Apraxia and the Neurophysiology of Motor Control

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Apraxia and the neurophysiology of motor control

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Liepmann's description of apraxia still serves as a point of reference for neurological thinking about higher motor function. The extent to which this model is accessible to interpretation in the light of current neurophysiological knowledge has been examined. Planning and programming processes have been distinguished, and the separate roles of striopallidal and cerebellar loops in the generation of these processes have been considered in relation to the contribution of parietal and frontal association areas. It is emphasized that a new taxonomy of action is imperative in order that neurophysiological and psychological approaches can be correlated in the study of higher motor function and its pathological disorders.

1. Introduction

In a colloquium on the motor aspects of behaviour, Teuber (1974) remarked, 'we would probably divide the audience into those who prefer working from the ground up . . . and those who would rather work from the top down; and some of us, perhaps most of us, really believe one has to do both. I, for one, have always felt that we must keep the highest achievements of motor organisation before us . . . if we want to come to terms, properly, with more elementary ones. Besides, complexity and simplicity are relative affairs.'

This approach recognizes that each scientific discipline advances at its own pace, in accordance with its theoretical models, its methodology and its instruments, defining its objects of study as it apprehends them. Physiology, for example, traditionally works 'from the ground up'; neurology generally starts at the top. These contrasting perspectives immediately raise the problem of interdisciplinary convergence with the inevitable gaps that occur and that are progressively bridged by the continually developing neurosciences.

The physiology of movement has now expanded impressively into contemporary studies of the neurobiology of motor control. Recent reviews (Evarts 1975; Jasper 1975; Brooks 1979) illustrate the growing need for this sector to develop new theoretical models that can accommodate the continually expanding body of experimental facts and that can suggest new ways of tracking the functional capacities of the nervous machinery.

For its part, the neurological disorders of motor functions have generally found a natural articulation with pathophysiology at the lower levels of the hierarchy of motor regulation. At the higher level, however, until very recently, it provided no more than a crude, functional frame of reference for the interpretation of the findings of physiological research. Conversely, the clinical categorization of higher motor disorders calls for a neurophysiologically based approach to the study of the underlying neural processes. Let us consider first the contribution of neuropathology to the understanding of higher motor functions.

2. THE NEUROPATHOLOGY OF APRAXIA

Apraxia offers an illuminating example of the impact of disordered functions on the study of mechanism and their eventual localization in nervous structures. Liepmann's remarkable description of apraxia still serves as a point of reference for any attempt to define the operations that intervene between the mental representation that a subject has of his body and of the physical world that surrounds it, and the intentional triggering of an appropriate action directed within the framework of that reality. Liepmann's thinking, inspired in part by his predecessors (see the recent comprehensive review by Signoret & North (1979)) and to a large extent by his own acute analysis of a single case (Liepmann 1900), invoked concepts that are still relevant to neuropsychological and neurophysiological interpretation. Three main issues will be considered here.

(a) *Voluntary and automatic control of action*

First, there is the crucial distinction between the automatic and the voluntary, described by Jackson in 1866 and illustrated by those patients who have lost the ability to carry out certain intentional acts in response to a command whereas they can produce them automatically in the context of natural usage. The specificity of the voluntary and deliberate, as opposed to the automatic, act remains a powerful challenge to physiological research.

(b) *Command of gestural activity: a serial model*

The strength of Liepmann's systematic analysis of apraxic disorders lies in its proposal of a functional serial model of gestural activity on three levels (see figure 1). These are as follows. (i) A veritable repertoire of 'kinetic formulae', acquired by experience and 'engrammed' in the premotor region where selective destruction could amputate the action of a well defined element in this repertoire: this is *motor apraxia*. (ii) Transactional activity, mobilizing 'kinetic formulae', under the influence of an idea, of an 'image of the act' to be realized: *ideomotor apraxia*. In this condition, a correct 'image' of the action is preserved and an intact repertoire of 'kinetic formulae' (that can be mobilized appropriately in a familiar context) is available but the patients are unable to release the action intentionally. The parietal area is assumed to be directly implicated in this transactional activity. (iii) A mental representation (schema) of the action, which includes an operational plan of the succession of movement sequences implicated in complex activity: *ideatory apraxia*.

How far is this model accessible to interpretation in the light of current neurophysiological knowledge?

(c) *The diversity of apraxic disorder*

The nosology of apraxic syndromes requires a taxonomy of motor activities that can be analysed in functional and neuroanatomical terms.

Two main disorders were recognized early, and later confirmed as the most frequently encountered. The first concerns the incapacity to use correctly a familiar object; it was initially described as 'apraxia' but later as 'apractognosia' (Morlaas 1928), thus reflecting the intrication of gnostic and praxic function and the difficulty of identifying a pure apraxia. The second disorder involves communicative gestures of which the status resembles that of the conventional signs of language. The term 'motor asymbolia' was proposed as equivalent to sensory asymbolia (agnosia) but in the motor sphere. It assumed the loss of some kind of 'memory images of movement' necessary for the intentional evocation and initiation of movement otherwise

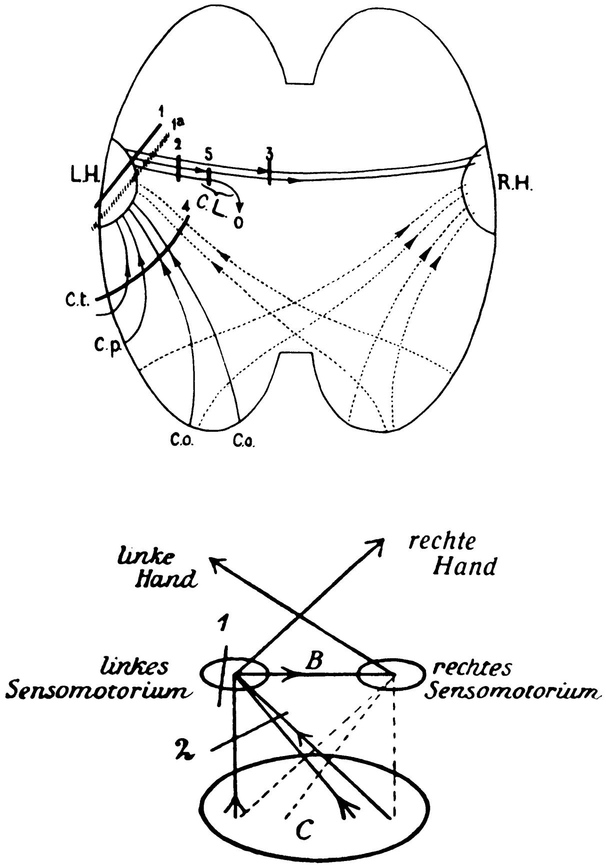

Figure 1. The first horizontal schema of Liepmann (1900) is presented on the lower figure. A lesion in the left 'sensomotorium' (1) gives rise to a left hemiplegia and aphasia and to a motor apraxia of the left hand, due to the absence of information linking the left and right 'sensomotorium' via callosal connections (B). A lesion disconnecting the cortical areas (C), where kinaesthetic images are engrammed, and the left sensomotorium leads to an ideomotor apraxia of the right hand. The upper sketch presents the second horizontal schema of Liepmann (1920), which is more elaborate. Lesions resulting in a dyspraxia of the left hand may be localized in the cortical motor representation (1) in the left hemisphere (L.H.), by interruption of callosal connections (2, 3) with the right hemisphere (R.H.), or by interrupting connections (4) between occipital (C.o.), parietal (C.p.), temporal (C.t.) association cortex and the motor cortex of the left hemisphere. The right hand is paralysed after lesions at 1 and 2, whereas a melokinetic apraxia is produced by a less severe lesion at 1a. A lesion at 4 causes an ideomotor apraxia and more diffuse, posterior lesions produce ideatory disorders. A lesion of the internal capsule (5) leads to paralysis of the right hand without dyspraxia of the left hand.

automatically mobilized in another context. The crucial role of the left, 'speaking', hemisphere in these disorders was clearly recognized by Liepmann and amply confirmed by later studies.

Into this preliminary nosology of the apraxias, however, we must incorporate additional disorders: the so-called 'specialized motor apraxias' (concerned with the loss of a very specific acquired skill like writing or playing a musical instrument) and the more frequently described anarthria and bucco-facial apraxia, all involving lesions of the premotor areas; locomotor apraxia, also associated with frontal lesions; dressing apraxia, characteristically related to posterior lesions of the right hemisphere; and constructional apraxia, often occurring with posterior lesions of either hemisphere but more frequent and perhaps more severe in right-sided lesions. Thus, clinical data identify specific motor disorders, linked with known lesions. These in turn can be analysed in relation to concepts of hemispheric asymmetry of function, for example the putative role of the left hemisphere in the control of action sequences and that of the right hemisphere in monitoring spatial relations. The heuristic value, for neuroanatomical and neurophysiological research, of this functional individuation of praxic disorders remains to be explored.

3. The contribution of neurophysiology

Contemporary research in the physiology of movement is now providing detailed knowledge of the internal working of a motor machinery of which the general organizational principles were clearly recognized, a century ago, by Jackson (1932). The 'ground up' approach of classical neurophysiology works from the study of combinations of elementary units, at the lowest level of organization, to that of the higher-order functional ensembles. A major change, however, in the concept of central motor control (Evarts 1975) involves a clear and progressive shift away from the tendency to think in terms of control by individual components, towards a viewpoint already stressed by Jackson as 'the relations of different divisions of the central nervous system to one another and to parts of the body'. In addition, the influence of cybernetic control models and of information-processing theories has provided new paradigms for experiment and interpretation of data.

Within this framework, neurophysiology has a contribution to make, based on a general sketch of the organization of motor control.

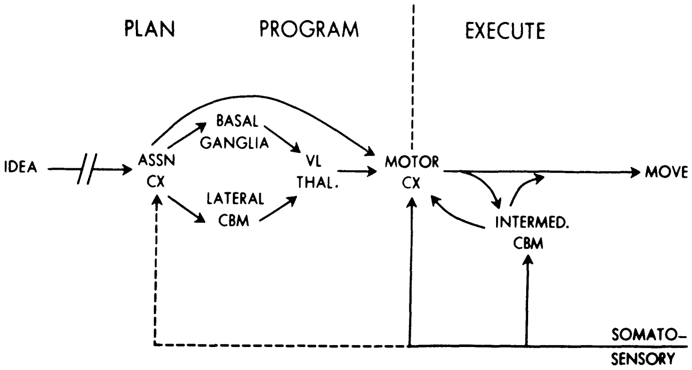

Figure 2. Proposed sequence of events in initiating movement. Schematic flow chart of nervous command. See text for details. (From Allen & Tsukahara (1974).)

(a) *A schema of motor control*

A schema that reflects much contemporary neurophysiological thinking and research on motor functions was developed by Allen & Tsukahara (1974). Basically it represents a three-stage model of control (figure 2).

(i) The *executive level* at the lowest stage is controlled by the motor cortex with the contribution of the palaeocerebellar loop (pars intermedia) to update the evolving movement. This updating is based on the motor command and on the proprioceptive description of limb position and velocity in accordance with the contextual requirements of postural adjustment. In addition to the numerous reflex loops from the spinal and mid-brain levels, a direct follow-up correction can be performed by the motor cortex itself.

(ii) At the second level, we find the *planning and programming* of the activities of the motor cortex. Cortical association areas (mainly frontal and parietal) contribute to this operation through two main loops. The first loop involves the cerebellar hemispheres (which compose about 90% of the human cerebellum): it receives an impressive number of input fibres (20 million) from most association areas but including mainly the motor cortex itself (area 6 and area 4) via the pontine nuclei and inferior olive; and it sends its output back to the motor cortex via the ventrolateral nucleus of the thalamus. Such a high-speed loop is thought to be involved in the initiation of intentional commands. The second loop involves the basal ganglia which, like the cerebellar hemispheres, do not receive proprioceptive input and receive most of their inputs from extensive association areas (frontal, parietal and temporal regions) as well as from area 4 (via the substantia nigra) and from the midline thalamic nuclei and limbic structures. According to this schema, both the cerebellar hemispheres and the basal ganglia are involved in planning and programming the activation of motor cortex rather than in the regulation of the evolving movement.

(iii) Finally, at the third level, we search for the events that precede planning and are presumed to influence (still mysteriously) the activity of association areas. In the schema, they are represented by the term 'idea' with a characteristic blank that interrupts the arrow pointing from 'idea' to association areas, an indication of the perplexity of motor physiologists in this largely untouched field of higher cerebral functions.

Fortunately, however, the apraxic disorders described in Liepmann's schema seem to involve mainly, if not exclusively, the second level of the physiological schema, although the rather vague concept of 'idea' may contain at least part of the story, as we shall see later. Let us now try to interrelate the basic issues of Liepmann's schema and current neurophysiological knowledge.

(*b*) *Kinetic formula versus motor program*

The concept of 'kinetic formulae' conceived of as motor engrams composing a repertoire of building blocks, from which the organism must draw for its performance, is clearly an important component of the notion of *motor program* widely used by neurophysiologists (see Brooks 1979). Such a repertoire of available prewired neuronal circuits does undoubtedly exist. Since Hess's pioneering work, it has been frequently demonstrated that electrical stimulation at specific 'nodal' loci in the brain stem and medulla structures (the command neurons of invertebrate physiology) effectively triggers complex and well organized movements belonging to the basic behavioural repertoire of the species (locomotion, orientation, and alimentary, communicative or defensive behaviour). Such wired programs can be called up and run without the assistance of the sensorimotor loops. Without entering here into the semantic difficulties (see MacKay 1980) posed by the term 'program' borrowed from the vocabulary of computer science (where it describes precisely the 'soft' set of instructions that is not inscribed in the 'hard' wiring), it may be noted that the very ambiguity of this new concept enriches its heuristic value for contemporary neurophysiological thinking (Young 1978; Arbib 1981).

The 'labelled lines' coding – in computer terms, the 'addressing' of instructions – is of paramount importance in the organization of nervous activity. The genetic prewiring of the basic circuitry of inborn activities may rightly be considered as an expression of anticipatory 'programs of action'; program means literally 'written in advance'. Most of the programs studied by physiologists belong to this category of prewired inborn circuits that are usually untouched by apraxic disorders. 'Kinetic formulae', however, belong unequivocally to the class of learned programs. Are then learned programs engrammed differently? Learning new motor skills may proceed by a structural reorganization of existing wired programs, by recombination of them or by organization, *de novo*, of new patterns of connectivity. The domain of motor learning is still relatively unexplored by neurophysiology although widely scanned by behavioural studies (cf. Paillard 1978).

(i) *The role of the pyramidal tract*

An intriguing question concerns the role of the pyramidal tract in the channelling of such new programs (if cortically engrammed) to the executive level of subcortical structure (cf. Paillard 1978). Classical observations have shown that normal motor habits or newly acquired conditioned responses are unaffected by pyramidotomy (cf. Wiesendanger 1969). Some interesting experiments by Ioffe (1973) with dogs showed that only those conditioned motor responses that were compatible with the inbuilt repertory of posturo-kinetic activities were preserved after pyramidotomy. We do not yet know whether long practice of a newly acquired motor activity, requiring a remodelling of programmed circuits at subcortical levels, could produce a stabilized, automatic response that would survive pyramidal section. It has recently been shown that a lesion of the ventralis lateralis (the thalamic relay for outputs of the strio-pallidal and cerebellar loops to the motor cortex) prevents the learning of a visuomotor placing task in cat, whereas the same lesion, performed after acquisition, does not interfere with performance (Fabre & Buser 1980).

It has, however, been observed in man after transposition of muscle tendons of the lower limb, which disrupts primitive patterns of coordination of synergistic units at the spinal level, that the newly learned pattern of muscular activity is, after training, performed correctly by the patient only when under voluntary control. The old pattern, now unadaptive, inevitably reappears in automatic uncontrolled walking (cf. Paillard 1960).

(ii) *The triggering of learned programs*

The next question concerns the possibility of triggering such learned programs by electrical stimulation at an appropriate site. Penfield & Rasmussen (1950) stressed the fact that electrical stimulation of premotor areas (the putative site of kinetic engrams) – in either awake man or monkey – has never activated the engrams of learned, organized skilled acts such as writing or speaking, as might have been predicted from the existence of local wired circuitry. Only crude unorganized local movement or inarticulate sounds were obtained. Command neurons, or at least command nets, controlling such programs, as in inborn programs, are clearly not present in this area. The activation of such newly acquired circuits seems to require a particular configuration of patterned input that artificial electrical stimulation is unable to reproduce (see a review by Paillard 1960). The identification in parietal association areas of neurons related neither to sensory nor to motor function but involved in some kind of 'command function' (Mountcastle 1976) might offer a way of tracking the origin of the flow of command signals that mobilize elsewhere the engrams of a learned motor act. These neurons, however, do not represent in any way an equivalent of the 'command neurons' identified in invertebrates (Kupferman & Weiss 1978).

Alternatively, we might suppose that learned programs are not prewired ready to work like inborn programs but that they have to be built afresh when invoked by programming processes whose precentral structures would only be the relay station for their channelling to the cortical motor keyboard.

(*c*) *The link between association area and motor cortex*

This is the next important issue of Liepmann's schema. The symptomatology of *ideomotor apraxia* seems to involve specifically some transactional activity linking an intact 'representation of the act' (motor image) with an existing 'kinetic formula'. What could be the neural support of such a transactional process?

(i) *Cerebellar versus striopallidal loops*

Returning to the physiological model of motor control, one crucial feature emerges. The most obvious and important connections between cortical association areas and motor cortex seem to be through the striatal and the cerebellar loops. Both are known to take part in the planning and programming of action. Space does not allow a detailed account here of these two loops that have attracted (especially the cerebellar loops) a considerable amount of research during the last decade, although their functions still remain largely conjectural (Massion & Sasaki 1979).

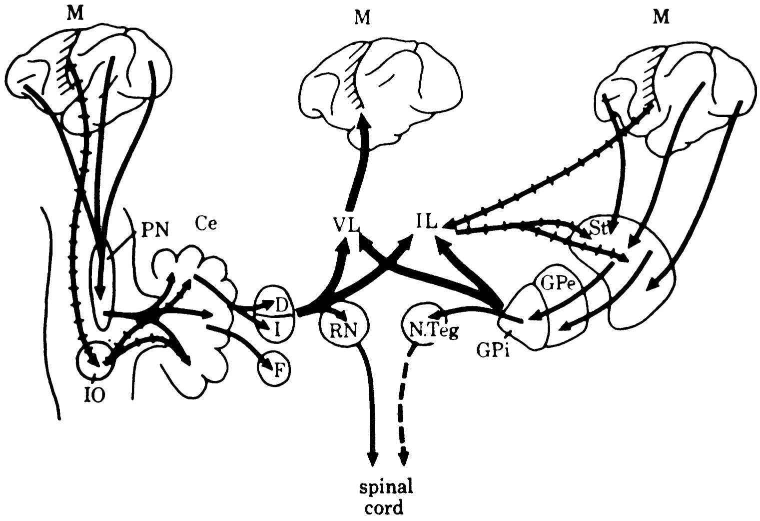

Figure 3. Diagram from Kemp & Powell (1971) illustrating the two re-entrance loops connecting cortical association areas to motor cortex (M) via subcortical structures: on the left, the neocerebellar loop involves the position nuclei (PN), the inferior olive (IO), the lateral cerebellum (Ce), the dentate (D) and interpositus (I) nuclei projecting to the motor cortex via the ventro-lateral (VL) thalamic nucleus, and down to the spinal cord via the rubrum (RN); on the right, the striato-pallidal loop includes the striatum (St), the external segment (GPe) and the internal segment (GPi) of the globus pallidus projecting to the motor cortex via the ventro-lateral nucleus (VL), and down to brain stem structures via the tegmentalis nucleus (N.Teg). Both loops converge into the thalamic intralaminar structures (IL) that, in turn, project to the striatum. See comments in the text.

Both loops contribute to neural events preceding the executive part of action and channel their output mainly to premotor areas (area 6). Both receive their largest afferent inputs from cortical association areas and are relatively 'open loop' as to their direct afferentation by proprioceptive input, in contrast to the palaeostructures of the cerebellum involved at the lower stage. Some contrasting features should, however, be emphasized forthe understanding of their specific contribution to the transactional activity assumed to be disturbed in ideomotor apraxia.

First, the high-speed running of the neocerebellar loop has to be compared with the slower processing of the neostriate loop. It has been claimed that the former loop chiefly contributes to the updating of evolving movements (Eccles 1967) initiated by the motor cortex and more specially those concerned with skilled hand-use, including upper limb movements and their associated postural adjustments required for the positioning of the hand in visual space (Massion & Sasaki 1979). Activity in the dentate nucleus, just before pyramidal tract discharge, has been interpreted as reflecting a pretuning of the components of the loop, preparatory to its subsequent intervention in updating the running of a program released by the motor cortex. Cerebellar neurons do not discharge in pronounced fashion during the fore-period of a planned movement in a delayed task (Strick 1978) or in simple tasks, whatever their speed of execution (Tach 1970*a*, *b*). In contrast, basal ganglia neurons discharge about as early as those of the association cortex during the fore-period of a task (Soltysik *et al.* 1975). On that basis, and considering many other data, one would assign to the cerebellum a relatively 'downstream' place in the hierarchy of the motor-programming process (Brooks 1979).

Moreover, contrary to earlier views, it has been established by the current, more sophisticated, neuroanatomical tracing methods that the great majority of the numerous fibres composing the cortico-pontine tract (that feeds the neocerebellar loop) originate from motor and premotor cortical areas (Wiesendanger *et al.* 1979). In contrast, the main input of basal ganglia, in addition to the numerous small fibres connecting it to widely extensive association areas, originates from the median and intralaminar nuclei of the thalamus (see figure 3) and from limbic structures, thus linking them to the stream of impulses that are presumed to initiate action in the context of interests, values and needs.

It has recently been suggested that the basal ganglia are involved in higher-level cognitive activity (Öberg & Divac 1979), and reexamination of Parkinsonian disorders (Marsden 1980) has suggested that the basal ganglia are involved, in the automatic and selective calling-up of learned motor plans although there is no evidence that motor plans are stored in the basal ganglia. Similarly, despite theoretical (Marr 1969) and numerous experimental efforts to elucidate the contribution of cerebellar structures to the motor-learning process (Gilbert & Tach 1977), its role as a depository of such learned motor programs has not been compellingly demonstrated; clinical teaching suggests that cerebellar patients suffer neither the loss of motor memory nor the loss of the ability to learn new motor tasks within the limits of their impaired execution (Holmes 1939). We therefore have to distinguish clearly between inbuilt programs and a programming and planning process.

(ii) *Programming and planning*

Two lines of approach may help to clarify this issue.

(1) *The computer science approach.* Several levels of program sophistication, derived from studies of artificial intelligence, may be envisaged (Arbib 1981). *Programs*, written in the 'machine language' directly controlling basic computing operations, are certainly comparable with the basic prewired circuits at the lowest level of nervous organization that can produce, given appropriate input, a predictable output. They can execute stereotyped, predetermined sequences when triggered 'straight-line' without the assistance of a regulatory loop. They can also exhibit some flexibility of execution when incorporated with on-line testing of internal (postural state) and external (resistance to overcome) constraints. This is obviously the role of the palaeocerebellar loop and of the numerous servo-assistance loops of the spinal machinery. The hierarchical organization of lower-order into higher-order programs is now well recognized in the functional working out of the nervous system, as in computing technology, in order to

reduce the number of degrees of freedom to be controlled for robot manipulation. An explanation of this hierarchy may be sought in Sherringtonian terms – an anatomically based linkage of reflex neuronal circuits – or within the framework of functional 'coordinative structures' (Bernstein 1967; Gel'fand *et al.* 1973). On this choice will depend the explanatory hypotheses that must accommodate the fundamental adaptive properties of machinery that shows a remarkable capacity to bring back its basic executive unit within the narrow range of its regulative feedback operations after a shift outside prompted by the requirements of an intended action. The concept of 'coordinative structures', despite its theoretical appeal (Turvey *et al.* 1978; Kelso & Tuller 1981), still emerges at too abstract a level for direct linkage with neural analysis.

A first range of adaptive capacity certainly exists at this basic level of programmed units by means of *feedforward adjustment* involving cerebellar mechanisms; several experimental models are now being extensively studied (e.g. adaptation to a load, change of gain in the vestibulo-ocular reflex, anticipatory postural reflexes). A greater range of adaptive possibilities appears with *executive programs* that run a sequence of synergic actions of which the composition and ordering may depend on the on-line testing of internal and external requirements. The learned programs controlled by the neocerebellar loops seem to belong to this category. Whether they involve, as in computer technology, a 'high-level' language that has to be translated into the 'machine language' of the lower stage remains to be clarified. It is at an upper stage of computation that we encounter planning operations.

Planning represents, in computer technology, a higher-order process. It covers operations designed to find a solution tailored to the current situation and involving context-dependent interpretation. The solution is expressed only in general terms and not as a detailed specification of the executive program to be released (Miller *et al.* 1960). In this sense, planning does depend on a higher-order program not directly translatable into machine language but requires a programming process taking into account internal goals and external circumstances and generating 'plans' for the coordinated control of existing executive programs. As Arbib (1981) has emphasized, one of the aims of future research must be to distinguish more effectively the planning operations within cortical structures and to understand the signal flow that this planning must impose upon regions that modulate it. This leads back to questions as to the possible role of the basal ganglia and of the association areas in the planning process of learned actions. An experimental approach to this problem, however, requires an operational definition of the structure of plans.

(2) *The approach of experimental psychology*. The basic requirements of planning are, first, the selective choice of the set of executive programs to be coordinated to achieve the intended goal and, second, fixing the order and the agenda of their activation. The study of apraxia suggests that these requirements may be independently disordered by pathology: the ideomotor syndrome involves mainly the first and the ideatory syndrome the second condition.

The hope of linking planning operations with experimentally testable neural operations certainly requires a better understanding of its chronology and of its possible analysis into separate component suboperations. Here the methods of cognitive psychology, derived from information-processing models, make their contribution. Assuming that planning is not directly involved in the embodiment of executive commands at the lower level of motor organization, we can consider its operations as intervening before the invocation of executive programs by the decision to act. In fact, a performance can be delayed in its execution but nevertheless leave the subject ready for the occurrence of a trigger signal. In other words, planning can be conceived of as staged operations that resolve into the so-called 'preparatory set' of psychologists; it corresponds then to some 'readiness to act' in accordance with a predictable trigger signal or the anticipated sensory consequences of the intended act, and in conformity with a selected strategy.

Thus plans deal with a relatively predictable future, at various timescales and with various contextual frames. The precision of the plan is then closely related to the degree of uncertainty foreseen by the specific requirements of organizing action. Psychological experimentation is now directed to the relation between this uncertainty and the rapidity with which an action is performed, in an attempt to specify the limiting factors of the planning process. The many provisional specifications from which the plan is composed take into account predictions concerning the time and place of the intended action, the appropriate strategies, the environmental constraints to be overcome by pretuning of feedback loops, and the sensory consequences of the activity required to stop action or to change the plan (A. Semjen, unpublished 1978). A segmentation of the provisional components of the plan along these lines might provide both a useful framework for future neurophysiological investigations that are matched with behavioural tests (Requin 1980*a*) and a contribution to the nosology of pathological disorders (see figure 4). In fact, contemporary neurophysiological debate is of relevance in this context.

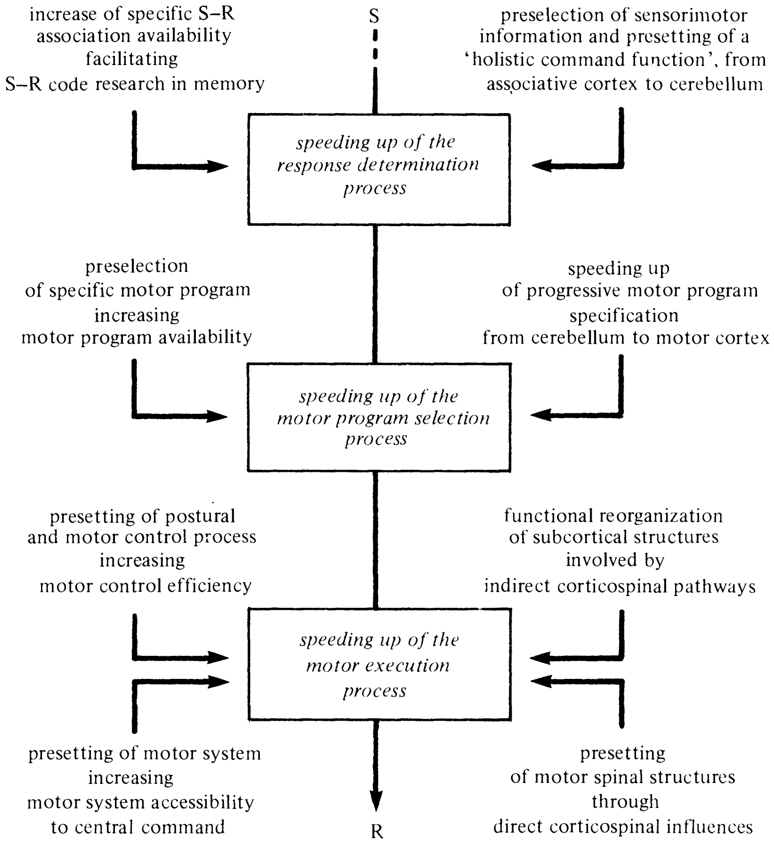

FIGURE 4. Organization of the preparatory processes in a three-stage serial model of motor activity. Consequences of the preparatory process are indicated for each of the three stages in the central frame of the figure. Corresponding hypothetical mechanisms are suggested on the left at the psychological, and on right at the neurophysiological, level of analysis (Requin 1980*b*).

(iii) *Role of the basal ganglia in the planning process*

The functional role of these structures is still a matter of controversy, and many aspects of the physiological organization of the striatum are still in doubt despite extensive research during the 70 years that have elapsed since the basal nuclei were first recognized as being implicated in movement disorders (Wilson 1932).

Although pharmacological, biochemical and histological work has contributed much to our anatomical knowledge of the input and output connections of the basal nuclei and also to the alleviation of Parkinson's Disease, it has so far provided little information about the intrinsic cellular organization of this structure of which the complexity, in contrast with the highly regular architecture of the cerebellum, is more comparable with that of cortical association areas. Classical electrophysiological and ablation studies have failed to reveal the basic functions of these cortical areas; the increasingly used technique of chronic extracellular recording from awake, freely moving animals, has been more productive.

Single-cell recording from neurons in different areas of the striato-pallidal complex yield several new lines of evidence. Data obtained from the globus pallidus and substantia nigra, which receive the main efferent projections of the caudate nucleus and putamen and are therefore assumed to give a good picture of the functional output of these structures, show that the activity of a number of these neurons is related to identifiable movements made by the alert monkey. It provides clear support for the view that one function of neostriatal neurons is in the control of movement (Marsden 1980).

From a comprehensive review of the available data and from their own investigations of the activity of neurons in the neostriatum and related structures in the alert animal, Rolls *et al.* (1979) have tentatively put forward the following hypothetical account of the whole chaining of activity. Neurons at the first stage of processing are related to sensory information received from many cortical areas and concerning events in the environment. A second large population of neurons selectively detects those events that are relevant to the animal's performance. Whether this selection is intrinsic to the neostriatum or performed elsewhere is, as yet, unclear. A third category of neurons has the function of 'enabling' the animal and preparing it to respond. 'These neurones would be activated via the preceding neurones by those events which are cues for the performance of the particular task in which the animal was to become engaged, and would be deactivated when the task is complete. They are task-related and not strictly related to a specific movement.' Finally, a fourth set of neurons, mainly found in the globus pallidus and substantia nigra, are definitely movement-related, dealing mainly with arm and head positioning and related chiefly to the grasping of external objects by hand and mouth (Rolls *et al.* 1979). 'They may be concerned with the initiation and execution of motor responses.'

This account is certainly consistent with clinical data and also with current hypotheses as to the functions of the basal ganglia. Three such notions are of special relevance for the attempt to trace the nervous substrate of planning processes.

First, Denny-Brown & Yanagisawa (1976) have interpreted the deficits produced by neostriatal damage in terms of 'the absence of the activating set, preparatory to motor performance oriented to the environment', in accordance with an earlier view (Denny-Brown & Chambers 1958) comparing the basal ganglia to a 'clearing house' that accumulated samples of continuing cortically projected activity and that could, on a competitive basis, facilitate some while suppressing the remainder. Second, Marsden (1980) has recently developed the idea that the basal ganglia are chiefly responsible for 'the automatic execution of learned motor plans', making a clear distinction between plan and programs, the latter being preserved in Parkinson's disease; the patient shows a specific defect in executing simultaneous or sequential motor programs and is unable to initiate predictive action (e.g. fast ballistic movements). Third, Cools (1980) has underlined the fact that experimentally induced dysfunctioning of striatal structures in animals 'alters the assessment of the relative priority of self-generated and externally produced constraints on the selection of new behavioural patterns, without affecting the sensory, motor and sensorimotor capacities required to perform the behavioural patterns themselves': a view consonant with earlier observations by Gybels *et al.* (1967).

It is interesting that the defects shown in animals, which influence the ordering and sequencing of events before the programming of motor patterns, are analogous to those occurring in patients with Parkinson's Disease. These patients are impaired in the ordering and sequencing of simple motor acts and, interestingly, are reported to show defects in the sequencing of concepts at the cognitive level. If confirmed, such findings represent an important advance in our understanding of the functions of the basal ganglia. Whatever the future development of this new and exciting field of research, the data so far point to the basal ganglia loop as one of the main channels linking cortical association areas to motor structures and involving them directly in the planning process. It follows that the specific contribution of frontal and parietal areas can only be understood in relation to operations processed at this level, for which they probably provide the relevant input.

(iv) *Parietal versus frontal association areas*

The involvement of these association areas in the anticipatory process, which is part of the planning of action, is attested by characteristic changes in the electrical activity of the brain during voluntary action. The study of event-related potentials offers extensive new opportunities to investigate brain activity in relation to sensorimotor and cognitive function (see Desmedt 1979). A slow negative wave, developing over the frontal region and expanding widely to the parietal areas, has been described in man during the period before the spontaneous initiation of a voluntary movement (Kornhuber & Deecke 1965). It is followed by premotor and motor potentials just preceding the first signs of electromyographic activity. The so-called 'readiness potential' begins as early as 1 s before overt motor activity. It is correlated with the laterality of the limb used and with the force specifications of the act. The origin – neural, glial, or vascular – of the underlying mechanism is still controversial. The functional significance of this wave for the planning process is not yet fully understood. The slow wave, however, clearly correlates with unit activity recorded in frontal and parietal areas, thereby marking the contribution of these regions to the evolving process.

The parietal area is the main cortical region where lesions may be associated with ideomotor apraxia. If we assume that the basal ganglia are involved in a process of preselection between optional strategies, we are then led to postulate that the parietal area may contribute to the initiation and orientation of this preselection. Accordingly, we would expect association areas to be involved in the integration of the multimodal sensory information necessary for the elaboration of the plan of intended action (the morphosynthesis of Denny-Brown (1966)).

The posterior parietal association cortex of monkey consists of Brodmann's areas 5 and 7. Its rostral part is mainly concerned with the integration of somatosensory and proprioceptive information relating the relative position of body segments to their movement, whereas the posterior part is dominated by the integration of visual information about events located in the external environment (Hyvarinen 1982). Clinical observations, experimental lesion studies in animals, and recent detailed research on single cell activity (see Lynch 1980) indicate that the parietal lobes may form a substrate for a complex interface between sensory and motor function under the control of attentional gating mechanisms, in which motivational state and past experience play a role.

Area 7 seems to be involved in the selection of perceptual cues relevant to the intended goal. This selection is oriented by limbic information (Mesulam *et al.* 1977) about the internal state and motivational incentives of the organism. Whether the matching function between the significant cues and the motor process is located in this area is unclear. Movement-related cells in this region, however, show activity that is not closely related to the parametric details of the associated movement. The eyes, the hand and the mouth are the main collectors of information, and the last two are the main effectors of skilled movement in primates. Movement-related cells are chiefly concerned with the activity of these three organs directed within the proximal visual space and over the body surface. Arm projection, manipulation of objects, mouth prehension, eye fixation, eye tracking and saccadic eye movements are correlated with specific single cell activity, provided that the monkey directs his interest to the relevant cues (visual or tactile). Visuo-manual reaching, hand-to-mouth or hand-to-body activity, and orientation of the eyes to visual or tactile cues indicate the specific, although not clearly topographic, organization of this area for monitoring such specific sensorimotor activities (Hyvarinen 1982).

These data are consistent with clinical observations of the sequelae of lesions in this area, including optic ataxia, oculo-motor apraxia, disorders in manipulating familiar objects, dressing apraxia, as well as the classical attentional deficit in visual and somaesthetic modalities known as sensory neglect. The assessment of the behavioural relevance of the perceptual context and of the location of the stimulus in extrapersonal space appears to be a main function of the parietal region. As summarized by Mountcastle (1978*a*), 'the parietal lobe, together with the distributed system of which it is a central node, generates an internal neural construction of immediately surrounding space, of the location and movements of objects within it in relation to body position, and of the position and movements of the body in relation to that immediately surrounding space. The region appears in general to be concerned with continually updating information regarding the relation between internal and external coordinate systems.'

These properties are clearly associated with the posterior right hemisphere in man and account for early neurological statements concerning the importance of the integrity of a body scheme that provides our acts and our perceptions with the special frame in which they acquire their spatial organization.

The frontal area may share with the parietal region many common features although it differs in some important respects. First, it is worth noting the important cortico-cortical linkage between both areas. The arcuate sulcus is the main frontal area reciprocally connected with areas 5 and 7. Although not as extensively studied as the parietal region, unit recording in this arcuate region reveals the presence of movement-related units belonging to the same category as that found in the parietal region, with a special emphasis on eye-movement control in area 8. Like the parietal area, the frontal zone receives extensive projections form the limbic structures and is similarly modulated by selective attentional processes. Unilateral neglect after lesions in the arcuate region in primates is not unlike that produced by lesions of multimodal parietal association areas in man.

In contrast, the spatial context of motor organization, which is so characteristic of parietal function, is not so directly involved in frontal function. If the selection of spatial perceptual cues appropriate for the intended motor action is the main function of the former, then it would seem that a selection of purpose, on the basis of biological or higher-order priorities, characterizes frontal function. Thus, monkeys with bilateral frontal damage do not respond efficiently to reinforcement and thus are prevented from guiding their behaviour in terms of reward and punishment. It would therefore be interesting to have more data about the response of parietal monkeys to reinforcement. It has been reported, however, that parietal units are not reward-dependent (Perret *et al.* 1978).

Studies of nervous activity in the frontal areas in the unanaesthetized animal have only recently been undertaken. From an experiment using sensory matching with delayed lever-press, it was concluded that 'the prefrontal cortex is related to a sensorial attention mechanism to the visual stimulus which enables correct choice of the behaviour to be rewarded' (Kubota *et al.* 1980). Another study, based on delayed conditional discrimination, led to the suggestion that 'prefrontal units may be related to the meaning of the stimulus independent of its physical properties' (Watanàbe 1981). In addition, the tight anatomical connections between frontal, hypothalamic and limbic structures, combined with the clinical and physiological evidence of the effects of frontal lesions, support the view that frontal structures perform 'the environmentally related "gating" of diencephalic innate behaviours' (Damasio 1979) under the control of acquired, higher-order values.

Both frontal and parietal association areas afferent the neostriatum. Task-related cells identified in this region, and visuo-motor disorders associated with its lesions, are clearly related to parietal function. But the main symptomatology of neostriatal lesions presents a closer similarity with that of the frontal lobes. The original findings of Jacobsen (1935) still dominate this area of study: the delayed response and the delayed alternation tasks are impaired in animals after frontal as well as caudate lesions. If the former is clearly linked to an impairment of sustained attention (the characteristic distractability of some frontal patients), the latter refers more closely to a difficulty in overcoming an established response (changing the goal). This is reminiscent of the perseveration exhibited by patients who rigidly adhere to one criterion and ignore other instructions (Luria 1969). The inability to shift from one strategy to another has been demonstrated in both monkey (Mishkin 1964) and man (Milner 1964), as well as a difficulty in disregarding the familiar aspect of a stimulus when an unfamiliar one requires attention (Perret 1974). As in striato-pallidal syndromes, patients with frontal-lobe damage may perform movements slowly, have difficulty in initiating or stopping action and in carrying out more than one motor program at a time.

One additional feature of frontal-lobe dysfunction is of interest here: it concerns the patient's inability to assess the value of each new action. The patient seldom verifies to what extent his action matches the original intent; he may verbally comment on his errors but fail to control them. This points to the existence of an evaluation process based on a matching operation between perceptual feedback of the continuing operation and a representation of the expected consequences of the action. The concept of corollary discharge, involved in the preparatory process that the motor plan exerts on the sensory system to tune it in accordance with the expected consequences of the intended act, was introduced by Teuber (1966); he postulated a special role for the frontal lobes in this function.

As to the ordering and sequencing of action, which are important aspects of the planning process and of its derangements in ideatory apraxia, we still lack compelling evidence regarding the neurophysiological mechanisms involved, despite Lashley's (1951) remarkable theoretical analysis. It has been reported, however, that monkeys with lesions of premotor cortex, area 6, and the arcuate sulcus are poor at carrying out sequences of actions that they can correctly perform individually (Deuel 1977). In man, the dominant role of the speaking hemisphere in sequential motor activity has been demonstrated (Kimura & Archibald 1974), without uncovering the nature and localization of the underlying mechanisms. It is interesting, however, that deficits in movement sequencing have recently been observed after damage to the supplementary motor area, the function of which remains largely undiscovered. It has nevertheless been shown, by measurements of local rates of cerebral blood flow in man during rapid finger movement tasks, that the supplementary motor area is highly active when the subject is instructed to imagine a complex sequence of movements whereas premotor and sensorimotor areas react in turn, once the movement is executed (Roland *et al.* 1980). The supplementary motor area appears to be more involved in complex than in single movements. Thus it may have a special role in the planning process for complex sequences of movements. A similar view derives from single unit recording. Visual cues, which provide an instruction regarding the planning of the next movement (e.g. how to reach at a food reward behind an obstacle), cause the activity of single neurons in this region to change in character. Similar observations have been made in the premotor cortex. Removal of this area renders a monkey incapable of reaching around a transparent obstacle to obtain a reward; the animal tries to project his arm directly toward the food reward, bumping against the transparent sheet (Moll & Kuypers 1977). Planning of the detour is clearly deficient whereas direct reaching is automatically triggered.

Summarizing the evidence so far, we may tentatively contrast the respective roles of frontal and parietal association areas in the planning of purposeful activity as follows: the frontal areas may chiefly contribute to the gathering of information, from both external cues and memory store, that is necessary for the choice of the goal in relation to interest, priorities or values, and the anticipated consequences of the action; the parietal areas are more involved in the selection of behavioural cues – especially spatial ones – in relation to body state and to the postural and environmental requirements of the intended act, in so far as its specific purpose has been prescribed by frontal processes. Both channel their information to striato-pallidal structures where, depending on the goal specification and the contextual requirements of the action, a strategy will be selected. The subsequent tuning process of the sensorimotor executive structures will then enable the execution of the act. Outcomes of action are evaluated, rated good or bad, conforming or not conforming to the expected consequences of the action. According to this matching process, which is mainly dependent on frontal function (in so far as higher-order criteria and purposive goals are concerned), the action will cease or the strategy, even the goal, will be changed.

Two remaining questions challenge neurophysiological explanation: the neurological concept of 'motor image' and the vexed problem of 'will'.

(v) *The concept of 'motor image'*

The idea of anticipatory mental events, specifically associated with the voluntary initiation of goal-directed activity, developed early in philosophical and psychological thinking. In 1812, Buchanan put forward the basic concept of 'an idea of action to be performed' as constituting, together with the 'desire of performing it' and 'its execution', 'the three principal and essential components of the process of "volition"'. A profuse literature has since been devoted to the elaboration of these concepts (reviewed by Kimble & Perlmuter 1970). The 'kinetic souvenirs', the 'motor image' and the 'idea of the act', invoked by Liepmann in his analysis of the apraxias, relate directly to these concepts and to their lucid formulation by William James (1890).

As we have no idea of what a 'representation' would look like in the nervous circuitry of the parietal lobe, it is as yet difficult to substantiate the hypotheses emerging from psychological studies (reviewed by Greenwald 1970). These include the notion of 'perceptual traces which may later function as images to be compared with feedback from attempts to reproduce those responses' (Adams 1968) and that of a 'response repertory facilitating incipient responses matching that of previously rewarded responses' (Mowrer 1960). It is not yet clear how such notions can be instantiated at a neurophysiological level although it is obvious that kinaesthetic information, derived from evolving action, is available in the parietal region. Let us now consider possible approaches to this problem.

First, a recent study of event-related potentials in two patients with a classical ideomotor apraxia, is apposite (Ragot *et al.* 1982). It is known that a late, positive component of the visually evoked potential has been identified at a latency of approximately 300 ms (the P_{300} wave), but only when a perceptual identification task is required of the subject. Furthermore, it has been shown that the amplitude of the wave is highly correlated with the time required to elicit a motor response to the stimulus. Interestingly, in the study mentioned above, the P_{300} latency showed no marked increase in the apraxic patients (compared with the control group), whereas reaction time was considerably increased. Consequently the authors suggested that the perceptual process preceding decision-making is left intact in ideomotor apraxia and that, in accordance with Liepmann's scheme, only the motor command stage is impaired. These results point in a promising direction although their support for the notion that the motor image was intact is limited by the simplicity of the motor tasks employed.

Second, insult to the nervous structures in the parietal region that are involved in the ideomotor process should logically result in the suppression or disruption of neural processing occurring in the lesioned area. But, in accordance with Liepmann's view of ideomotor apraxia, the circuitry in which 'the images of kinaesthetic representation of movement' are presumed to be engrammed must be preserved. Two interpretations follow: either the 'representation of the act' is not localized here or, alternatively, a class of overlearned, skilled actions are so automatically linked to familiar context as to render ideomotor mediation unnecessary for their activation. A verbal command to act, characteristically disclosing disorders of praxis, may introduce a new level of signals, deprived of contextual cues, that make this mediation imperative.

Third, there have been somewhat different formulations of the concept of anticipatory images: compare the notion of a 'motor image' (James 1890), using traces of kinaesthetic feedback from previous instrumental action to foresee the proprioceptive information to be generated by the planned act, with that of an anticipatory 'goal image', using a representation of previous information that signals the successful achievement of the goal, thereby allowing a selection of 'habits' (Hull 1931). Far from being mutually exclusive, as claimed by Greenwald (1970), such processes may well have a complementary role in generating the plan of action: a 'goal representation', based on contextual information about external and internal cues and associated with previous reinforced behavioural activities, that is able to select a general class of possible strategies; and a 'motor representation' based on contextual, kinaesthetic and visual information related to previous, effective motor actions.

If so, it would be tempting to assign 'goal representation' to frontal association areas and the motor sketch of future acts to the parietal region. Then the motor schema would assist the basal ganglia to preselect, with reference to the spatial requirements of the act, a given strategy from among the ensemble of possibilities prescribed by the goal image, as invoked by previous experience and drive requirements. This would explain how, despite a parietal lesion, the goal image may activate a presetting of action that permits its automatic triggering in a drive-dependent context, whereas a verbal command, deprived of this context, fails to initiate a voluntary release of the same action, although this action is clearly envisaged at the goal level. The validity of a distinction between goal specification and motor schema, as two different factors of the planning process, merits further experimentation.

(vi) *The concept of volition*

The concepts of will, purpose, intention, attention and consciousness, stemming from nineteenth century psychology and largely neglected thereafter, now re-emerge in psychological research (see Kimble & Perlmuter 1970) and contemporary neuroscience (Buser & Rougeul-Buser 1978) as relevant to theories of motor behaviour. Liepmann himself set apart from the apraxias those motor disturbances due to a 'paralysis of the will'. They are now described by contemporary neurologists as 'negligence motrice' (Castaigne *et al.* 1970, 1972). Unable to mobilize their limbs spontaneously, in the absence of motor, sensory or attentional deficits, the patients can nevertheless perform movements, albeit laboriously under strict attentional control when actively encouraged by the physician. This appears to be a specific difficulty in initiating movement which, unlike apraxia, deprives the patient of the automatic execution of movement that can nevertheless be voluntarily triggered in the conditions described above. It represents for motor initiative what attentional mechanisms are for perception. It is associated with frontal or parietal lesions and also damage to thalamic, diencephalic nuclei and limbic structures. Together with the syndrome of akinesis and mutism, this disorder is probably related to a general disturbance of motor awareness.

Cerebral lesions affect voluntary rather than automatic action (Jackson 1932), suggesting that the latter may normally be performed without the direct participation of the higher cortical centres. A recent study, using cerebral blood flow measurement in man (Roland *et al.* 1980) showed that special cerebral mechanisms are required for the performance of voluntary movements but are not necessary for the maintenance of a fixed posture or for the performance of automatic movement. Penfield's earlier claim (see review by Paillard 1960) that the stream of volitional impulses might originate in subcortical regions (centrencephalic structures) has been supported by recent studies of the role of intralaminar and medial nuclei of the thalamus, traditionally considered to be the main channel for the selective distribution of activation throughout cortical structures.

The fact, however, that a voluntary act is the one that can be voluntarily inhibited raises the important question as to the origin of this inhibitory control, of which the potency and rapidity of intervention are striking features. Compelling neurophysiological evidence is still lacking but the basal ganglia might be mainly involved in this inhibitory control. Further advances can be expected from the labelling of single-cell activity, with regard to its specific dependence on features such as attention, task, reward, spatial or temporal dimensions, movement command, sensory input and goal meaning. The recent impetus in studies of the attentional process could well open fascinating new issues (Mountcastle 1978*b*), when combined with psychological expertise (Posner & Boies 1971). Voluntary, conscious and intentional components of behaviour, as opposed to automatic, unconscious control of action, can be related to the attentional demand that they require for their execution (Posner & Cohen 1980). This approach encourages the search for what Teuber (1974) called 'the physiological marker of voluntariness'.

Finally, the study of the motivational process is gathering important new data about the role of limbic structures in the functioning of the cerebral cognitive agency. The anatomical and physiological identification of circuits linking limbic structures to the frontal and inferior parietal lobe may contribute to our understanding of the specific role of association cortex in the organization and control of voluntary movement. Mesulam & Geschwind (1978) suggested that these circuits provide the anatomical basis of many pathological derangements of attentional processes and of affect. The disruption of neural connections between neocortex and the limbic system 'may interfere with the ability of neocortex to initiate, coordinate or inhibit complex behavior in response to the proper limbic cues'. These limbic structures have long been considered as the chief mediator of motivational processes and drives in relation to the system of values, inherited or acquired, that orient the activity of the organism and take part in the purposeful selection of goal-directed behaviour (Vanderwolf 1971).

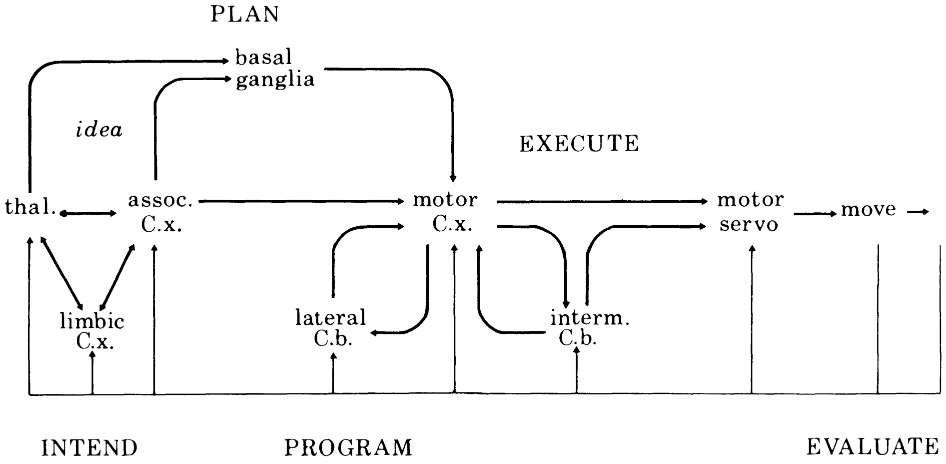

FIGURE 5. Revised and completed schema of Allen & Tsukahara (1974). See comments in the text.

4. OVERVIEW AND PERSPECTIVES

Neurophysiological analysis is founded on the assumption that any action system may be described in terms of the brain functions that support it and the processing operations to which it gives rise. While recognizing that pathological disorders, due to the breakdown of components of the system, seldom split it along clean functional lines, a processing model must be attempted if the gap between neurological and neurophysiological data is to be bridged (see figure 5).

(a) *Levels of control*

Elaborated motor performances are produced at the *executive* level by hierarchically prewired circuits that are strictly controlled to operate only at the required time and they adjust to postural requirements and environmental constraints through predetermined internal and external feedback loops. The older part of the cerebellum plays a major role in this regulation. The evaluation of performance with respect to the intended goal depends there chiefly on exteroceptive cues but possibly also on proprioceptive cues, both of which are conveyed to central structures. The motor cortex, in its pyramidal and extrapyramidal contribution, plays an integral part at this executive level. The main unsolved problem concerns the *embodiment of learned programs*. Either direct corticomotoneural pathways bypass the inborn repertory of motor programs at brain-stem and spinal level (clearly the case for manual and vocal skills) or corticospinal commands may generate new sets of coordinated activity by tuning existing programs (Paillard 1978). In either case, it has to be established how and where these new programs are printed as stable, new circuits that can be automatically triggered. Durable feedforward changes, involving the contribution of the cerebellar loop for their establishment, have been demonstrated but there is as yet no convincing evidence of the existence of a repertory of newly acquired skills, whether in the cerebellum, basal ganglia or cortical structures; nor has it yet been proved that permanent changes of the inbuilt circuitry exist.

Programming is an active process that involves the presetting and pretuning of conditional loops before the decision to act. This programming process demands precise structural prescriptions (including the configuration of muscles to be activated), time specifications (date and sequential ordering), and force requirements (speed and amplitude). In the case of delay, this pretuning can be preserved, but as soon as the decision to act is taken, the *neocerebellar loop* is involved in starting this programming process, updating during its execution, and stopping. The palaeocerebellar loop, for its part, assists the evolving action, with special reference to postural constraints.

Planning involves principally the striato-pallidal loop, enabling the selection of the most suitable program in accordance with the intended goal, the initial state of the body and its expected postural changes, and the estimated requirements of the task in intra- and extrapersonal space.

The frontal lobe may be chiefly involved in the definition of the goal in accordance with drive and the priorities supplied by limbic information, and in the evaluation of the achievement of the act in relation to the initial predictions of its sensory consequences. It is here, predominantly, that the meaning of task cues is processed, thus defining, in general terms, a class of possible strategies.

The parietal lobe is mainly concerned with contextual, postural requirements and spatial constraints, as defined by proprioceptive and exteroceptive cues. It is the location and spatial configuration of task cues that are chiefly involved, thus contributing to the choice of one motor strategy from among the class of possible strategies delimited by the requirements of the goal.

Finally, *attentional processes* are required to prepare, initiate, and control voluntary action. They involve thalamic (intralaminar and median nuclei) and limbic structures. At this higher level, however, there is as yet no clear understanding of how 'motor schema', 'goal image', or 'repertory of values' are stored in brain circuitry. Recognition of patterned input is assumed to be essential for the decision process. A dynamic flow of information would then be distributed through a matrix of functional modules, the interconnections of which organize a cascade of layered, 'control surfaces' (Arbib 1976). The inferior parietal lobule contains a number of discrete cytoarchitectonic areas (Pandya & Seltzer 1978). These probably maintain different, if overlapping, extrinsic connections. Such local differentiation is compatible with the further parcellation of areas into functional modules. Modular redundancy, reflected in the columnar organization of these areas, imposes the concept of parallel processing within a complex distributive system (Mountcastle 1978*a*), the integrated output of which converges on subcortical structures.

(*b*) *The dynamic patterning of nervous command*

The higher levels control their own particular function and parameters, maintaining with each other more heterarchical than hierarchical relations. The more general specifications of the performance subordinate and modulate the more specific: contextual specifications (goal, postural and spatial constraints) determine the strategy that, in turn, involves a selection of program prescriptions which then specify the servo-assisted execution of the program. 'Each loop operates as a loop receiving sensory information pertaining to the movement under execution and information about states of other loops in the system' (MacKay 1980). The three important parameters – muscle selection, timing specification and force regulation – are then adjusted by the program of action derived from conscious goal selection (see figure 6).

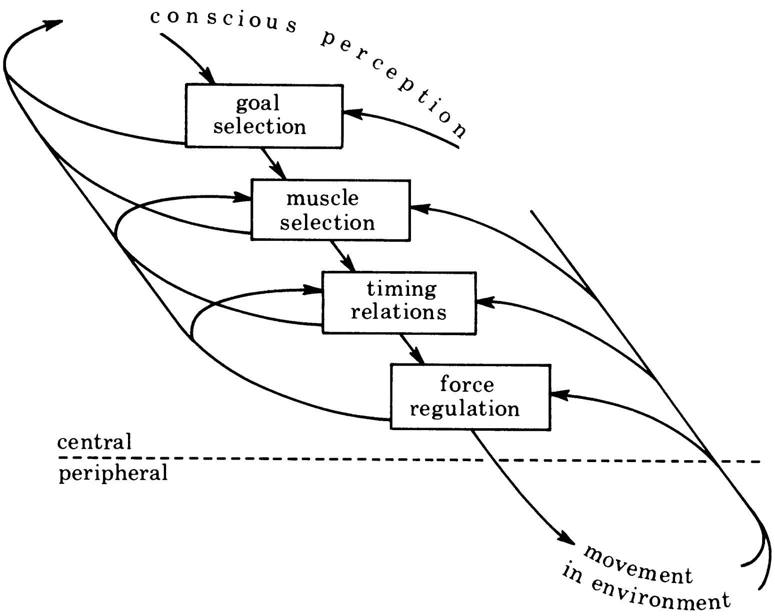

Figure 6. Simplified schematic diagram illustrating the hierarchical structure of a generalized motor program (MacKay 1980).

Planning and programming therefore consist of tuning conditional loops, closed within the nervous system or in the external environment, that control the flow of information through the nervous system and its peripheral transducers (muscle and sense organs). We are then led to conceive of the patterning of nervous command, at each level of its generation, as the result of a continuous stream of nerve impulses; these are carried along a variety of circular paths (Paillard 1960) that result in automatically driven movement, to produce the sensory input that has to be matched with the goal specifications (MacKay 1978).

(*c*) *Taxonomic requirements*

Further progress in our understanding of higher nervous functions, and consequently of their disorders, including the apraxias, depends on our capacity to analyse behaviour within well defined situational constraints and then to formulate an appropriate taxonomy of action (Phillips 1977).

The identification of the elementary operations that intervene at each level of motor control obviously depends on the choice of descriptive model. Various descriptions of apraxic disorders have been proposed (Hécaen 1968; De Ajuriaguerra & Tissot 1969; Roy 1978; Signoret & North 1979; Kelso & Tuller 1981). The criteria used in neurology, neurophysiology and psychology tend to vary but, at least in apraxia, the schema put forward for the organization of intentional gesture is compatible with the models of neurophysiology and information-processing theory. A variety of experimental paradigms can be borrowed directly from behavioural studies; others must be designed *de novo*. Whatever the choice, prescriptions of programming must be specified and experimentally controlled.

In practice, investigation of the programming stage of motor behaviour would take into account its reliance on three-dimensional coding – timing specification, muscle selection and force regulation – that probably involve separate mechanisms. The systematic study of this stage therefore requires a task that clamps one parameter and leaves the others responsive to the planning of action, so that the cost of planning, in relation to the requirements of the program, can be explored. At the planning stage, experimental conditions must be designed that force the subject to change strategies to achieve a given goal. In addition, tasks requiring the subject to use both hands simultaneously offer the opportunity to study the capacity to share voluntary control over the planning process (Kelso *et al.* 1979). Also, complex tasks, involving the ordering and serial sequencing of successive components, are of special interest in order to investigate how timing requirements have to be handled at the planning stage. Finally, it would be informative to explore how the 'meaning' of the intended task might influence the planning process, for instance, in conditions where the same sequence of muscular activity is used in various contexts with different behavioural significance. In general, the affordance of situational context on the intentional triggering of action has to be systematically examined.

5. Conclusion

Recent advances in the neurophysiology of higher motor function have led to a turning-point where, as already envisaged by Sherrington (cited by Paillard 1978) at the beginning of the century, important new developments can now be expected from cross-fertilization in the neurosciences. This has been aptly summarized by Mountcastle: 'It has been clear for a long time that the quantitative study of behavior traditionally the domain of the Psychologist, and of neural events in the brain, called "Neurophysiology" are conceptually different approaches to what are generically the same set of problems. . . . What is new is that it is now possible to combine in one experiment the methods and concepts of each to yield a deeper insight into the brain mechanisms that govern behavior than is possible with either alone.' Accordingly, clinical neurology and the neuropsychology of higher cognitive and motor function should be among the principal beneficiaries of this promising interdisciplinary convergence.

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