This chapter reviewed the ample evidence for zombie agents—highly specialized, sensory-motor agents that work quite well without giving rise to phenomenal sensations. The hallmarks of a zombie agent are (1) rapid, reflex-like processing, (2) a narrow but specific input domain, (3) a specific behavior, and (4) the lack of access to working memory.

In the visual domain, Milner and Goodale argue for two distinct processing strategies, vision-for-action and vision-for-perception, implemented by networks within the dorsal and the ventral pathways, respectively. Because the job of visuo-motor agents is to grasp or point at things, they need to encode the actual distance between the body and these objects, their size, and other metric measures. The vision-for-perception mode mediates conscious vision. It must recognize things, no matter their size, orientation, or location. This explains why zombie agents access more veridical information about spatial relationships in the world than perception can. That is, while you may not see what is really out there, your motor system does. Prominent examples of such dissociations include tracking targets with the eyes, adjusting body posture, estimating the steepness of hills, and night walking.

Zombie agents control your eyes, hands, feet, and posture, and rapidly transduce sensory input into stereotypical motor output. They might even trigger aggressive or sexual behaviors when getting a whiff of the right stuff. All, however, bypass consciousness. This is the zombie you.

So far, I have said nothing about the differences between zombie and conscious processing modes at the neuronal level. The forward propagating netwave triggered by a brief sensory input may be too transient to be sufficient for the NCC, but can mediate zombie behaviors. What is needed for conscious perception is enough time for feedback activity from frontal areas to build up stable coalitions. I will expand on this theme in Section 15.3.

Dissociations between conscious and nonconscious behaviors can be more prominent in sickness. This is the topic of the next chapter.

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13.1 | VISUAL AGNOSIA

Pure agnosia, a relatively rare condition, is defined as a failure of recognition that cannot be attributed to elementary sensory defects (e.g., retinal deficits), mental or linguistic deterioration, or attentional disturbances. It is often limited to one sensory modality. Typically, a visual agnosia patient can't recognize a set of keys on a chain dangling in front of her. If she grasps them or if they are jingled, she immediately knows what they are.
Poetically termed *Seelenblindheit* (literally, blindness of the soul), it was rechristened agnosia by Freud and this term has remained. All sorts of quirky subcategories exist, including the inability to perceive color (*achromatopsia*; Section 8.2), the loss of motion perception (*akinetopsia*; Section 8.3), the inability to identify faces (*prosopagnosia*; Section 8.5), and *Capgras syndrome*, in which the patient insists that a loved one, say his wife, has been taken over by an imposter who looks, talks, and feels exactly the way she used to do before the alien replaced her.1

The often quite localized brain damage associated with agnosia implies that the NCC for specific perceptual attributes, such as color, motion, faces, or the feeling of familiarity, is restricted to a part of the cerebral cortex; that is, a particular brain region is an essential node for the perceptual trait in question. Armed with single-cell data from the monkey, Francis and I have hypothesized that the NCC in these essential nodes is based on an explicit, columnar representation (Section 2.2).

Take D.F., a patient who suffers from profound object agnosia, caused by a near-fatal carbon monoxide poisoning at age 34. The oxygen deprivation led to widespread and irreversible damage throughout her brain.2

D.F. does not recognize most objects by sight, but does well when she can touch them. She can’t tell whether a pencil held in front of her is horizontal or vertical, whether she is looking at a square or a triangle, and she is unable to copy simple drawings (Figure 13.1). Yet she is not blind. She can see colors, she can identify some objects by their distinct texture or color (e.g., a yellow banana), and she can sketch things from memory. D.F. walks around on her own, steps over blocks placed in her way, and catches a ball or a wooden stick thrown to her. D.F. grasps objects placed in front of her with considerable accuracy and confidence, even though she cannot see them in any meaningful manner. She doesn’t see the orientation of an elongated slot, she can’t talk about it, nor can she rotate her hand to match its slant. When she has to place a card into the slot from arm’s length away, however, she easily complies, rotating her hand to the correct orientation as soon as it starts to move toward the slot (Figure 13.2). She can even do this when the light goes off as soon as she initiates this action. In other words, the patient couldn’t have used visual feedback to guide her hand.

D.F. scales her grip to the size of the objects she has to pick up, even though she can’t tell a smaller from a larger one. The larger the object, the bigger the opening between thumb and fingers. When D.F. reaches for an object two sec-

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1Fahm (1990); Damasio, Tranel, and Rizzo (2000); Bauer and Demery (2003); and the exhaustive Grüsser and Landis (1991) review the clinical literature on agnosia.

2The original case report is Milner et al. (1991). See the two monographs by Milner and Goodale (1995) and by Goodale and Milner (2004) for details. Brain imaging confirms that the damage impacted D.F.'s ventral pathway much more than it did her dorsal one.
Chapter Thirteen | Agnosia, Blindsight, Epilepsy, and Sleep-Walking

First brought up in Section 12.2, it implies that the networks responsible for D.F.'s reaching movements do not access working memory. Indeed, why should they? Because they are concerned with the here and now, they operate purely on-line.

What this particular agnosia patient vividly exemplifies is that visual form and object information may be lost to consciousness yet continues to shape behavior. Milner and Goodale formulated their innovative two-visual-streams framework in light of D.F.'s pattern of lost and retained faculties: one for conscious vision and one for translating retinal input into motor actions without evoking any sensations. This is an elaboration of the Ungerleider-Mishkin “where” versus “what” distinction (Section 7.5); Milner and Goodale argue that the neuronal substrate for nonconscious, visuo-motor actions is located along the dorsal, vision-for-action pathway, while object detection and other tasks involving visual consciousness rely on the ventral, vision-for-perception pathway that has been severely compromised by anoxia in D.F. (Figure 7.3).

13.2 | BLINDSIGHT

Blindsight is an unusual condition in which the patient points at a target or correctly guesses its color or orientation, while strenuously denying any visual sensation. Different from visual agnosia, the patient doesn’t see anything in the affected field of view. It is a strange syndrome that elicited disbelief and howls of derision when first reported in the literature. Due to the concerted and decades-long research efforts of the neuropsychologists Larry Weiskrantz and Alan Cowey at Oxford University and Petra Stoerig at the University of Düsseldorf in Germany, the initial criticisms have been adequately addressed. The syndrome proves the existence, in a handful of individuals, of some limited visuo-motor behaviors without seeing. Hence the oxymoronic name blindsight.1

Blindsight results from damage to the primary visual cortex. Consequently, the patient fails to see anything in the hemifield opposite the impaired V1. He is blind there. Nevertheless, he can still point roughly in the direction of a bright light: “I have no idea where the target is, but it could be over there.” One such patient, G.Y., can nearly always surmise in which direction a spot of light is moving. If either the motion is too slow or the contrast of the target is too low, performance falls to chance levels—that is, the patient is truly guessing. Conversely, if the motion is salient enough, an ill-defined conscious percept may be reported, akin to “waving your hand in front of your eyes when they are closed,” in the words of one patient. Other blindsight patients can distinguish a cross from a circle, a horizontal line from a vertical line, or can correctly guess which one of two colors is present.

Some blindsighted individuals can grasp at objects, even though they can’t see their form. Their performance improves as the lag between stimulus and response decreases.2 That is, the faster the patient points at an unseen target, the better her performance (recall that imposing a two-second delay eliminated D.F.’s grip-scaling abilities). The subliminal stimulus representations in blindsight are fragile. Without feedback from the perceptual system, the performance can’t tolerate significant delays.

It is important to emphasize that blindsight patients don’t have normal visual abilities without consciousness. There is no evidence that they can track one of several independently moving targets, can process information pertaining to multiple objects, or can recognize complex pictures. Most importantly, because they don’t see, they can’t use visual information to plan. If forced to, a blindsight patient might be able to correctly guess whether or not a water bottle was present in the blind field, but he wouldn’t use this information to plan how to cross a desert. Information in the blind field is not used in any spontaneous, intentional manner. Blindsight patients, therefore, are a far cry from the philosopher’s zombie.

In blindsight, the dominant retinal output—the geniculo-cortical channel—has lost its terminus in V1. So, how does visual information reach the motor regions? The most likely route involves the ganglion cell axons that connect the retina to the superior colliculus (Figure 3.6). From there, the information moves through the pulvinar of the thalamus into extrastriate visual cortices, bypassing the damaged V1.3

I expressed in the opening page of Chapter 8 my belief that this subcortical bypass into the visual hierarchy is too weak (either in amplitude or in duration) to correlate neurons along the ventral stream for long enough into the necessary coalition to be sufficient for a percept. This pathway may be sufficient, though, to trigger some behavior under impoverished conditions (e.g., only a single target is presented, as in almost all blindsight studies). I suspect that when confronted with a more complex scene, the representations of the

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1These delay manipulations and their effect on reaching in D.F. and in normals are described in Goodale, Jakobson, and Kellogg (1994) (see also Bridgeman, Peery, and Anand, 1997). Hu and Goodale (2000) further characterized the different computations underlying direct and pantoimnic grasping in healthy subjects.

2The original report was based on patients with visual field defects who could detect stimuli in their blind field by pointing at them while denying seeing them (Röppl, Held, and Frost, 1973). The most up-to-date reference is the extensive monograph by Weiskrantz (1997), while Cowey and Weiskrantz (1996) offer concise summaries. Kenridge, Heywood, and Stoerig (1991) and Weiskrantz (1996) provide a taste of the ongoing controversy surrounding blindsight patients.

3Pererin and Rossetti (1996), Rossetti (1998), and A. Cowey (personal communication).

4Minor projections from the LGN into V2 or higher cortical areas are another possibility.
manifestations, are generalized seizures (grand mal) that engulf the entire brain, with associated convulsions—the rhythmic tightening and relaxing of the muscles—and total loss of consciousness.8

Of more interest to students of consciousness are focal or partial seizures that begin in or involve one part of the brain. In a simple partial seizure, consciousness is not impaired. The characteristic rhythmic twitching might be limited to a limb and the patient can experience strange tastes, smells, or feelings. These symptoms, referred to as an aura, can be harbingers of worse things to come, when the simple seizure turns into a complex one.

Complex partial seizures are characterized by a murring or loss of awareness, accompanied by automatisms, such as chewing, smacking the lips, coordinated hand and arm movements—as if directing an imaginary orchestra—laughing, or acting scared, fiddling with clothes, verbal utterings, and so on. If not restrained, the person may wander about and “wake up” far from home or the hospital. Usually, the patient has no memory of any events during the seizure. Once the attack is over, some patients lapse into an exhausted sleep or a period of confusion while others are almost instantly fully responsive, as if a switch had been thrown.9 Complex focal seizures often occur in the temporal lobe and last for a few minutes.

A patient experiencing one of these seizures may seem conscious, given her behavior and apparent display of emotions. On a subsequent attack, however, the same, though not identical, motor manifestations return. The patient once again smiles and tries to leave her bed. She behaves like an actor during an audition who goes through the same scene again and again, each time laughing on cue. After observing a few of these individuals, automatisms can readily be distinguished from conscious behaviors by their unnatural, forced, and obsessive quality.

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8 A seizure can be defined as an episode of hyper-synchronous, self-maintained neuronal discharge. Affected neurons fire in an elevated and highly synchronized manner, instead of in their usual disjoint and sparse fashion.

9 I recommend Fried (1997) or Elger (2000) for an introduction to complex, partial seizures, Penfield and Jasper (1954) as the classic treatise on the subject, and Oxbury, Polkey, and Duchowny (2000) for its exhaustive coverage. For two thoughtful essays on the loss of consciousness during seizures, see Groarke, Olivier, and Ives (1980) and Groarke (1986). I am struck by the extreme heterogeneity of seizures (simple and complex seizures, absences, myoclonic seizures, and generalized tonic-clonic ones). Their origin, and their consequent spread to other brain areas, varies tremendously, too, as do their duration, semiology, and associated auras. I am here mainly referring to focal seizures in the temporal lobes of adults. Another fascinating topic is absence seizures or petit mal. These are brief periods of wakeful unconsciousness. Most common in children, an absence seizure suddenly interrupts ongoing mental and physical activity for a few seconds, during which the child will stare motionless into space, before returning abruptly to the land of the living. Absence seizures involve the generation of abnormal oscillatory discharges in thalamocortical circuitry (Crunelli and Leresche, 2002). They can occur frequently, do not go hand-in-hand with muscle activity, and could be studied profitably using event-related fMRI.

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7 Cowey and Stoerig (1995). Stoerig, Zontanou, and Cowey (2002) followed up their seminal animal experiment by comparing one of these monkeys with four patients with unilateral field defects. This validated their approach, in the sense that the monkey and the people responded in similar ways, serving as a potent reminder that the neuroanatomy and psychology of vision is similar in both species.
Some patients can interact, at least in a limited manner, with their environment during these brief episodes. One patient can answer some routine questions. Another reported an attack while riding his bicycle to work. After setting out in the morning, he would occasionally find himself riding back home along his usual route, all while having a seizure. Others engage in nocturnal wanderings. All of this raises the question whether these automatism are remnants of zombie agents that animate the otherwise unconscious patient.

The extent to which consciousness is actually lost during the seizure is difficult to verify in a clinical setting. One way to assess whether anything but zombie behaviors are present is to evaluate working memory during a fit. As mentioned in Chapters 11 and 12, the ability to store and make use of information over many seconds is a hallmark of conscious processes, and one not enjoyed by zombies.

It seems plausible that automatism occur whenever the aberrant electrical discharges destroy the coalitions making up the NCC, while the neural activity underlying zombie behaviors is more resistant to such interference. Clinical evidence suggests a hemispheric bias. Partial seizures with left or bilateral temporal lobe involvement are more likely to interfere with consciousness than right temporal lobe seizures.

To study automatism in any rigorous manner is not easy. They occur unpredictably, and the patient may be unable to carry out the previously rehearsed motor program because his brain won't cooperate. What is clear is that some fairly elaborate sensory-motor behaviors are retained without much, if any, conscious sensation.

### 13.4 SLEEPWALKING

What about people who walk in their sleep? Are zombie agents at work during somnambulistic activities, which range from the prosaic—sitting up in bed and mumbling incomprehensibly— to the more unusual—dressing and undressing, going to the bathroom, and moving furniture— to the bizarre—climbing out of the window or driving a car? Sleepwalkers sure seem unconscious while they stumble around their bedrooms, don't respond when spoken to, and don't recall anything out of the ordinary the next morning.

Sleepwalking episodes last from a fraction of a minute to half an hour. They are more frequent in children than in adults, occur during the nonrapid eye movement phase of sleep, and leave no explicit, conscious recollection upon waking.

Sleepwalkers display zombie traits instantly recognizable by any horror movie fan—absence of sensations and feelings, glazed eyes, the extraordinary strength, and the clumsy movements. To wit,

They appeared, although in a state of frenzy and intense autonomic arousal, as automatons unaware of what they were doing and unresponsive to stimuli from their environment.

Occasionally, sleepwalkers turn violent and become a danger to themselves, their sleeping partners, and others. Rarely, this has ended in death. When these cases come to trial, some sort of noninsane automatism defense is invoked, based on the argument that the defendant wasn't himself when the homicide occurred. By today's medico-legal standards concerned with conscious intention, a sleepwalker is like a zombie, a person with limited behavioral repertoire and no conscious sensation.

Little is known about the spectrum of behaviors expressed during somnambulism. Within the visual modality, is saliency-based attention operating? Probably. Can the sleepwalker pay (top-down) attention to events or objects? Probably not. Is working memory functioning? Unlikely. Are the sensory-motor agents that control eye movements, posture, reaching, and gait, active? Probably, to some extent.

What are the underlying pathological mechanisms? Because sleepwalking occurs in deep sleep, the low level of arousal signals from the brainstem may be

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10 Pedley and Guilleminault (1977).

11 Suppose, for instance, that the patient hears either a low- or a high-pitch tone during the attack. A few seconds later, the same or a different tone is presented again and the patient has to point with his hand, arm, or head to the ground if the tones are the same and point to the skyward if the tones are different. To perform this experiment, the patient must remember the task instructions during the seizure, have at least some limited degree of control over his limbs, and be able to hear the tones (M. Karlten, T. Grunwald, and C. Koch, personal communication).

12 Ebner et al. (1995), Inoue and Mihara (1998), and Lux et al. (2002). The left hemisphere is usually dominant for language (Chapter 17), prompting speculation about a relationship between loss of consciousness as measured at the bedside and aphasia. In at least one case (C. Elger, personal communication), loss of responsiveness occurred following a focal seizure in the left temporal lobe of a patient whose right hemisphere was dominant for language (as assessed by the Wada test). Thus, the link between behavior and no consciousness and the left temporal lobe transcends language. The minimal brain structures that must be engulfed by the epileptic discharges for consciousness to be lost remain unknown (Reeves, 1985).

13 Sleepwalkers are characteristically deep sleepers, are hard to awaken, and have low dream recall. For reviews on sleepwalking, see Kaveny et al. (1990); Massand, Popul, and Weilburg (1995); and Vgontzas and Kales (1999). The possible relationship to consciousness is discussed in Neonsou et al. (2000).

14 Something Shakespeare recognized long ago. In Act V, Scene I of Macbeth, the doctor notes that Lady Macbeth is wandering around. He says, "You see, her eyes are open," to which the Lady in Waiting comments, "Ay, but their sense is shut." See also Jacobson et al. (1965).

15 Page 738 in Moldofsky et al. (1995).

16 For a thoughtful discussion of one sleepwalking case that resulted in mayhem and death, and the attendant legal implications, see Broughton et al. (1994). Other examples are discussed by Moldofsky et al. (1995) and Schenck and Mahowald (1998).
insufficient to support the sustained feedback activity needed for a dominant coalition to establish itself as NCC (that is, the NCC's of Chapter 5 are not fully present), but adequate to mediate the more transient, feedforward activity sufficient to power zombie agents. Answering these questions in any decisive manner will be difficult unless a method to reliably induce sleepwalking in human volunteers, monkeys, or mice is discovered.

13.5 ZOMBIE AGENTS AND THE NCC

Now that you've learned there's an army of nonconscious zombie agents in your head, how does this aid the quest to understand the NCC?

First, it puts to rest the idea that the computational complexity of a sensory-motor task can separate nonconscious from conscious actions in any straightforward manner. Zombie agents mediate nontrivial motor programs, and not mere reflexes. Imagine, for example, the web of processes necessary to evaluate the optical flow patterns impinging upon the eyes, combining this with information from the vestibular sense, and adjusting the skeletal-muscular system accordingly to retain an upright posture. As long as these procedures occur over and over, however, they can be learned (Section 11.2) by the cortex in collusion with the basal ganglia. Any distinction between nonconscious and conscious processes has to take this learning aspect into account, something I will develop more fully in the next chapter.

Second, what about the pathways underlying zombie actions? One possibility is that they might be physically distinct and separate from the networks that generate the NCC. That is, neural activity in some regions of the brain mediates behavior without consciousness while activity elsewhere is sufficient for sensations. Milner and Goodale argue forcefully that vision-for-action is carried out by the dorsal pathway while vision-for-perception is consigned to the ventral pathway. Another possibility is that the same network operates in two distinct modes. One is based on a transient net-wave that originates in the sensory periphery (e.g., in the retina) and quickly moves through the various cortical processing stages until it triggers a stereotyped nonconscious response. This traveling net-wave is too ephemeral to leave elevated firing in its wake. This would be the zombie mode of action in which the brain operates in what is essentially a feedforward manner, without significant active feedback (Table 5.1).

If the input is more sustained and is boosted by top-down attention, on the other hand, a sort of standing wave or resonance might be created in the network, with vital contributions from the feedback pathways. Both local and more global feedback could cause neurons to synchronize their spiking activity above and beyond the degree of synchronization that results from the sensory input by itself. This increases their postsynaptic punch compared to when they fire independently. A powerful coalition of neurons could be assembled in this manner, able to project its influence to the far reaches of the cortex and below. This would be the slow mode that underlies conscious perception.

Although these ideas are still inchoate, they can guide more detailed investigations. Given the highly variable nature and time course of the neurological deficits discussed here and the ethical limits on human experimentation, such investigations need to focus on appropriate animal models. Studying patients is essential to fully characterizing the phenomenology of deficits as they relate to consciousness. But untangling the underlying neuronal circuits demands interventions that selectively target discrete cellular components in the vast fields of the brain, and this can't be done in humans.

13.6 A TURING TEST FOR CONSCIOUSNESS?

In 1950, the mathematician Alan Turing published a paper in which he considered the question of "Can machines think?" within the framework of an imitation game. Known today as a Turing Test, it involves carrying on an extended conversation with an entity, via typed natural language, on a variety of arbitrary topics, from the mundane to the esoteric. If, after a while, the observer is unable to decide whether this entity has been interacting with a machine or a human, it must be deemed intelligent. The Turing Test offers a practical means to gauge progress in designing intelligent machines. A similar operational means to distinguish automatic zombie behaviors from those that require consciousness would be desirable.

Of tremendous significance is the evidence from normals, D.F., and blindsight patients that a delay of more than a few seconds virtually eliminates many of their zombie behaviors. In Chapter 11, I surmised that sophisticated actions that require the retention of information over seconds, such as trace conditioning or working memory, might be further litmus tests. Jointly, such a battery of operations might distinguish automatic from conscious behaviors.

Take your favorite sensory-motor routine in some species and enforce a waiting period of a few seconds between the sensory input and the execution of an action. If the subject can't perform the task with the delay, it was probably mediated by a zombie agent. If the organism's performance is only marginally affected by the delay, then the input must have been stored in some sort of intermediate, short-term buffer, implying some measure of consciousness. If the subject can be successfully distracted during this interval by a suit-

able salient stimulus (e.g., flashing lights), it would reinforce the conclusion that attention was involved in actively maintaining information during the delay period.

Dogs, probably like all mammals, easily pass this test. Think of hiding a bone well out of sight and teaching the dog to sit still until you tell her to “go fetch the bone.”

This is not meant to be an infallible test, but good enough to be practical and useful at the bedside or in the laboratory. Of course, such tests are irrelevant for investigating questions of machine consciousness, since computers, robots, and other man-made artifacts are constrained by radically different forces than biological organisms.

13.7 | Recapitulation

In this chapter I examined pathologies that offer tantalizing glimpses of what humans can do without consciousness.

In visual agnosia, patients lose one or more specific aspects of visual perception (color, motion, faces, form). Patient D.F. is a case in point. She can't identify objects or recognize their shape or form. Yet she retains remarkable visual reactivity: she can slide her hand into a slit of variable orientation, she can grasp things appropriately, and she can walk around without bumping into objects placed in her way. Blindsight patients are blind in part of their visual field, but they can, if forced to, point to a bright light, move their eyes toward it, guess at the color of an invisible stimulus, and so on.

Some of these behaviors break down if a delay of several seconds is interposed between stimulus presentation and action, suggesting that these patients lack the necessary facilities to store information for more than a few seconds. The delay test proposed here constitutes a practical means to experimentally differentiate zombie agents from conscious systems in animals, babies, or severely disabled patients.

Some patients with complex, focal seizures or people who sleepwalk exhibit fairly elaborate learned motor patterns; they wander around, move furniture, or drive a car. They typically don't respond to verbal commands nor do they recall events that occurred during their episodes. These automatisms follow an internal program that can be influenced—to a limited extent—by the environment.

What underlies the distinction between automatic behaviors and those that rely on consciousness? Conceptually simplest is the possibility that distinct networks mediate zombie and conscious actions. Zombie agents might live outside the cortex proper as well as in the dorsal stream, while conscious visual perception is mediated by the ventral pathway. Like a long-time married couple, they each have their own vices and virtues, but manage to harmoniously work together.

Alternatively, the same network may operate in two modes. A transient net-wave is propelled from the sensory periphery through the cortical hierarchy to the output stages. This happens so quickly that each neuron contributes only a few spikes, leaving no long-lasting activity in the wake of the net-wave. This is sufficient to initiate stereotypical actions without any associated sensation. If the input is more sustained or is boosted by a top-down attentional bias, on the other hand, it sets up long-lasting reverberatory activity that is powerful enough to generate the coalitions sufficient for conscious perception.

If zombies are so wonderful, what is the function of consciousness? Why bother with consciousness at all? In tackling these questions next, I'll have to grapple with the two concepts central to the mind-body problem—qualia and meaning.