2}$. The modified model then is:

$$\mathbf{F}_{c}: \begin{pmatrix} Q \\ P \end{pmatrix} \mapsto \begin{pmatrix} 1 \\ c(Q, P) \end{pmatrix} \begin{pmatrix} Q+P \\ \mathbf{D}(\alpha) P \end{pmatrix}, \tag{4}$$

where τ has the same meaning as before, but the rotation angle α is randomly chosen from the interval $[-\gamma_a, +\gamma_a]$. In words: the AGENT proceeds moving

in its former direction, $P' \in K_+(P)$, if this direction is GOOD, otherwise the movement of the robot is reversed, $P' \in K_-(P)$. The random variable α represents the "internal noise" of the robot in that its new direction is randomly chosen within $K_+(P)$ or $K_-(P)$. If $a = P^2$, the dynamics due to (3) is completely deterministic, i.e. the robot either maintains or precisely reverses its former direction.

The opening angle α can be regarded as a measure for the "flexibility" of the robot: for a = 0, the motion of the robot is restricted to only one dimension, while for a > 0, the robot can move in two dimensions. Therefore for $\gamma_a = 0$, the robot reaches the target only if its initial direction is appropriate, while for $\gamma_a > 0$, the robots reaches the source almost surely. Thereby the opening angle only affects the convergence rate, i.e. the mean time the robot needs to reach the target, Krüger & Reimann¹⁵, in that, more precisely the robot's distance from the source decreases linearly in time like

$$\sim const \cdot t + o(t),$$

the constant being a convex function of the opening angle $\gamma_a > 0$.

To induce noise to a (deterministic) system, a Langevin force term is commonly *added*, which has to fulfill certain stochastic properties (vanishing average and δ -correlation). In fact, mapping (3) does not describe a diffusion process of Langevin type, in that the modulation of the random walk in (3) is *multiplicative*:

$$F_c(Q,P) = c_*(Q,P) \cdot F(Q,P), \qquad c_*(Q,P) := (1, c(Q,P))^T.$$
 (5)

Therefore, F_c can not be written as a random walk F to which an external driving force is superimposed. In fact, the dynamics of the AGENT may be regarded as a random dynamics in a gradient field - but this gradient field is internally defined, rather than externally: By the mapping $g: U \to X$, the external signalling space U is mapped to an internal space X, on which the "force term" is defined. Therefore, this force can be regarded as being "generated" by the system itself.

3 Homing and escaping behavior

In the following, only some particular properties of the dynamics of the abovedefined system (3) will be considered. The model proposed represents an extension and generalization of the approach mentioned by O.E. Holland and C.R. Melhuich¹⁶. A mathematical analysis of the mapping including the existence and the stability of the invariant set is beyond the scope of this work.

Figure 3 displays the trajectory of the agent (as defined above) due to the mapping F_c , in the presence of a signalling field emitted by some source located inside the circle. The simulation was done for a very simple model. As signalling source, we defined a light bulb of constant illumination. Accordingly,

the strength of received signals follows a monotonously decreasing function of the distance between the AGENT and the signal source. The action of the AGENT's effectors, e.g. the motor, is assumed to leave the velocity unchanged. Moreover, the effects of received on the internal state was assumed strictly monotonous. Since the homeostatic range was assumed to consist of only one point, the circle displayed in Figure 3 represents all positions in which the internal state is homeostatic.

It is well-known that diffusion processes in a plane have the mixing property according to which each the trajectory of a diffusive substance will meet any arbitrarily small neighborhood of any point in the plane after a sufficient long time. Therefore, "homing behavior", i.e. reaching the circle, is trivially achieved by a random-walk dynamics. In fact, as apparent from the Figure 6, the agent reaches the circle after a couple of iteration steps, its time development being due to F_c . For the same reason, a diffusing AGENT will leave every disc of finite radius after some time $t \approx d^{-2}$. According to the dynamics defined by F_c , the AGENT will not escape from a finite neighborhood of the target, but will remain in some finite distance to the circle for all time. In fact, this is a major difference between the purely random walk F and the "weighted random walk" F_c . Actually, the spatial trajectory of the AGENT shows an oscillation around the circle, its amplitude being dependent on the initial velocity of the agent.

— Figure 3 to be inserted about here —

In contrast to a purely random walk, the trajectory due to F_c is composed by "perturbed" straight lines, the perturbation being due to the randomness in the choice of the rotation matrix. Nevertheless, segments have to be lines (in the mean), because according to the weight function a certain direction is maintained as long as this direction is "good", otherwise the direction will be reversed. Even seemingly "straight" trajectories can not be expected in pure random walks, in fact, the mean curvature of random trajectories is related to the range out of which the rotation angle α is taken.

— Figure 4 to be inserted about here —

4 Obstacle avoidance as a simple consequence

The AGENT shows obstacle avoidance as a direct result of its definition. No further assumptions had to be made. The reason for this seemingly non-trivial behavior is in fact simple.

For the simulation, a non-transparent obstacle was inserted into the light field and, as a zero-order approximation, the "shadow region" behind this obstacle was assumed to have zero intensity (see Figure 5). Therefore, the AGENT receives the same amount of light at any position in the shadow region. According to the "homeostasis" requirement posed, the intensity measured by the AGENT must be non-zero. Therefore, the AGENT "escapes" this shadow region, as it escapes infinity. Further, according to (2), its mean forward-direction of its motion is maintained until it reaches the boundary of the shadow region, up to stochastical perturbations. At the shadow border, the intensity measured has some non-zero value according to which the AGENT's becomes directed towards the source as in the former cases, avoiding the shadow region. This establishes a simple obstacle avoiding behavior as an immediate result of the basic properties proposed. In particular, no modifications of the initial model (3) were made.

— Figure 5 to be inserted about here —

Due to the assumption about the shadow region of having zero intensity, the possibility exists that due its initial direction, the AGENT may hit the obstacle. But this "failure" is due to the crude and physically unreasonable model: In fact, the intensity distribution of light behind an obstacle is not constant but exhibits a gradient which increases towards the boundary of the shadow region. Therefore in a more realistic setting, the AGENT's motion is in fact directed even inside the shadow region.

5 Conclusion

We proposed a new system architecture for AGENTS due to biological systems. Its essential new feature is the existence of an intermediary component, by which the functional properties of the AGENT are changed during its dynamics. In this paper, only the modification of the effectory pole was considered, the modulation of the AGENT's sensory pole, sensitization and/or desensitization has been considered elsewhere, Reimann⁹. The goal of this internal dynamics is to maintain the internal state of the AGENT within given bounds for almost all times. This requirement has been shown to direct the random motion of the system. The system's control is realized by a kind of *internal* feed-back, according to which to the external loop $\mathbf{E} \to \mathcal{E} \to \mathbf{S} \to \mathbf{E}$, a second loop $\mathbf{E} \to \mathcal{E} \to \mathbf{S} \to \mathbf{I} \to \mathbf{E}$ is superimposed in parallel¹. The model was shown not to be of *Langevin* types, but is due to a *multiplicatively* modified random walk. This parallel architecture not only establishes a fast and reliable homing behavior, but also causes obstacle avoidance as an immediate result. Both, the hardware equipment, as well as the computational effort needed is minimal, homing behavior as well as obstacle avoidance were achieved without using any directional information, in fact the AGENT's design is rotationally symmetric and its sensor is omni-directional, i.e. it only measures scalar values like intensities.

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 $^{{}^{1}\}mathcal{E}$ denotes the environment of the AGENT

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Figure 1: Schematic representation of a reflex-arc showing two parallel pathways, one which is the sensory-effectory coupling between sensory ganglia cells \mathbf{S} and moto-neurons \mathbf{E} , whereas in parallel interneurons \mathbf{I} are activated due to the activation of the sensible ganglion cell altering the postsynaptic state of the motoneurons inhibitorilly.



Figure 2: Schematic representation of an AGENT, \mathbf{E} and \mathbf{S} denoting its effectory and sensory pole, respectively, while \mathbf{I} denotes its intermediary component. The intermediary component functions by modulating the signal transducing pathway \mathbf{F} along the pathway \mathbf{M} .



Figure 3: Phaseplot of the weighted random motion of the agent and the timedevelopment of its spatial distance from the source, due to equation 3. The AGENT's initial position is close to the source.



Figure 4: Phaseplot of the weighted random motion of the agent and the timedevelopment of its spatial distance from the source, due to equation 3. The AGENT's initial position is far away from the source.



Figure 5: Obstacle avoidance due to (3). The left picture shows a phaseplot of the weighted random motion of the AGENT, while the right one displays the time-evolution of its spatial distance from the source.