Functions and Mechanisms: A Perspectivalist View

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Abstract. Though the mechanical philosophy is traditionally associated with the rejection of teleological description and explanation, the theories of the contemporary physiological sciences, such as neuroscience, are replete with both functional and mechanistic descriptions. I explore the relationship between these two stances, showing how functional description contributes to the search for mechanisms. I discuss three ways that functional descriptions contribute to the explanations and mechanistic theories in contemporary neuroscience: as a way of tersely indicating an etiological explanation, as a way of framing constitutive explanations, and as a way of explaining the item by situating it within a higher-level mechanisms. This account of functional description is ineliminably perspectival in the sense that it relies ultimately on decisions by an observer about what matters or is of interest in the system that they study.

1. Introduction. In its most austere and demanding forms, the mechanical philosophy insists on a disenchanted world explicable without remainder in terms of basic causal principles. Though mechanical philosophers differ from one another about which causal principles are fundamental (size, shape, and motion for Descartes, attraction and repulsion for du Bois-Reymond, conservation of energy for Helmholz), they univocally reject explanations that appeal to vital forces and final causes. Austere views such as these are commonly associated with the idea that the mechanical world is an aimless machine, churning blindly, without its own end or purpose, and also with the apparent historical opposition between functions and mechanisms as conceptual tools for understanding the natural world.[2]

Contrast this historical opposition of function and mechanism with the state of play in early 21st century physiological sciences, such as neuroscience. In such fields, the language of mechanism is literally ubiquitous, and most scientists continue to demand

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[2] Westfall describes the world of the mechanical philosophy as a "lifeless field knowing only brute blow of inert chunks of matter" (1973, 31;see also Westfall 1971; Shapin 1996). Historians have suggested that the opposition of mechanism and Aristotelian explanations in terms of forms and final causes oversimplifies the diversity of perspectives one finds in the 17th Century and beyond. See especially Allen (2005); Des Chene (2005); Osler (2001).

that adequate explanations reveal the hidden mechanisms by which things work (Bechtel and Richardson 1993; Craver 2002; 2007; Craver and Darden 2001; Machamer, Darden, and Craver 2000). Yet the mechanical philosophy embodied in the explanatory practices of 21st century physiological sciences embraces functional descriptions as well. Consider some recent titles:

*MicroRNAs: Genomics, biogenesis, mechanism, and function (Bartel 2004)

*Mechanism and function of formins in the control of actin assembly (Goode and Eck 2007)

*Serpin structure, mechanism, and function (Gettins 2002)

*Mechanisms and functional implications of adult neurogenesis (Zhao et al. 2008)

Theoretical terms such as vesicle, neurotransmitter, receptor, channel, and ocular dominance column are conspicuously functional, describing entities not in terms size, shape, and motion but in terms of their job or role in the behavior of a system. This intermingling of functional and mechanistic description is not limited to molecular and cellular phenomena. The doctrine of localization of function, a cornerstone of contemporary (mechanistic) neuroscience claims that discrete brain regions and brain mechanisms perform distinct functions.

This happy coexistence of functional and mechanistic description in our contemporary physiological sciences suggests that the concept of mechanism, the concept of function, or both are significantly different from the way they were understood within the mechanical philosophy of the 17th Century. In this paper, I embrace a form of perspectivalism about both functions and mechanisms, one result of which is to narrow this historical gap. Like early advocates of the mechanical philosophy insisted, I claim that the causal structure of the world is disenchanted and purposeless. Mechanistic and functional descriptions, in contrast, presuppose a vantage point on the causal structure of the world, a stance taken by intentional creatures when they single out certain preferred behaviors as worthy of explanation. Specifically, talk of functions and final causes is not legitimized by or reduced to privileged kinds of etiological histories (though some functions have such histories) or to certain special effects of the item in question. Rather, they are imposed from without by creatures seeking to understand how a given phenomenon of interest is situated in the causal structure of the world.

The philosophical project surrounding functions and mechanisms so conceived is not to find a way of building them into the causal bedrock of the world, but of understanding the essential role that these notions play in physiological sciences such as neuroscience and beyond. One project is to understand how functional and mechanistic descriptions are related to one another in physiological sciences. In particular, I stress the roles that functional description plays in the effort to construct multilevel mechanistic theories (Craver 2002). A second project is to show how functional descriptions can be explanatory even when there is no etiological story to tell about how the functional item came to be. I argue that functional description can serve as a form of causal-mechanical explanation; it is a means of situating an item in the causal structure of the world (Salmon 1984). A third project is to make explicit the evidential criteria by which functional and mechanistic descriptions are evaluated. I argue that functional attributions are contentful to the extent that they can be cashed out in a detailed description of how an item is organized into a higher-level mechanism, and that one has good evidence for one's functional description to the extent that one can show how the item is organized into the mechanism. Functional description, in short, is a means of integrating an item into a hierarchical nexus of mechanisms. This account (like Craver 2001) is inspired by Cummins' (1975; 1983; 2001) and Toulmin's (1975) discussions of functions. My goal is to situate this "analytic account" with respect to the contemporary mechanical philosophy.

2. What Makes a Neurotransmitter a Neurotransmitter? Let us begin with a simple and uncontroversial example of a functional description in the neurosciences: the neurotransmitter. To call something a neurotransmitter is to suggest that it is the kind of thing that can be used to send signals from one cell to another. Consider the evidence required to establish that a given chemical substance is a neurotransmitter.

Table 1. Six traditional criteria for identifying a neurotransmitter.

- 1. The chemical must be present in the presynaptic terminal.
- 2. The chemical must be released by the presynaptic terminal in amounts sufficient to exert its supposed action on the post-synaptic neuron (or organ). Release should be

dependent upon inward calcium current and the degree of depolarization of the axon terminal during the action potential.

- 3. Exogenous application of the chemical substance in concentrations reasonably close to those found endogenously must mimic exactly the effect of endogenously released neurotransmitter.
- 4. The chemical must be synthesized in the presynaptic cell.
- 5. There must be some means of removing the chemical from the site of action (the synaptic cleft.
- 6. The effects of the putative neurotransmitter should be mimicked by known pharmacological agonists and should be blocked by known antagonists for that neurotransmitter.

Table 1 lists the six criteria that appear in most introductory neuroscience texts. Although one or more of these criteria are violated for some known neurotransmitters (especially amino acid transmitters like glutamate), they nonetheless represent well the kinds of evidence used to evaluate functional descriptions. Specifically, they are designed to show that the putative neurotransmitter is situated in the mechanisms of the synapse (see Figure 1) in such a way that it can plausibly act as a means of intracellular communication. The first criterion is that the transmitter must be present in the axon terminal. In Figure 3, the transmitter is shown stored within circular vesicles floating in the cytoplasm of the axon terminal (C). This criterion is relevant because the axon terminal is the paradigmatic starting point for the mechanisms of intraneuronal communication. The second criterion requires that the substance should be released in a calcium- and depolarization- dependent manner, and should be released in amounts sufficient to exert its supposed action on the post-synaptic cell. The release of the substance should be calcium- and depolarization- dependent because the mechanisms of neurotransmitter release are typically taken to begin with the depolarization of the axon terminal (thus linking the chemical signal across synapses to the intraneuronal electrical signal that the synapse is to preserve). Depolarization typically effects neurotransmitter release by opening voltage-sensitive Ca^{2+} channels in the axon terminal (See Figure 3; items 1-6). Furthermore, if the substance is to act as a synaptic signal, it must be released under physiologically relevant conditions in sufficient quantities that it can produce effects in the post-synaptic cell. (See Figure 3; items 10-12). The third criterion requires

that these post-synaptic effects be produced by both exogenous and endogenous application of the chemical. This criterion tests the causal relevance of the substance to the activity in the post-synaptic cell. Evidence concerning pre-synaptic synthesis, the fourth criterion, is required to show that the chemical's production is subject to mechanisms of regulation typical of neurotransmitter systems. The fifth criterion, that there is some mechanism for removing neurotransmitter from the cleft, is required because the tight relationship between action potentials in the pre-synaptic cell and transmitter concentration in the cleft would not hold if there were no mechanisms for disposing of "excess" transmitter in a timely fashion (See Figure 3; items 7, 8, and 8a). Finally, the putative post-synaptic effect of the chemical substance should be mimicked or blocked by pharmacological agents known to activate or impede the post-synaptic receptors for that substance. Again, this criterion is required to test the causal relationship between the presence of the substance in the cleft and the post-synaptic response. If agonists cannot mimic the substance, then one has some reason to doubt whether the substance itself is responsible for the post-synaptic effect. If interfering with post-synaptic receptors does not block the effect, then the substance at least does not act in a manner typical of neurotransmitters.

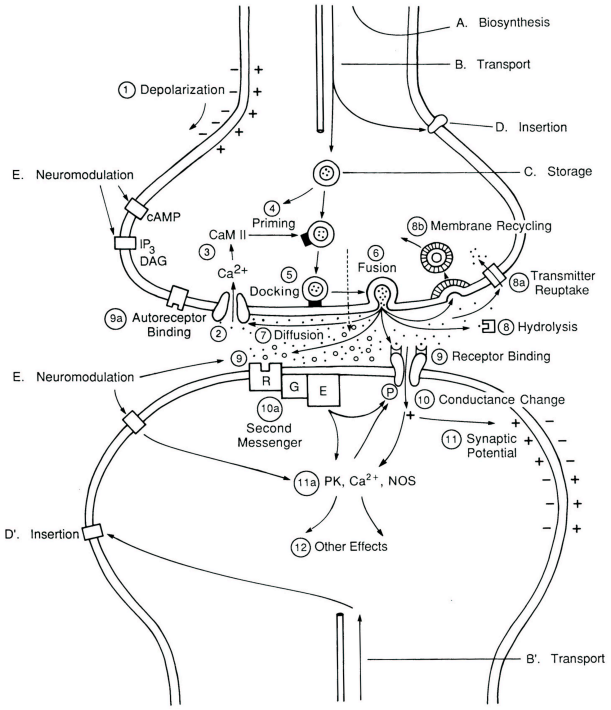

Figure 1. Mechanisms in the synapse (Reprinted from Shepherd 1994).

So what makes a neurotransmitter a neurotransmitter? To presage the discussion of Section 6, note that the criteria express no commitments about the developmental or evolutionary origins of the molecule in question. For those who embrace an adaptational view of functions, to claim that a substance has the function of mediating communication between cells (as evidenced by the six criteria) involves asserting (i) that the chemical substance came to be at this synapse because it can mediate communication between cells, and (ii) that the chemical substance is capable of mediating communication between cells. Although criteria 1-6 are clearly designed to satisfy some requirement like (ii), precisely none of them address (i). Indeed, it appears that the evidential order of things is the other way around: the evidence for (ii) is the best reason for believing (i). Regardless of how the molecule came to be used as a neurotransmitter (by drift, exaptation, evolution, chance, or divine fiat), so long as it satisfies criteria 1-6, it still functions as a neurotransmitter at the synapse.

Second, to presage the discussion of Section 7, criteria 1-6 go well beyond merely exhibiting an input-output relationship. After all, nothing is "put into" or "put out of" the neurotransmitter (except in synthesis and enzymatic degradation). And although some of the criteria (especially 3-6) do address relationships that might be represented in an input-output function, the others do not. Rather, the criteria are designed to show that a given chemical substance is situated among the mechanisms of chemical transmission in such a way that it can fulfill the role of a neurotransmitter. This involves not merely specifying some IO relation of the chemical substance but, in addition, showing that the exercise of the capacities thus described is organized into the mechanisms for regulating the chemical's synthesis, release, and removal from the synapse. Were one merely to describe how neurotransmitters are synthesized or how they bind to post-synaptic receptors, one would not have evidence that the substance functions as a neurotransmitter. The function "neurotransmitter" reaches out into the mechanisms of the synapse to include other details about the mechanisms in the pre- and post-synaptic cell. To describe a neurotransmitter so locally would be like describing a spark-plug as having

the function of making sparks; it would describe it in isolation from its mechanistic environment.

In short, criteria 1-6 concern neither the history of the substance, nor its local interactions with other parts of the cell, but rather how the substance is situated within the mechanism of synaptic communication. To describe a substance as a neurotransmitter is to describe how it fits into a containing system (Cummins 1975) or a mechanism (Toulmin 1975).

3. Mechanisms. But what is a mechanism? This question has received intense philosophical discussion over the last decade (see Machamer, Darden, and Craver 2000; compare, e.g., Bechtel and Abramson 2005; Bechtel and Richardson 1993; Burian 1996; Darden 2006; Glennan 1996; 2002; Salmon 1984; Thagard 2000; Wimsatt 1976). I prefer my own account (Craver 2007), which is a descendant of the account in Machamer et al. (2000) but supplemented with a view of causal relevance owing to Woodward (2003).

Roughly, a mechanism is a collection of entities and activities organized such that they give rise to the behavior of a mechanism as a whole. Entities are objects, such as neurotransmitters and cells. These entities are characterized in terms of structural properties, such as their size, conformation, and material constituents, and in terms of the relations with other entities in the mechanism (their locations, relative motions, forces). Activities are the things the entities do, such as binding to receptors and generating action potentials. Activities, on my Woodwardian interpretation, are typically characterized by and a set of generalizations concerning the properties and organizational features required for an activity to occur and the consequences of such occurrence. Such generalizations describe, for example, the properties that are required for different activities to occur (e.g., molecular conformations), the sphere of influence of the activity (e.g., obeying the inverse square law), its direction of action (e.g., linearly or at right angles), and so on. The activities in neuroscience and physiology tend to be mechanistically explicable. The neuronal activities of generating and propagating action potentials, for example, can be explained in terms of the activities of ions and proteins in the cell membrane.

These entities and activities are organized together spatially, temporally, and actively such that they give rise to the phenomenon to be explained. Forms of spatial organization include the size, shape, location, orientation, and compartmentalization of

the various parts of the mechanism. Forms of temporal organization include the orders, rates, and durations of the various activities. Active organization is a matter of which entities act and interact with one another, for example, whether they are organized in series or in parallel, and whether they involve cycles and feedback loops. Mechanism schemas, texts or diagrams that describe mechanisms at various grains, describe the relevant properties of the entities and detail the overall organization of the mechanism by virtue of which it gives rise to the behavior of the mechanism as a whole. The components are bound together into a single mechanism in part because of the causal interactions among them and, more fundamentally, because of their relevance to the behavior of the mechanism as a whole.

This notion of mechanism, exemplified time and again in contemporary biology, physiology, and neuroscience texts, clearly breaks with the historic association of mechanism with a set of basic and catholic causal principles. The kinds of activity that appear in contemporary mechanistic explanations are for more diverse than austere mechanists would allow. Furthermore, mechanisms need not be deterministic (probabilistic mechanisms are common) or sequential (they may involve feedback, forks, joins, and causal loops). Though descriptions of mechanisms must start and end somewhere, mechanisms themselves might have no clear beginning or end, and often run in cycles that are only artificially described as working from start to finish (such as the Krebs cycle or the mechanisms underlying circadian rhythms). This liberalization of the concept of mechanism has expanded the explanatory potential of the mechanical worldview while trading away only the Enthusiasm of austere mechanical philosophers.

As an example of a mechanism, consider how the NMDA receptor/ionophore complex works. This mechanism is named for what it does. It is a receptor because it binds the neurotransmitter glutamate (and pharmacological agents that mimic glutamate, such as N-methyl D-aspartate (NMDA)). It is an ionophore because when it binds to glutamate, it forms an ion channel traversing the membrane of the neuron. Activation of the NMDA receptor is a means of transforming an extra-cellular chemical signal (born by neurotransmitters) and an intracellular electrical signal (born by ion fluxes in the cell) into an intracellular chemical signal (born by intracellular ions and molecules). It can thus be described as working from beginning to end. The extra-cellular chemical signal comes

in the form of neurotransmitters (glutamate and glycine) that bind to extra-cellular binding sites. When they so bind, the protein changes its conformation, exposing a channel through its center. Under resting electrical conditions of the postsynaptic cell, the ion channel is blocked by positively charged magnesium (Mg^{2+}) ions held in place by electrical attraction and repulsion. When the post-synaptic cell depolarizes (as when it is in an excited state), the cell becomes less negative (and eventually positive) with respect to the extra-cellular fluid. The electrical forces holding the Mg^{2+} in the channel weaken, and the Mg^{2+} ions drift out of the channel, allowing Ca^{2+} (the intracellular chemical "signal") to diffuse into the cell.

This brief description includes the entities (e.g., glutamate, binding sites, channels, ions, membranes) and activities (e.g., binding, blocking, repelling, depolarizing) that constitute how the mechanism works. The activities can be characterized in terms of more or less invariant change-relating generalizations specifying, for example, that glutamate binding changes the pore's conformation, that depolarization removes the Mg^{2+} blockade, or that opening the channel allows Ca^{2+} to flow into the cell. The components are organized spatially (e.g., the channel spans the membrane), temporally (e.g., depolarization precedes the release of the Mg^{2+} ions) and actively (e.g., the transmitter binds to the receptor). The organization of these parts gives rise to the behavior of the mechanism as a whole: a highly regulated gating of Ca^{2+} currents across the membrane. One could make the mechanism behave differently or not at all by intervening to change these components or to alter their characteristic organization.

This mechanism (as with all mechanisms as the contemporary mechanical philosophy describes them) is explicitly defined in terms of what it does. The mechanism works from beginning to end, where the end is not what the mechanism invariably does but what we think it is supposed to do. Mutant NMDA receptors, for example, might not work this way, and even perfectly healthy and "normal" NMDA receptors might fail to open under the appropriate conditions if only because the molecular movements involved in channel opening are stochastic. Perhaps it is true that most or all NMDA receptors work in this way, but if so, this is an accidental fact added to the functional description, not something constitutive of its functioning as such. One might describe the behavior of

a most irregular mechanism (such as the mechanism of neurotransmitter release), or even a mechanism that has exactly one instance, in exactly the same way. The sense of "normal" here is thus not synonymous with "universal" or "regular" or "typical" but instead should be understood as specifying how the receptor works when glutamate synapses work as *they* normally do, and so on, until the hierarchy ends in some behavior that that the scientist is interested, for whatever reason, in explaining.

This teleological feature of mechanistic description is also implicit in the fact that mechanisms such as the NMDA receptor mechanism are bounded: a judgment has been made about which entities, activities, and organizational features are in the mechanism and which are not. The world does not come pre-chunked into mechanisms; it takes considerable effort to carve mechanisms out of the busy and buzzing confusion that constitutes the causal structure of the world. This is not always so obvious. Some mechanisms are entirely contained within physical compartments, such as a nucleus, or a cell membrane, or skin. Transcription (typically) happens within the nucleus, and translation occurs in the cytoplasm. However, mechanisms more frequently transgress compartmental boundaries. The description of the mechanism of the NMDA receptor, for example, relies crucially on the fact that some components of the mechanism are inside the membrane and some are outside and allows the intracellular and extracellular concentrations of ions to be different, allows a diffusion gradient to be set up between them. Even the simple act of carving such a mechanism into working parts, as opposed to mere spatio-temporal pieces that might be produced by slicing, dicing, or spiral cutting, requires some principle by which one can recognize a difference between ways of chunking that are relevant to some end and those that are not (Kauffman 1971).

Austere mechanists, eschewing final and formal causes from their explanations in favor of corpuscles operating blindly by motion and contact, lacked principles to define the unity of a machine, organ, or organism. Descartes at times favors principles of spatial organization: the parts are within spatial boundaries, they move together, and they can be transported together from one place to another while maintaining fixed relative positions with the other components (see Des Chene 2001). Others (such as Salamone De Caus) appeal to contact among the parts. Contemporary physiologists recognize counterexamples to each of these suggestions. I have already noted that mechanisms

frequently defy tidy physical boundaries (although every mechanism can, trivially, be circumscribed). Parts of mechanisms also often move in separate directions (as any multiple-pulley system illustrates). Some mechanisms are more ephemeral than others; they work only as components happen to come into the appropriate spatial arrangement.[3] For example, in many biochemical cascades, the relevant reactions could happen anywhere in the cytoplasm. Such mechanisms lack stable spatial relations; they could not be picked up and carried from one place to the next. Those accustomed to drawing and studying tidy diagrams of mechanisms in physiology textbooks can temporarily forget that bodies and cells, for example, are bubbling stews of entities and activities, and that it takes considerable scientific effort, abstraction, and idealization to distinguish components from contraband, activities from incidental interactions, and causes from background conditions. And this filtering process requires (essentially) fixing on some behavior, process, or *function* for which a mechanistic explanation will be sought (see Craver 2007, ch. 4).

In a slogan, mechanisms are the mechanisms of the things that they do. The entities and activities that are part of the mechanism are those that are relevant to that function or to the end-state, the final product that the mechanism, by its very nature, ultimately produces. Relevance here should be understood in part in terms of relations of mutual manipulability between a component and some behavior of a mechanism that one seeks to understand; in short, a component is relevant to a behavior of the mechanism as a whole if one can manipulate the behavior of the mechanism as a whole by intervening on the component (as in lesion experiments or electrical stimulation) and one can manipulate the behavior of the component by intervening to stimulate or inhibit the behavior of the mechanism as a whole (see Craver 2007, Ch.4, Section 8). Furthermore, we divide a system into parts in part by deciding first what needs to be done in order for the mechanism as a whole to behave as it normally does. The NMDA receptor's function (to turn a joint chemical and electrical stimulus into a change in intracellular Ca^{2+} concentration) determines which features of the channel structure are especially important or necessary for just that role. From the perspectivalist view adopted here, these judgments of normality continue upward until they are grounded ultimately in the

[3] See Glennan (2009).

judgment or interests of an observer. Functions, on this view, are roles within mechanisms, defined ultimately in terms of a topping-off point selected for its relevance to observer interests and perspectives. Mechanisms come into view as entities and activities organized to perform such functions.[4]

4. Levels of Mechanisms. One reason neuroscientists are interested in the NMDA receptor is because its behavior is a component in the mechanism of Long Term Potentiation (LTP). LTP is one of the means by which certain neurons in the central nervous system (CNS) strengthen their connections (synapses) with one another. When the pre-synaptic neuron (the one that releases neurotransmitters) and the post-synaptic neuron (the one containing the NMDA receptor) are simultaneously active, the synapse is strengthened (LTP is "induced"). When the pre-synaptic neuron is active, it releases glutamate (and glycine) into the synaptic cleft. The post-synaptic cell is active when it is depolarized from its resting electrical potential. These two factors, recall, are the crucial set-up conditions for the opening of the NMDA receptor. The termination condition (the influx of Ca^{2+}) is a crucial stage in the induction of LTP. Many neuroscientists believe that LTP is a crucial activity in the mechanisms of some kinds of learning and memory. For example, LTP is a component in the mechanisms of spatial map formation in the hippocampus (a medial temporal lobe structure), and these spatial maps are thought to be components in the mechanisms of spatial memory, the ability to learn to navigate through novel environments. The NMDA receptor (an entity) and LTP (an activity) are also thought to be involved mechanisms that "top off" in drug addiction (Kauer et al. 2007) and Alzheimer's disease (Rowan et al. 2003), which could in no compelling sense be described as adaptations.

All these theories describe mechanisms at multiple levels of organization. The activity of the NMDA receptor is of interest by virtue of the fact that it is a stage in the mechanisms of LTP induction, which constitutes a stage in the mechanism of spatial map formation, and so on. Such multiply embedded hierarchies are usefully thought of as levels of mechanisms: they are part-whole relations with the additional restriction that the

[4] Given the hierarchical embedding of mechanisms to be discussed below, functional description is often appropriate both for the behaviors of mechanisms as a whole (either because they have been privileged as such by an observer or because they play a role in a higher-level system that is so privileged) and for the roles of the parts in producing that behavior.

parts are components organized together to produce the behavior of the mechanism as a whole. To be at a lower (-1 or -m) level just is to be one of the components organized into the mechanism as a whole, which constitutes the higher (+1 or +n) level. Of course, there are other useful notions of "level" in biology (tracking e.g., objects of different sizes, the phenomena in different theories, the domains of different sciences, and the targets of different techniques). However, levels of mechanisms capture a common notion of level, and one that is especially relevant to thinking about the relationship between functions and mechanisms in physiological sciences.

Different scientists top off their mechanistic hierarchies in different highest-level activities. Some biologists are interested in, for example, channel structure and how channels open and close. They aren't especially interested in system-level mechanisms or cognition. Some are interested in cognition, others in social bonding, and others in ecological systems. Sometimes biologists direct their attention toward mechanisms and functions that contribute to an organism's fitness. Sometimes they want to know how diseases work, how toxins kill cells, or how pollutants change the dynamics of an ecosystem. Differences in topping off points reflect differences in interest and emphasis, and these differences are reflected in the mechanistic theories that different scientists, fields or traditions use to explain the phenomena in their domain. It is by reference to these historically, individually, and disciplinarily relative topping off points that the relevance of lower-level components is determined. The choice of a topping-off level selectively focuses the researcher's attention upon certain lower-level mechanisms and not others. It can lead researchers to carve the system into altogether different parts, as Kauffman (1971) and Wimsatt (1974) emphasize. In our working example, an antecedent interest in spatial memory focuses the investigator's attention upon the mechanisms of spatial map formation, LTP, and the mechanisms of NMDA receptor activation. The mechanisms of NMDA receptor activation have also been hypothesized to play crucial roles in hierarchies that top off in the progression of Huntington's, the psychological effects of PCP abuse, the mechanisms of programmed cell death, and the mechanisms of chronic pain. The choice of a topping-off point is a crucial step in filtering the causal nexus to yield a properly mechanistic nexus. This is why I am a perspectivalist about functions and mechanisms.

I hasten to emphasize that this perspectivalism has limits. Ultimately, the causal structure of the world, facts about what variables make a difference to which others and which entities and activities exist and occur, allow only some perspectives to fit. It is an empirical question whether a system exhibits the behavior that one is trying to explain. It is an empirical matter whether a given entity, activity, or organizational feature exists, and whether it is in fact relevant (in the sense sketched above) to the phenomenon thus described. My point is that the actual causal structures of the body, the brain, and the cell are bewilderingly complex and reticulate. This is why it is such a significant scientific achievement (Haugleand 1998) to properly characterize a function and to generate a multilevel description of mechanisms that accommodates all of the data about the parts, activities, and organizational features at multiple levels and weaves them into a coherent image of how something works. That said, there are many ways of decomposing such bewilderingly complex bits of the causal nexus into intelligible units, and the identification of functions and mechanisms is crucial for bringing intelligible order to such a causal stew. They are crucial steps, that is, in providing explanations.

5. Explanation: The Mechanist's Stance. Logical empiricist philosophers of science (such as Hempel 1965) once thought that explanations are arguments showing that a description of the phenomenon to be explained follows from statements describing covering laws and relevant conditions. This elegant and powerful view faded from currency because arguments and explanations have different criteria of adequacy; inferential subsumption under general laws is neither necessary nor sufficient for an adequate explanation (see Salmon 1989; Craver 2007, Ch. 2). Rather, to explain a phenomenon is to show how it is situated in the causal nexus (Salmon 1984). More plainly, explanations reveal the causal structure of the world. I embrace this view of explanation, but not Salmon's view of the causation.

For Salmon, the causal nexus is composed of causal processes (understood as space-time continuants bearing conserved quantities) that interact with one another when they intersect one another in space-time and exchange conserved quantities (Salmon 1994; Dowe 2000). This view embodies the boldness and simplicity of the earliest statements of the mechanical philosophy, but it is not ideal for thinking about sciences such as neuroscience. The process view emphasizes relatively fundamental kinds of

causal interaction (those that involve collisions or charges, for example). Physiological activities such as the opening of an ion channel, the transcription of DNA, and the formation of spatial maps are much too complex for tidy description from this perspective. Furthermore, the process view requires that causal processes intersect one another in causal interactions. This means that the causal nexus so conceived has no room for causation by omission or double-prevention (e.g., inhibiting an inhibitor), forms of causation that are literally ubiquitous in the physiological sciences (for a fuller discussion, see Craver 2007). Depolarization of the post-synaptic cell causes Ca^{2+} to enter through the NMDA receptor, but it does so by removing a process (Mg^{2+} ions) that prevents the ions' flow.

So we must understand the causal nexus more liberally in physiological sciences. As discussed briefly above, the interactions and activities (following Woodward's (2003) account of actual causation) can be understood in terms of relations by which the value of one variable (standing for a property, or the presence or absence of an object, or the occurrence or non-occurrence of an activity) depends upon the value of another. Such dependency amounts to the fact that one can change the value of the first variable by intervening to change the value of the second (given certain restrictions on the intervention). On this view, the causal nexus can be represented roughly as a set of variables related by generalizations that remain stable (or invariant) when one intervenes to change the value of the variables in the generalization. Such a view explicitly allows for causal interactions above the fundamental level (as there is no metaphysical restriction on the kinds of objects or properties that might enter into a causal relationship) and has no difficulty accommodating causation by omissions and preventions. This view also comports nicely with the kinds of experiment one uses to test causal claims. One intervenes to change the putative cause and detects the changes, if any, in the effect variable under those controlled conditions. Explanation on this view is a matter of revealing causal dependency relations of this sort or, for the explanation of singular events, tracing the productive relations among the entities and activities that make such change-relating generalizations true. Either way, this view remains true to Salmon's overall vision: to explain an event or phenomenon is to show how it is situated in the causal structure of the world.

Salmon recognizes two ways of situating a phenomenon in the causal structure of the world: an etiological form of explanation, in which one explains a phenomenon by tracing its antecedent causes, and a constitutive explanation, in which one explains a phenomenon by revealing its internal causal structure. In order to accommodate the diverse explanatory roles played by functional description in physiological sciences, it is necessary to add a third, contextual variety of causal-mechanical explanation, further liberating the contemporary mechanical philosophy from its historical strictures and recognizing within that philosophy an essential place for functions in our effort to make the causal structure of the world intelligible.

6. Etiological Explanation and Adaptational Functions. Etiological explanations are typically offered in response to questions concerning the origins of some item, its path of development, or its historical trajectory. Salmon represents this etiological aspect of mechanistic explanation in the bottom portion of Figure 2. The figure illustrates the *backward looking* character of etiological explanations; such explanations highlight the pathway connecting relevant set-up conditions in the past, through intermediate stages of activity, to the item to be explained.

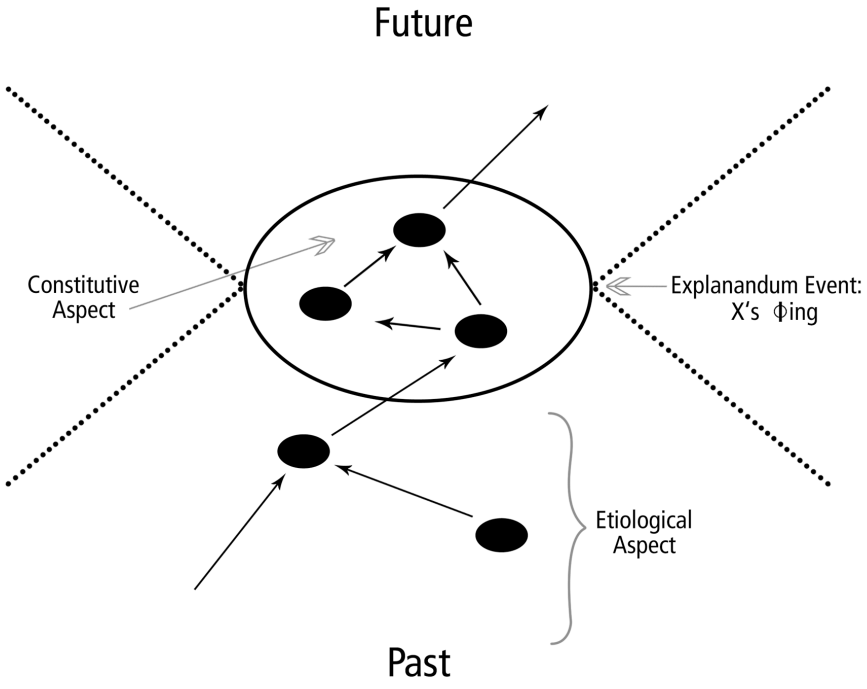

Figure 2. Constitutive and etiological aspects of causal-mechanical explanation.

Some philosophers and scientists reserve the term function for traits, properties, and activities that are adaptations (Ruse 1971; Wimsatt 1972; Brandon 1990). Churchland and Sejnowski claim that this use captures the sense of "function" used in neuroscience (1992; 69; see also Bechtel 1989).[5] I focus on Wright's classic formulation:

The function of X is Z means:

(a) X is there because it does Z,

(b) Z is a consequence (or result) of X's being there (1973, 161).

In the standard biological case, (a) is embellished as a natural-selection story roughly to the effect that heritable traits of type X, by virtue of their doing Z, increased the likelihood that organisms bearing traits of type X would survive and/or reproduce, and as a result contributed to the preservation of traits of type X in a given population. The NMDA receptor has the adaptational function of mediating cellular "signals" if and only if the NMDA receptor allows these signals to be mediated and was preserved in organisms because it did so in the past. One advantage of identifying biological functions with adaptations is that doing so can often accommodate the intuition that a trait's function explains its presence. Asked why the mouse has NMDA receptors, it may be correct to respond that the NMDA receptors are there *because* they mediate certain chemical signals. The "because" in this sentence is the "because" of efficient causation: adaptational explanation is an example of the *etiological* type of mechanistic explanation.

The ability to make sense of this kind of functional explanation is a notable advantage of causal-mechanical models of explanation relative to covering-law models. The puzzle of functional explanation is to accommodate the intuition that a trait's presence can be explained by appeal to what it does. We explain why we have NMDA receptors by appeal to their role in learning and memory, for example. A defender of the covering-law model of explanation faces the challenge of showing that one can derive or otherwise infer the presence of a particular type of trait from what the trait allows the organism to do. However, as Hempel recognized, the fact that the same function can be

[5] They also note that the function of an item is its "job," and that any apparent teleology in the sense of function is "eliminable or reducible without remainder in an evolutionary framework." For classic adaptational accounts, see Brandon 1990, Millikan (1984), Neander (1991), Ruse (1971), Wimsatt (1972), and Wright (1973). Garson (2008) provides a recent review.

produced by multiple functionally equivalent types of trait always brings the explanatory argument up short: at most one can infer that one of the functional traits exists, not the functional type that one seeks to explain. The causal-mechanical view, on this understanding, thus proposes to show that the surface character of functional explanation (that an item's presence can be explained by adverting to its function) can be translated into an etiological framework that describes a selective developmental or evolutionary process. One describes the mechanisms beginning with the first appearance of the trait and ending with its contribution to survival and reproduction. This is well and good, so long as we bear in mind that the translation is only approximate. The presence of an item in an organism now is explained in terms of the behavior of items of the same type in the past. The token effects of the trait now, however, do not explain the presence of the item. The teleology preserved in the assimilation of functions to adaptations thus breaks with the Aristotelian idea of a goal or purpose as a cause of behavior over and above its constitutive and etiologial explanations. (There is reason to doubt that Aristotle held this view of final causes; see Leunissen 2007) The assimilation of functional language to language about kinds of causal histories on this understanding is eliminative, not reductive. To ascribe a function is a shorthand way of describing it as having a certain kind of history and, one might say, nothing more than that. The adaptational function of an item makes no further contribution to how an item is situated in the causal structure of the world.

Functional descriptions, however, are sometimes said to have a normative dimension (see, for example, Neander 1991; Wimsatt 1972; Wright 1973). The functional description distinguishes an item's preferential behavior (its *proper function*) from the item's myriad non-functional effects in a system (e.g., a receptor deforms the lipid bilayer of the cell membrane), the myriad things that the item might function *as* (e.g., a target for pharmacological intervention), and the many ways that an item might malfunction (e.g., a mutation causes a receptor not to bind with a neurotransmitter). Functional descriptions thus conceived describe how things ought to work rather than how they in fact work. This way of thinking about functions fits naturally with the idea that creatures have been designed; the function of the receptor is the purpose for which the demiurge created it and arranged it just so. And perhaps this manner of speaking can be translated into our

post-theistic biology by putting evolution by natural selection in the role of a divine maker: roughly, an item's function is that effect in virtue of which its type has been preserved in a species. Thus Wimsatt claims, "Given the operation of differential selective processes, it is possible to show that any given system resulting from this process has all the relevant logical features of purposiveness and teleology" (1972, 16). Selective processes, it is said, define goal states within higher-level systems or preferable states of individual or species-level traits (Hull 1974). If so, one can reduce facts about how a physiological item *ought to* behave to facts about how items of that type *do* or *did* behave.

To make good on the proposed reduction one should be able to form an argument that begins with premises describing the selective history of an item and concludes with statements about its goals purposes, preferential states. However, I know of no successful argument that begins with premises about what causes what and ends with conclusions about what ought to be the case: either the "ought" is smuggled into one of the premises, or the proposed derivation relies on obvious "tricks" peculiar to formal logic, or the notion appears from out of the blue in the conclusion. (See Russell 2010 for a critical examination of some clever attempts to derive an ought from an is). Machamer (1977) reconstructs the proposed argument linking facts about an item's having been selected for some behavior to conclusions stating that the item's behavior is good, preferable, or a goal state. He demonstrates convincingly that this argument succeeds only if one tacitly presumes the existence of a higher-level containing system the behavior of which is good, preferable, or a goal state. One must presume, for example, that it is preferable that an organism should live and reproduce, or that the species ought to survive, or that one ought to live some conception of the good life. But these shoulds and oughts, on the perspectival view recommended here, are ultimately projections of our interests or preferences. The causal structure of the world does not ground talk of goals, purposes, and preferential states. Such things are "queer" in a mechanistic world (in Mackie's phrase; 1977) because they are "fraught with ought" to borrow from Sellars (see 2007).

Schaffner (1993) develops a line of arguments suggesting that selection is neither necessary nor sufficient to make talk of goals, purposes, and preferential states appropriate. One can understand the growth of thunderstorms, the fine-grained sand on a

beach, and the momentum of a pachinko ball at the bottom of a pachinko machine in terms of selection mechanisms (see Schaffner's cloner example), but few are willing to assign functions (in a non-perspectival sense) to thunderstorms, the fineness of a grain of sand on the beach, or the momentum of a pachinko ball. Further, as has often been noted, the etiological reduction of functions prevents one from assigning functions to traits on which selection has not yet acted. The first NMDA receptor did not have a function according to this view because a trait can have a function only in the second generation (after it has contributed to fitness), and only if the first NMDA receptor in fact manifested its dispositions in a way that contributed to the organism's reproduction. The perspectivalist avoids this consequence.

None of this is news. I emphasize it not because evolutionary thinking is out of place in physiological sciences such as neuroscience. Indeed, in trying to find an intelligible picture of what organisms are doing and how they do it, it is most useful to consider the selective forces that have likely shaped their development. (I say likely because of phenomena such as drift and exaptation; see Gould and Lewontin 1978; Gould and Vrba 1982.) Evolutionary thinking can be heuristically useful as a guide to creative thinking about what an organism or organ is doing, the conditions under which it is suited to work, and about its apparent failure to work optimally, as one would expect had it been created by a benevolent, omniscient, and omnipotent designer.

I reiterate these objections to the normative implications of selective etiologies for two reasons. First, neuroscience and physiology have goals that would be hampered by the general acceptance of such a proprietary notion of function. Much of physiological science such as neuroscience is driven not by the goal of understanding how the nervous system functions when it is working properly but rather by the goal of understanding how it can fail and how such failures might be predicted and controlled. One can describe the function of items in the mechanisms for replication of DNA, or for anoxic cell death, or for the production of cancer. When one describes an oncogene as an oncogene, one is describing it functionally without being committed to the idea that the oncogene survived in virtue of being an oncogene. Indeed, it would seem likely that it survived in spite of the fact that it functions as an oncogene. Likewise, a researcher hoping to build a robotic interface with someone's motor cortex, for example, might hunt for signals that can be

commandeered for the purposes of moving the arm even if such signals were not at all part of anything that evolution by natural selection might have considered. These are as much a part of the mechanisms of the brain as are those parts and mechanisms that have been selected for their effects. Researchers approaching the brain from such a translational perspective will see functions where the advocate of adaptational functions does not.[6]

Second, if anyone has an obligation to think slowly and pedantically about the normative implications of evolutionary biology, it is philosophers of biology. Those who claim to find in evolutionary biology a means of distinguishing the good from the bad, the healthy from the diseased, the deleterious from the beneficial, owe a compelling argument for the ability of evolutionary processes to ground such conclusions. The perspectivalist refuses to pass the buck for these normative judgments to evolutionary biology. When we make such normative judgments, says the perspectivalist, we are responsible for justifying them. Evolution cannot do that work for us.

7. Constitutive Explanations and IO Functions. Consider a second role played by functional description in mechanistic neuroscience and physiology. According to this view, a function is a mapping from inputs to outputs in conformity with a rule. Call these input-output (IO) functions. Sometimes Cummins describes functions this way. For example, he says that functions are capacities, where capacities are, "specified by giving a special law linking precipitating conditions to manifestations— i.e., by specifying input-output conditions" (1983, 53). IO functions characterize the activity of some item without reference either to its context or to its internal complexities. In forming such a description, one draws a conceptual dividing line at the spatial boundary of the object or activity and recognizes a limited number of specific *interfaces* across that boundary—more or less well-defined interactions with items outside of that boundary (see Haugeland 1998). For example, in describing the IO function of the NMDA receptor, one begins by parsing it from its environment at its spatial boundary and characterizing the relevant interfaces (interactions) across that boundary. Three significant interfaces between the NMDA receptor and its environment are the binding of glutamate and

[6] Analyses of the concept of function in terms of current ability or propensity to survive and reproduce (e.g., Bigelow and Pargetter 1987; Boorse 1976; Canfield 1964) likewise fail to accommodate many of the perfectly legitimate uses of functional language that can be found in neuroscience.

glycine to the receptor, the blocking action of Mg^{2+} ions, and the influx of Ca^{2+} through the channel pore.

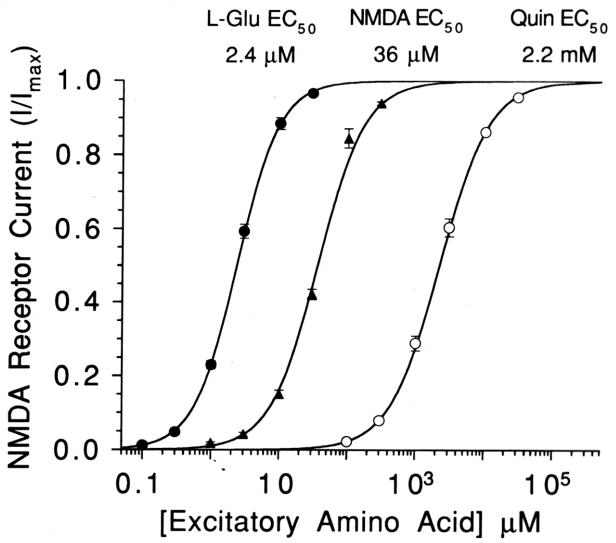

Figure 3a. NMDA receptor current as a function of concentration of excitatory amino acids (Reprinted from Mayer et al. 1991).

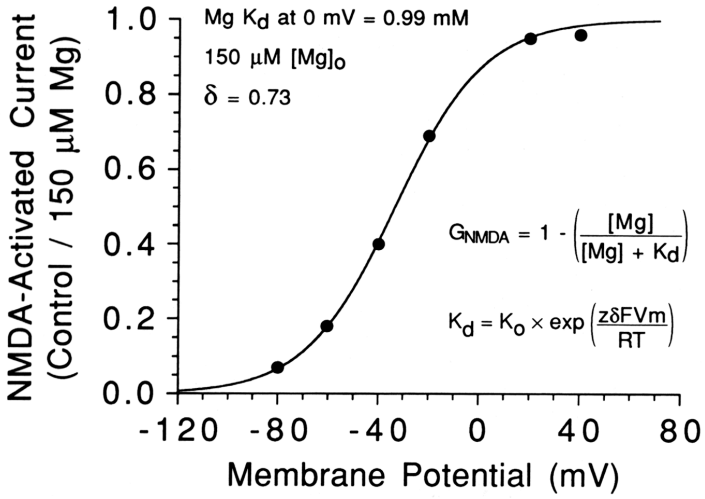

Figure 3b. NMDA activated current as a function of membrane potential. (Reprinted from Mayer, et al. 1991)

IO functions can sometimes be characterized mathematically. Two examples are represented graphically in Figures 3a and 3b (taken from Mayer et al. 1991). The first figure depicts a typical dose-response curve relating concentrations of agonists (glutamate and pharmacological agonists) to the current of Ca^{2+} flowing into the post-

synaptic cell in the absence of Mg^{2+}. The second of these characterizes changes in that influx of Ca^{2+} as a function of post-synaptic depolarization in a medium with extremely high concentrations of Mg^{2+}. Both can be understood, as suggested above, as causal generalizations that are invariant under interventions: one intervenes to change the concentration of neurotransmitter in the synapse and detects changes in the current flowing through the channel, as in Figure 3a, or one holds Mg^{2+} concentrations constant while varying the membrane voltage and recording the current through the channel, as in Figure 3b. Clearly neither of these invariant change-relating generalizations (cf. Glennan 2002; Craver 2007) characterizes completely the activation of the NMDA receptor (i.e., its function), and each characterizes it only under highly constrained conditions (e.g., experimentally gerrymandered levels of Mg^{2+}). Rather, these IO functions and others like them combine to form a complex description of the behavior of the NMDA receptor.

This *complex IO function* plays two crucial roles for the physiological scientist beyond providing a precise characterization of the phenomenon. First, such abstract description affords the scientist descriptive leverage over the messy details of the constitutive mechanism that produces the complex IO function. One can speak of the activation of the NMDA receptor without going into the complex and poorly understood details of protein chemistry, and one can speak of LTP induction without detailing the intricate pattern of molecular activities responsible that induce LTP. IO functions are also descriptive tools for dealing with the multiple realizability of most biological functions: that is, for dealing with individual, strain, and species differences. The same IO function might be instantiated by a number of different mechanisms.

Complex IO functions are also important for characterizing the phenomena for which one will seek *constitutive explanations*, the second aspect of causal-mechanical explanation that Salmon recognizes (cf. Bechtel and Richardson 1993). In constitutive (as opposed to etiological) explanations one explains an event by revealing its internal causal structure. Instead of revealing the causes by which the NMDA receptor is activated, developed, or evolved (as in etiological explanations), one describes the relevant causal structure internal to NMDA receptor, the entities, activities, and organizational features in virtue of which it activates when neurotransmitters are present and the postsynaptic cell is depolarized. Such explanations have been called explanation by decomposition,

functional analysis, and explanation by reverse engineering. Constitutive explanations are downward-looking in the sense that they describe the internal mechanisms— organized lower (-1 or -m) level activities and entities— by virtue of which some aspect of the complex IO function is produced. They situate an item in the causal nexus by detailing the lower-level mechanism that produces those aspects of the complex IO function. The ellipse in the center of Figure 2 represents this type of explanation. Constitutive explanations are sought when one wants to know how something works or wants to know the "hidden" mechanism by virtue of which an item does something of interest. The explanation of the opening of the NMDA receptor in Section 3 is an example of this constitutive form of mechanistic explanation. That explanation is tailored to account for the IO functions represented in Figures 3a and 3b and the myriad others like them. It is in this sense that the complex IO function frames the constitutive explanation; they define the relevant input-output relationships that the internal mechanism must be capable of performing.

The language of inputs and outputs that characterize the behavior of the NMDA receptor does not apply straight-forwardly to the example of neurotransmitters with which we began. Neurotransmitters are not mechanisms for transforming inputs into outputs, at least as commonly conceived. One can describe the synthesis and release of neurotransmitters this way, and one can describe their effects on post-synaptic receptors this way (as in Figure 3a), but the molecule itself seems to be a passive participant in these change-relating generalizations that describe how the molecule is situated as a component within a higher-level mechanism. Perhaps one could characterize features of the molecule's environment, such as temperature or pH as inputs, and one could characterize the molecule's conformation as an output. But this manner of speaking is strained and to my knowledge would not be adopted by scientists. The function of the neurotransmitter, in other words, is primarily understood contextually.

8. Contextual Functions. Consider four ways of describing the heart's role in the circulatory system. The heart:

(i) distributes oxygen and calories to the body;

(ii) pumps blood through the circulatory system;

(iii) expels blood; and

(iv) contracts.

Descriptions i-iii are contextual (or "wide") in varying degrees; they each describe things that the heart could not do by itself without being organized together with other entities and/or activities. The heart cannot expel blood (iii) without blood, and the expulsion of blood will only circulate it (ii) if the veins and arteries are appropriately organized. Even then, the heart cannot distribute oxygen and calories (i) in the absence of oxygen and calories. A description of the heart's mechanistic role function is contextual to the extent that it makes explicit reference to objects other than the heart itself and its parts. Reference to objects beyond the boundaries of the heart, notice, is not required in describing (iv) the heart's contraction. In describing the heart as contracting, one makes no implicit commitments concerning the mechanistic context in which this activity is embedded. One offers an isolated description of the sort described in the preceding section.

The same can be said of our description of neurotransmitters. Glutamate, for example, might be described as a molecule that:

(i*) mediates spatial cognition.

(ii*) carries a chemical signal.

(iii*) binds to a post-synaptic receptor

(iv*) has a characteristic primary sequence and conformation.

Again, descriptions i* to iii* are wide. When one speaks of dopamine or serotonin as neurotransmitters that regulate emotion, control movement, or underlie addiction, one describes the molecule contextually as a component in a larger system. Contextual descriptions of this sort describe some part and its activities in terms of the contribution it makes to a higher (+1 or +n) level mechanism. Such descriptions tacitly refer to the fact that if one were to, for example, intervene to change neurotransmitter levels, one could influence the behavior of such higher-level systems. Cummins writes that, "to ascribe a function to something is to ascribe a capacity to it that is singled out by its role in an analysis of some capacity of a containing system." (Cummins 1983, 99), and we should add that functional characterizations often describe those capacities in a manner that includes wider and wider regions of the causal structure of the system under consideration, as in items i-iiii. There is a difference, after all, between knowing that

spark-plugs produce sparks and knowing how that sparking is situated in the mechanisms of an engine. In the former case, we describe the spark-plug's IO function; in the latter we describe its role contextually. Contextual functions are not simply capacities (IO functions) *picked out* by their place in a higher-level mechanism; rather, they are descriptions of the activity of some item in terms of how it is organized into the workings of a higher-level mechanism. One and the same token sparking of a spark plug may be said to be an instance of sparking, of igniting an explosion, of pushing a piston, and of turning the drive shaft depending on how much of the item's context in the causal nexus one includes in the description. There is no firm dividing line between IO functions and role functions; the distinction depends upon where one draws the boundary lines around an object. My point here is that contextual descriptions are invariably richer than their IO counterparts, making clear how a given IO function is situated in some other system that we care about.

Contextual, isolated, and constitutive descriptions should not be seen as corresponding to divisions in the furniture of the world. They should rather be thought of as distinct perspectives on a hierarchy of levels of mechanisms. As Lycan puts it, "See Nature as hierarchically organized in this way and the 'function/structure' distinction goes relative: something is a role as opposed to an occupant, a functional state as opposed to a realizer, or vice versa, only modulo a designated level of nature" (1990, 78; cf. Churchlands and Sejnowski 1993, 18-27). I put it like this: see the world as a mechanistic hierarchy and the distinction between a contextual (+1 or +n) function, an isolated behavior (0), and its constitutive (-1 or -m) mechanism goes relative to a perspective on level in that hierarchy.[7] One cannot describe an item's role, in the broad sense intended here, without describing the place of its IO function in some more inclusive mechanism.

[7] Stephen Toulmin makes this point most beautifully: "There is no clear division of natural processes in the real world, into 'functions' on the one hand and 'mechanisms' on the other. Rather, we draw a distinction between the functional and mechanistic *aspects* of any natural process, in one context or another; and whatever can be viewed as a mechanism, form one point of view and in one context, can alternatively be seen as a function, from another point of view or in another context. Indeed, the very *organization* of organisms— the organization that is sometimes described as though it simply involved a 'hierarchy' of progressive larger structures— can be better viewed as involving a 'ladder' of progressively more complex systems. All of these systems, whatever their levels of complexity, need to be analyzed and understood in terms of the functions they serve and also of the mechanisms they call into play. And when we shift the focus of our attention from one level of analysis to another— from one fineness of grain to another— even those very processes which began by presenting themselves to us under the guise of 'mechanisms' will be

There is thus a need to recognize a third form of mechanistic explanation beyond those recognized by Salmon: *contextual explanation* (Craver 2001). Sometimes a neuroscientist or physiologist is ignorant of what a given item does or is good for, and this leads her to search for a higher-level mechanism within which it has a role. The answer to such a request for explanation comes in the form of a description of how an item is situated in a higher-level mechanism. The process of situating an item in a higher (+1 or +n) level mechanism involves showing how it is organized (spatially, temporally, and actively) into the higher-level mechanism. Contextual explanations are characteristically outward looking and upward looking. They are outward looking because they refer to components outside of the item to be explained and they are upward looking because they contextualize that item within the behaviors of a higher-level mechanism. Mechanistic explanation, at least as we now understand it, is thus not synonymous with downward-looking, reductive explanation (though constitutive explanations are reductive in the sense that they explain wholes in terms of parts); there are also upward-looking mechanistic explanations.

To return to the example with which began, the neurotransmitter has to be released in correlation with the electrical properties of the cell, has to be cleared from the cleft, has to act on post-synaptic receptors, and has to exhibit the kinds of active organization within a mechanism revealed by the other criteria in Table 1. The concept of a neurotransmitter, as one of our well-articulated concepts in contemporary neuroscience, provides a model of a contentful functional ascription and of the kinds of evidence by which such ascriptions are to be evaluated. It helps to show precisely what is unsatisfying about glib comments that, for example, dopamine is a happiness neurotransmitter. For in the first place, dopamine does many things in the brain, and associating it with just one of those functions already represents a perspectival simplification of how dopamine fits in the causal nexus of the nervous system. In the second place, such a description implicitly appeals to complex higher-level-mechanisms-we-know-not-precisely-what. One might know that one can regulate one's emotional state by regulating dopamine levels and remain largely ignorant of the complex mechanisms by virtue of which that effect is mediated. We add content to such terse and gestural functional descriptions by revealing

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transformed into 'functions.' (Toulmin 1975, 53)

the entities, activities, and organizational features by which dopamine contributes to the regulation of emotions. This perspective on functional attribution suggests a regulative ideal in formulating functional attributions: they are contentful and precise to the extent that they explicitly make claims about how an item is situated in its causal context. It is by reference to the evidence for such organization (as is the case for neurotransmitters) that functional attributions are evaluated. Similar remarks apply to common ways of talking about brain regions and genes, for example. When one talks about a gene for aggression or a brain region for decision-making, one is speaking gesturally about how an item fits into a higher-level mechanism, and we make progress in fleshing out the content of such gestures to the extent that we build descriptions of how the item is situated in the causal nexus.

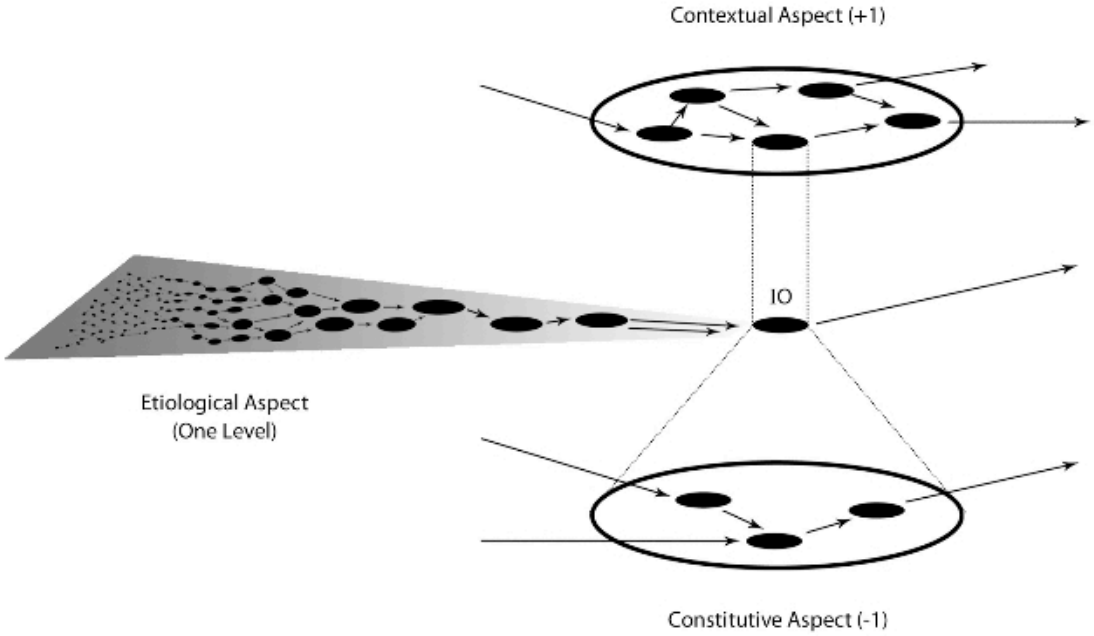

Figure 4. Constitutive, contextual, and etiological aspects of causal-mechanical explanation.

8. Conclusion. Three explanatory perspectives are illustrated in Figure 4, each of which should be acceptable those who embrace the mechanistic philosophy as we now know it. The figure, which is intended to replace Salmon's useful diagram of aspects of causal-mechanical explanation, depicts two levels (the top and bottom circles) in a mechanistic hierarchy flanking a complex IO function in the middle. The past and future portions of

the causal nexus are to the left and right of the hierarchy, respectively. For each type of explanation, the explanandum is some aspect of the complex IO function in the middle; call it E.

Etiological explanations trace the pathway of entities and activities terminating in E; they explain how E came to be there, came to pass, or came to have some property. Such an explanation is shown on the left hand side of Figure 4. It is represented as a single level for simplicity, though any complex etiological explanation will typically span multiple levels as well. Explanation in terms of natural selection is a type of etiological explanation, one that requires an understanding of genes, organs, organisms, populations, and ecosystems. Adaptational explanations are *backward looking*. They are also legitimate answers to a causal reading of the question "Why is E there?"

Constitutive explanations explain how E works. They are *downward looking* in that they situate E with respect to the portion of the causal nexus at a lower (-1 or -m) level in a hierarchy of mechanisms. E is a "black box," but if we look within, we find that it is composed of the entities and activities at that level. Complex IO functions are especially useful for describing E without reference to such messy details, but they also frame internal mechanistic explanations; it is a requirement on the adequacy of such explanations that they account (more or less) for the input-output functions of the mechanism as a whole.

Finally, contextual explanations are *upward looking*; they situate E with respect to the portion of the causal nexus in a higher (+1 or +n) level in a hierarchy of mechanisms. This is why it is explanatory to cite E's role-function; contextual role-descriptions provide a more or less terse description of how E is related to the other entities and activities in a higher-level mechanism. They are therefore legitimate answers to a second reading of the question, "Why is E there?" in that they show what the item does as a component in a higher-level mechanism.

In the contemporary mechanical philosophy, functional and mechanistic description work in tandem to bring intelligible order to complex systems. By identifying functions within such systems, one approaches the system with some set of interests and perspectives in mind. One might be interested in understanding how parts of organisms work, how they break or become diseased, or how they might be commandeered for our

own purposes. Regardless of which perspective one takes, the identification of functions is a crucial step in the discovery of mechanisms. We no longer speak of mechanisms simpliciter, but rather as mechanisms *for* some behavior. Mechanistic descriptions thus come loaded with teleological content concerning the role, goal, purpose, or preferred behavior of the mechanism. This teleogical loading cannot be reduced to features of the causal structure of the word, but it is ineliminable from our physiological, and particularly neural, sciences, precisely because their central goal is to make the busy and buzzing confusion of complex systems intelligible and, in some cases, usable.

Daniel Dennett (1987) suggests that we make the world intelligible by taking different stances: the intentional stance, the design stance, and the physical stance. My discussion has been about three ways of making things intelligible within a kind of mechanistic design stance, liberated from Dennettian associations with adaptationism and optimality: a stance that there is a behavior that the mechanism as a whole exhibits (that it is the mechanism *of* a behavior) and that the components of the mechanism are organized and interact such that they exhibit its overall behavior. Whether the teleology of our contemporary mechanical world-view is ultimately reducible to features of the causal structure of the world thus depends on whether the ability to *take* a stance with respect to a system can be situated without remainder within the causal structure of the world. And here we have a, perhaps *the*, central puzzle that any properly mechanical understanding of mind must some-day face.

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