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Swimming in the Rat:

Analysis of Locomotor Performance in Comparison to Stepping

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Summary. Swimming in a mammalian quadruped, the rat, is analyzed in kinematic (joint angles) and electromyographic (EMG) terms. Data were collected on the movements of the hip, knee, ankle, and toe joints and three principle extensors and three flexors of the right hindlimb and compared with similar data collected on the same rats during treadmill stepping. The flexion, or protraction phase of swimming and stepping had many elements in common, including a similarity of EMG activity patterns and corresponding limb movements. However, in the extension, or retraction phase, there were notable differences. Although joint-extensor muscles were all coactive in both conditions, the brevity of the swimming extensor phase precluded the characteristic variation in EMG activity levels seen in the extensors in stepping. The flexors, in particular semitendinosus (ST), exhibited bursts of activity at the end of the extensor phase of swimming which were not present during the comparable period of stepping. The extra burst in ST produced a very rapid knee flexion at this time. Whereas the range of hip joint movement was similar in the two conditions, the ranges of the knee and ankle joints were expanded during swimming.

Overall, the evidence suggests that swimming is a very rapid form of a basic locomotor pattern in which the extensors are driven to their maximum contraction rate. The extra extension of the limb derives from the absence of ground reaction forces, allowing the knee and ankle joints to fully extend. The added bursts in the flexors remain to be explained. A discussion of these results in terms of current theories of single limb locomotor pattern generation is presented.

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Only brief mention of swimming in mammalian quadrupeds has, to the authors' knowledge, been published. Yet many quadrupeds, including rats, inhabit areas around water where they may be observed adeptly swimming. Cats, the favored quadruped for locomotor studies, are not renowned for their aquatic predisposition which may explain why only one group has looked at swimming in this animal.

Miller and van der Burg (1973) have reported that the "yield" phase of stepping is not present in swimming cats due to the absence of interaction of the limb with the ground. More significantly, Miller et al. (1975) have found that data from swimming cats support their hypothesis that there are "two fundamental patterns of cyclical locomotor movement based on the alternation of flexion and extension in each limb: alternate and in-phase locomotion." However, they show very little joint angle and no EMG data.

It might be assumed that swimming and stepping are based on a similar neural organization, but that swimming movements may be "simpler" than stepping movements because of the absence of rapid changes in load during the ground contact phase (Lundberg 1969). It is currently accepted that central, pre-programmed commands determine the main features of locomotor behavior (Grillner 1975; Wetzel et al. 1976), and that sensory feedback is utilized to some extent in modifying ongoing locomotor movements (Duysens and Stein 1978). The mechanisms for modification of the central locomotor commands are not known. It is likely that alterations

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in exteroceptive and proprioceptive feedback engendered by a change from terrestrial to aquatic locomotion could produce important differences in the motor output in the two cases which might lend insight into the mechanisms. This study demonstrates differences between stepping and swimming, particularly in the execution of the extension phase.

Methods

The methods used to analyze swimming in the rat were basically the same as described in a previous paper on treadmill locomotion (Gruner et al. 1980). The same 11 rats were used. While the rats were being trained on the treadmill for stepping, they were also trained for approximately 2 weeks to swim from one end to the other of a 16″ × 10″ × 60″ tank, one side of which was clear plexiglass. The water was at room temperature, about 23° C. As a control for surgical artifacts, movies were taken of the rats swimming before implantation of EMG electrodes. No changes in swimming patterns were observed after implantation.

After recording on the treadmill was completed, each rat was placed at one end of the swimming tank. As the rat swam to the other end it was photographed at 64 frames/s, and EMG's were recorded on paper at 100 mm/s and, for some rats, on magnetic tape at 15 in/s. Both raw (30–90 Hz) and rectified-filtered records (Garland et al. 1972) were made on the polygraph. EMG's were monitored on an oscilloscope to determine and maintain optimum recording levels.

Records were analyzed as described in the previous paper except for the following points. The pelvis angle was defined as the angle between a line parallel to the water surface and a line tangent to the back at a point above the hip joint.

The errors in joint angle analysis have also been discussed previously (Gruner et al. 1980). Skin slippage over the joints was still minimal, but blurring became more serious in determining the positions of the ankle and toe joints in swimming during the extension or retraction phase. However, it should only have added another ±1° of error to the knee, ankle, and toe angles, which should not affect the conclusions below. Latencies of joint angle maxima and minima were first determined from the joint angles. They were then double-checked by directly observing the motion of the joint in the movie and determining its apparent time of reversal. This procedure should limit the error in latency determination to one frame, or approximately 7% of the average cycle duration.

The muscles recorded from were the biceps femoris (BF; hip extensor, knee flexor), iliacus (IL; hip flexor), vastus lateralis (VL; knee extensor), semitendinosus (ST; knee flexor, hip extensor), lateral gastrocnemius (G; ankle extensor, knee flexor), and tibialis anterior (TA; ankle flexor). In both stepping and swimming the muscles could be classified into two groups based on their EMG patterns. The BF, VL, and G behaved primarily as joint extensors, and the IL, ST, and TA as flexors. The muscles will be discussed mainly in these terms except where secondary functions of the double joints muscles appear important.

Results

General Features of EMG's

The swim cycle was divided into two phases for descriptive purposes. Power stroke (PS) was defined as extension of the limb from minimum to maximum hip angle, and return stroke (RS) as flexion of the limb from hip maximum to minimum (Fig. 4). Representative raw (30–10 kHz) EMG recordings from the flexors and extensors of two rats during swimming appear in Fig. 1. The EMG amplitudes were in the neighborhood of 500–1,000 μV. The extensors BF, VL, and G produced single, short (30–50 ms) bursts during the PS phase of each cycle (Fig. 1C). There was overlapping of flexor and extensor activity, chiefly of IL and ST with BF, VL, and G (Figs. 1A, B).

The records in Fig. 1 suggest that there are two distinct periods of activity in each of the flexors IL, ST, and TA. The most obvious case was ST which showed a prominent burst coincident with the extensors in the latter half of powerstroke, and which will be denoted as the ST PS burst. After a brief pause, a second period of activity began which lasted until the middle of return stroke (the ST RS burst). Note that the PS burst in ST is usually greater in amplitude than the RS burst.

In TA (Figs. 1A, B, C) there was invariably a small degree of activity at the same time as the ST PS burst. While it is doubtful that this was entirely an artifact, the extensor digitorum longus, which lies alongside the TA, also showed a double bursting pattern with a strong PS burst similar to ST (Gruner, unpubl.). The onset of the main period of TA activity, the TA RS burst, occurred on the average 10–20 ms after the ST RS burst (Figs. 1A and B) and outlasted it by about 50 ms.

Activity in IL is difficult to interpret (Figs. 1A and B). There was usually a high-frequency "burst" of variable amplitude (denoted by "*") coincident with the ST PS burst. This burst sometimes disappeared altogether for as yet undetermined reasons. Although it may seem unwarranted to assert that this is really a distinct period of activity, it can be seen that when activity in IL does occur simultaneously with the ST PS burst, there is invariably a brief pause in IL activity of a few ms duration exactly at the termination of the ST PS burst. This pause demarcates what will be referred to as the IL PS burst from the ensuing RS burst. Return stroke activity in IL is further complicated by an apparent reciprocal relationship to the amplitude of ST RS activity (Figs. 1A and B): IL activity is distinctly greater during the pause between the ST PS and RS bursts than at the peak of the ST RS burst, and greater still after the end of the ST RS burst. This reciprocal relationship does not seem to apply to the IL PS burst.

To summarize, the extensors showed single, synchronous bursts during power stroke. The flexors, particularly ST, showed two periods of activity: the

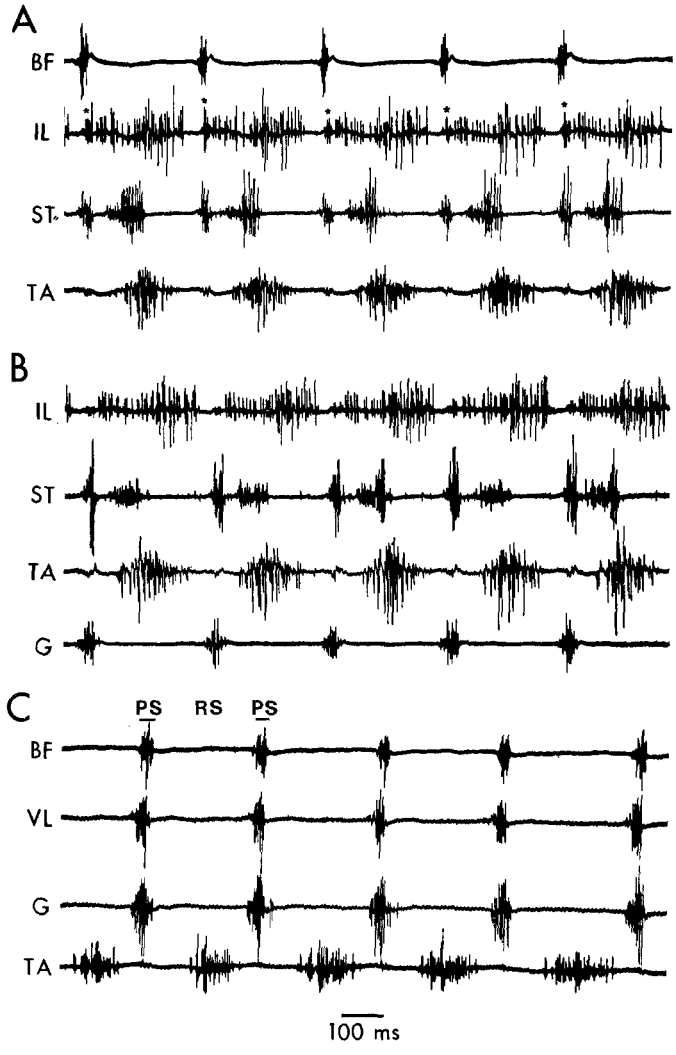

Fig. 1A–C. Raw EMG recordings of two normal rats swimming (**A** and **B** from one rat, **C** from another). In **A** the IL PS bursts are denoted by "*". Note in **A** and **B** the PS bursts in the flexors ST and IL temporally overlap those of the extensors BF and G. Activity in IL occurring after the termination of the ST PS burst is reciprocally related to ST activity. In **C** approximate periods of power stroke (PS) and return stroke (RS) are indicated; synchronous activity occurs in BF, VL, and G during PS. Note indications of PS bursts in TA in **A, B** and **C.** BF, biceps femoris; G, gastrocnemius; IL, iliacus; ST, semitendinosus; TA, tibialis anterior; VL, vastus lateralis

PS burst, which overlapped the extensor PS bursts, and the RS burst. The flexor RS bursts began and ended at different times for each muscle, the order of onsets was IL – ST – TA, while the order of offsets was ST – TA – IL.

General Features of Limb Movement and Muscle Activity

Synchronized joint angle and filtered EMG data for one rat is shown in Fig. 2. Eight cycles, each beginning with hip minimum (vertical lines) are plotted. Swim cycles were typically about 200 ms long. The pelvic angle did not exhibit any rhythmic fluctuations, and was in the range of about 32 ± 8° for all animals.

The hip, knee, ankle, and toe angle curves each exhibited one period of extension and one of flexion per cycle (i.e., each curve had only one minimum and maximum). The knee began to extend near the middle of the return stroke phase, followed by the toe, ankle, and hip joints. The toe angle increased to 180° (straight out) at the onset of power stroke and remained there until return stroke had begun. In return stroke the phalanges extended by sequentially "unrolling" themselves so that they presented minimum resistance to forward motion.

The filtered EMG patterns in Fig. 2 show the same features described in Fig. 1. Extensor activity consisted of short, nearly synchronous bursts in the power stroke phase. The IL showed three distinct periods of activity, two initital bursts (the PS burst, denoted by "*" in the first cycle, followed immediately by "early" RS activity), and a phase of gradually increasing activity which ceased at the end of the cycle ("late" RS activity). Little or no TA activity was observed during powerstroke in this rat.

Locomotor Features Revealed by Cycle Averaging

To facilitate comparisons among rats, eight swim cycles per rat were analyzed and the curves for each joint or muscle were normalized to constant duration and averaged together (Fig. 3), revealing consistent features of the joint angle curves. There was often a plateau, or slowing, of flexion in the hip angle curves during mid return stroke. In each case knee flexion began slightly ahead of that in the hip and ankle and was more rapid, reaching a minimum at mid return stroke. After the ankle angle reached maximum extension, flexion in this joint was very slow in most cases until about mid return stroke. In each animal extension of the ankle began after the knee and ahead of the hip.

Figure 4 is a composite of typical joint angle and EMG records from Fig. 3 showing hip, knee, and ankle joint angle records and flexor and extensor EMG's for each of these joints. The figures above each set of curves represent the positions of the bones of the rat's hindlimb at the time indicated by the short vertical line beneath the hip joint. Although the records in Fig. 4 were taken from three different rats, the temporal relations are very close to what was seen within individual rats. This figure illustrates all of the important features of rat swimming.

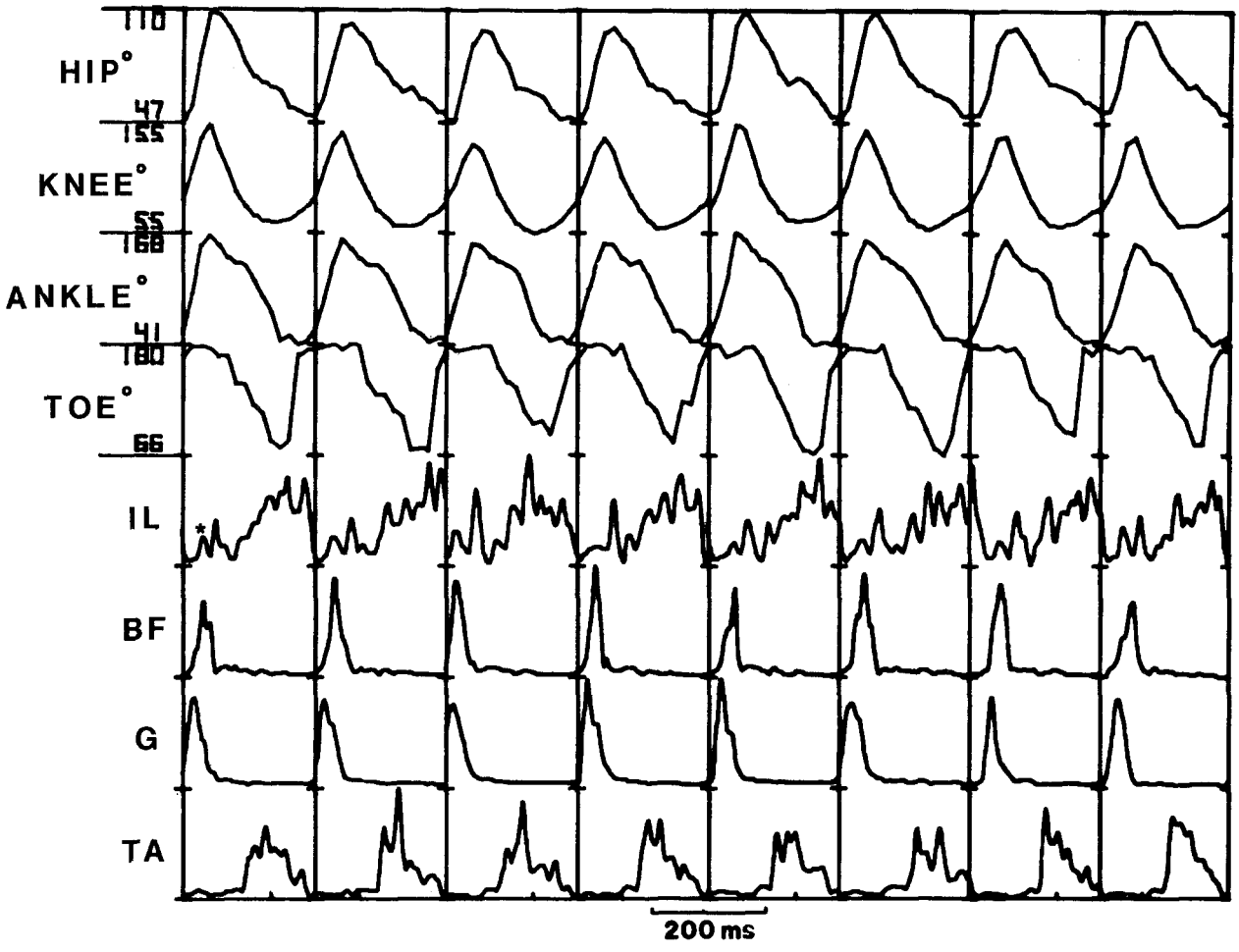

Fig. 2. Joint angle and filtered EMG data for rat 13.3 during unrestrained swimming. Eight swim cycles, each beginning with hip minimum (vertical lines) are shown; cycles were not all consecutive. The power stroke phase was defined as hip minimum to maximum; return stroke as maximum to minimum (cf. Fig. 4). The IL PS burst in the first cycle is indicated by "*". Minimum and maximum values for joint angle curves are given at the left margin (i.e., 47–110° for pelvis). Amplitude of EMG's is arbitrary but constant for each muscle

Correlation of Limb Movements and Muscle Activity

As a basis for correlating limb movement and muscle activity, a quantitative analysis of the joint angle curves was made by determining the minimum and maximum of each curve (the joint angle *turning points*) for eight swim cycles of each rat. The individual means (m) and standard deviations (sd) of both the latency and amplitude of each turning point were then calculated for 11 rats. In Fig. 4, the rectangles centered around the turning points represent the mean latency ±1 unit of standard deviation (normalized with respect to cycle duration) and the mean amplitude ±1 unit of standard deviation. The varition in amplitude of the hip minimum is denoted by horizontal bars on the vertical line at phase 0 since the mean and standard deviation of this latency is zero by definition.

The within-group mean (M) and standard deviation (SD) of the within-group mean were computed along with the *mean* of the individual standard deviations ($\overline{SD}$). The SD then is a measure of the variability of the means among animals in a group, while the $\overline{SD}$ is a measure of the mean variation of the turning point values within a group.

Limb Extension and Extensor Activity. The knee, ankle, and hip joint curves increased to maxima at mean latencies of 49, 59, and 62 ms, respectively (Table 1). The ankle and hip latencies were not significantly different, but the knee peaked significantly earlier ($p < 0.005$, t-test) than the ankle or hip.

To better define the relationship between extensor EMG onset and onset of joint extension, the average EMG and joint extension latencies with respect to hip minimum were computed for each rat in which the extensor in question was implanted. In

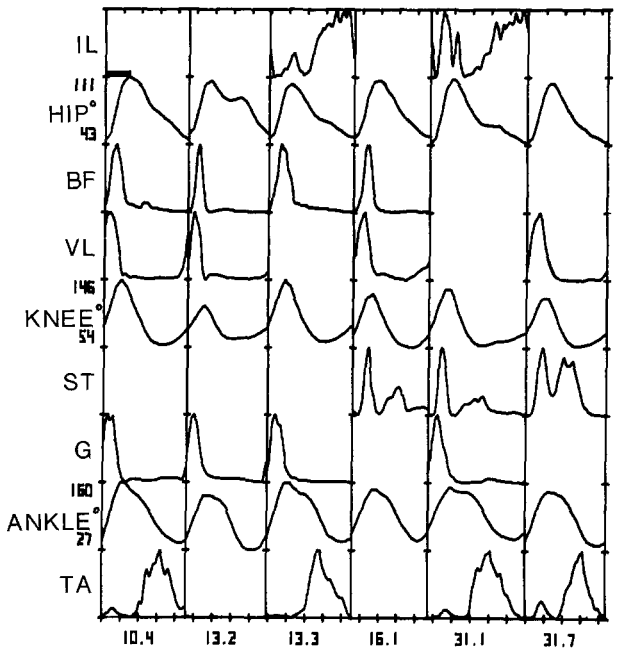

Fig. 3. Average swimming joint angle and EMG curves for six rats. Each curve is the average of eight normalized cycle curves (see Methods). Each column contains the joint angle and EMG curves of one rat (number at bottom of column). Minimum and maximum values for joint curves are given at left margin (i.e., 43–111° for hip). The power stroke phase is indicated in the first column by a horizontal bar at the top of the hip angle curve. Divisions along horizontal axes are 50 ms

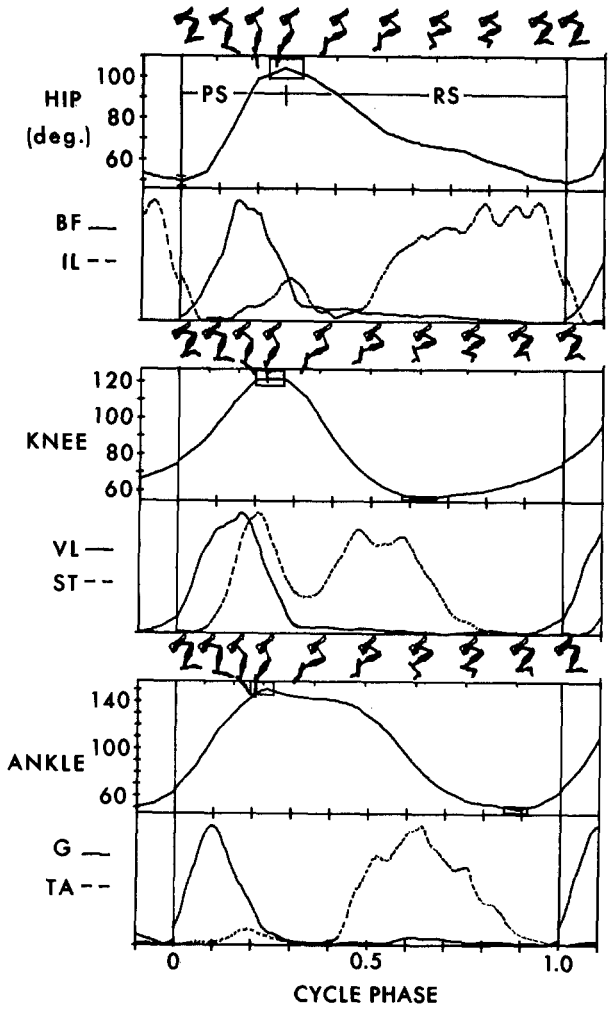

Fig. 4. Typical average joint angle and EMG curves from three rats, 13.3, 31.7, and 31.1 (top to bottom) replotted from Fig. 3. Solid EMG curves are extensors, dashed curves are flexors. The initial and final parts of the curves have been replotted at the end and beginning, respectively, of the curves so that phases 0 and 1.0 (vertical lines) denote hip minimum. The figures above each set of curves represent the positions of the bones of the rat's hindlimb as determined by the angles of the joints at the time indicated by the short vertical line beneath the hip joint. Rectangles around joint angle turning points denote ±1 units of standard deviation about the latency (horizontal) and joint angle (vertical) means

each case (Figs. 2–4), extensor onset followed extension of the joint it primarily affected. The knee, e.g., began to extend some 50 ms before VL activity onset.

The order in which the joints began to extend was the same as that of the respective extensor EMG onsets. The latencies of onset for the three extensors were significantly different from each other ($p < 0.05$; one-tailed t-test on the average latencies for each rat). The cessation of VL activity was significantly earlier than that of G ($p < 0.05$), and in every animal where both VL and BF were implanted VL activity also ceased before that of BF.

Limb Flexion and Flexor Activity. Knee flexion in return stroke began slightly earlier and was more rapid than in either the hip or ankle joints. Joint angle and EMG records (Figs. 2–4) showed that the ST PS burst started about 20 ms ahead of knee flexion. Knee flexion lasted about 100 ms, with ST activity ending 20–30 ms later.

The IL PS burst occurred 10–20 ms ahead of hip flexion and was closely followed by the earlier RS burst which was usually larger. A 5–10 ms pause separated early and late RS activity. The onset of late IL RS activity was usually within ±5 ms of the end of the ST PS burst, or 20–30 ms after ST onset. At the end of the cycle, IL activity began to decline rapidly about 10 ms before the onset of hip extension.

Ankle extension stopped abruptly at the end of power stroke, at the same time as hip extension. In most animals there was only limited ankle flexion for the next 50 ms. A small TA PS burst usually occurred at the end of ankle extension (Fig. 4). The main period of TA activity, the RS burst, began an average of 25 ms (n = 9) after hip maximum (onset of return stroke). There was a noticeable increase in the rate of ankle flexion (Figs. 2–4) in most animals about 10 ms

Table 1. Mean latencies and amplitudes of joint angle turning points for rats swimming (n = 11)

Turning Point	Latency M	SD	$\overline{SD}$	Amplitude M	SD	$\overline{SD}$
Toe max	44	9.5	21.3	179	0.3	0.6
Knee max	49	4.7	7.4	126	18.1	7.0
Ankle max	59	6.7	10.3	146	10.1	4.9
Hip max	62	4.6	8.8	107	6.3	4.5
Knee min	147	16.3	14.6	55	7.0	2.7
Toe min	168	16.0	12.1	73	8.5	8.3
Ankle min	194	19.5	11.3	40	7.8	3.0
Cycle duration	221	20.9	6.7	45	5.3	2.5

later. Figure 3 shows that in rats 31.1 and 31.7, the TA RS burst followed the ST RS burst by 20 ms or more.

Discussion

The major concerns of this discussion will be first to examine swimming in more detail, and then compare swimming and stepping to discern common features which might reflect invariant properties of locomotor behavior.

Relations Between Joint Movement and Muscle Activity in Swimming

Joint Extension and Extensor Activity. Joint extension preceded onset of the respective extensor muscle in each case. The "premature" extension in the hip and ankle joints could be accounted for by forces exerted by the water against the limb which would act to extend them (Fig. 4). In the hip, e.g., extension preceded BF activity by a few milliseconds. However, the angular acceleration of this joint often increased dramatically about 15 ms (1 frame) after the onset of the cycle (Figs. 2–4). This situation would be expected if hip extension was initially passive until tension produced by BF activity took over 10–15 ms after the EMG onset.

Extension in the knee, however, must have been active since it was directed against the force of the water, and in addition may have had to counteract tension in the ST (Fig. 4). It seems likely that there must have been activity in another knee extensor, such as vastus medialis or rectus femoris, at this time.

Joint Flexion and Flexor Activity. The flexors usually showed activity in both the power and return stroke phases. Power stroke bursts in IL, ST, and TA were fairly uniform in onset latency and duration and had similar activity profiles, while the RS bursts each showed characteristic patterns of onset latency, duration, and activity profiles.

The early and rapid knee flexion apparently resulted from a combination of factors, the most important of which was the large ST PS burst which followed the VL burst by about 20 ms and peaked about 20 ms ahead of knee flexion. Also, activity declined slightly earlier in VL than in BF. Since BF has a secondary knee flexor action, it could have contributed to knee flexion. Finally, activity in IL and especially TA at the beginning of return stroke was weak relative to that in ST.

The advanced flexion in the knee relative to that in the hip or ankle might have produced significant propulsion at the end of power stroke. In Fig. 4, between the fourth and fifth hindlimb drawings from the left above the knee curve, one can see that knee flexion causes the foot to move from directly under the hip joint to considerably behind the hip, while the hip and ankle joints flex very little. Since the orientation of the foot is maintained nearly perpendicular to the direction of motion, this flexion should propel the animal forward.

In the hip joint, inititial flexion was evidently produced partly by the IL PS and early RS bursts. The hip extensor activity of ST at this time would have only minimally opposed hip flexion. Previous results for stepping (Gruner et al. 1980) indicated that other muscles are probably also important in initiating hip flexion.

The rate of hip flexion often declined about mid return stroke (cf. rats 13.3 and 31.1 in Fig. 3). This slowing corresponded well with a decrease in amplitude in IL RS activity during the period of ST RS activity (i.e., the pause between the early and late RS bursts). A longer period of decreased activity in rat 31.1 than in rat 13.3 was associated with a more pronounced retardation of hip flexion. Moreover, rat 31.1 showed large PS and early RS bursts along with a particularly rapid initial hip flexion.

Extension of the ankle stopped abruptly at the end of power stroke, apparently at its mechanical limit (150°). There was only limited flexion for the next 50 ms, possibly because the small TA PS burst and/or EDL PS activity was insufficient to produce more than limited flexion against tension in the ankle extensors. About ⅓ of the way through the return stroke the TA RS burst occurred and ankle flexion

increased dramatically. This RS burst ended some 20 ms before the end of the cycle, about the same time ankle extension began.

Comparison of Swimming and Stepping

To facilitate the comparison of swimming and stepping, it will be helpful to equate the periods of rearward extension of the hindlimb in swimming (power stroke) and stepping (stance) and refer to them as *retraction*, while forward flexion, or return stroke and swing, will be termed protraction. These definitions are based on movement of the hip joint.

Phase Durations. Although power stroke was one-tenth the duration of stance, the return stroke and swing durations were nearly equal. In one rat (13.3), 25 step and 25 swim cycles were divided into retraction and protraction phases using the onset of BF activity as the start of retraction and IL onset (or the IL RS burst onset in swimming) as the start of protraction. The mean durations were: power stroke, 48 ± 3 ms; stance 435 ± 77 ms; return stroke, 184 ± 7 ms; and swing, 193 ± 23 ms. The difference between return stroke and swing durations was not significant ($p > 0.05$).

A probably explanation for the shorter retraction duration in swimming is the greater EMG amplitudes (500–1,000 μV in swimming vs. 50–500 μV in stepping) for all muscles. The more intense the extensor contraction, the greater the rate of limb retraction and the shorter its duration. A short retraction is advantageous in swimming since the thrust generated by an object (e.g., the hindlimb) moving through water increases as the square of the velocity (Gray 1968).

Joint Angles. It has been suggested (Pearson and Duysens 1975; Grillner and Rossignol 1978) that protraction may be initiated when the hip joint reaches a certain position. A comparison of hip joint angles in swimming and stepping showed that the limits of hip flexion and extension were nearly identical (Table 1; Table 1 of Gruner et al. 1980). Indeed, for the last 10–20 ms of hip extension (about the time it would take a limb afferent signal to reach the spinal cord) the angular rate of extension in swimming and stepping were nearly equal (Fig. 4). Equalization of the rate of angular displacement of the femur during the afferent conduction delay would insure that protraction would be initiated at the same hip angle in both conditions. Maximum hip extension could not trigger the flexor PS bursts, however, since they began at least 30 ms prior to this event.

The extent of joint flexion in the knee and ankle joints was greater during swimming than stepping by about 10°. This hyperflexion would bring the limb closer to the body and lower its hydrodynamic resistance during protraction. The 15–20° greater extension of the knee and ankle in swimming allows the maximum surface area of the hindleg to be used during the power stroke.

EMG's. As a means of comparing EMG's in swimming and stepping, average EMG curves of all rats (Fig. 3) were divided into retraction and protraction components based on the latency of the maximum of the corresponding average hip angle curve. All curves were normalized to a constant duration and corresponding curves from several rats were averaged together. In Fig. 5 two major differences in swimming and stepping EMG's are apparent: first, the characteristic, asymmetric EMG waveforms of stepping were reduced to simple bell shaped curves in swimming; and second, extra bursts in some flexors occurred during retraction in swimming. On the other hand, the onset latencies of the extensor bursts occurred in the same order – VL, G, BF – in both swimming and stepping (Gruner et al. 1980; Gruner 1978). Flexor activity in the protraction phases of swimming and stepping was also very similar in both latency of termination and duration.

Modifications in Locomotor Control in the Conversion from Stepping to Swimming

Central Control of Locomotion. The central control of locomotion can be divided into supraspinal and spinal components. Results of experiments mainly in the mesencephalic cat (Boylls 1975; Orlovsky and Shik 1976; Shik and Orlovsky 1976) suggest that supraspinal activity primarily affects the amplitude of muscular activity. Spinal centers (spinal locomotor generators) then control the timing of muscular activity by direct activation of muscles, and by gating supraspinal, propriospinal, and afferent inputs.

The increased extensor amplitudes which produced the short retraction period of swimming can be explained by an increase in activity from supraspinal locomotor "command" centers, just as stimulation of the "locomotor generator region" of the brainstem in mesencephalic cats results in greater extensor activity during stance (Severin et al. 1967). The onset of extensor activity in stepping is apparently centrally timed, since in cats it is not dramatically affected by deafferentation (Grillner and Zangger 1979). In swimming, knee extension began considerably ahead of VL onset in spite of the greater hydrodynamic resistance to knee extension at the end of RS, indicating a lack of proprioceptive influence on the timing of VL activity.

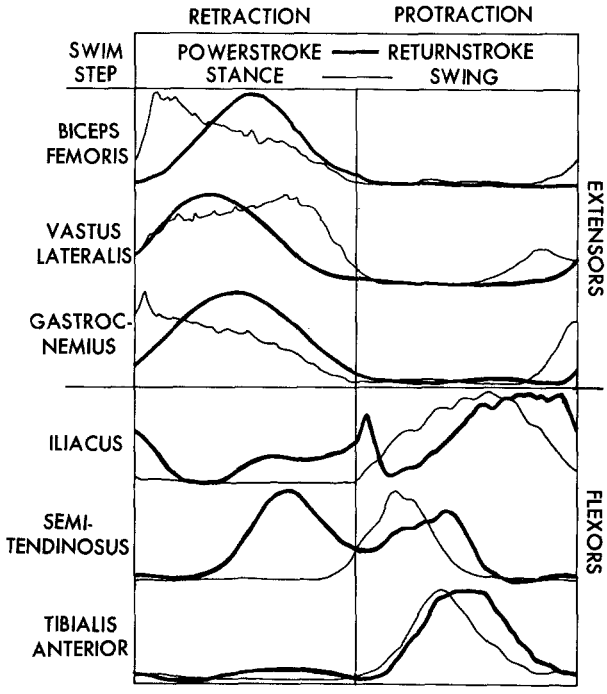

Fig. 5. Comparison of normalized EMG activity during retraction and protraction phases of swimming (heavy lines) and stepping (thin lines). See text for explanation

In cat locomotion, several flexors, such as ST (but not TA or IL), show a burst in early stance which increases with locomotor speed (Engberg and Lundberg 1969) and are also independent of afferentation. If one allows for the fact that in cat trotting the hip minimum (used to define onset of retraction in swimming) occurs appreciably ahead of foot contact, it appears that this "stance phase" burst in cat trotting is analogous to the ST PS burst. The flexor bursts could arise from increased intensity of locomotor command center activity (Perret and Cabelguen 1976), release from suppression by vestibulospinal activity (Orlovsky and Pavlova 1972; Gruner et al. 1980), or changes in central relationships between flexor and extensor spinal motor centers. These data do not favor any specific mechanism.

Inspection of flexor EMG's (Fig. 5) indicates that the PS and RS bursts have different origins. While the PS bursts are synchronous and have no analog in stepping, the RS bursts have characteristic durations and latencies similar to the flexor bursts in stepping. The tentative differentiation of IL activity into PS, early RS, and late RS components is based partly on the apparently antagonistic relationship between ST and IL RS activity. The reason for such antagonism between ST and IL have not been reported in the rat or cat (Eccles and Lundberg 1958). There is a possibility, however, that the early IL RS burst is reflexely evoked by stretching of the hip flexor-knee extensor rectus femoris by ST PS activity (Eccles and Lundberg 1958).

Modifications of Locomotor Activity by Afferent Feedback. The complex pattern of extensor activity in stepping is partly the result of a "shaping" of motoneuron activity by exteroceptive and proprioceptive feedback (Lundberg 1969; Grillner 1975; Grillner and Zangger 1975, 1979; Wetzel et al. 1976; Goldberger 1977). It is difficult to infer anything about the influence of peripheral afferents on extensor activity during power stroke since the period of activity was very brief (20–30 ms), and the extensors appeared to be driven at their maximal rate of contraction. The fact that the protraction durations of swimming and stepping were nearly equal was evidently due to the increased amplitudes of flexor EMG's. This compensation could be a specific adaptation to the "braking" effect of the water on the limb mediated by proprioceptive feedback (Orlovsky and Shik 1965). However, flexor activity was also greater at higher rates of stepping induced by brainstem stimulation in mesencephalic cats (Severin et al. 1967).

Conclusion

There are both significant differences and similarities in the features of terrestrial and aquatic locomotion. The present data can be explained most easily by assuming that the main features of locomotion in both cases are generated by a "common" locomotor program which is modified in accordance with the particular configuration of afferent feedback.

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