Chapter 2
Neurobiological Foundations of Action Planning and Execution

In the mid-1800s, Phineas Gage worked on the construction of railroads in the United States, and his job as a foreman was to get rid of annoying masses of stone by blowing them up with explosives. Unfortunately, during one explosion, his **frontal cortex** was pierced by a chisel. In 1868, the physician John M. Harlow describes in great detail how Gage's serious wound was treated successfully, and how he started to work again after a few months. However, colleagues and superiors had to conclude that he was "no longer Gage": he lacked motivation, had difficulty in making plans, and showed strong **personality changes**, which were not in his benefit. Nevertheless, he was still able to work, and so he took a job in a horse stable; however, he found it increasingly difficult to develop action plans and to translate them into appropriate actions. Harlow described Gage as a person who always made plans for future activities, only to abandon them and to replace them with other, apparently better plans.

The analysis of the case, and especially the skull of Phineas Gage, has made substantial contributions to our **understanding** of the interplay between the human brain, cognitive processes, and action control (cf. Sect. 2.6.2). In fact, the actual performance of cognitive functions is usually understood best when they cease to exist for some reason, be it through lack of exercise, natural aging, illness, or accidents. This does not just apply to perception and memory, but also to the planning of actions and action control.

Particularly interesting in this context, are patients who show deficits in the planning or execution of actions, for example, as a result of **brain lesions**. This is interesting because the failure of control of action in patients with specific, accurately described lesions in the brain can give us preliminary insights into which brain areas are involved with action control. Additionally, results from **physiological animal experiments** and **neuroimaging methods** (Box 2.4) have contributed to a better understanding of the neuronal foundation of human action control. Although the mapping and understanding of the neuronal basis of the processes of action planning and action control is currently not as detailed as, say, that of the visual cortex,

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it is becoming clear that successful planning, initiation, and execution of actions require an intact functional loop, which encompasses the **frontal cortex**, the **premotor** and **motor cortices**, the **basal ganglia**, and the **cerebellum**. All of these areas (as well as many others that will not be treated here, from a didactic viewpoint) make specific contributions to action control.

When we attempt to describe the most important contributions of these areas in the text that follows, we should not forget that it is the interplay and integration of these areas that produces effective actions. The performance of a given brain area must always be viewed in conjunction with the **functional loop** to which it contributes. Therefore, we do not have the intention to present a comprehensive overview of neuroscientific research into human action control. Instead, we would merely like to point out some properties of neuronal information processing that have direct consequences for a psychological understanding of action control, and to discuss the actual functions of the neuroanatomical functions that are important for action control.

To aid in orientation and get a grasp of where in the human brain the areas we will discuss are located, we can use a **map of the brain** which was published by the German neurologist Korbinian Brodmann in 1909 (Fig. 2.1). On the basis of his cyto-architectonic studies, Brodmann divided the cortex into 52 areas, known today as **Brodmann's areas (BA)**. A series of these areas is generally considered to have functionally separate roles in cerebral information processing. In the next section, we will first have a look at the question of how these different anatomical areas actually communicate with each other.

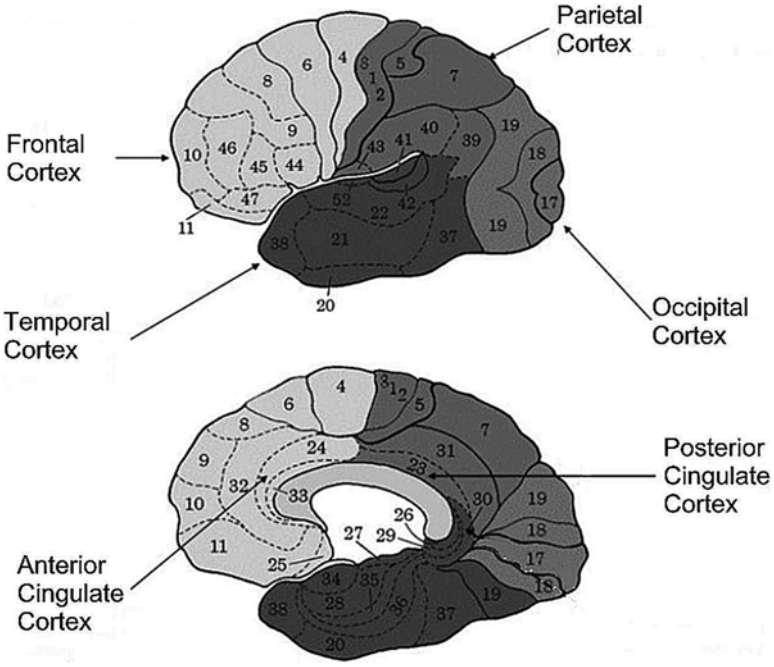

Fig. 2.1 Brodmann's (1909) map of the human brain

2.1 Neuronal Communication

The smallest functional unit of the brain is the **nerve cell** or the **neuron**. There are about 100 billion of them in a human brain. The number of neurons generally remains fairly constant from birth to way beyond the 65th year of age. A neuron has a cell body (soma) with relatively short projections (dendrites), which take up information from other neurons as input, and lead it to the soma. A neuron also has a relatively long projection (the axon), which conducts electrical impulses from the soma to the dendrites of other neurons. The site where the axon of a neuron comes into contact with the dendrite of another neuron is called a **synapse**. When the electrical impulse which is conducted through the axon exceeds a certain threshold, a chemical messenger (called a neurotransmitter) is released at the end of the axon. When the neurotransmitter at the synapse contacts the dendrite of the second and other proximal neurons, the electrical impulse is carried on by the second and a host of other neurons.

Single neurons appear to be highly specialized. This is suggested by studies in which extremely thin microelectrodes are inserted into the brains of animals. When the soma of an active neuron lies close to the electrode's tip, the minute electrical potentials that are caused by the activity are registered by the electrode. This signal is then amplified acoustically, for example, so that the activity of the neuron can be heard: the stronger the electrical activation of the neuron, the louder the noise. When visual or auditory stimuli are now presented to the animal, it becomes clear that single neurons are tuned to process highly specific information: some cells respond exclusively to the shape or orientation of objects, some exclusively to visible motion in a specific direction. Some cells in the auditory cortex respond to tones of a specific frequency, others to tones of a specific volume, and others yet respond to tones that change their frequency and get higher or lower. Other cells respond to faces, some to specific faces, others to all faces that are oriented in a specific direction. Then there are cells which are active when an animal moves in a particular way, but also when the animal observes that movement in another animal. So, when a neuron is confronted with the specific stimulus information it is sensitive to, it reacts with an **increase in activity** and signals in that way, that at that very moment, highly specific information is available, for example, the movement of an object in a given direction. The only thing this specific neuron "knows" is that, for example, something moves in the specific direction for which it is specialized, that is, it operates completely **feature specific**. It merely codes this one feature, without "knowing" anything about the other features of this object that is moving. It has no information about the object's shape, color, size, or identity, that is, about features that are coded in other cortical brain areas, which are often located relatively far away and are occasionally organized in a different manner (so-called distributed coding of features).

The **principle of distributed coding** of features is very applicable to the processing of visual information: the various features of visual stimuli are coded into different cortical color, form, orienting, and motion maps (DeYoe and van Essen 1988).

Apparently, it also applies to coding the various features of action. It has been shown in monkeys, for example, that the direction, expenditure of energy, and range of a movement are coded in a distributed manner; in humans, comparable indications for the duration, expenditure of energy, and the effector, with which a movement is executed (see review by Hommel and Elsner 2009). The principle of distributed coding in separate modules offers a range of **evolutionary benefits**: phylogenetically, it allows for a continuous adaptation and the steady expansion of the brain, as individual modules can be modified, added, or eliminated, without the entire brain having to be "rebuilt." Ontogenetically, it gives rise to a comparable measure of tolerance to damage of the brain (occasionally reversible due to plasticity of the brain), which often manifests itself as the loss of subprocesses, which does not necessarily interfere with the functioning of the entire cortex (see Box 2.1).

Box 2.1 Plasticity of the Brain

How exactly do specific cortical areas come to be and how do neuronal networks are connected in the way they are? The fact that our brains resemble both those of other humans and those of primates quite well suggests that genetic wiring plans play a large part in the development of the brain. However, genes do not determine everything because the structure of our brain is largely dependent on experiences. This is demonstrated by classical experiments of the Nobel prize winners David Hubel and Torsten Wiesel, who bandaged up one eye of kittens before they came into contact with direct light (Hubel and Wiesel 1963). These kittens could do everything they liked, but could only use one eye to do so. After multiple months, the scientists took the bandages off, and assessed the neuronal connections between the two eyes and the brain. The surprising result was that the bandaged eye, although **optically intact**, was not connected with the visual areas of the brain. It was **functionally blind**. Clearly, under these circumstances the neurons had formed networks in such a manner that connections had only established between the retinal cells of the seeing eye and the visual cortex.

These early tests with animals have illustrated an important fact: neurons do not form networks based on a fixed blueprint, but based on a **functional, activity-dependent scheme**. Which connections are established, must be at least partially fixed in genetic codes; for example, retinal cells in the eye only connect with cells in the visual cortex in the occipital lobe of the brain, and not with neurons in other parts of the brain, such as the motor cortex. But, apart from that, neuronal networks are generally plastic and flexible, and they adapt continually to the organism and his or her activities through modification, installation, and elimination of connections. Numerous clinical and experimental studies show that this does not just apply to the developing brains of babies and children, but to those of fully developed adults too.

(continued)

Box 2.1 (continued)

It can be shown in animal experiments that loss of specific neuronal populations can be **compensated** in a surprisingly short time, as other neuronal populations take over the functions of lost populations. Sanes et al. (1992) bisected the nerve that innervates the musculature of whiskers in rats. This led to a loss of the neuronal populations in the primary motor cortex that are responsible for control of the whiskers. Within hours, the neuronal network that controls movements of the facial muscles was reorganized in such a manner that neurons in neighboring areas of the motor cortex replaced the lost neuronal populations.

Pascual-Leone et al. (1993) have shown that the size of the finger area in the motor cortex varies depending on activity level: while the finger areas of blind persons who have little expertise in reading Braille are roughly the same size for both hands, in blind Braille experts, the cortical area that represents the "reading" finger is larger than the corresponding area for the finger of the other hand. Complementary to such observations, which indicate that neuronal representations in the motor cortex can **expand** activity-dependently, it has also been demonstrated that motor areas in the brain **decrease** in size when abilities to move are restricted either temporarily or for a longer period of time. Liepert et al. (1995) have studied patients whose ankles were restricted in freedom of movement, without a peripheral nerve lesion being present. They found that the motor areas responsible for control of the damaged ankle were smaller than the corresponding areas responsible for the non-afflicted ankle.

These observations indicate that neuronal representations are plastic and can adapt flexibly to the circumstances and activities of the organism. In which **temporal dimensions** such adaption processes can be completed, can be studied with experiments, in which participants acquire motor skills. Pascual-Leone et al. (1995) had their participants execute movement sequences of five fingers on the keys of a piano over the course of 5 days, and they analyzed the changes in hand representations in the motor cortex. The spatial expansion of the hand area increased as expertise in executing the movement sequences increased. That this growth was actually caused by the acquisition of a skill and not, for example, a random side effect of merely moving the fingers of one hand repetitively was demonstrated by the observation that isolated, non-sequential movements of the fingers were not associated with expansion of the hand area.

The plasticity of the human brain is also demonstrated in the often remarkable successes in the rehabilitation of stroke victims. **Strokes** (**cerebrovascular accidents**) are caused most often by a lack of perfusion in the brain following the obstruction of blood vessels and less often by a hemorrhage (e.g., following an accident). A lack of perfusion leads to a disruption in the oxygen supply to the brain, which results in the death of many nerve cells in the brain. The consequences can be motor disabilities in, for example, the arm, hand, leg or feet on one side of the body as well as loss of speech.

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Box 2.1 (continued)
These disabilities often restrict a victim's performance of everyday chores in the long term. Almost all of the victim's **motor activities** like, for example, opening doors, getting dressed, reading the newspaper, brushing teeth, and playing cards can often only be performed with the unaffected arm.

Additionally, many patients suffer from stroke-induced **speech disorders** (aphasias) which can be manifested during writing, reading, comprehending, or speaking. Aphasias are caused by damage to the neuronal populations which participate in speech production (Broca's speech area; BA44 and BA45) and/or comprehension of speech (Wernicke's area; BA42 and BA22). Damage to Broca's area mainly leads to problems with speech production accompanied by generally intact speech comprehension, while damage to Wernicke's area is characterized by generally intact speech production while speech comprehension processes are interfered with (review by Kolb and Whishaw 1996). During the rehabilitation of such disturbances, surprising improvements can often be attained. The ability to speak, for example, can be recovered, or paralyses can disappear almost completely. It is the plasticity of the human brain, its ability to adapt and modify neuronal structures continuously, so that neurons in other brain regions can manage to take over the functions of damaged areas (Hallett 2001), that makes these remarkable types of recovery possible.

Problems arise when this system represents multiple different features that are represented **at the same time**—which is always the case in daily life. In these cases, the problem is to distinguish which features belong to which perceptual and action events. To illustrate the problem, imagine a table with two pieces of fruit on it: on your left, quite close to you, is a green, not entirely ripe apple; to the right, a bit farther away, is a red strawberry. Now imagine you want to grasp both fruits at the same time, the strawberry with your right hand, to eat it, and the apple with your left hand, to put it away. How is this scenario represented neuronally? Probably like this: The information that emanates from both fruits activates a great number of neurons, which signal, for example, that the following features are available: *red, green, left, right, large, small, nearby, faraway, sweet, sour*. What we perceive, however, is not a bunch of separate, unconnected features, but a coherent whole, namely a red strawberry, which is on a table in front of us, to the right of a somewhat unripe apple. The preparation of the movements of the two hands also contains a number of feature-based codes, such as *left, right, nearby, faraway*, and many more. Movements, too, are not represented phenomenologically as single features or elements, but as coherent events, namely as the action plan that the right hand will grasp the strawberry, say, while the left hand will grab the apple.

Now, how can a system which is based on the principle of distributed representations distinguish which activated codes belong to which perceptual or action event? Do the features *red, right,* and *small* belong to the same fruit? Do the movement features *right* (for the hand) and *faraway* (for the amplitude of the movement) go together? Should the right hand make the larger and the left hand the smaller move-

ment, or should it be the other way around? The solution to the problem probably requires the integration or binding of related cognitive or cortical feature codes (Singer 1994). How might this binding work?

A simple **solution** would be presented if the brain would have an area where the codes which are represented in a distributed manner would be collected and assembled; in other words, a center like the pineal gland from the Cartesian tradition, to which Descartes ascribed the function of central processing of afferent and efferent processes. However, such a **center** does not exist in the brains of humans and other higher species.

Another solution to the binding problem is based on the idea that spatially distributed populations of neurons, which encode different information, can **communicate** with one another. Individual neurons communicate with a great number of other neurons and form so-called **functional networks**. This proceeds via synapses, through which the axon of a given neuron makes contact with the dendrites of other neurons. At birth, every neuron has about 2500 synapses. In the first 3 years of life, their number increases massively (up to about 15,000 synapses per neuron), only to return to the numbers common in the adult brain (10,000–20,000); this happens somewhere between the tenth year of life and puberty (*synaptic pruning,* Huttenlocher 1994). Therefore, our brain consists of an incredibly complicated network of nerve cells, which are each in direct contact with thousands of other nerve cells through synapses. Most synapses are excitatory in nature (meaning they carry on the stimulation); some are inhibitory and preclude an uncontrolled stimulation in the cluster of neurons.

In recent years, a (however still controversial solution) to the binding problem is being discussed increasingly. Von der Malsburg (1995) proposed that spatially distributed neuronal populations, which encode separate aspects of the same stimulus, **synchronize their discharge patterns in time** and thereby signal which of the activated codes belong together and which codes do not. In fact, single-cell research with cats and monkeys has shown that neuronal populations in separate parts of the cortex, which are sometimes removed quite far from each other, do couple their activities in time. In monkeys, synchronized activity between the premotor and motor cortices and between neurons in the motor and somatosensory areas before initiating a finger movement was found. In cats, temporal synchronization between neurons of the visual and the parietal cortex as well as between neurons in the parietal and the motor cortex was found.

In humans, such temporal synchronizations of neuronal clusters can be measured with electro-encephalogram (EEG). The temporal couplings between distributed neuronal clusters that are reported in animal research are accompanied by **oscillations** in the beta (13–20 Hz) and/or gamma bands (30–80 Hz) and these oscillations can be extracted from the EEG frequency spectrum by using so-called wavelet-analyses. In such experiments, it can be shown that EEG oscillations can occur in relation with both perceptual and action processes. For example, Tallon-Baudry and Bertrand (1999) found an increase of oscillatory activity in the gamma range when their participants observed visual stimulus configurations. EEG oscillations also occur in connection with actions. Pfurtscheller et al. (1994) found gamma oscillations directly prior to the onset of movement of the left or right index finger, the right toe, or the tongue; these oscillations were found in the somatosensory cortex, where these body

parts are represented (see Sect. 2.2). In short, ballistic movements (i.e., short, rapid movements which cannot be interrupted), the oscillatory activity starts directly before the execution of a movement and ends with the onset of the movement. In slow, guided movements, the oscillations can last throughout the execution of the movement (Kristeva-Feige et al. 1993).

Now that we have sketched how the brain is built and how it works, we will turn to the question of which cortical and subcortical structures are **involved** in the planning and execution of action and which **part** they play (Fig. 2.2). We will see that the prefrontal cortex is always involved when we act in a goal-directed manner. The neuronal networks of the primary motor and lateral premotor cortices are responsible for the execution of movements. The supplementary motor area (SMA) is concerned with the planning of actions and the sequencing of single action elements. The dorsolateral prefrontal cortex (DLPFC) represents the goal of an action and is responsible for the activation, implementation, and configuration of executive control processes which coordinate our actions and adapt them to changing conditions. The anterior cingulate cortex (ACC) monitors our actions and their success, and signals the DLPFC when updating of action goals would be beneficial. The selection of actions in an interplay with the DLPFC considers expected rewards. These are computed or made available by the orbitofrontal cortex (OFC). We will also see that subcortical structures play a decisive role in the control of actions: Expected

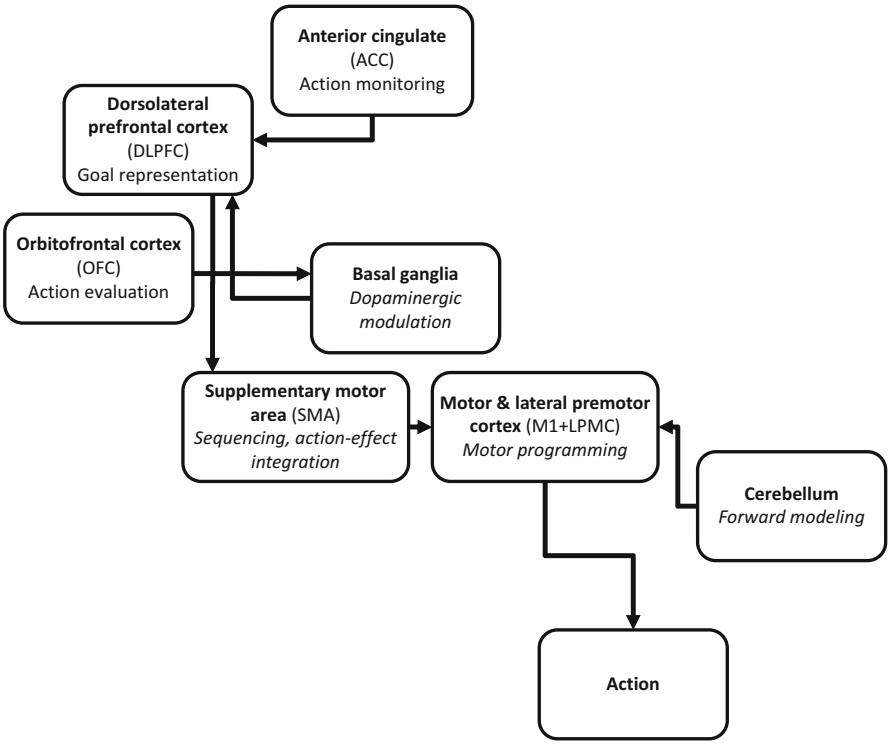

Fig. 2.2 Overview of the major contributions of various cortical and subcortical structures to the control of human actions

rewards influence the dopamine production in the basal ganglia, which modulate the workings of the DLPFC. Finally, accurate and fluid execution of movements depends on intact structures of the cerebellum (small brain), which monitors the success of concrete movement elements on the basis of forward models.

2.2 Primary Motor Cortex and Lateral Premotor Cortex (BA4/6)

Near the end of the nineteenth century, the German neurologists Gustav Fritsch and Eduard Hitzig discovered that electrical stimulation of a dog's cortex caused muscle contractions on the contralateral side: When the right cortex was stimulated, muscles on the left side of the body moved, and stimulation of the left cortex led to muscle contractions on the right side of the body. At approximately the same time, the English neurologist Hughlings Jackson discovered that epileptic insults are caused by lesions in the cortical motor area in the large brains. In the beginning of the twentieth century, the English neuropsychologist Charles Sherrington demonstrated that it is easiest to induce muscle contractions in monkeys when the electrodes are placed in the gyrus praecentralis in either of the hemispheres of the large brain. This area is today known as the **primary motor cortex (M1)** (Fig. 2.3).

M1 is found in the central areas of the two hemispheres (BA4) and borders on the sensory areas (i.e., on the somatosensory cortex). Numerous observations suggest that M1 is an important **coordinating point where cognition and motor activity join** and that its functioning is crucial for the execution of **movements**. For example,

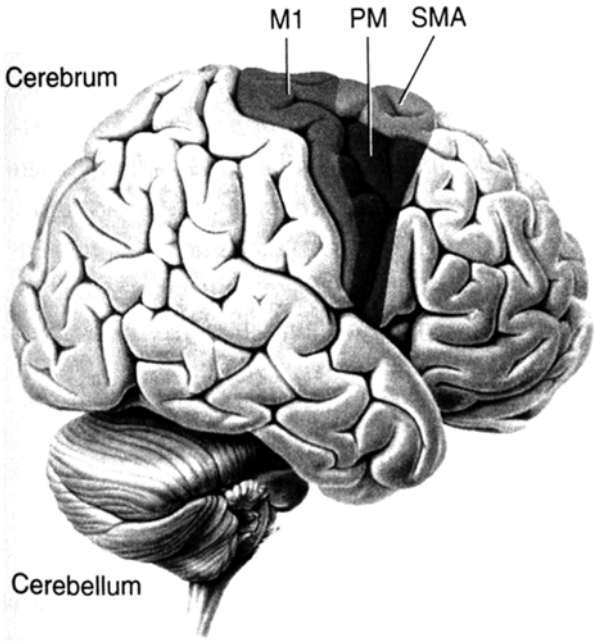

Fig. 2.3 Motor areas of the human cortex (adapted from Konczak 2008; with permission from Spektrum Akademischer Verlag)

patients with damage in just the motor areas have little difficulty with remembering action goals, maintain them, or switch between them. However, they have massive problems with executing bodily movements to realize these goals successfully. Depending on which hemisphere of the brain is afflicted, lesser (paresis) or more severe (plegia) paralyses of the limbs on the **contralateral side of the body** are manifested. If the left hemisphere is damaged, effectors on the right side of the body are paralyzed and vice versa: Effectors on the left side are paralyzed when brain damage is localized to the right hemisphere. Therefore, each half of the motor cortex controls the contralateral effectors. (This applies, at least, to parts of the facial mimic and the hands, but not for control of the movements of the feet.)

2.2.1 Motor Homunculus

In the 1930s, the Canadian neurosurgeon Wilder Penfield started to electrically stimulate various areas of the cortex of patients, whose skulls were opened for surgery. As there are no touch and pain cells in the brain, this process was painless for the patients. Penfield discovered that stimulation of the gyrus postcentralis induced **tactile sensations** in specific parts of the body. Furthermore, he found that these stimulation points in the brain were not scattered haphazardly throughout the brain, but were actually organized according to a **systematic map**. He also concluded that such a map was not present for the surface of the body. In a neighboring area of the brain, in the gyrus praecentralis, a similar map exists, which represents the skeletal musculature of the body. Depending on which part of this map he stimulated, contractions of specific muscle groups occurred. When he stimulated regions near the very top near the central sulcus (furrow) that separates the two brain hemispheres (medially), contractions of muscles in the contralateral leg were induced, while stimulation more to the sides, in the lateral motor cortex, resulted in movements of the hands or facial musculature. The systematic mapping of the primary motor cortex demonstrated that this brain region entails a somatotopic map of the complete skeletal musculature. This map was named the **motor homunculus** (cf. Sect. 1.2.4), which lies opposite to its sensory equivalent (the somatosensory homunculus on the other side of the central sulcus).

As is shown in Fig. 2.4, this representation is strongly distorted. Particularly important parts of the motion apparatus like the hands and mouth are strongly overrepresented while other parts like the trunk are strongly underrepresented. This is probably because the size of the cortical fields is not associated with the size of the innervated muscles, but with the complexity of the motor functions we have at our disposal. This would explain how the hand, with which a large number of different operations can be performed, can be represented by a much larger area than the foot, which is stereotypically used for locomotion only. These kinds of **somatotopic maps** are also located in cortical areas directly anterior to M1. The medially located area near the central sulcus that separates the two hemispheres was designated **supplementary motor area (SMA)** by Penfield (medial BA6, cf. Sect. 2.3); the area lateral to that, **premotor cortex (PM)** (lateral BA6). PM and M1 cooperate intensely, and much information that M1 receives is modulated by the PM.

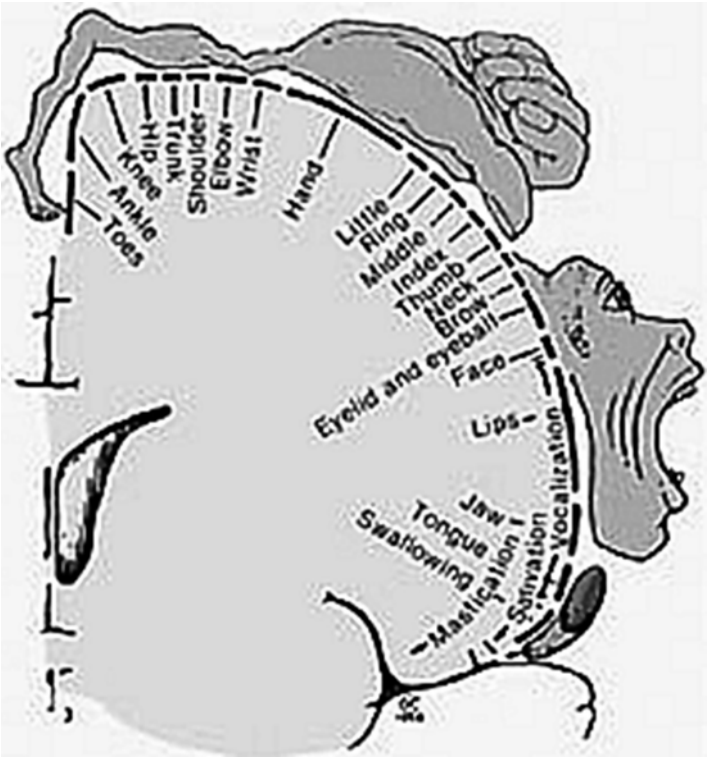

Fig. 2.4 Motor homunculus. Adapted from Penfield & Rasmussen, 1950, with permission from Gale, a part of Cengage Learning Inc.

Which function could the motor homunculus have? What do the neurons that constitute it represent? The classical answer to this question is it represents the skeletal musculature of the body, and controls the activity of the various muscles of the bodily periphery. Therefore, the motor homunculus was seen as a type of a marionette's control bar to which the muscles are connected as with threads, and on which they will act like a puppeteer who operates a marionette. For this to work, there should be a 1:1 connection between, for example, M1 neurons to a specific group of muscle fibers. This is clearly not the case, however. Although it is possible to distinguish, for example, leg, hand, and face areas in a somatotopic map, there is no differentiation within these areas: it was impossible to demonstrate a somatotopic representation of the hand muscles when the hand area of primates was stimulated systematically (Schieber 1999). Therefore, repetitive stimulation of the same M1 neurons during various motor tasks activates various muscle fibers (Georgopoulos et al. 1999). Furthermore, various muscles can be represented in the same area in M1 and various areas in M1 can activate the same group of muscle fibers (e.g., Penfield and Boldrey 1937). Therefore, somatotopic maps in M1 do *not* appear to represent the skeletal musculature of the body and do not appear to be the address where control of the various muscular fiber groups takes place.

Box 2.2 Stimulating the Primary Motor and Premotor Cortex

Graziano et al. (2002) electrically stimulated various areas within the primary motor and premotor cortex of two monkeys. Unlike the classical studies by Penfield, in which stimulation of short durations was used (ca. 50 ms), these authors stimulated the neurons of the motor cortex for 500 ms. Instead of contractions of single muscles, they evoked **fluent, spatially and temporally well-coordinated movements that were directed at spatial goals** under these conditions. The stimulation of a specific area, for example, led the monkey to close its finger, move its hand to its head, and then open its mouth. This movement sequence occurred reliably, independent of the hand's position when stimulation was commenced. When neighboring areas were stimulated, the same motion sequence was initiated, but with one significant difference: depending on the site of the stimulation, the hand assumed **different goal positions** after finishing the movement, a little more below or farther away from the midline of the body.

So, the (longer-term) electrical stimulation of neuronal populations in the motor cortex and lateral premotor cortex appears to evoke relatively complex movements towards spatially specific goals. This might mean that the somatotopic maps of the motor cortex do not correspond to specific muscle groups, but to positions in space, that is, to potential targets of movements in the grasp or manipulation space close to the body. In fact, Graziano et al. found a very **close relationship** between the stimulated areas in the brain and the spatial targets of the movements evoked by the stimulation. They stimulated the motor cortex in the right hemisphere in eight different positions within the hand-arm area and found, again independent of the starting position, eight different end positions of the hand (Fig. 2.5; the circle on the sketched brain hemisphere shows the area in which the stimulation took place). The left hand of the monkey moved to a position in the upper, middle, or lower grasping space, either to the right side of the body (ipsilateral to the stimulated hemisphere), to the middle of the body, or to the left side of the body (contralateral to the stimulated hemisphere). Through further measurements and stimulation of the motor and premotor areas (around the areas marked by letters in Fig. 2.5), Graziano et al. were able to demonstrate that a whole series of complex movements actions is represented in **somatotopic maps**. While stimulation of one area evoked hand movements towards the middle of the body at chest height, combined with a precision grip, a fist, an open hand with spread fingers, or a rotation, stimulation of another area led to hand movements to the snout of the monkey, combined with a precision grip and the opening of the mouth.

These observations demonstrate that the neuronal populations of the lateral premotor and the primary motor cortex control complex and coordinated movements. It is particularly interesting that these movements kept occurring consistently and unaltered, even after hundreds of stimulations. Even when an obstruction was placed between the hand and the target position, the movement did not change, so that the hand would hit the obstruction and kept

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Box 2.2 (continued)
applying pressure to it for as long as the stimulation continued. The induced movements were even independent of what the monkey was doing: they occurred when it sat still, moved spontaneously, grasped a piece of a fruit, or was even anesthetized.

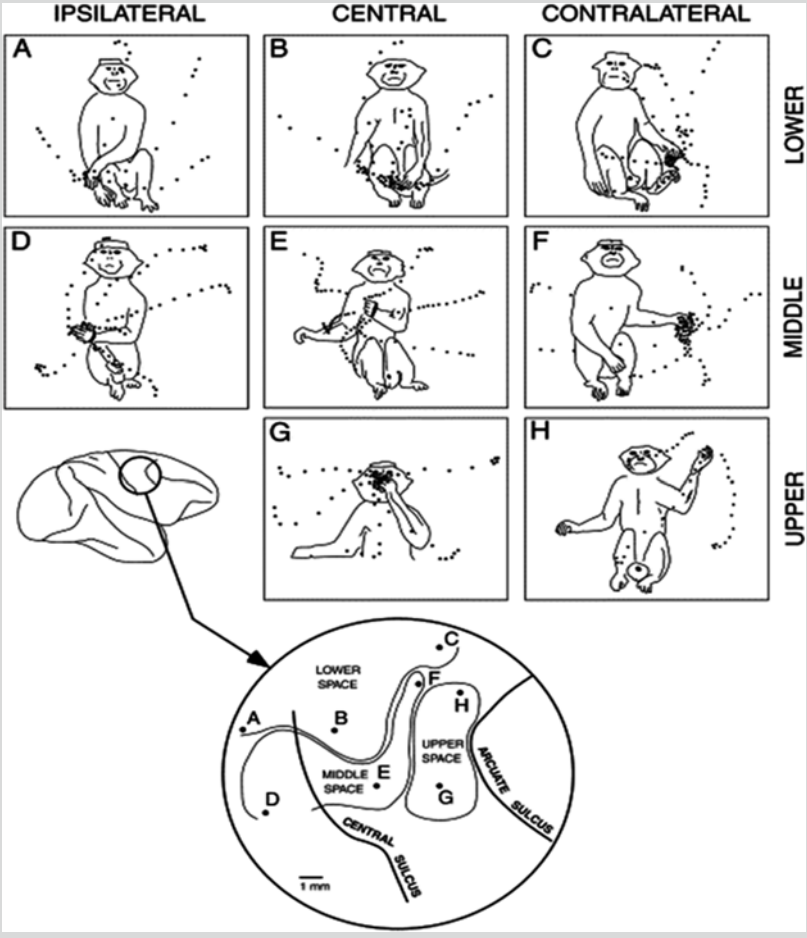

Fig. 2.5 Various hand postures induced by stimulating monkey motor cortex. Adapted from Graziano et al. 2002, with permission from Elsevier

So, if the neurons in M1 and PM do not control single muscles, what *do* they code? Which information do they make available? Research with primates suggests that they encode a sequence of **motor parameters** such as the direction and speed of movements, the position of joint angles, and muscle force (see above), and that they can **directly control** the course of more complex movements. The motor cortex and parts of the premotor cortex (which is also involved in the perception of movements, see Box 2.3) can be seen as the **final station in the action control of humans** and provide the actual muscular activation. These brain areas do not take part in the adaptive planning themselves: they eventually execute what has been planned by other cortical systems. One of the systems that is concerned with the planning of movements is the so-called SMA, to which we turn next.

Box 2.3 Mirror Neurons

The premotor cortex was typically ascribed functions related to action planning and control. This made it all the more surprising when neurons with sensorimotor properties were discovered in the premotor cortex of monkeys (di Pellegrino et al. 1992), which serve both perception and action. These so-called mirror neurons were not just active when a monkey performed a grasping movement itself, but also when it observed the same movement in a member of its species, or even in a human being. The activity of these neurons is highly specific and clearly related to action; these neurons are active only when a specific grasping motion with a specific target object is made (review by Rizzolatti and Craighero 2004). A comparable shared system for the performance and observation of movements appears to exist in humans too (Decety and Grezes 1999). Fadiga et al. (1995) have demonstrated that humans who observe others perform a given action, activate the same muscles they would use to perform that action. The neuronal network which is activated by the observation of actions in humans encompasses, besides the premotor cortex, parietal areas and the superior temporal sulcus (STS) (Grafton et al. 1996; Rizzolatti et al. 1996), but not the primary cortex and the SMA.

These observations have created quite a sensation; partially rightfully so, and partially not rightfully so. Unrightfully so in cases where the existence of mirror neurons is interpreted as an **explanation of a number of hard-to-explain phenomena**, such as imitation learning, empathy, or the understanding of fellow humans. For example, when the perception of another person's action activated one's own mirror neurons, this might explain why that action would be easy to imitate and why people often do so, possibly unconsciously. Of course, it is entirely possible that mirror neurons are involved in these cognitive performances, but their bare existence cannot be considered a satisfactory explanation.

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Box 2.3 (continued)
In fact, how can one's own motor neurons actually represent another person's visual action effects in one's own motor functions, while the other person often has a completely different body structure, and must therefore use completely different muscles in a completely different way to perform the same action? In a certain sense, mirror neurons do not solve any theoretical problems, nor do they offer a better functional understanding of the mechanisms that lie at their foundation.

However, there is a number of reasons why the discovery of mirror neurons justifies the sensation it caused. One reason is that the existence of mirror neurons indicates the **close relation between action and perception**, a relation that is overlooked in many textbooks, not to mention research. When the pure observation of an action leads to the activation of motor areas, the question arises if or to what extent action-related knowledge influences the observation. That it does do so, is demonstrated by, for example, the fMRI study by Calvo-Merino et al. (2005). They studied professional ballet dancers, Capoeira dancers, and laymen who did not master either of these dancing styles, and presented sequences of ballet and Capoeira movements to all three groups. The neuronal mirror system was only activated during the observation of these movements when the observing person had mastered the dancing style himself. Further evidence for the role of one's own motor expertise in the perception of movements is provided by the fMRI study of Grèzes et al. (2004). Here, participants saw videos of people (with blurred faces) who lifted a weight, and in some videos, the participants themselves were shown. The mirror systems were activated significantly earlier when participants observed their own movements in the video.

A further ground that makes the discovery of mirror neurons interesting has to do with the **relation between the perception of oneself and others**, which could receive a whole new theoretical meaning in the light of mirror neurons. Let us consider how mirror neurons can produce the relationship between an observed action and an action that is performed by oneself. One possibility is that we first obtain the systematic relationship between our own movement and the sensory consequences thereof (Sects. 2.3 and 2.4). We generally experience our own movements proprioceptively or kinesthetically and, to a certain extent, also visually. In general, perception is multimodal, however, and as children we quickly learn to generalize between modalities (Spelke 1976): we see what things feel like, and we feel what they look like. These two learning processes—the association of motor commands and sensory effects on the one hand, and generalization over modalities on the other hand—are sufficient to establish a mirror system that responds equally strongly to perceived and one's own movements and that depends on one's own experiences (see also Keysers and Perrett 2004).

2.3 Supplementary Motor Area (BA6 Medial)

The SMA is the medial part of the premotor cortex and is important for the **selection, planning,** and **sequencing** of goal-directed actions (Chaps. 5–7). The SMA also appears to be important for the perception of the intentionality of an action. Together with the lateral part of the premotor cortex, the SMA is one of the most important information sources for the primary motor cortex.

2.3.1 Role of the SMA in the Sequencing of Action Elements

Evidence for a decisive role of the SMA in the sequencing of movements is derived from **patient studies, fMRI and TMS studies in healthy participants, and animal research**. For example, patients with unilateral lesions in the SMA have deficits in the execution of sequential movements with the contralateral arm, or difficulties in reproducing rhythms from memory (Dick et al. 1986; Halsband et al. 1993). In healthy persons, the SMA is much more active during self-initiated—as compared to stimulus-induced—movements (Deiber et al. 1999), and disruption of the SMA through targeted transcranial magnetic stimulation (TMS)-pulses produces errors in the generation of complicated movement sequences (Pascual-Leone et al. 2000).

Tanji and Shima (1994) were able to demonstrate the existence of different **types of neurons** in the SMA of monkeys, neuron types that apparently encode different aspects of the sequencing of movements. The monkeys learned to execute various movements, and then had to string them together from memory in various successions. Three types of SMA neurons could be identified:

- Neurons that fired during the preparation of a movement sequence
- Neurons that were only active in the interval between two movements
- Neurons that appeared to represent the succession of single movements

In a follow-up study, Shima and Tanji (1998) temporarily disabled the neuronal population of the SMA pharmacologically, and found that the monkeys made more errors in the execution of movement sequences from memory under these circumstances, but not when the to-be-executed movements were signaled by visual cues.

2.3.2 Role of the SMA in the Integration of Actions

Besides its key role in the sequencing of movements, the SMA is also involved significantly with the **encoding** of intentions and action goals and the **selection** of intentional actions. Goal directed actions are movements which are executed for the purpose of producing very specific, intended effects: one operates the light switch

to turn on the light, rides a bicycle to reach another area, talks to convey a message to others. Intentions and goals are therefore focused on the relationship between movement patterns and desired outcomes (Chap. 3).

The SMA also appears to play an important role in the **integration** of movements and effects. For example, in the study by Elsner et al. (2002) participants first acquired new auditory action effects when they pressed buttons that produced specific tones. Later, they were instructed to wait for the presentation of another tone, while they were in a **Positron emission tomography** (PET) scanner that measured their brain activity (cf. Box 2.4). During this waiting period, the action effects which were acquired previously, that is, the tones that were previously elicited by button presses, were also presented. The auditory action effects activated not just the auditory cortex, but the SMA and the hippocampus (a structure that is important for episodic memory) too. This observation was replicated recently by Melcher et al. (2008) in a fMRI experiment. This showed that acquired action effects are integrated with the associated movement patterns, and that this integration produced a connection between the SMA and the sensory representations in the episodic memory. As we will see in later chapters, this connection constitutes an important **condition** for the **selection of movement patterns** based on the number of effects that can be attained with them. In other words: it is this connection that allows us to perform goal-directed actions.

Box 2.4 Methods to Study Brain Processes

The classical method to study the active brain is to record the fluctuations in potentials on the surface of the skull, which occur before, during, or after a sensory, motor, or psychological stimulus or reaction in the EEG. The largest contribution to potential fluctuations is made by spontaneous activity of cortical neurons. This changes immediately when the brain is occupied with the processing of a stimulus or the preparation of a motor response. This causes systematic activation patterns that can be averaged across many trials and made visible in an **evoked potential** (also known as event related potential, ERP).

The evoked potentials are extracted for the spontaneous EEG through averaging across a series of responses (generally, a couple dozen) to visual or acoustic signals. They are classified according to their polarity, positive or negative, and their time of occurrence. For example, the first positive part of the potential (generally in the range from 90 to 140 ms) is designated P1, a component that is associated with early stimulus processing. The specification of polarity and latency alone do not define potentials sufficiently: the area(s) on the skull from which the different evoked potentials are recorded should be added. This provides information on which cortical areas are involved in the processing of stimuli or the performing of specific tasks (albeit with fairly poor spatial resolution). In sum, evoked potentials have an excellent temporal resolution (in the millisecond range) and provide valuable

(continued)

Box 2.4 (continued)
information about the **electrical activity** that accompanies perceptual and action-related processes.

The traditional shortcoming of evoked potentials, their poor spatial resolution, can be countered by **imaging techniques** that have been developed in recent years. **Positron emission tomography (PET)** and **functional magnetic resonance imaging (fMRI)** are relatively new techniques for the imaging of brain areas that are activated function-dependently. PET is based on the measuring of radioactive marker substances that are injected into the bloodstream beforehand. These radioactive markers are used more strongly in actively metabolizing cells, in other words, cells that are engaged in performing a given task. A positron detector, which is placed around the head, counts the emitted particles so that a computer can identify areas of stronger or weaker radiation. PET attains a spatial resolution which allows localization in the millimeter range, but has a very poor temporal resolution (in the range of multiple seconds, up to 10 s).

The particular **benefit** of functional magnetic resonance imaging (fMRI, also known as functional nuclear spin tomography) is that radioactive marker substances are not needed. The only downside is that the processes whose brain activation is studied (e.g., reading, arithmetic, finger movements) must be performed in the small space that is offered by an MRI scanner.

The MRI technique uses the fact that our brain, like other body tissues, consists for a significant percentage of water. This fact is used to **image** the structures of the brain: the hydrogen molecules in our brains possess magnetic properties; each of their atoms functions as a magnetic dipole. When these dipoles are put in a magnetic field, they align with the surrounding magnetic field, just like a compass needle. For this to happen, an extremely strong **magnetic field** is necessary. Typically, MRI scanners use magnetic fields that are 50,000 times as strong as the magnetic field of the earth. When the alignment of magnetic dipoles is disrupted through high-frequency energy impulses, after which they return to their previous aligned position, impulses that are recorded and then amplified, arise. These signals allow the identification of hydrogen molecules and the assessment of their relative proportion in various brain areas. As other body tissues, the brain consists of 70 % of water, and various areas in the brain have different proportions of water. Nerve cells, for example, are relatively rich in water, while the myelin sheath that covers the axon is relatively poor in water. This generates **intensity differences** between signals from different tissue types, which are used to identify various structures in the brain in relatively high levels of detail.

Up to this point in our description, MRI presents us with an image of the **architecture of the brain**. With their high spatial resolution of less than 0.5 mm, MRI images show that the surface of the brain is not particularly spectacular, but that it is richly structured internally. However, no matter how detailed these images are, they provide no clues as to what extent brain areas are involved in performing various tasks. To draw conclusions like that, one would have to observe the brain as it is working, so to speak, and to analyze the

continued

Box 2.4 (continued)
task- and function-dependent activity of various brain areas. This is possible through functional magnetic resonance imaging (fMRI), which indirectly measures the **metabolism** of the brain. To understand how the brain's metabolic activity can serve as an indicator of the activation of neuronal populations, we must first have a look at the functional units that constitute the brain.

The basic functional unit of the brain is the **neuron** (Sect. 2.1). A neuron has a cell body, with relatively short projections, dendrites, which take up information from other neurons as input, and lead it to the soma. A neuron also has a relatively long projection, the axon, which conducts electrical impulses from the soma to the dendrites of other neurons. The site where the axon of a neuron comes into contact with the dendrite of another neuron is called a **synapse**. When the electrical impulse which is conducted through the axon exceeds a certain threshold, a chemical **neurotransmitter** is released at the end of the axon. When the neurotransmitter at the synapse contacts the dendrite of the second and other proximal neurons, the electrical impulse is carried on by the second and a host of other neurons, which form a network together. It is important to note that after they have been released, neurotransmitters are "recycled" and are transported back into the neuron. This process requires energy, which has a consequence: the brain's **perfusion** increases locally (the so-called hemodynamic response), to ensure that sufficient amounts of oxygen are available.

This effect is utilized to identify the areas of the brain that are particularly **active** during the performance of a given task: the properties of the dipoles of the hydrogen atoms depend, amongst other factors, on the amount of oxygen in the surrounding blood. Local changes in the amount of oxygen follow increased neuronal activity with a short delay, so that differences in the intensity of signals from the dipoles can be indirectly used to identify those regions of the brain that are responsible for the increased oxygen requirements.

Take, for example, a classical experiment in which participants had to move their fingers ("tapping"). Such movements are prepared in the premotor and primary motor cortices and are led to the motor neurons in the spinal cord after modification by the extrapyramidal system and the cerebellum. To display the neuronal activations in fMRI, participants had to tap his fingers for 30 s while in the MRI scanner, then remain still for another 30 s. During this time, the scanner recorded a few hundred images of both movement phases. These were then averaged and showed, after complicated post-processing, the distribution of neuronal activity during finger movements of the right hand.

While interpreting fMRI data, one should understand that it is not actually the neuronal activation that is being displayed, but a **surrogate**, the hemodynamic response to increased energy requirements *as a result of* neuronal activity. This surrogate is much **slower** (it is developed during multiple seconds after setting of a task) than the neuronal activation, which lasts less than 100 ms. Therefore, there is always a delay of up to 6 s between the time point of neuronal activation and the hemodynamic response. This would not be a problem if the hemodynamic

(continued)

Box 2.4 (continued)
response would always start after a fixed interval following the neuronal activation. However, this is not the case at all. There are indications that the time point at which the hemodynamic response sets in varies between persons, brain regions, and tasks. Therefore, one cannot be certain that the measured hemodynamic reaction actually represents the neuronal activity that is elicited by a given task, or whether it reflects activity that started later.

These problems of interpretation can be neutralized through different experimental designs. For starters, one can use a **block design**: intermix blocks in which the participant performs the task of interest and in which he performs no task or, even better, a control task. The control task should be constructed in such a manner that it is completely similar to the experimental task, but does not evoke the cognitive processes of interest that are associated with the experimental task. This allows researchers to subtract the neural activations from the control task from those of the experimental task, and thus isolate the activity of those brain areas which are associated with the cognitive processes of interest.

Secondly, one can choose a so-called **parametric design** and vary the manipulation of interest systematically in intensity. When a given brain area is systematically affected by this manipulation, the extent of its activation should vary systematically with the intensity of the manipulation. To illustrate: in a study on motivation, one could vary the incentive for solving a problem in equidistant steps and test which brain area is more activated during stronger incentives.

Thirdly, one can present stimulus configurations as isolated stimuli with sufficiently long time intervals, so that individual reactions to single stimuli can be identified. Such **event-related designs** evade potentially confounding factors such as fatigue and habituation, which are common during repetitive stimulation.

The methods to study brain processes that we have sketched up to this point, all attempt to open a window onto the brain and to observe it during its "everyday" activities without attempting to intervene in these activities from the outside. But this is not the only way to study the workings of the brain. The other possibility is to artificially simulate the different structures and to observe the sensory and motor consequences of external stimulation. This path is followed by **stimulation studies** that are generally performed with animals, although occasionally also with humans, whose skull caps are opened, for example, because of neural surgery.

The electrical simulation of neurons in the cortex goes back to Fritsch and Hitzig (1870) who used electrodes to stimulate the surface of the cortex and demonstrated that the motor cortex of dogs shows a somatotopic organization. These observations were confirmed in monkeys and humans during the next decades. Asanuma et al. (1976) developed the method when they simulated

continued

Box 2.4 (continued)
neurons through microelectrodes with low intensity. This technique was used in many experiments, in which it was shown that short (often shorter than 50 ms) stimulation of neurons in the motor cortex evokes muscle activity, which is in fact the contraction of single or multiple groups of muscle fibers.

Graziano et al. (2002) used **longer stimulation** (500 ms) in addition to short simulation and this longer stimulation was close to the time monkeys need to execute arm or hand movements. They found that under these conditions, complicated, well-coordinated movements were sold the monkeys. The contractions of single muscle fibers may well have been the beginning of these longer movement sequences that were evoked by longer stimulation.

In such studies, narrowly defined areas of the cortex were stimulated locally, under the assumption that stimulating a relatively small number of neurons locally would spread to widely branching networks of neurons, that control the action. However, the electrical stimulation of neuronal populations does not resemble actual biological or physiological processes. Therefore, any and all effects should be interpreted with caution. For example, it is possible that external stimulation generates an artificial, unnatural activation pattern in the affected neuronal population. This method can be convincing when the evoked movements resemble biological movements that can be associated with known functions of the brain area that is being studied.

A less invasive method to stimulate the cortex externally is **transcranial magnetic stimulation (TMS)**. With this technique, the electrical activity of cortical neurons is influenced through a magnetic field, which is created by a coil that can be placed in various positions on the surface of the skull. Depending on such stimulation parameters as duration and intensity, local neuronal populations of the brain can be inhibited in a temporally precise manner (this would lead to a, so to speak, transient, functional lesions of the neuronal population), but they can also be activated. For example, when the magnetic field is located over the visual areas of the occipital cortex near V5, an area that is assumed to be important for the perception of movement, then it can be demonstrated that TMS selectively disrupts the perception of the motion direction of an object, without influencing its identification at the same time. When neurons in the motor cortex are stimulated, it can be shown in reaction time experiments that TMS shortly before the execution of a movement delays that movement, without influencing its form. Finally, it can be demonstrated that stimulation of the SMA selectively disrupts the execution of complex motion sequences, but not the execution of simpler sequences.

Further evidence for the role of the SMA in the integration of movements and their effects is presented by Haggard et al. (2002). They could first demonstrate that participants systematically underestimate the timing of the effects of actions that they produced themselves: even when an effect occurred after a delay, participants still experienced it as occurring temporally close to the action. This observation also suggests an integration of action and action effects, which led to a temporal illusion here. Interestingly, this illusion breaks down when the activity of the SMA is interrupted through targeted TMS pulses (Haggard et al. 2002). Therefore, the SMA appears to be important both for the integration of action effects and for specific aspects of the experiencing of intentionality. This conclusion is supported by work of Lau et al. (2004). Here, participants made spontaneous finger movements and they were instructed to pay attention either to external stimuli, or to their own intentions. Activation of the SMA was greater in the latter condition, so the greater intentionality of the action corresponded to a stronger contribution of the SMA.

Patient studies suggest a strong relation between intentionality and SMA too. Patients with damage to the SMA often respond to objects in their surroundings with actions that are not accompanied by feelings of intentionality. For example, they may grasp a pen that lies in front of them and start to write, apparently unintentionally. Highly comparable is the so-called **alien hand syndrome**, in which patients know that they are executing particular movements, but cannot report on plans to perform this movement (Marcel 2003).

The observations of Fried et al. (1991) are very interesting in this context: they stimulated the SMA of epileptic patients directly during preoperative neurosurgical measures. During low-intensity stimulation, patients indicated that they occasionally felt the desire to move a specific limb. When stimulation of a greater intensity was applied to the very same area, actual contractions of the limb were observed. This observation, like the alien hand syndrome, indicates a close connection between the subjective experience of intentions and the neuronal processes in the SMA.

2.4 Cerebellum

The SMA is involved in the integration of actions and their consequences. This brain area is important because it allows the selection of alternative actions depending on the intended effect, as well as the evaluation of the consequence of an action by comparing the intended and actually obtained effects (Chap. 9). These effects which we have spoken of up to this point, and which are generally related to intentions, are relatively abstract when compared to the concrete muscular activities and motor parameters of the movements that are necessary to attain these effects. Where does the **information** to control these activities and to specify their parameters come from? It seems plausible that the **cerebellum** makes this information available.

The cerebellum is located below the large brain, in the occipital cortex (the lobe in the back of the head). Damage to the cerebellum does not result in complete failure of motor functions, but is manifested by problems in **coordinating movement processes**. Luciani (1891), for example, studied swimming movements of dogs in which one of the cerebellar hemispheres was removed. He observed that the basic movement pattern was retained in all four paws. However, the coordination of the two paws located ipsilaterally to the lesion was disrupted: their movements were irregular, uncoordinated, and lost their fluidity.

2.4.1 Consequences of Damage to the Cerebellum

Clinical studies by the British neurologist Gordon Holmes of soldiers who suffered gunshot wounds to the cerebellum during the First World War showed that the cerebellum is involved with the regulation of **muscle tone**, with the control of **support and gait motor function**, and with the **coordination** of movement segments (Holmes 1917, 1939). Small lesions of the cerebellum can be compensated fairly well, while larger lesions can cause so-called **ataxic motion disorders**. **Ataxia** describes a lack of coordination, which can be manifested in the motor skills of the eyes, speech, torso, and extremities. Dissymmetric movements would be an example: healthy persons are generally able to close their eyes and move their arms from the side of their torso in such a manner that the tips of their index fingers touch in the center of their torso without many problems. Patients with cerebellar lesions are unable to generate motion impulses that make the two hands move in synchrony spatially and temporally during such pointing movements. This often leads to over- and undershooting movements of the fingers.

These patients often have problems with speech, which is manifested as slowed, halting speech with poor articulation and uneven stressing of syllables. Beyond that, they have problems with the execution of fast, alternating movements, which require a rapid switching between agonists and antagonists, often have an insecure, wide-legged gait, and occasionally show a(n intentional) tremor, which, contrary to the resting tremor in Parkinson's disease patients, occurs during the execution of a movement, especially during its final phase, where the accuracy requirements are often greatest.

2.4.2 Cognitive Functions of the Cerebellum

The cerebellum has many recurrent (that is, reciprocal, interactive) connections with almost all areas of the cerebral cortex (Middleton and Strick 2000). It receives input from the motor cortex and almost all sensory areas in the cerebral cortex. Through ascending projections in the spinal cord, it receives proprioceptive information about the conditions of the skeletal musculature and about the current

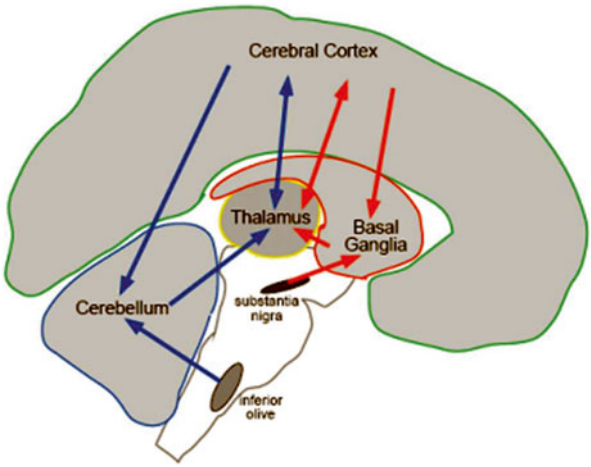

Fig. 2.6 Connections between the cerebellum and various cortical and subcortical structures of the human brain (from Doya 1999; adapted with permission from Elsevier)

positions of the various joints. It projects through the thalamus to the motor areas of the large brain and to further areas of the prefrontal parietal and temporal cortices (Fig. 2.6). This strong networking suggests that the cerebellum is involved with other cognitive processes besides its motor tasks.

In addition to the control of action, **two** partially related **functions** of the cerebellum are of particular importance: predicting the sensory consequences of concrete movements (so-called forward modeling; Box 2.5), and the control of motor learning (Sect. 9.4). Evidence for the involvement of the cerebellum in motor learning is provided, for example, by Imamizu et al. (2000). These authors had participants in a fMRI study follow a moving stimulus with a mouse, and to attempt to cover it with the mouse cursor. The requirement of motor learning was induced through a manipulation of the relationship between the movements of the mouse and the mouse cursor so that, for example, moving the mouse upwards led the mouse cursor to move 120° to the right. To actually move the cursor upwards, participants had to move the mouse 120° to the left. In this condition, activity in the cerebellum was considerably greater than in a control condition. Given increasing practice with the oddly behaving mouse, activity declined proportionally to the reduction of motion errors of the participants (i.e., the difference between the position of the moving stimulus and the mouse cursor). This suggests that the cerebellum is involved with the **acquisition of new motor models**. However, even after the participants had learned to use the new mouse, activity of some areas in the cerebellum was increased as compared with the control condition, possibly because the newly acquired models in this task should be maintained actively.

Box 2.5 Forward and Inverse Models of Action Control

The concept of forward modeling stems from systems theory, as it is applied, for example, in engineering in the construction of robots. For example, assume that you are attempting to grasp an object, such as a coffee cup that is placed in front of you, for the first time in your life. In such a case, no motor experience is available to you, and so, you have no other choice but to try out various movements on a trial and error basis. As your experience increases, you will, however, acquire the successful movement, so **motor learning** takes place. In the future you will be able to reliably execute the desired grasping motion.

Expressed in terms of systems theory or cybernetics, you can thereby transfer a **desired** state into an **actual** state (Fig. 2.7). At the beginning of the movement, the desired state is compared to the actual state and the difference (estimated state error) is computed. This activates a motor control structure that, in turn, passes on instructions to the motor system (see outer loop in Fig. 2.7). The activity of the motor system leads to perceivable sensory changes: you can see and feel how your hand moves towards the cup. This changed state is compared to the desired state; when they resemble each other (so when the estimated error is close to zero), then the action is finished, otherwise the whole loop is reiterated until the desired state is attained. You may be familiar with this principle from central heating: the heating is kept active until the desired temperature is reached.

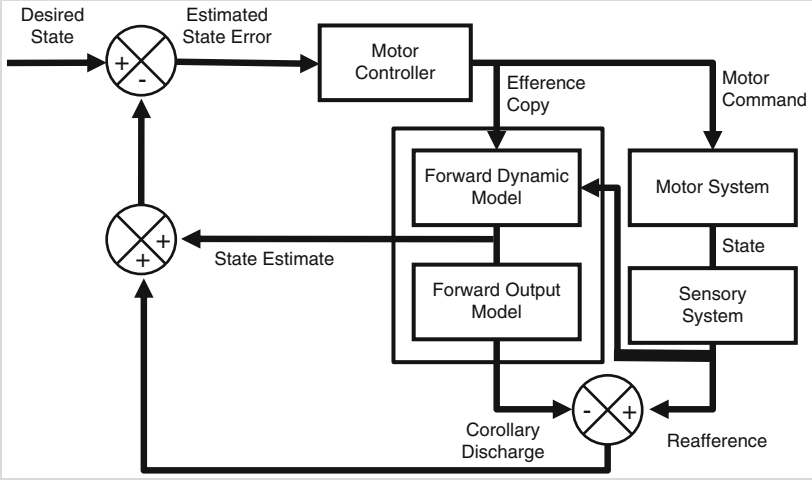

Fig. 2.7 The principles underlying forward modeling (after Wolpert et al. 1998. © Cell Publishing)

(continued)

Box 2.5 (continued)

Going through such loops again and again in the course of motor learning allows the learner to discover and acquire knowledge about systematic relationships between the motor commands (or rather the motor activities that result from them) and their sensory consequences. The acquisition of these relationships allows two things: the forward modeling and the inverse modeling of the relevant action. **Inverse models** provide information about which motor commands are required to achieve specific movement effects—like how the hand can be controlled to grasp a coffee cup in a specific location. The relationships between motor commands and sensory consequences are used here to determine the former on the basis of the latter. **Forward models** serve, conversely, to predict sensory consequences on the basis of motor commands (Wolpert et al. 1998).

What purpose could such forward models serve? Why would one even want to predict sensory consequences? There are a number of reasons for that. Most importantly, a prediction allows us to check internally, before a movement even starts, whether the selected motor commands will actually realize the intended movement (i.e., the desired sensory effects) and, if necessary, make corrections to the motor commands. You have probably experienced breaking off or not even having initiated a movement or the uttering of a word, simply because it somehow seemed wrong. It is very likely that the prediction of (in this case) apparently incorrect sensory consequences played a part in that. In the depicted model, this possibility has been taken into account: a copy of the motor command (so-called efference copy, a term introduced by Von Holst and Mittelstaedt 1950; cf. Box 9.2 on the **Reafference Principle**) is used to predict the execution of a movement in parallel with motor control and execution of the likely sensory consequences. This prediction can be compared directly to the desired goal state and, when the outcome is negative (that is, when the estimated error is large), passed on directly to the motor control structures and be used to change the motor commands there.

The possibility to monitor the performance of a movement and to correct it if necessary, independently of its actual consequences, facilitates action control in many situations (cf. Chap. 9). Sensory feedback about movements (e.g., visual feedback about the position of a moved hand) is not available until a considerable amount of time (usually multiple hundreds of milliseconds) has passed after the movement has ended; that depends on neuronal transfer and processing times. If the next movement elements would have to wait for this feedback first (in which it is ensured that the previous movement has been terminated correctly), then more complex movement terminations would last very long, would not look particularly smooth, and would be very difficult to control. In reality, the elements of an action are superimposed (cf. Chap. 7). This is clearly visible in grasping, where the hand already opens

(continued)

Box 2.5 (continued)
itself and adjusts its position to the size of the object as it moves towards the object that is to be grasped (Chap. 6). Now, when the putative outcomes of a movement are available through forward modeling even before it is terminated, then subsequent movements can already be planned and partially initiated. Both the planning of various movement elements as well as their execution can therefore overlap temporally, and the complete movement termination will become very efficient and smooth.

A further advantage of forward modeling is the possibility to control motor learning (Sect. 9.4). Hitherto, we have assumed that forward modeling will lead to the same outcome as the actual movement, so that the predicted and the actual states are identical at the end of a movement. At the start of the acquisition of a movement, that is not always the case, as the predictions are often fragmentary and unreliable due to lack of practice and experience. In such a case, if the outcome does not correspond with the outcome of a movement, so when, to use system theoretical jargon, an error has been detected, then this is an indication that the movement has not yet been sufficiently learned motorically. In other words, the identity of forward modeled predictions and actual movement outcomes signals successful motor learning, while differences, in contrast, signal further learning needs (Doya 2000; Wolpert et al. 1998). In this sense, a comparison of forward modeling and movement can take on the role of a trainer, without requiring the judgement of other persons.

2.5 Basal Ganglia

The basal ganglia are a collection of **subcortical nuclei** (nucleus caudatus and putamen, together known as the striatum, globus pallidus, substantia nigra, and nucleus subthalamicus), which receive strong afferent projections from not just the motor areas of the large brain, but also from the frontal eye fields, the limbic system, and the orbitofrontal and prefrontal cortices. In the basal ganglia, signals from the various brain areas remain topographically separated, are processed in parallel, and are sent back to the original cortical areas (Alexander 1995). That the basal ganglia are connected with not just the motor areas of the large brain, but also with the limbic and associative systems, indicates that they are not involved exclusively with **voluntary motor skills**. A particularly important function of the basal ganglia is the production of dopamine, a very influential neurotransmitter that modulates numerous cognitive and motor processes.

2.5.1 Consequences of Damage to the Basal Ganglia

Damage to the basal ganglia leads to a number of disruptions in **voluntary motor skills**, which are manifested in different manners, depending on which structures are damaged: a strong retardation of movements (bradykinesia), which is reflected by increased reaction times to visual and acoustic stimuli and altered speed profiles in goal-directed movements; a rest tremor (involuntary, rhythmic oscillations of the limbs); and a heightened stiffness of the musculature (rigor) can be the result of damage to the substantia nigra. Disruptions of this kind often occur in **Parkinson's disease** (morbus Parkinson), one of the most common neurological ailments (famous Parkinson's patients are the late pope John Paul II and the ex-boxing champion Muhammad Ali). The neurophysiological cause of the disease is the massive loss of dopamine-producing neurons in the substantia nigra. Damage in the area of the striatum is causes of **Huntington's disease**, which is manifested by massive gait and postural instabilities. Finally, lesions of the nucleus subthalamicus lead to involuntary heavy, large-amplitude movements.

Both Parkinson's and Huntington's diseases are explained by damage to the basal ganglia (and the accompanying dopaminergic failure regulation), which leads to a **disturbed balance** between excitatory and inhibitory influences on the frontal cortex (Alexander et al. 1990). According this conception, the execution of voluntary action is blocked by Parkinson's patients, while the inhibitory influences are increased drastically, which suppresses the initiation of voluntary movements, or at least makes their initiation harder. In Huntington's patients, the reverse is true: here the inhibitory influence of the basal ganglia is reduced drastically, which lowers the threshold to initiate motor activities dramatically. This would account for the common execution of involuntary, fast, and choppy movements.

2.5.2 Cognitive Functions of the Basal Ganglia

Dopamine that is produced in the basal ganglia does not only influence action regulation directly, it also modulates the acquisition of cognitive and motor skills. The unexpected success of an action leads to a temporary increase of the dopamine level, while an unexpected failure leads to reduction of the level (Schultz 1998). This makes information available to learning processes, about whether a given action was beneficial or detrimental, and whether it should be learned or avoided in the future. Groundbreaking research by Schultz et al. (1993) supports these hypotheses. These researchers have, for example, trained monkeys in operant conditioning paradigm, to grasp for objects following the presentation of visual stimuli; the monkeys were rewarded with fruit juice. Dopamine-producing neurons were very active during the starting phase of the training, when the monkeys received their reward following a successful performance. Following sufficient training, these neurons started to fire when the visual stimulus was presented, and they adjusted their

activity when a reward was received. Therefore, the activity of the dopaminergic neurons first signaled the rewards that were unexpected in the beginning of the training, then, later, increasingly signaled the expectation of a future reward (for comparable observations in humans, see Haruno et al. 2004). According to Doya (2000), based on experience, the basal ganglia can maintain information about which rewards are to be expected under the current circumstances and which action alternatives are associated with which rewards. Such a system can play a decisive role in the evaluation and selection of actions, and bias the action alternative that offers the greatest reward.

2.6 Prefrontal Cortex

The frontal and in particular the prefrontal cortex are relatively voluminous in humans as compared to other species. For example, in dogs, the frontal cortex makes up about 7 % of the cortex, in monkeys about 17 %, and in humans, more than 30 %. Until fairly recently, this part of the brain was considered to be quite mysterious. By now, however, it has become clear that the neuronal populations of the frontal cortex are not just involved with the primary processing of light and sound stimuli, olfaction, taste, and tactile sensations, but mainly with so-called **executive functions**. When a human develops plans, makes judgments, forms intentions and transforms them into actions, then the frontal cortex is active. Besides this, the frontal cortex is crucial for **working memory**. This is a type of short-term storage that is required, for example, when people converse; to understand a spoken sentence, one must recall at the end what was said at the beginning. Such short-term memory is also required at a party, where one should remember who is already greeted, and who is yet to be greeted. More importantly, the frontal cortex checks the **processes** that occur on the way from intention to action, and recognizes **possible errors**. Even before you put sugar (instead of grated Parmesan cheese) on your spaghetti, your frontal cortex starts corrective measures and steers your hand away from the sugar and towards the Parmesan cheese.

That the frontal region of the human brain is involved with intentions, and their conversion into actions, is corroborated by people with **damage** to this region. They often act thoughtlessly and prematurely, often change plans, ignore important information, and often commit errors without noticing it. Unless motor areas are affected, these patients are not noticeably or visibly disabled. However, their behavior is often curiously **inflexible and environmentally dependent**: they have problems with planning actions, remembering action goals, and switching between action goals (Burgess 2000). The control of their actions appears to have shifted to the outside world, so to speak, so that the confrontation with objects leads to the execution of actions that are often associated with those objects: many patients smoke when they encounter cigarettes, drink when they see a glass of liquid, and grasp or manipulate objects without an apparent goal (see Sect. 3.1.2). Altogether, their long-term actions appear to be **less intentional**; they appear to lose interest in attaining specific goals,

and to merely respond to external stimuli. We will now discuss three particularly important areas of the (pre)frontal cortex and outline their significant contributions to human action planning.

2.6.1 Dorsolateral Prefrontal Cortex (BA9/46)

The **dorsolateral prefrontal cortex (DLPFC)** is connected with almost all areas of the human brain, especially with the basal ganglia, the hippocampus, and the temporal, parietal, and occipital cortices. Its mode of operation depends strongly on **dopamine**, which is delivered to the DLPFC through the so-called mesocortical pathway (that originates in the ventral tegmentum). The DLPFC is the human brain area that requires the longest time to mature (often up to young adulthood) and that degenerates particularly quickly through aging processes and is therefore responsible for many side effects of aging.

Two important, strongly overlapping **functions** of action control are ascribed to the DLPFC. According to an older interpretation (Goldman-Rakic 1987), this structure serves as **working memory** in the meaning of Baddeley (1986). This was based on the repeated observations from cell recordings in animal research, that neurons in the DLFPC are activated through use of the working memory. In a typical task, monkeys were shown target positions for eye or hand movements, but there had to be a delay between these cues and the actual movements—these target positions had to be kept in working memory. It was demonstrated that neurons in the DLPFC fired more strongly during this **retention** interval. Therefore, it would seem plausible that these neurons represented either the target positions themselves, or actively maintained other representations during the retention interval.

More recent interpretations suggest that the DLPFC is mainly involved in **cognitive control** (Miller and Cohen 2001). It represents the goals of cognitive and motor actions and actively supports all processes that are relevant for the execution of these actions. In other words, the DLPFC could be the **neural correlate of the human will**. In fact, a number of neuroimaging studies have shown that the DLFPC is activated especially during the preparation of a new task and also that the more difficult the task, the stronger the participant's will must be to complete the task. MacDonald et al. (2000) presented Stroop stimuli, that is, color words that are presented in incongruent colors (for example, the word "red" in a green color, see also Sect. 9.3). In numerous trials, participants either had to read the word, which ought to be a natural reaction, or name the color, which is a complex assignment, as in these cases, reading of the word has to be inhibited. The activation of the DLPFC clearly increased during the preparation for the upcoming trial, especially during the preparation of the difficult color-naming trials. When we assume that the DLPFC is necessary to actively maintain action goals and to guarantee them the necessary influence on the relevant cognitive processes, then it ought to be clear why frontal lesions lead to large deficits in prioritizing, organizing, and coordinating various actions (Burgess et al. 2000).

In a liberal interpretation, the two assumed functions of the DLPFC are not mutually exclusive, by the way. For example, the concept of working memory does not suggest that it is a type of container into which the contents of our thoughts are transferred. Much more plausible is the assumption that activation in the DLPFC leads to **maintenance of relevant representations** in the sensory or thought areas. The codes of the working memory are therefore not necessarily copies of representations from other areas of the brain, but merely **pointers** to these representations. Action goals can function in the same way, that is, they can indicate or refer to the processes that are vital for attaining a specific goal and thereby actively maintain them.

2.6.2 Orbitofrontal Cortex (BA10-14/47)

The neuroanatomical localization of the **orbitofrontal cortex (OFC)** is still under discussion (Kringelbach and Rolls 2004), and its borders and their relation to neighboring areas are viewed different by various authors; some authors consider the OFC and the ACC (Sect. 2.6.3) to be one area, and they refer to it as **ventromedial cortex**. The OFC receives information from all sensory systems, such as the hippocampus, the amygdala, and the cingular cortex, but also from other areas of the prefrontal cortex. It sends information to, amongst other areas, the striatum (where it might possibly influence dopamine production), to the amygdala, entorhinal cortex, the hippocampus, and the inferior temporal cortex.

On the one hand, the OFC is clearly involved in **affective processes**, and it is assumed that it plays a crucial role in the **association of stimulus features** and the rewards (or punishments) that are associated with them (Rolls 1999). On the other hand, the OFC is important for **action planning**. The first clues for this were provided by the extensive analysis of the brain lesion of Phineas Gage, which we discussed at the start of this chapter. You will remember that the frontal cortex of Mr. Gage was pierced by a chisel during an explosion. Fortunately, both Gage's skull and the chisel have been retained, which allowed Hanna Damasio and colleagues (1994) to reconstruct the injury exactly in a computer simulation.

According to the reconstruction, the OFC was particularly damaged, which suggests that the difficulties Gage had in planning may well have been caused by the lesion of this brain area. This raises the question of to what extent the OFC is involved in action planning, and how this involvement can be united with the role the OFC plays in affective processing. The explanation of this association rests on the assumption that action planning involves **distinguishing** between alternative stimuli (Rolls 1999) or actions (Damasio 1994) and that these distinctions are made on the basis of the reward someone is trying to obtain. Damasio (1994) assumes that every action is associated with a representation of its **affective consequences**. This way, we learn what it "feels like" (or would feel like) to perform a specific action. These so-called **somatic markers**, that is, the representations of expected affective bodily reactions, allow for relatively fast, often also intuitive decisions; one simply chooses the action that "feels best." More recent studies of patients with OFC damage support this

notion. These patients especially have problems with making risky decisions, when compared with healthy persons or people with lesions of other brain areas (Bechara et al. 1998); also, these patients do not demonstrate the typical sweating response prior to making a very risky decision that is common in healthy persons.

2.6.3 Anterior Cingulate Cortex (BA24)

The name of the cingular cortex (*cingulum* is Latin for belt) is derived from the fact that it wraps around the corpus callosum (the connection between the two cortical hemispheres) like a belt. It is part of the **limbic system** that plays an important role in the emergence of emotions and the regulation of memory and behavior. The ACC receives afferent signals mainly from thalamic nuclei and sends efferent signals to other areas of the prefrontal cortex, the anterior nucleus and other limbic areas. The ACC is thought to be important for **monitoring action control**, for which it collaborates closely with the DLPFC. Evidence from Botvinick et al. (2001) suggests that the ACC registers conflicts between stimulus and response alternatives, and strengthens the representations of the action goal in the DLFPC (Sect. 9.3). This **strengthening of a target**, in turn, leads to **increased attention** to the information relevant to the action (Egner and Hirsch 2005).

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