Ability to Learn Grammar Laid to Gene by Researcher. This 1992 headline appeared not in a supermarket tabloid but in an Associated Press news story, based on a report at the annual meeting of the principal scientific association in the United States. The report had summarized evidence that Specific Language Impairment runs in families, focusing on the British family we met in Chapter 2 in which the inheritance pattern is particularly clear. The syndicated columnists James J. Kilpatrick and Erma Bombeck were incredulous. Kilpatrick’s column began:

Better Grammar through Genetics

Researchers made a stunning announcement the other day at a meeting of the American Association for the Advancement of Science. Are you ready? Genetic biologists have identified the grammar gene.

Yes! It appears from a news account that Steven Pinker of MIT and Myrna Gopnik of McGill University have solved a puzzle that has baffled teachers of English for years. Some pupils master grammar with no more than a few moans of protest. Others, given the same instruction, persist in saying that

Susie invited her and I to the party. It is all a matter of heredity. This we can handle.

A single dominant gene, the biologists believe, controls the ability to learn grammar. A child who says “them marbles is mine” is not necessarily stupid. He has all his marbles. The child is simply a little short on chromosomes.

It boggles the mind. Before long the researchers will isolate the gene that controls spelling . . . [the column continues] . . . neatness . . . The read-a-book gene . . . a gene to turn down the boom box . . . another to turn off the TV . . . politeness . . . chores . . . homework . . .

Bombeck wrote:

Poor Grammar? It Are in the Genes

It was not much of a surprise to read that kids who are unable to learn grammar are missing a dominant gene . . . At one time in his career, my husband taught high school English. He had 37 grammar-gene deficients in his class at one time. What do you think the odds of that happening are? They didn’t have a clue where they were. A comma could have been a petroglyph. A subjective complement was something you said to a friend when her hair came out right. A dangling participle was not their problem . . .

Where is that class of young people today, you ask? They are all major sports figures, rock stars and television personalities who make millions spewing out words such as “bummer,” “radical” and “awesome” and thinking they are complete sentences.

The syndicated columns, third-hand newspaper stories, editorial cartoons, and radio shows following the symposium gave me a quick education about how scientific discoveries get addled by journalists working under deadline pressure. To set the record straight: the discovery of the family with the inherited language disorder belongs to Gopnik; the reporter who generously shared the credit with me was
confused by the fact that I chaired the session and thus introduced Gopnik to the audience. No grammar gene was identified; a defective gene was inferred, from the way the syndrome runs in the family. A single gene is thought to disrupt grammar, but that does not mean a single gene controls grammar. (Removing the distributor wire prevents a car from moving, but that does not mean a car is controlled by its distributor wire.) And of course, what is disrupted is the ability to converse normally in everyday English, not the ability to learn the standard written dialect in school.

But even when they know the facts, many people share the columnists' incredulity. Could there really be a gene tied to something as specific as grammar? The very idea is an assault on the deeply rooted belief that the brain is a general-purpose learning device, void and without form prior to experience of the surrounding culture. And if there are grammar genes, what do they do? Build the grammar organ, presumably—a metaphor, from Chomsky, that many find just as preposterous.

But if there is a language instinct, it has to be embodied somewhere in the brain, and those brain circuits must have been prepared for their role by the genes that built them. What kind of evidence could show that there are genes that build parts of brains that control grammar? The ever-expanding toolkit of the geneticist and neurobiologist is mostly useless. Most people do not want their brains impaled by electrodes, injected with chemicals, rearranged by surgery, or removed for slicing and staining. (As Woody Allen said, “The brain is my second-favorite organ.”) So the biology of language remains poorly understood. But accidents of nature and ingenious indirect techniques have allowed neurolinguists to learn a surprising amount. Let’s try to home in on the putative grammar gene, beginning with a bird’s-eye view of the brain and zooming in on smaller and smaller components.

We can narrow down our search at the outset by throwing away half the brain. In 1861 the French physician Paul Broca dissected the brain of an aphasic patient who had been nicknamed “Tan” by hospital workers because that was the only syllable he uttered. Broca discovered a large cyst producing a lesion in Tan’s left hemisphere. The next eight cases of aphasia he observed also had left-hemisphere lesions, too many to be attributed to chance. Broca concluded that “the faculty for articulate language” resides in the left hemisphere.

In the 130 years since, Broca’s conclusion has been confirmed by many kinds of evidence. Some of it comes from the convenient fact that the right half of the body and of perceptual space is controlled by the left hemisphere of the brain and vice versa. Many people with aphasia suffer weakness or paralysis on the right side, including Tan and the recovered aphasic of Chapter 2, who awoke thinking that he had slept on his right arm. The link is summed up in Psalms 137:5–6:

If I forget thee, O Jerusalem, let my right hand forget her cunning.

If I do not remember thee, let my tongue cleave to the roof of my mouth.

Normal people recognize words more accurately when the words are flashed to the right side of their visual field than when they are flashed to the left, even when the language is Hebrew, which is written from right to left. When different words are presented simultaneously to the two ears, the person can make out the word coming into the right ear better. In some cases of otherwise incurable epilepsy, surgeons disconnect the two cerebral hemispheres by cutting the bundle of fibers running between them. After surgery the patients live completely normal lives, except for a subtlety discovered by the neuroscientist Michael Gazzaniga: when the patients are kept still, they can describe events taking place in their right visual field and can name objects in their right hand, but cannot describe events taking place in their left visual field or name objects placed in their left hand (though the right hemisphere can display its awareness of those events by nonverbal means like gesturing and pointing). The left half of their world has been disconnected from their language center.

When neuroscientists look directly at the brain, using a variety of techniques, they can actually see language in action in the left hemi-
The anatomy of the normal brain—its bulges and creases—is slightly asymmetrical. In some of the regions associated with language, the differences are large enough to be seen with the naked eye. Computerized Axial Tomography (CT or CAT) and Magnetic Resonance Imaging (MRI) use a computer algorithm to reconstruct a picture of the living brain in cross-section. Aphasics’ brains almost always show lesions in the left hemisphere. Neurologists can temporarily paralyze one hemisphere by injecting sodium amytal into the carotid artery. A patient with a sleeping right hemisphere can talk; a patient with a sleeping left hemisphere cannot. During brain surgery, patients can remain conscious under local anesthetic because the brain has no pain receptors. The neurosurgeon Wilder Penfield found that small electric shocks to certain parts of the left hemisphere could silence the patient in mid-sentence. (Neurosurgeons do these manipulations not out of curiosity but to be sure that they are not cutting out vital parts of the brain along with the diseased ones.) In a technique used on normal research subjects, electrodes are pasted all over the scalp, and the subjects’ electroencephalograms (EEG’s) are recorded as they read or hear words. There are recognizable jumps in the electrical signal that are synchronized with each word, and they are more prominent in the electrodes pasted on the left side of the skull than in those on the right (though this finding is tricky to interpret, because an electrical signal generated deep in one part of the brain can radiate out of another part).

In a new technique called Positron Emission Tomography (PET), a volunteer is injected with mildly radioactive glucose or water, or inhales a radioactive gas, comparable in dosage to a chest X-ray, and puts his head inside a ring of gamma-ray detectors. The parts of the brain that are more active burn more glucose and have more oxygenated blood sent their way. Computer algorithms can reconstruct which parts of the brain are working harder from the pattern of radiation that emanates from the head. An actual picture of metabolic activity within a slice of the brain can be displayed in a computer-generated photograph, with the more active areas showing up in bright reds and yellows, the quiet areas in dark indigos. By subtracting an image of the brain when its owner is watching meaningless patterns or listening to meaningless sounds from an image when the owner is understanding words or speech, one can see which areas of the brain “light up” during language processing. The hot spots, as expected, are on the left side.

What exactly is engaging the left hemisphere? It is not merely speechlike sounds, or wordlike shapes, or movements of the mouth, but abstract language. Most aphasic people—Mr. Ford from Chapter 2, for example—can blow out candles and suck on straws, but their writing suffers as much as their speech; this shows that it is not mouth control but language control that is damaged. Some aphasics remain fine singers, and many are superb at swearing. In perception, it has long been known that tones are discriminated better when they are played to the left ear, which is connected most strongly to the right hemisphere. But this is only true if the tones are perceived as musical sounds like hums; when the ears are Chinese or Thai and the same tones are features of phonemes, the advantage is to the right ear and the left hemisphere it feeds.

If a person is asked to shadow someone else’s speech (repeat it as the talker is talking) and, simultaneously, to tap a finger to the right or the left hand, the person has a harder time tapping with the right finger than with the left, because the right finger competes with language for the resources of the left hemisphere. Remarkably, the psychologist Ursula Bellugi and her colleagues have shown that the same thing happens when deaf people shadow one-handed signs in American Sign Language: they find it harder to tap with their right finger than with their left finger. The gestures must be tying up the left hemispheres, but it is not because they are gestures; it is because they are linguistic gestures. When a person (either a signer or a speaker) has to shadow a goodbye wave, a thumbs-up sign, or a meaningless gesticulation, the fingers of the right hand and the left hand are slowed down equally.

The study of aphasia in the deaf leads to a similar conclusion. Deaf signers with damage to their left hemispheres suffer from forms of sign aphasia that are virtually identical to the aphasia of hearing
victims with similar lesions. For example, Mr. Ford’s sign-language counterparts are unimpaired at nonlinguistic tasks that place similar demands on the eyes and hands, such as gesturing, pantomiming, recognizing faces, and copying designs. Injuries to the right hemisphere of deaf signers produce the opposite pattern: they remain flawless at signing but have difficulty performing visuospatial tasks, just like hearing patients with injured right hemispheres. It is a fascinating discovery. The right hemisphere is known to specialize in visuospatial abilities, so one might have expected that sign language, which depends on visuospatial abilities, would be computed in the right hemisphere. Bellugi’s findings show that language, whether by ear and mouth or by eye and hand, is controlled by the left hemisphere. The left hemisphere must be handling the abstract rules and trees underlying language, the grammar and the dictionary and the anatomy of words, and not merely the sounds and the mouthings at the surface.

Why is language so lopsided? A better question is, why is the rest of a person so symmetrical? Symmetry is an inherently improbable arrangement of matter. If you were to fill in the squares of an $8 \times 8$ checkerboard at random, the odds are less than one in a billion that the pattern would be bilaterally symmetrical. The molecules of life are asymmetrical, as are most plants and many animals. Making a body bilaterally symmetrical is difficult and expensive. Symmetry is so demanding that among animals with a symmetrical design, any disease or weakness can disrupt it. As a result, organisms from scorpion flies to barn swallows to human beings find symmetry sexy (a sign of a fit potential mate) and gross asymmetry a sign of deformity. There must be something in an animal’s lifestyle that makes a symmetrical design worth its price. The crucial lifestyle feature is mobility: the species with bilaterally symmetrical body plans are the ones that are designed to move in straight lines. The reasons are obvious. A creature with an asymmetrical body would veer off in circles, and a creature with asymmetrical sense organs would eccentrically monitor one side of its body even though equally interesting things can happen on either side.

Though locomoting organisms are symmetrical side-to-side, they are not (apart from Dr. Dolittle’s Push-mi-pull-you) symmetrical front-and-back. Thrusters apply force best in one direction, so it is easier to build a vehicle that can move in one direction and turn than a vehicle that can move equally well in forward and reverse (or that can scoot off in any direction at all, like a flying saucer). Organisms are not symmetrical up-and-down because gravity makes up different from down.

The symmetry in sensory and motor organs is reflected in the brain, most of which, at least in nonhumans, is dedicated to processing sensation and programming action. The brain is divided into maps of visual, auditory, and motor space that literally reproduce the structure of real space: if you move over a small amount in the brain, you find neurons that correspond to a neighboring region of the world as the animal senses it. So a symmetrical body and a symmetrical perceptual world is controlled by a brain that is itself almost perfectly symmetrical.

No biologist has explained why the left brain controls right space and vice versa. It took a psycholinguist, Marcel Kinsbourne, to come up with the only speculation that is even remotely plausible. All bilaterally symmetrical invertebrates (worms, insects, and so on) have the more straightforward arrangement in which the left side of the central nervous system controls the left side of the body and the right side controls the right side. Most likely, the invertebrate that was the ancestor of the chordates (animals with a stiffening rod around their spinal cords, including fish, amphibians, birds, reptiles, and mammals) had this arrangement as well. But all the chordates have “contralateral” control: right brain controls left body and left brain controls right body. What could have led to the rewiring? Here is Kinsbourne’s idea. Imagine that you are a creature with the left-brain-left-body arrangement. Now turn your head around to look behind you, a full 180 degrees back, like an owl. (Stop at 180 degrees; don’t go around and around like the girl in The Exorcist.) Now imagine that your head is stuck in that position. Your nerve cables have been given a half-twist, so the left brain would control your right body and vice versa.

Now, Kinsbourne is not suggesting that some primordial rubber-
necker literally got its head stuck, but that changes in the genetic instructions for building the creature resulted in the half-twist during embryonic development—a torsion that one can actually see happening during the development of snails and some flies. This may sound like a perverse way to build an organism, but evolution does it all the time, because it never works from a fresh drawing board but has to tinker with what is already around. For example, our sadistically designed S-shaped spines are the product of bending and straightening the arched backbones of our quadrupedal forebears. The Picascoesque face of the flounder was the product of warping the head of a kind of fish that had opted to cling sideways to the ocean floor, bringing around the eye that had been staring uselessly into the sand. Since Kinsbourne’s hypothetical creature left no fossils and has been extinct for over half a billion years, no one knows why it would have undergone the rotation. (Perhaps one of its ancestors had changed its posture, like the flounder, and subsequently righted itself. Evolution, which has no foresight, may have put its head back into alignment with its body by giving the head another quarter-twist in the same direction, rather than by the more sensible route of undoing the original quarter-twist.) But it does not really matter; Kinsbourne is only proposing that such a rotation must have taken place; he is not claiming he can reconstruct why it happened. (In the case of the snail, where the rotation is accompanied by a bending, like one of the arms of a pretzel, scientists are more knowledgeable. As my old biology textbook explains, “While the head and foot remain stationary, the visceral mass is rotated through an angle of 180°, so that the anus ... is carried upward and finally comes to lie [above] the head. . . . The advantages of this arrangement are clear enough in an animal that lives in a shell with only one opening.”)

In support of the theory, Kinsbourne notes that invertebrates have their main neural cables laid along their bellies and their hearts in their backs, whereas chordates have their neural cables laid along their backs and their hearts in their chests. This is exactly what one would expect from a 180-degree head-to-body turn in the transition from one group to the other, and Kinsbourne could not find any reports of an animal that has only one or two out of the three reversals that his theory says must have happened together. Major changes in body architecture affect the entire design of the animal and can be very difficult to undo. We are the descendants of that twisted creature, and half a billion years later, a stroke in the left hemisphere leaves the right arm tingling.

The benefits of a symmetrical body plan all have to do with sensing and moving in the bilaterally indifferent environment. For body systems that do not interact directly with the environment, the symmetrical blueprint can be overridden. Internal organs such as the heart, liver, and stomach are good examples; they are not in contact with the layout of the external world, and they are grossly asymmetrical. The same thing happens on a much smaller scale in the microscopic circuitry of the brain.

Think about the act of deliberately manipulating some captive object. The actions are not being keyed to the environment; the manipulator is putting the object anywhere it wants. So the organism’s forelimbs, and the brain centers controlling them, do not have to be symmetrical in order to react to events appearing unpredictably on one side or the other; they can be tailored to whatever configuration is most efficient to carry out the action. Manipulating an object often benefits from a division of labor between the limbs, one holding the object, the other acting on it. The result is the asymmetrical claws of lobsters, and the asymmetrical brains that control paws and hands in a variety of species. Humans are by far the most adept manipulators in the animal kingdom, and we are the species that displays the strongest and most consistent limb preference. Ninety percent of people in all societies and periods in history are right-handed, and most are thought to possess one or two copies of a dominant gene that imposes the right-hand (left-brain) bias. Possessors of two copies of the recessive version of the gene develop without this strong right-hand bias; they turn into the rest of the right-handers and into the left-handers and ambidextrics.

Processing information that is spread out over time but not space is another function where symmetry serves no purpose. Given a cer-
tain amount of neural tissue necessary to perform such a function, it makes more sense to put it all in one place with short interconnections, rather than have half of it communicate with the other half over a slow, noisy, long-distance connection between the hemispheres. Thus the control of song is strongly lateralized in the left hemispheres of many birds, and the production and recognition of calls and squeaks is somewhat lateralized in monkeys, dolphins, and mice.

Human language may have been concentrated in one hemisphere because it, too, is coordinated in time but not environmental space: words are strung together in order but do not have to be aimed in various directions. Possibly, the hemisphere that already contained computational microcircuitry necessary for control of the fine, deliberate, sequential manipulation of captive objects was the most natural place in which to put language, which also requires sequential control. In the lineage leading to humans, that happened to be the left hemisphere. Many cognitive psychologists believe that a variety of mental processes requiring sequential coordination and arrangement of parts co-reside in the left hemisphere, such as recognizing and imagining multipart objects and engaging in step-by-step logical reasoning. Gaztanaga, testing the two hemispheres of a split-brain patient separately, found that the newly isolated left hemisphere had the same IQ as the entire connected brain before surgery!

Linguistically, most left-handers are not mirror images of the righty majority. The left hemisphere controls language in virtually all right-handers (97%), but the right hemisphere controls language in a minority of left-handers, only about 19%. The rest have language in the left hemisphere (68%) or redundantly in both. In all of these lefties, language is more evenly distributed between the hemispheres than it is in righties, and thus the lefties are more likely to withstand a stroke on one side of the brain without suffering from aphasia. There is some evidence that left-handers, though better at mathematical, spatial, and artistic activities, are more susceptible to language impairment, dyslexia, and stuttering. Even righties with left-handed relatives (presumably, those righties possessing only one copy of the dominant right-bias gene) appear to parse sentences in subtly different ways than pure righties.

Language, of course, does not use up the entire left half of the brain. Broca observed that Tan’s brain was mushy and deformed in the regions immediately above the Sylvian fissure—the huge cleavage that separates the distinctively human temporal lobe from the rest of the brain. The area in which Tan’s damage began is now called Broca’s area, and several other anatomical regions hugging both sides of the Sylvian fissure affect language when they are damaged. The most prominent are shown as the large gray blobs in the diagram (see page 314). In about 98% of the cases where brain damage leads to language problems, the damage is somewhere on the banks of the Sylvian fissure of the left hemisphere. Penfield found that most of the spots that disrupted language when he stimulated them were there, too. Though the language areas appear to be separated by large gulfs, this may be an illusion. The cerebral cortex (gray matter) is a large sheet of two-dimensional tissue that has been wadded up to fit inside the spherical skull. Just as crumpling a newspaper can appear to scramble the pictures and text, a side view of a brain is a misleading picture of which regions are adjacent. Gazzaniga’s coworkers have developed a technique that uses MRI pictures of brain slices to reconstruct what the person’s cortex would look like if somehow it could be unwrinkled into a flat sheet. They found that all the areas that have been implicated in language are adjacent in one continuous territory. This region of the cortex, the left perisylvian region, can be considered to be the language organ.

Let us zoom in closer. Tan and Mr. Ford, in whom Broca’s area was damaged, suffered from a syndrome of slow, labored, ungrammatical speech called Broca’s aphasia. Here is another example, from a man called Peter Hogan. In the first passage he describes what brought him into the hospital; in the second, his former job in a paper mill:

Yes . . . ah . . . Monday . . . ah . . . Dad and Peter Hogan, and Dad . . . ah . . . hospital . . . and ah . . . Wednesday . . . Wednes-
day nine o'clock and ah Thursday . . . ten o'clock ah doctors . . . two . . . two . . . an doctors and . . . ah . . . teeth . . . yah . . . And a doctor an girl . . . and gums, an I.

Lower Falls . . . Maine . . . Paper. Four hundred tons a day! And ah . . . sulphur machines, and ah . . . wood . . . Two weeks and eight hours. Eight hours . . . no! Twelve hours, fifteen hours . . . workin . . . workin . . . workin! Yes, and ah . . . sulphur. Sulphur and . . . Ah wood. Ah . . . handlin! And ah sick, four years ago.

Broca's area is adjacent to the part of the motor-control strip dedicated to the jaws, lip, and tongue, and it was once thought that Broca's area is involved in the production of language (though obviously not speech per se, because writing and signing are just as affected). But the area seems to be implicated in grammatical processing in general. A defect in grammar will be most obvious in the output, because any slip will lead to a sentence that is conspicuously defective. Comprehension, on the other hand, can often exploit the redundancy in speech to come up with sensible interpretations with little in the way of actual parsing. For example, one can understand *The dog bit the man* or *The apple that the boy is eating is red* just by knowing that dogs bite men, boys eat apples, and apples are red. Even *The car pushes the truck* can be guessed at because the cause is mentioned before the effect. For a century, Broca's aphasics fooled neurologists by using shortcuts. Their trickery was finally unmasked when psycholinguists asked them to act out sentences that could be understood only by their syntax, like *The car is pushed by the truck* or *The girl whom the boy is pushing is tall*. The patients gave the correct interpretation half the time and its opposite half the time—a mental coin flip.

There are other reasons to believe that the front portion of the perisylvian cortex, where Broca's area is found, is involved in grammatical processing. When people read a sentence, electrodes pasted over the front of their left hemispheres pick up distinctive patterns of electrical activity at the point in the sentence at which it becomes ungrammatical. Those electrodes also pick up changes during the portions of a sentence in which a moved phrase must be held in memory while the reader awaits its trace, like *What did you say (trace) to John?* Several studies using PET and other techniques to measure blood flow have shown that this region lights up when people listen to speech in a language they know, tell stories, or understand complex sentences. Various control tasks and subtractions confirm that it is processing the structure of sentences, not just thinking about their content, that engages this general area. A recent and very carefully designed experiment by Karin Stromswold and the neurologists David Caplan and Nat Alpert obtained an even more precise picture; it showed one circumscribed part of Broca's area lighting up.

So is Broca's area the grammar organ? Not really. Damage to Broca's area alone usually does not produce long-lasting severe aphasia; the surrounding areas and underlying white matter (which connects Broca's area to other brain regions) must be damaged as well. Sometimes symptoms of Broca's aphasia can be produced by a stroke or Parkinson’s disease that damages the basal ganglia, complex neural centers buried inside the frontal lobes that are otherwise needed for skilled movement. The labored speech output of Broca's aphasics may be distinct from the lack of grammar in their speech, and may impli-
cate not Broca’s area but hidden parts of the cortex nearby that tend to be damaged by the same lesions. And, most surprisingly of all, some kinds of grammatical abilities seem to survive damage to Broca’s area. When asked to distinguish grammatical from ungrammatical sentences, some Broca’s aphasics can detect even subtle violations of the rules of syntax, as in pairs like these:

- John was finally kissed Louise.
- John was finally kissed by Louise.
- I want you will go to the store now.
- I want you to go to the store now.
- Did the old man enjoying the view?
- Did the old man enjoy the view?

Still, aphasics do not detect all ungrammaticalities, nor do all aphasics detect them, so the role of Broca’s area in language is maddeningly unclear. Perhaps the area underlies grammatical processing by converting messages in mentalese into grammatical structures and vice versa, in part by communicating via the basal ganglia with the prefrontal lobes, which subserve abstract reasoning and knowledge.

Broca’s area is also connected by a band of fibers to a second language organ, Wernicke’s area. Damage to Wernicke’s area produces a very different syndrome of aphasia. Howard Gardner describes his encounter with a Mr. Gorgan:

> “What brings you to the hospital?” I asked the 72-year-old retired butcher four weeks after his admission to the hospital.

> “Boy, I’m sweating, I’m awful nervous, you know, once in a while I get caught up, I can’t mention the tarripoi, a month ago, quite a little, I’ve done a lot well, I impose a lot, while, on the other hand, you know what I mean, I have to run around, look it over, trebbin and all that sort of stuff.”

> I attempted several times to break in, but was unable to do so against this relentlessly steady and rapid outflow. Finally, I put up my hand, rested it on Gorgan’s shoulder, and was able to gain a moment’s reprieve.

> “Thank you, Mr. Gorgan. I want to ask you a few—”

> “Oh sure, go ahead, any old think you want. If I could I would. Oh, I’m taking the word the wrong way to say, all of the barbers here whenever they stop you it’s going around and around, if you know what I mean, that is tying and tying for repucer, repuceration, well, we were trying the best that we could while another time it was with the beds over there the same thing . . .”

Wernicke’s aphasia is in some ways the complement of Broca’s. Patients utter fluent streams of more-or-less grammatical phrases, but their speech makes no sense and is filled with neologisms and word substitutions. Unlike many Broca’s patients, Wernicke’s patients have consistent difficulty naming objects; they come up with related words or distortions of the sound of the correct one:

- table: “chair”
- elbow: “knee”
- clip: “plick”
- butter: “rubber”
- ceiling: “leasing”
- ankle: “ankley, no mankle, no kankle”
- comb: “close, saw it, cit it, cut, the comb, the came”
- paper: “piece of handkerchief, pauper, hand pepper, piece of hand paper”
- fork: “tonsil, teller, tongue, fung”

A striking symptom of Wernicke’s aphasia is that the patients show few signs of comprehending the speech around them. In a third kind of aphasia, the connection between Wernicke’s area and Broca’s is damaged, and these patients are unable to repeat sentences. In a fourth kind, Broca’s and Wernicke’s and the link between them are intact but they are an island cut off from the rest of the cortex, and these patients eerily repeat what they hear without understanding it or ever speaking spontaneously. For these reasons, and because Wernicke’s area is adjacent to the part of the cortex that processes sound,
The area was once thought to underlie language comprehension. But that would not explain why the speech of these patients sounds so psychotic. Wernicke's area seems to have a role in looking up words and funneling them to other areas, notably Broca's, that assemble or parse them syntactically. Wernicke's aphasia, perhaps, is the product of an intact Broca's area madly churning out phrases without the intended message and intended words that Wernicke's area ordinarily supplies. But to be honest, no one really knows what either Broca's area or Wernicke's area is for.

Wernicke's area, together with the two shaded areas adjacent to it in the diagram (the angular and supramarginal gyri), sit at the crossroads of three lobes of the brain, and hence are ideally suited to integrating streams of information about visual shapes, sounds, bodily sensations (from the "somatosensory" strip), and spatial relations (from the parietal lobe). It would be a logical place to store links between the sounds of words and the appearance and geometry of what they refer to. Indeed, damage to this general vicinity often causes a syndrome that is called anomia, though a more mnemonic label might be "no-name-ia," which is literally what it means. The neuropsychologist Kathleen Baynes describes "HW," a business executive who suffered a stroke in this general area. He is highly intelligent, articulate, and conversationally adept but finds it virtually impossible to retrieve nouns from his mental dictionary, though he can understand them. Here is how he responded when Baynes asked him to describe a picture of a boy falling from a stool as he reaches into a jar on a shelf and hands a cookie to his sister:

First of all this is falling down, just about, and is gonna fall down and they're both getting something to eat . . . but the trouble is this is gonna let go and they're both gonna fall down . . . I can't see well enough but I believe that either she or will have some food that's not good for you and she's to get some for her, too . . . and that you get it there because they shouldn't go up there and get it unless you tell them that they could have it. And so this is falling down and for sure there's one they're going to have for food and, and this didn't come out right, the, uh, the stuff that's uh, good for, it's not good for you but it, but you love, um mum mum [smacks lips] . . . and that so they've . . . see that, I can't see whether it's in there or not . . . I think she's saying, I want two or three, I want one, I think, I think so, and so, so she's gonna get this one for sure it's gonna fall down there or whatever, she's gonna get that one and, and there, he's gonna get one himself or more, it all depends with this when they fall down . . . and when it falls down there's no problem, all they got to do is fix it and go right back up and get some more.

HW uses noun phrases perfectly but cannot retrieve the nouns to put inside them: he uses pronouns, gerunds like falling down, and a few generic nouns like food and stuff, referring to particular objects with convoluted circumlocutions. Verbs tend to pose less of a problem for anomics; they are much harder for Broca's aphasics, presumably because verbs are intimately linked to syntax.

There are other indications that these regions in the rear of the perisylvian are implicated in storing and retrieving words. When people read perfectly grammatical sentences and come across a word that makes no sense, like The boys heard Joe's orange about Africa, electrodes pasted near the back of the skull pick up a change in their EEG's (although, as I have mentioned, it is only a guess that the blips are coming from below the electrodes). When people put their heads in the PET scanner, this general part of the brain lights up when they hear words (and pseudo-words, like tweal) and even when they read words on a screen and have to decide whether the words rhyme—a task requiring them to imagine the word's sounds.

A very gross anatomy of the language sub-organs within the perisylvian might be: front of the perisylvian (including Broca's area), grammatical processing; rear of the perisylvian (including Wernicke's and the three-lobe junction), the sounds of words, especially nouns, and some aspects of their meaning. Can we zoom in still closer, and locate
smaller areas of brain that carry out more circumscribed language tasks? The answer is no and yes. No, there are no smaller patches of brain that one can draw a line around and label as some linguistic module—at least, not today. But yes, there must be portions of cortex that carry out circumscribed tasks, because brain damage can lead to language deficits that are startlingly specific. It is an intriguing paradox.

Here are some examples. Although impairments of what I have been calling the sixth sense, speech perception, can arise from damage to most areas of the left perisylvian (and speech perception causes several parts of the perisylvian to light up in PET studies), there is a specific syndrome called Pure Word Deafness that is exactly what it sounds like: the patients can read and speak, and can recognize environmental sounds like music, slamming doors, and animal cries, but cannot recognize spoken words; words are as meaningless as if they were from a foreign language. Among patients with problems in grammar, some do not display the halting articulation of Broca’s aphasia but produce fluent ungrammatical speech. Some aphasics leave out verbs, inflections, and function words; others use the wrong ones. Some cannot comprehend complicated sentences involving traces (like The man who the woman kissed (trace) hugged the child) but can comprehend complex sentences involving reflexives (like The girl said that the woman washed herself). Other patients do the reverse. There are Italian patients who mangle their language’s inflectional suffixes (similar to the -ing, -s, and -ed of English) but are almost flawlessly with its derivational suffixes (similar to -able, -ness, and -er).

The mental thesaurus, in particular, is sometimes torn into pieces with clean edges. Among anomic patients (those who have trouble using nouns), different patients have problems with different kinds of nouns. Some can use concrete nouns but not abstract nouns. Some can use abstract nouns but not concrete nouns. Some can use nouns for nonliving things but have trouble with nouns for living things; others can use nouns for living things but have trouble with nouns for nonliving things. Some can name animals and vegetables but not foods, body parts, clothing, vehicles, or furniture. There are patients who have trouble with nouns for anything but animals, patients who cannot name body parts, patients who cannot name objects typically found indoors, patients who cannot name colors, and patients who have trouble with proper names. One patient could not name fruits or vegetables: he could name an abacus and a sphinx but not an apple or a peach. The psychologist Edgar Zurif, jesting the neurologist’s habit of giving a fancy name to every syndrome, has suggested that it be called anomia for bananas, or “banananomia.”

Does this mean that the brain has a produce section? No one has found one, nor centers for inflections, traces, phonology, and so on. Pinning brain areas to mental functions has been frustrating. Frequently one finds two patients with lesions in the same general area but with different kinds of impairment, or two patients with the same impairment but lesions in different areas. Sometimes a circumscribed impairment, like the inability to name animals, can be caused by massive lesions, brain-wide degeneration, or a blow to the head. And about ten percent of the time a patient with a lesion in the general vicinity of Wernicke’s area can have a Broca-like aphasia, and a patient with lesions near Broca’s area can have a Wernicke-like aphasia.

Why has it been so hard to draw an atlas of the brain with areas for different parts of language? According to one school of thought, it is because there aren’t any; the brain is a meatloaf. Except for sensation and movement, mental processes are patterns of neuronal activity that are widely distributed, hologram-style, all over the brain. But the meatloaf theory is hard to reconcile with the amazingly specific deficits of many brain-damaged patients, and it is becoming obsolete in this “decade of the brain.” Using tools that are getting more sophisticated each month, neurobiologists are charting vast territories that once bore the unhelpful label “association cortex” in the old textbooks, and are delineating dozens of new regions with their own functions or styles of processing, like visual areas specializing in object shape, spatial layout, color, 3D stereo-vision, simple motion, and complex motion.

For all we know, the brain might have regions dedicated to processes as specific as noun phrases and metrical trees; our methods for
studying the human brain are still so crude that we would be unable to find them. Perhaps the regions look like little polka dots or blobs or stripes scattered around the general language areas of the brain. They might be irregularly shaped squiggles, like gerrymandered political districts. In different people, the regions might be pulled and stretched onto different bulges and folds of the brain. (All of these arrangements are found in brain systems we understand better, like the visual system.) If so, the enormous bomb craters that we call brain lesions, and the blurry snapshots we call PET scans, would leave their whereabouts unknown.

There is already some evidence that the linguistic brain might be organized in this tortuous way. The neurosurgeon George Ojemann, following up on Penfield's methods, electrically stimulated different sites in conscious, exposed brains. He found that stimulating within a site no more than a few millimeters across could disrupt a single function, like repeating or completing a sentence, naming an object, or reading a word. But these dots were scattered over the brain (largely, but not exclusively, in the perisylvian regions) and were found in different places in different individuals.

From the standpoint of what the brain is designed to do, it would not be surprising if language subcenters are idiosyncratically tangled or scattered over the cortex. The brain is a special kind of organ, the organ of computation, and unlike an organ that moves stuff around in the physical world such as the hip or the heart, the brain does not need its functional parts to have nice cohesive shapes. As long as the connectivity of the neural microcircuitry is preserved, its parts can be put in different places and do the same thing, just as the wires connecting a set of electrical components can be haphazardly stuffed into a cabinet, or the headquarters of a corporation can be located anywhere if it has good communication links to its plants and warehouses. This seems especially true of words: lesions or electrical stimulation over wide areas of the brain can cause naming difficulties. A word is a bundle of different kinds of information. Perhaps each word is like a hub that can be positioned anywhere in a large region, as long as its spokes extend to the parts of the brain storing its sound, its syntax, its logic, and the appearance of the things it stands for.

The developing brain may take advantage of the disembodied nature of computation to position language circuits with some degree of flexibility. Say a variety of brain areas have the potential to grow the precise wiring diagrams for language components. An initial bias causes the circuits to be laid down in their typical sites; the alternative sites are then suppressed. But if those first sites get damaged within a certain critical period, the circuits can grow elsewhere. Many neurologists believe that this is why the language centers are located in unexpected places in a significant minority of people. Birth is traumatic, and not just for the familiar psychological reasons. The birth canal squeezes the baby's head like a lemon, and newborns frequently suffer small strokes and other brain insults. Adults with anomalous language areas may be the recovered victims of these primal injuries. Now that MRI machines are common in brain research centers, visiting journalists and philosophers are sometimes given pictures of their brains to take home as a souvenir. Occasionally the picture will reveal a walnut-sized dent, which, aside from some teasing from friends who say they knew it all along, bespeaks no ill effects.

There are other reasons why language functions have been so hard to pin down in the brain. Some kinds of linguistic knowledge might be stored in multiple copies, some of higher quality than others, in several places. Also, by the time stroke victims can be tested systematically, they have often recovered some of their facility with language, in part by compensating with general reasoning abilities. And neurologists are not like electronics technicians who can wiggle a probe into the input or output line of some component to isolate its function. They must tap the whole patient via his or her eyes and ears and mouth and hands, and there are many computational waystations between the stimulus they present and the response they observe. For example, naming an object involves recognizing it, looking up its entry in the mental dictionary, accessing its pronunciation, articulating it, and perhaps also monitoring the output for errors by
listening to it. A naming problem could arise if any of these processes tripped up.

There is some hope that we will have better localization of mental processes soon, because more precise brain-imaging technologies are rapidly being developed. One example is Functional MRI, which can measure—with much more precision than PET—how hard the different parts of the brain are working during different kinds of mental activity. Another is Magneto-Encephalography, which is like EEG but can pinpoint the part of the brain that an electromagnetic signal is coming from.

We will never understand language organs and grammar genes by looking only for postage-stamp-sized blobs of brain. The computations underlying mental life are caused by the wiring of the intricate networks that make up the cortex, networks with millions of neurons, each neuron connected to thousands of others, operating in thousandths of a second. What would we see if we could crank up the microscope and peer into the microcircuitry of the language areas? No one knows, but I would like to give you an educated guess. Ironically, this is both the aspect of the language instinct that we know the least about and the aspect that is the most important, because it is there that the actual causes of speaking and understanding lie. I will present you with a dramatization of what grammatical information processing might be like from a neuron's-eye view.

It is not something that you should take particularly seriously; it is simply a demonstration that the language instinct is compatible in principle with the billiard-ball causality of the physical universe, not just mysticism dressed up in a biological metaphor.

Neural network modeling is based on a simplified toy neuron. This neuron can do just a few things. It can be active or inactive. When active, it sends a signal down its axon (output wire) to the other cells it is connected to; the connections are called synapses. Synapses can be excitatory or inhibitory and can have various degrees of strength. The neuron at the receiving end adds up any signals coming in from excitatory synapses, subtracts any signals coming in from inhibitory synapses, and if the sum exceeds a threshold, the receiving neuron becomes active itself.

A network of these toy neurons, if large enough, can serve as a computer, calculating the answer to any problem that can be specified precisely, just like the page-crawling Turing machine in Chapter 3 that could deduce that Socrates is mortal. That is because toy neurons can be wired together in a few simple ways that turn them into "logic gates," devices that can compute the logical relations "and," "or," and "not" that underlie deduction. The meaning of the logical relation "and" is that the statement "A and B" is true if A is true and if B is true. An AND gate that computes that relation would be one that turns itself on if all of its inputs are on. If we assume that the threshold for our toy neurons is .5, then a set of incoming synapses whose weights are each less than .5 but that sum to greater than .5, say .4 and .4, will function as an AND gate, such as the one on the left here:

\[
\text{AND} \quad \text{OR} \quad \text{NOT}
\]

The meaning of the logical relation "or" is that a statement "A or B" is true if A is true or if B is true. Thus an OR gate must turn on if at least one of its inputs is on. To implement it, each synaptic weight must be greater than the neuron's threshold, say .6, like the middle circuit in the diagram. Finally, the meaning of the logical relation "not" is that a statement "Not A" is true if A is false, and vice versa. Thus a NOT gate should turn its output off if its input is on, and vice versa. It is implemented by an inhibitory synapse, shown on the right, whose negative weight is sufficient to turn off an output neuron that is otherwise always on.

Here is how a network of neurons might compute a moderately complex grammatical rule. The English inflection -s as in *Bill walks* is a suffix that should be applied under the following conditions: when the subject is in the third person AND singular AND the action is in the present tense AND is done habitually (this is its "aspect," in lingo)—
but NOT if the verb is irregular like do, have, say, or be (for example, we say Bill is, not Bill be's). A network of neural gates that computes these logical relations looks like this:

First, there is a bank of neurons standing for inflectional features on the lower left. The relevant ones are connected via an AND gate to a neuron that stands for the combination third person, singular number, present tense, and habitual aspect (labeled "3sph"). That neuron exciting a neuron corresponding to the -s inflection, which in turn excites the neuron corresponding to the phoneme z in a bank of neurons that represent the pronunciations of suffixes. If the verb is regular, this is all the computation that is needed for the suffix; the pronunciation of the stem, as specified in the mental dictionary, is simply copied over verbatim to the stem neurons by connections I have not drawn in. (That is, the form for to hit is just hit + s; the form for to wug is just wug + s.) For irregular verbs like be, this process must be blocked, or else the neural network would produce the incorrect be's. So the 3sph combination neuron also sends a signal to a neuron that stands for the entire irregular form is. If the person whose brain we are modeling is intending to use the verb be, a neuron standing for the verb be is already active, and it, too, sends activation to the is neuron. Because the two inputs to is are connected as an AND gate, both must be on to activate is. That is, if and only if the person is thinking of be and third-person-singular-present-habitual at the same time, the is neuron is activated. The is neuron inhibits the -s inflection via a NOT gate formed by an inhibitory synapse, preventing is's or be's, but activates the vowel i and the consonant z in the bank of neurons standing for the stem. (Obviously I have omitted many neurons and many connections to the rest of the brain.)

I have hand-wired this network, but the connections are specific to English and in a real brain would have to have been learned. Continuing our neural network fantasy for a while, try to imagine what this network might look like in a baby. Pretend that each of the pools of neurons is innately there. But wherever I have drawn an arrow from a single neuron in one pool to a single neuron in another, imagine a suite of arrows, from every neuron in one pool to every neuron in another. This corresponds to the child innately "expecting" there to be, say, suffixes for persons, numbers, tenses, and aspects, as well as possible irregular words for those combinations, but not knowing exactly which combinations, suffixes, or irregulars are found in the particular language. Learning them corresponds to strengthening some of the synapses at the arrowheads (the ones I happen to have drawn in) and letting the others stay invisible. This could work as follows. Imagine that when the infant hears a word with a z in its suffix, the z neuron in the suffix pool at the right edge of the diagram gets activated, and when the infant thinks of third person, singular number, present tense, and habitual aspect (parts of his construal of the event), those four neurons at the left edge get activated, too. If the activation spreads backwards as well as forwards, and if a synapse gets strengthened every time it is activated at the same time that its output neuron is already active, then all the synapses lining the paths between "3rd," "singular," "present," "habitual" at one end, and "z" at the other end, get strengthened. Repeat the experience enough
times, and the partly specified neonate network gets tuned into the adult one I have pictured.

Let's zoom in even closer. What primal solderer laid down the pools of neurons and the innate potential connections among them? This is one of the hottest topics in contemporary neuroscience, and we are beginning to get the glimmerings of how embryonic brains get wired. Not the language areas of humans, of course, but the eyeballs of fruit flies and the thalamuses of ferrets and the visual cortices of cats and monkeys. Neurons destined for particular cortical areas are born in specific areas along the walls of the ventricles, the fluid-filled cavities at the center of the cerebral hemispheres. They then creep outward toward the skull into their final resting place in the cortex along guy wires formed by the glial cells (the support cells that, together with neurons, constitute the bulk of the brain). The connections between neurons in different regions of the cortex are often laid down when the intended target area releases some chemical, and the axons growing every which way from the source area "sniff out" that chemical and follow the direction in which its concentration increases, like plant roots growing toward sources of moisture and fertilizer. The axons also sense the presence of specific molecules on the glial surfaces on which they creep, and can steer themselves like Hansel and Gretel following the trail of bread crumbs. Once the axons reach the general vicinity of their target, more precise synaptic connections can be formed because the growing axons and the target neurons bear certain molecules on their surfaces that match each other like a lock and key and adhere in place. These initial connections are often quite sloppy, though, with neurons exuberantly sending out axons that grow toward, and connect to, all kinds of inappropriate targets. The inappropriate ones die off, either because their targets fail to provide some chemical necessary for their survival, or because the connections they form are not used enough once the brain turns on in fetal development.

Try to stay with me in this neuro-mythological quest: we are beginning to approach the "grammar genes." The molecules that guide, connect, and preserve neurons are proteins. A protein is specified by a gene, and a gene is a sequence of bases in the DNA string found in a chromosome. A gene is turned on by "transcription factors" and other regulatory molecules—gadgets that latch on to a sequence of bases somewhere on a DNA molecule and unzip a neighboring stretch, allowing that gene to be transcribed into RNA, which is then translated into protein. Generally these regulatory factors are themselves proteins, so the process of building an organism is an intricate cascade of DNA making proteins, some of which interact with other DNA to make more proteins, and so on. Small differences in the timing or amount of some protein can have large effects on the organism being built.

Thus a single gene rarely specifies some identifiable part of an organism. Instead, it specifies the release of some protein at specific times in development, an ingredient of an unfathomably complex recipe, usually having some effect in molding a suite of parts that are also affected by many other genes. Brain wiring in particular has a complex relationship to the genes that lay it down. A surface molecule may not be used in a single circuit but in many circuits, each guided by a specific combination. For example, if there are three proteins, X, Y, and Z, that can sit on a membrane, one axon might glue itself to a surface that has X and Y and not Z, and another might glue itself to a surface that has Y and Z but not X. Neuroscientists estimate that about thirty thousand genes, the majority of the human genome, are used to build the brain and nervous system.

And it all begins with a single cell, the fertilized egg. It contains two copies of each chromosome, one from the mother, one from the father. Each parental chromosome was originally assembled in the parents' gonads by randomly splicing together parts of the chromosomes of the two grandparents.

We have arrived at a point at which we can define what grammar genes would be. The grammar genes would be stretches of DNA that code for proteins, or trigger the transcription of proteins, in certain times and places in the brain, that guide, attract, or glue neurons into networks that, in combination with the synaptic tuning that takes
place during learning, are necessary to compute the solution to some grammatical problem (like choosing an affix or a word).

So do grammar genes really exist, or is the whole idea just loopy? Can we expect the scenario in the 1990 editorial cartoon by Brian Duffy? A pig, standing upright, asks a farmer, "What's for dinner? Not me, I hope." The farmer says to his companion, "That's the one that received the human gene implant."

For any grammar gene that exists in every human being, there is currently no way to verify its existence directly. As in many cases in biology, genes are easiest to identify when they correlate with some difference between individuals, often a difference implicated in some pathology.

We certainly know that there is something in the sperm and egg that affects the language abilities of the child that grows out of their union. Stuttering, dyslexia (a difficulty in reading that is often related to a difficulty in mentally snipping syllables into their phonemes), and Specific Language Impairment (SLI) all run in families. This does not prove that they are genetic (recipes and wealth also run in families), but these three syndromes probably are. In each case there is no plausible environmental agent that could act on afflicted family members while sparing the normal ones. And the syndromes are far more likely to affect both members of a pair of identical twins, who share an environment and all their DNA, than both members of a pair of fraternal twins, who share an environment and only half of their DNA. For example, identical four-year-old twins tend to mispronounce the same words more often than fraternal twins, and if a child has Specific Language Impairment, there is an eighty percent chance that an identical twin will have it too, but only a thirty-five percent chance that a fraternal twin will have it. It would be interesting to see whether adopted children resemble their biological family members, who share their DNA but not their environments. I am unaware of any adoption study that tests for SLI or dyslexia, but one study has found that a measure of early language ability in the first year of life (a measure that combines vocabulary, vocal imitation, word combinations, jabbering, and word comprehension) was correlated with the general cognitive ability and memory of the birth mother, but not of the adoptive mother or father.

The K family, three generations of SLI sufferers, whose members say things like Carol is cry in the church and can not deduce the plural of wug, is currently one of the most dramatic demonstrations that defects in grammatical abilities might be inherited. The attention-grabbing hypothesis about a single dominant autosomal gene is based on the following Mendelian reasoning. The syndrome is suspected of being genetic because there is no plausible environmental cause that would single out some family members and spare their agemates (in one case, one fraternal twin was affected, the other not), and because the syndrome has struck fifty-three percent of the family members but strikes no more than about three percent of the population at large. (In principle, the family could just have been unlucky; after all, they were not randomly selected from the population but came to the geneticists' attention only because of the high concentration of the syndrome. But it is unlikely.) A single gene is thought to be responsible because if several genes were responsible, each eroding language ability by a bit, there would be several degrees of disability among the family members, depending on how many of the damaging genes they inherited. But the syndrome seems to be all-or-none: the school system and family members all agree on who does and who does not have the impairment, and in most of Gopnik's tests the impaired members cluster together at the low end of the scale while the normal members cluster at the high end, with no overlap. The gene is thought to be autosomal (not on the X chromosome) and dominant because the syndrome struck males and females with equal frequency, and in all cases the spouse of an impaired parent, whether husband or wife, was normal. If the gene were recessive and autosomal, it would be necessary to have two impaired parents to inherit the syndrome. If it were recessive and on the X chromosome, only males would have it; females would be carriers. And if it were dominant and on the X chromosome, an impaired father would pass it on to all of his daughters and none of his sons, because sons get their X chromosome from their