those responses, but it surely helped you acquire them. Incidentally, superstitions are born this way. There is something Orwellian about the distribution of emotions in our world: All objects can get some emotional attachment, but some objects get far more than others. Our primary biological design skews our secondary acquisitions relative to the world around us.

The consequence of extending emotional value to objects that were not biologically prescribed to be emotionally laden is that the range of stimuli that can potentially induce emotions is infinite. In one way or another, most objects and situations lead to some emotional reaction, although some far more so than others. The emotional reaction may be weak or strong—and fortunately for us it is weak more often than not—but it is there nonetheless. Emotion and the biological machinery underlying it are the obligate accompaniment of behavior, conscious or not. Some level of emoting is the obligate accompaniment of thinking about oneself or about one's surroundings.

The pervasiveness of emotion in our development and subsequently in our everyday experience connects virtually every object or situation in our experience, by virtue of conditioning, to the fundamental values of homeostatic regulation: reward and punishment; pleasure or pain; approach or withdrawal; personal advantage or disadvantage; and, inevitably, good (in the sense of survival) or evil (in the sense of death). Whether we like it or not, this is the *natural* human condition. But when consciousness is available, feelings have their maximum impact, and individuals are also able to reflect and to plan. They have a means to control the pervasive tyranny of emotion: it is called reason. Ironically, of course, the engines of reason still require emotion, which means that the controlling power of reason is often modest.

Another important consequence of the pervasiveness of emotions is that virtually every image, actually perceived or recalled, is accompanied by some reaction from the apparatus of emotion. We will consider the importance of this fact when we discuss the mechanisms for the birth of consciousness in chapter 6.

Let me close this comment on inducers of emotions with a reminder of a tricky aspect of the induction process. So far, I have referred to direct inducers—thunder, snakes, happy memories. But emotions can be induced indirectly, and the inducer can produce its result in a somewhat negative fashion, by blocking the progress of an ongoing emotion. Here is an example. When, in the presence of a source of food or sex, an animal develops approach behavior and exhibits features of the emotion happiness, blocking its way and preventing it from achieving its goals will cause frustration and even anger, a very different emotion from happiness. The inducer of the anger is not the prospect of food or sex but rather the thwarting of the behavior that was leading the animal to the good prospect. Another example would be the sudden suspension of a situation of punishment—for instance, sustained pain—which would induce well-being and happiness. The purifying (cathartic) effect that all good tragedies should have, according to Aristotle, is based on the sudden suspension of a steadily induced state of fear and pity. Long after Aristotle, Alfred Hitchcock built a brilliant career on this simple biological arrangement, and Hollywood has never stopped banking on it. Whether we like it or not, we feel very comfortable after Janet Leigh stops screaming in the shower and lies quietly on the bathtub floor. As far as emotion goes, there is not much escape in the setup that nature prepared for us. We get it coming and we get it going.

The Mechanics of Emotion

From experience, you know that the responses that make up emotions are most varied. Some responses are easily apparent in yourself and in others. Think of the muscles in the face adopting the configurations that are typical of joy or sorrow or anger; or of the skin blanching as a reaction to bad news or flushing in a situation of embarrassment; or consider the body postures that signify joy, defiance, sadness, or discouragement; or the sweaty and clammy hands of apprehension; the racing heart associated with pride; or the slowing, near-stillness of the heart in terror.

Other responses are hidden from sight but no less important, such as the myriad changes that occur in organs other than blood vessels, skin, and heart. One example is the secretion of hormones such as cortisol that change the chemical profile of the internal milieu; or the secretion of peptides, such as β-endorphin or oxytocin, that alter the operation of several brain circuits. Another is the release of neurotransmitters, such as the monoamines, norepinephrine, serotonin, and dopamine. During emotions, neurons located in the hypothalamus, basal forebrain, and brain stem release those chemical substances in several regions of the brain up above and, by so doing, temporarily transform the mode of working for many neural circuits. Typical consequences of the increase or decrease of release of such transmitters include the sense we have of the mind processes speeding up or slowing down, not to mention the sense of pleasantness or unpleasantness that pervades mental experience. Such sensing is part of our feeling of an emotion.

Different emotions are produced by different brain systems. In the very same way that you can tell the difference between a facial expression of anger and a facial expression of joy, in the very same way in which you can feel the difference between sadness or happiness in your flesh, neuroscience is beginning to show us how different brain systems work to produce, say, anger or sadness or happiness.

The study of patients with neurological diseases and focal brain damage has yielded some of the most revealing results in this area, but these investigations are now being complemented by functional neuroimaging of individuals without neurological disease. I should note that the work with human subjects also permits a rich dialogue with investigators who are approaching some of these same problems in animals, another welcome novelty in this area of research.

The essence of the available findings can be summarized as follows. First, the brain induces emotions from a remarkably small number of brain sites. Most of them are located below the cerebral cortex and are known as subcortical. The main subcortical sites are in the brain-stem region, hypothalamus, and basal forebrain. One example is the region known as periaqueductal gray (PAG), which is a major coordinator of

emotional responses. The PAG acts via motor nuclei of the reticular formation and via the nuclei of cranial nerves, such as the nuclei of the vagus nerve.[14] Another important subcortical site is the amygdala. The induction sites in the cerebral cortex, the cortical sites, include sectors of the anterior cingulate region and of the ventromedial prefrontal region.

Second, these sites are involved in processing different emotions to varying degrees. We have recently shown, using PET imaging, that the induction and experience of sadness, anger, fear, and happiness lead to activation in several of the sites mentioned above, but that the pattern for each emotion is distinctive. For instance, sadness consistently activates the ventromedial prefrontal cortex, hypothalamus, and brain stem, while anger or fear activate neither the prefrontal cortex nor hypothalamus. Brain-stem activation is shared by all three emotions, but intense hypothalamic and ventromedial prefrontal activation appears specific to sadness.[15]

Third, some of these sites are also involved in the recognition of stimuli which signify certain emotions. For instance, a series of studies in my laboratory has shown that a structure known as the amygdala, which sits in the depth of each temporal lobe, is indispensable to

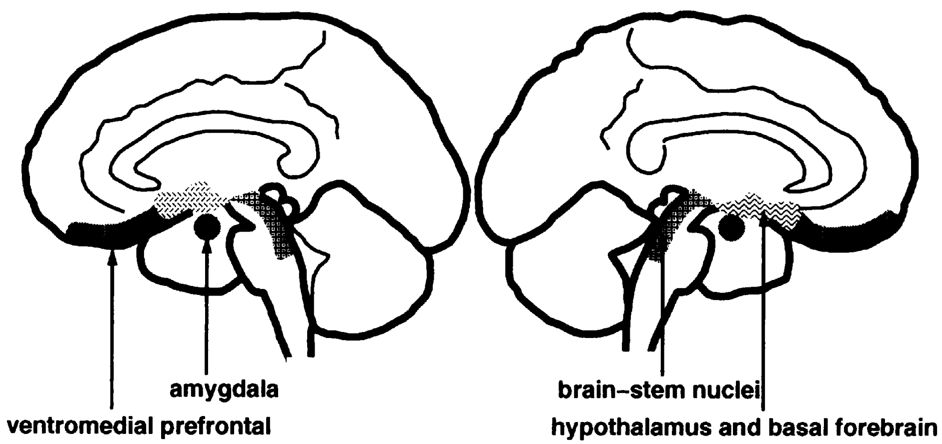

Figure 2.1. Principal emotion induction sites. Only one of these four sites is visible on the brain's surface (the ventromedial prefrontal region). The other regions are subcortical (see figure A.3 in the appendix for exact location). They are all located close to the brain's midline.

recognizing fear in facial expressions, to being conditioned to fear, and even to expressing fear. (In a parallel body of work, the studies of Joseph LeDoux and Michael Davis have shown that the amygdala is necessary for fear conditioning and revealed details of the circuitry involved in the process.[16]) The amygdala, however, has little interest in recognizing or learning about disgust or happiness. Importantly, other structures, just as specifically, are interested in those other emotions and not in fear.

The following description illustrates the fine etching of brain systems related to the production and recognition of emotion. It is but one among several examples that might be adduced to support the idea that there is no single brain center for processing emotions but rather discrete systems related to separate emotional patterns.

Have No Fear

Almost a decade ago, a young woman, to whom I shall refer as S, caught my attention because of the appearance of her brain CT scan. Unexpectedly, her scan revealed that both amygdalae, the one in the left and the one in the right temporal lobes, were almost entirely calcified. The appearance is striking. In a CT scan the normal brain shows up in myriad gray pixels, and the shade of gray defines the contours of the structures. But if a mineral like calcium has been deposited within the brain mass, the scan shows it as a bright milky white that you cannot possibly miss.

All around the two amygdalae, the brain of patient S was perfectly normal. But the amount of calcium deposition was such within the amygdalae that it was immediately apparent that little or no normal function of the neurons within the amygdalae could still take place. Each amygdala is very much a crossroads structure, with pathways from numerous cortical and subcortical regions ending in it and pathways emanating from it to just as many sites. The normal operations carried out by such profuse pathway cross signaling could simply not take place on either side of the brain of S. Nor was this a recent condition in her brain. The deposition of minerals within brain tissue takes

a long time to occur and the thorough and selective job we could witness in her brain had probably taken many years to accomplish, having begun within the first years of her life. For those who are curious about the causes behind the problem, I will say that S suffers from Urbach-Wiethe disease, a rare autosomal recessive condition characterized by abnormal depositions of calcium in the skin and throat. When the brain is affected by calcium deposits, the most frequently targeted structures are the amygdalae. Those patients often have seizures, fortunately not severe, and a minor seizure was indeed the reason why S first came to our care. We were able to help her and she has not had any seizures since.

My first impression of S was of a tall, slender, and extremely pleasant young woman. I was especially curious to find out about her learning and memory ability and about her social demeanor. The reason for this curiosity was twofold. There was considerable controversy at the

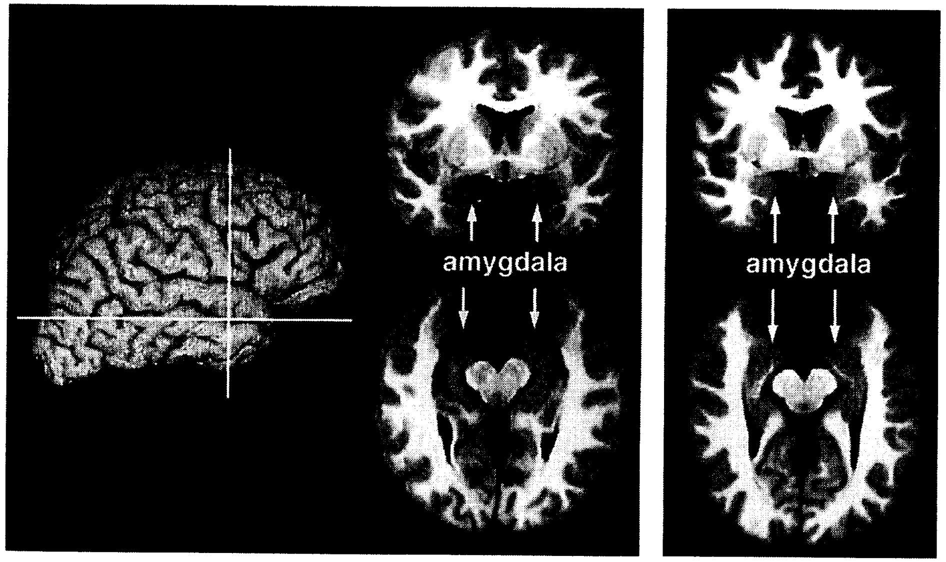

Figure 2.2. Bilateral damage of the amygdala in patient S (left panel) and normal amygdala (right panel). The sections were obtained along the two perpendicular planes shown by the white lines drawn over the brain's external surface. The black areas identified by the arrows are the damaged amygdalae. Compare with the normal amygdalae of a control brain shown in the exact same sections in the two panels on the right.

time regarding the contribution of the amygdalae to the learning of new facts, some investigators believing the amygdala was a vital partner to the hippocampus in the acquisition of new factual memory, other investigators believing it had little to contribute on that score. The curiosity regarding her demeanor was based on the fact that from studies involving nonhuman primates, it was known that the amygdala plays a role in social behaviors.[17]

I can make a long story short by telling you that there was nothing wrong whatsoever with S's ability to learn new facts. This was evident when I met her for only the second time and she clearly recognized me, smiled, and greeted me by name. Her one-shot learning of who I was, what my face looked like, and of my name was flawless. Numerous psychological tests would bear out this first impression, and that is precisely how things remain today. Years later, we were to show that a particular aspect of her learning was defective, but this had nothing to do with learning facts: it had to do with conditioning to unpleasant stimuli.[18]

Her social history, on the other hand, was exceptional. To put it in the simplest possible terms, I would say that S approached people and situations with a predominantly positive attitude. Others would actually say that her approach was excessively and inappropriately forthcoming. S was not only pleasant and cheerful, she seemed eager to interact with most anyone who would engage her in conversation, and several members of the clinical and research teams felt that the reserve and reticence one would have expected from her was simply lacking. For instance, shortly after an introduction, S would not shy away from hugging and touching. Make no mistake, her behavior caused no discomfort to anyone, but it was invariably perceived as a far cry from the standard behavior of a patient in her circumstances.

We were to learn that this very same attitude pervaded all areas of her life. She made friends easily, formed romantic attachments without difficulty, and had often been taken advantage of by those she trusted. On the other hand, she was and is a conscientious mother, and she tries hard to abide by social rules and be appreciated for her efforts. Human nature is indeed hard to describe and full of contradictions in the best of circumstances and the prime of health. It is almost impossible to do justice to it when we enter the realm of disease.

The first years of research on S yielded two important results. On the one hand, S did not have any problem learning facts. In fact, it was possible to say that her sensory perceptions, her movements, her language, and her basic intelligence were no different from those of an entirely healthy average individual in terms of elementary competence. On the other hand, her social behavior demonstrated a consistent skewing of her prevailing emotional tone. It was as if negative emotions such as fear and anger had been removed from her affective vocabulary, allowing the positive emotions to dominate her life, at least by greater frequency of occurrence if not by greater intensity. This was of special interest to me because I had noticed a similar pattern in patients with bilateral damage to the anterior sector of the temporal lobe, who, as a part of their large lesions, also had damage to the amygdalae. It was reasonable to hypothesize that their affective lopsidedness was traceable to damage in the amygdala.

All of these suppositions were to be turned into hard fact when Ralph Adolphs joined my laboratory. Using a variety of clever techniques in the investigation of several patients, some with damage to the amygdala and some with damage to other structures, Adolphs was able to determine that the affective lopsidedness was mostly caused by the impairment of one emotion: fear.[19]

Using a multidimensional scaling technique, Adolphs showed that S cannot consistently tell the expression of fear in another person's face, especially when the expression is ambiguous or other emotions are being expressed simultaneously. She has no such problem with the recognition of other facial expressions of emotion, namely, that of surprise which is, in many respects, similar in general configuration. Curiously, S, who has a remarkable gift for drawing and has good drafting skills, cannot draw a face that represents fear although she can draw faces that represent other emotions. When asked to mimic facial expressions of emotions she does so easily for the primary emotions but not for fear. Her attempts produce little change in her facial expression after which she confesses her complete failure. Again, she has no difficulty producing a facial expression of surprise. Lastly, S does not experience fear in the same way you or I would in a situation that would normally induce it. At a purely intellectual level she knows what fear is supposed to be, what should cause it, and even what one may do in situations of fear, but little or none of that intellectual baggage, so to speak, is of any use to her in the real world. The fearlessness of her nature, which is the result of the bilateral damage to her amygdalae, has prevented her from learning, throughout her young life, the significance of the unpleasant situations that all of us have lived through. As a result she has not learned the telltale signs that announce possible danger and possible unpleasantness, especially as they show up in the face of another person or in a situation. Nowhere has this been proved more clearly than in a recent study requiring a judgment of trustworthiness and approachability based on human faces.[20]

The experiment called for the judgment of one hundred human faces that had been previously rated by normal individuals as indicating varied degrees of trustworthiness and approachability. There were fifty faces that had been consistently judged as inspiring trust and fifty that were not. The selection of these faces was made by normal individuals who were asked a simple question: How would you rate this face on a scale of one to five, relative to the trustworthiness and approachability that the owner of the face inspires? Or, in other words, how eager would you be to approach the person with this particular face if you needed help?

Once the one hundred faces were properly distributed based on the ratings of the forty-six normal individuals, we turned to patients with brain damage. S was one of three patients with bilateral damage to the amygdala included in the study, but we also investigated the performance of seven patients with damage to either the left amygdala or right amygdala, three patients with damage to the hippocampus and an inability to learn new facts, and ten patients with damage elsewhere in the brain, i.e., outside the amygdala and outside the hippocampus. The results were far more remarkable than we expected.

S, along with other patients who also have damage to the amygdalae on both sides of the brain, looked at faces that you or I would consider trustworthy and classified them, quite correctly, as you or I would, as faces that one might approach in case of need. But when they looked at faces of which you or I would be suspicious, faces of persons that we would try to avoid, they judged them as equally trustworthy. The patients with damage to only one amygdala, the amnesic patients, and the other brain-damaged patients performed as normals do.

The inability to make sound social judgments, based on previous experience, of situations that are or are not conducive to one's welfare has important consequences for those who are so affected. Immersed in a secure Pollyanna world, these individuals cannot protect themselves against simple and not-so-simple social risks and are thus more vulnerable and less independent than we are. Their life histories testify to this chronic impairment as much as they testify to the paramount importance of emotion in the governance not just of simple creatures but of humans as well.

How It All Works

In a typical emotion, then, certain regions of the brain, which are part of a largely preset neural system related to emotions, send commands to other regions of the brain and to most everywhere in the body proper. The commands are sent via two routes. One route is the bloodstream, where the commands are sent in the form of chemical molecules that act on receptors in the cells which constitute body tissues. The other route consists of neuron pathways and the commands along this route take the form of electrochemical signals which act on other neurons or on muscular fibers or on organs (such as the adrenal gland) which in turn can release chemicals of their own into the bloodstream.

The result of these coordinated chemical and neural commands is a global change in the state of the organism. The organs which receive the commands change as a result of the command, and the muscles, whether the smooth muscles in a blood vessel or the striated muscles in the face, move as they are told to do. But the brain itself is changed just as remarkably. The release of substances such as monoamines and peptides from regions of nuclei in the brain stem and basal forebrain alters the mode of processing of numerous other brain circuits, triggers certain specific behaviors (for example, bonding, playing, crying), and modifies the signaling of body states to the brain. In other words, both the brain and the body proper are largely and profoundly affected by the set of commands although the origin of those commands is circumscribed to a relatively small brain area which responds to a particular content of the mental process. Now consider this: Beyond emotion, specifically described as the collection of responses I just outlined, two additional steps must take place before an emotion is *known*. The first is feeling, the imaging of the changes we just discussed. The second is the application of core consciousness to the entire set of phenomena. Knowing an emotion—feeling a feeling—only occurs at that point.

These events can be summarized by walking through the three key steps of the process:

1. Engagement of the organism by an inducer of emotion, for instance, a particular object processed visually, resulting in visual representations of the object. Imagine running into Aunt Maggie, whom you love and have not seen in a long time. Chances are you will immediately recognize Aunt Maggie, but even if you do not, or even before you do, the basic process of emotion will continue on to the next step.
2. Signals consequent to the processing of the object's image activate all the neural sites that are prepared to respond to the particular class of inducer to which the object belongs. The sites I am talking about—for instance, in the ventromedial prefrontal cortices, amygdala, and brain stem—have been preset innately, but past experience with things Maggie has modulated the manner in which they are likely to respond, for instance, the ease with which they will respond. By the way, Aunt Maggie is not traveling all over your brain in the form of a passport photo. She exists as a visual image, arising out of neural patterns generated by the interaction of several areas in early visual cortices, largely in occipital lobes. Signals consequent to the presence of her image travel elsewhere and do their job when parts of the brain that are interested in things Maggie respond to such signals.
3. As a result of step 2, emotion induction sites trigger a number of signals toward other brain sites (for instance, monoamine nuclei, somatosensory cortices, cingulate cortices) and toward the body (for instance, viscera, endocrine glands), as previously discussed. Under some circumstances the balance of responses may favor intrabrain circuitry and engage the body minimally. This is what I have called "as if body loop" responses.

The combined result of steps 1, 2, and 3 is a momentary and appropriate collection of responses to the circumstances causing the whole commotion: for instance, Aunt Maggie in sight; or the death of a friend announced; or nothing that you can tell consciously; or, if you are a baby bird in a high nest, the image of a large object flying overhead. Take the latter example. The baby bird has no idea that this is a predatory eagle, and no conscious sense of the danger of the situation. No thought process, in the proper meaning of the term, tells the baby bird to do what it does next, which is to crouch as low as possible in the nest, as quietly as possible, such that it may become invisible to the eagle. And yet, the steps of the process that I have just described were engaged: visual images were formed in the baby bird's visual brain, some sectors of the brain responded to the *kind* of visual image the brain formed, and all the appropriate responses, chemical and neural, autonomic and motor, were engaged at full tilt. The quiet and slow tinkering of evolution has done all the thinking for the baby bird, and its genetic system has dutifully transmitted it. With a little bit of help from mother bird and earlier circumstances, the minicon-cert of fear is ready to be played whenever the situation demands it. The fear response that you can see in a dog or a cat is executed in exactly the same manner, and so is the fear response you can examine in yourself when you walk at night on a dark street. That we, and at least the dog and the cat, can also come to know about the feelings caused by those emotions, thanks to consciousness, is another story.

In fact, you can find the basic configurations of emotions in simple organisms, even in unicellular organisms, and you will find yourself attributing emotions such as happiness or fear or anger to very simple creatures who, in all likelihood, have no feeling of such emotions in the sense that you or I do, creatures which are too simple to have a brain, or, having one, too rudimentary to have a mind. You make those attributions purely on the basis of the movements of the organism, the speed of each act, the number of acts per unit of time, the style of the movements, and so on. You can do the same thing with a simple chip moving about on a computer screen. Some jagged fast movements will appear "angry," harmonious but explosive jumps will look "joyous," recoiling motions will look "fearful." A video that depicts several geometric shapes moving about at different rates and holding varied relationships reliably elicits attributions of emotional state from normal adults and even children. The reason why you can anthropomorphize the chip or an animal so effectively is simple: emotion, as the word indicates, is about movement, about externalized behavior, about certain orchestrations of reactions to a given cause, within a given environment.[21]

Somewhere between the chip and your pet sits one of the living creatures that has most contributed to progress in neurobiology, a marine snail known as *Aplysia californica.* Eric Kandel and his colleagues have made great inroads in the study of memory using this very simple snail which may not have much of a mind but certainly has a scientifically decipherable nervous system and many interesting behaviors. Well, *Aplysia* may not have feelings as you or I do, but it has something not unlike emotions. Touch the gill of an *Aplysia,* and you will see the gill recoil swiftly and completely, while the heart rate of *Aplysia* goes up and it releases ink into the surroundings to confuse the enemy, a bit like James Bond when he is hotly pursued by Dr. No. *Aplysia* is emoting with a miniconcert of responses that is formally no different, only simpler, from the one that you or I could display under comparable circumstances. To the degree that *Aplysia* can represent its emotive state in the nervous system, it may have the makings of a feeling. We do not know whether *Aplysia* has feelings or not, but it is extremely difficult to imagine that *Aplysia* would know of such feelings if it does have them.[22]

Sharpening the Definition of Emotion: An Aside

What qualifies for an emotion? Does pain? Does a startle reflex? Neither does, but if not, why not? The closeness of these related phenomena calls for sharp distinctions but the differences tend to be ignored. Startle reflexes are part of the repertoire of regulatory responses available to complex organisms and are made up of simple behaviors (e.g., limb withdrawal). They may be included among the numerous and concerted responses that constitute an emotion—endocrine responses, multiple visceral responses, multiple musculoskeletal responses, and so on. But even the simple emotive behavior of the *Aplysia* is more complicated than a simple startle response.

Pain does not qualify for emotion, either. Pain is the consequence of a state of local dysfunction in a living tissue, the consequence of a stimulus—impending or actual tissue damage—which causes the sensation of pain but also causes regulatory responses such as reflexes and may also induce emotions on its own. In other words, emotions can be caused by the same stimulus that causes pain, but they are a different result from that same cause. Subsequently, we can come to know that we have pain and that we are having an emotion associated with it, provided there is consciousness.

When you picked up that hot plate the other day and burned the skin of your fingers, you had pain and might even have suffered from having it. Here is what happened to you, in the simplest neurobiological terms:

First, the heat activated a large number of thin and unmyelinated nerve fibers, known as C-fibers, available near the burn. (These fibers, which are distributed literally everywhere in the body, are evolutionarily old and are largely dedicated to carrying signals about internal body states, including those that will end up causing pain. They are called *unmyelinated* because they lack the insulating sheath known as myelin. Lightly myelinated fibers known as A-δ fibers travel along with C-fibers and perform a similar role. Together they are called *nociceptive* because they respond to stimuli that are potentially or actually damaging to living tissues.)

Second, the heat destroyed several thousand skin cells, and the destruction released a number of chemical substances in the area.

Third, several classes of white blood cell concerned with repairing tissue damage were called to the area, the call having come from some of the released chemicals (e.g., a peptide known as substance P and ions such as potassium).

Fourth, several of those chemicals activated nerve fibers on their own, joining their signaling voices to that of the heat itself.

Once the activation wave was started in the nerve fibers, it traveled to the spinal cord and a chain of signals was produced across several neurons (a neuron is a nerve cell) and several synapses (a synapse is the point where two neurons connect and transmit signals) along the appropriate pathways. The signals went all the way into the top levels of the nervous system: the brain stem, the thalamus, and even the cerebral cortex.

What happened as a result of the succession of signals? Ensembles of neurons located at several levels of the nervous system were temporarily activated and the activation produced a neural pattern, a sort of map of the signals related to the injury in your fingers. The central nervous system was now in possession of multiple and varied neural patterns of tissue damage selected according to the biological specifications of your nervous system and of the body proper with which it connects. The conditions needed to generate a sensation of pain had been met.

The question that I am leading to arrives at this point: Would one or all of those neural patterns of injured tissue be the same thing as

knowing that you had pain? And the answer is, not really. Knowing that you have pain requires something else that occurs *after* the neural patterns that correspond to the substrate of pain—the nociceptive signals—are displayed in the appropriate areas of the brain stem, thalamus, and cerebral cortex and generate an image of pain, a feeling of pain. But note that the "after" process to which I am referring is not beyond the brain, it is very much in the brain and, as far as I can fathom, is just as biophysical as the process that came before. Specifically, in the example above, it is a process that interrelates neural patterns of tissue damage with the neural patterns that stand for *you*, such that yet another neural pattern can arise—the neural pattern of you knowing, which is just another name for consciousness. If the latter interrelating process does not take place, you will never know that there was tissue damage in your organism—if there is no you and there is no knowing, there is no way for you to know, right?

Curiously, if there had been no you, i.e., if you were not conscious and if there had been no self and no knowing relative to hot plates and burning fingers, the wealthy machinery of your self-*less* brain would still have used the nociceptive neural patterns generated by tissue damage to produce a number of useful responses. For instance, the organism would have been able to withdraw the arm and hand from the source of heat within hundreds of milliseconds of the beginning of tissue damage, a reflex process mediated by the central nervous system. But notice that in the previous sentence I said "organism" rather than "you." Without knowing and self, it would not have been quite "you" withdrawing the arm. Under those circumstances, the reflex would belong to the organism but not necessarily to "you." Moreover, a number of emotional responses would be engaged automatically, producing changes in facial expression and posture, along with changes in heart rate and control of blood circulation—we do not learn to wince with pain, we just wince. Although all of these responses, simple and not so simple, occur reliably in comparable situations in all conscious human beings, consciousness is not needed at all for the responses to take place. For instance, many of these responses are present even in comatose patients in whom consciousness is suspended—one of the ways in which we neurologists evaluate the state of the nervous system in an unconscious patient consists of establishing whether the patient reacts with facial and limb movements to unpleasant stimuli such as rubbing the skin over the sternum.

Tissue damage causes neural patterns on the basis of which your organism is in a state of pain. If you are conscious, those same patterns can also allow *you* to know you have pain. But whether or not you are conscious, tissue damage and the ensuing sensory patterns also cause the variety of automated responses outlined above, from a simple limb withdrawal to a complicated negative emotion. In short, pain and emotion are not the same thing.

You may wonder how the above distinction can be made, and I can give you a large body of evidence in its support. I will begin with a fact that comes from direct experience, early in my training, of a patient in whom the dissociation between *pain as such* and *emotion caused by pain* was vividly patent.[23] The patient was suffering from a severe case of refractory trigeminal neuralgia, also known as tic douloureux. This is a condition involving the nerve that supplies signals for face sensation in which even innocent stimuli, such as a light touch of the skin of the face or a sudden breeze, trigger an excruciating pain. No medication would help this young man who could do little but crouch, immobilized, whenever the excruciating pain stabbed his flesh. As a last resort, the neurosurgeon Almeida Lima, who was also one of my first mentors, offered to operate on him, because producing small lesions in a specific sector of the frontal lobe had been shown to alleviate pain and was being used in last-resort situations such as this.

I will not forget seeing the patient on the day before the operation, afraid to make any movement that might trigger a new round of pain, and then seeing him two days after the operation, when we visited him on rounds; he had become an entirely different person, relaxed, happily absorbed in a game of cards with a companion in his hospital room. When Lima asked him about the pain, he looked up and said quite cheerfully that "the pains were the same," but that he felt fine now. I remember my surprise as Lima probed the man's state of mind a bit further. The operation had done little or nothing to the sensory patterns corresponding to local tissue dysfunction that were being supplied by the trigeminal system. The mental images of that tissue dysfunction were not altered and that is why the patient could report that the pains were the same. And yet the operation had been a success. It had certainly abolished the emotional reactions that the sensory patterns of tissue dysfunction had been engendering. Suffering was gone. The facial expression, the voice, and the general deportment of this man were not those one associates with pain.

This sort of dissociation between "pain sensation" and "pain affect" has been confirmed in studies of groups of patients who underwent surgical procedures for the management of pain. More recently, Pierre Rainville, who is now an investigator in my laboratory, has shown by means of a clever manipulation using hypnosis that pain sensation and pain affect are clearly separable. Hypnotic suggestions designed to influence pain affect specifically without altering pain sensation modulated cerebral activity within the cingulate cortex, the same overall region that neurosurgeons can damage to alleviate suffering from chronic and intractable pain. Rainville has also shown that when hypnotic suggestions were aimed at pain sensation rather than at the emotions associated with pain, not only were there changes in *both* unpleasantness and intensity ratings, but also there were changes in SI (the primary somatosensory cortex) and the cingulate cortex.[24] In brief: hypnotic suggestions aimed at the emotions that follow pain rather than at pain sensation reduced emotion but not pain sensation and also caused functional changes in cingulate cortex only; hypnotic suggestions aimed at pain sensation reduced *both* pain sensation and emotion, and caused functional changes in SI and in the cingulate cortex. Perhaps you have had the direct experience of what I am describing if you have ever taken beta-blockers to