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Lesions of the Precentral Gyrus in Nonhuman Primates: A Pre-Medline Bibliography

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Many contemporary investigators are unaware of the important papers involving lesions of the primate primary motor cortex published prior to those revealed by a computer search of the literature (i.e., papers published prior to about 1966). In order to increase awareness of these reports, we present here an annotated bibliography of these papers beginning with that of Ferrier and Yeo (1884). We provide evidence that these papers can provide valuable information on the function of the primate motor cortex and on recovery of behavior after brain lesions, and are also useful for sharpening the questions posed by more refined modern studies.

KEY WORDS: primary motor cortex; Brodmann's Area 4; motor control.

INTRODUCTION AND METHODS

Many contemporary investigators of primate functional neuroanatomy have only a limited awareness of the many reports in this area published prior to those revealed by a Medline search, which begins about 1966. This is unfortunate because there were many valuable and still useful papers on primate functional neuroanatomy published well before that year. These papers remain important for several reasons: First, knowledge of these early reports is essential in order to assure Institutional Review Boards that any

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planned primate lesion research does not duplicate prior work and thereby result in the unnecessary killing of these valuable animals. Second, many of the investigators who did this work were experimental neurologists who could directly relate their findings to the disorders observed in their human patients. Third, these investigators had greater latitude in their evaluation procedures than current animal use guidelines permit. For example, these investigators were allowed greater direct physical contact with their animals than are current investigators. Fourth, journals in earlier times provided more space to describe qualitative findings than current journals. Although the qualitative descriptions typical of these reports are observer-dependent and therefore not as replicable as quantitative measures, they can still be used to sharpen the questions addressed in modern studies. Furthermore, these descriptions often reveal details on subtle aspects of behavioral recovery that are not considered in contemporary studies. And, fifth, although the large and comparatively uncontrolled lesions used by some early investigators are difficult to replicate precisely, such lesions are more representative of the effects of some disease processes (e.g., strokes) than are some of the more modern limited lesions. Thus, in a recent paper, Friel and Nudo (1998) stated that the small lesion size used in their own study is not representative of the larger lesions that occur in human stroke patients.

In order to enhance current knowledge of early studies that involved lesions of all or part of the primate primary motor cortex (M1), which is confined to the precentral gyrus, we have compiled an annotated bibliography (Table I) of papers published prior to 1966, beginning with the earliest, the 1884 report of Ferrier and Yeo. We chose to initiate our bibliographic project with reports involving lesions of the primary motor cortex because this region continues to be investigated actively (Hoffman and Strick, 1995; Kubota, 1996; Nudo *et al.*, 1996; Friel and Nudo, 1998; Rouiller *et al.*, 1998; Schieber and Poliakov, 1998; Liu and Rouiller, 1999).

We obtained the references for this bibliography from citations within the articles themselves, Ruch's (1941) *Bibliographica Primatologica*, and our own extensive reference collections on this topic. We did not include in the bibliography reports that purposely involved simultaneous lesions of multiple motor areas (e.g., primary and supplementary motor cortices). Furthermore, we recognize that the lesions in many of these studies extended well beyond the precentral gyrus and that the cortical area believed to comprise the primary motor cortex decreased in extent during the time period covered by the bibliography. Although we attempted to include all relevant reports, the bibliography may be deficient pertaining to some important studies. Further, there were some very early references from non-English sources that might have relevant material that we were unable to obtain.

Table I. Bibliography of pre-1966 reports of primate precentral gyrus lesions

Reference & yr	Experiment[a]	Sp, age, sex[b]	Lesion[c]	M[a]	H[e]	Obs per[f]	Types of observations	Comments
Ferrier and Yeo (1884) (also Ferrier, 1886)	1(16)	ma, ns, ns	hl(u)	2	Y	8 m	General behavior; reflexes	No loss of sensation; all movements not affected equally
Luciani (1884) Luciani and Seppilli (1885) (see text)	1	ns	T	1	Y	6 m	General behavior	Concludes that motor zone is really a "sensory-motor" zone in that it received impulses from muscles
Ferrier (1886)	1 (see Fig. 113)	ns, ns, ns	f(u)	2	Y	2 m	General behavior	No loss of tactile sensation; no change in condition during survival period
Horsley and Schäfer (1888)	grp1	mo, ns, ns	T(u)	1	Y	7–8 d	General behavior	Extirpation better than cauterization; no loss of sensation
	1(7)	mo, ns, ns	T(u)	1	Y	19 d		
Munk (1892; in German)	grp1	mo, ns, ns	f or ne or he or hl(u)	1	Y	ns	General behavior; compensatory responses	Used the term "apes" which we assume refers to monkeys; compared results to those for dogs
Mott and Halliburton (1908)	1(1)	l, ns, ns	T(u)	1	Y	14 d	General behavior	Deficits are transitory or not apparent
	2(7)	l, ns, ns	f(u)	1	Y	14 d		
	3(8)	l, ns, ns	hl(u)	1	Y	14 d		
	4(9)	l, ns, ns	fa(u)	1	Y	14 d		
Lewy (1911; (in German)	grp1	mo, ns, ns	f(u)	1	Y	1 y	Muscle atrophy	Discusses compensation for paralysis

Table I. (*Continued.*)

Reference & yr	Experiment[a]	Sp, age, sex[b]	Lesion[c]	M[a]	H[e]	Obs per[f]	Types of observations	Comments
Trendelenburg (1911; in German)	grp1	r, ns, ns	f(u)	2	Y	3w	General behavior; effects of cauterization on dura	Abilities exist to compensate for deficits
Leyton and Sherrington (1917)	1	ch, a, ♂	f(u)	1	N	7 w	General behavior (especially forelimb) and reflexes	Thorough and extensive study of the motor cortex in anthropoid apes
	2	ch, ns, ♂	f(bs)	1	N	1 m		
	3	ch, im, ♀	hl(u)	1	N	1 m		
	4	ch, im, ♂	hl(u)	1	N	3 w		
	5	g, im, ♂	e(b)	1	N	6 d		
	6	ch, im, ♂	T(u)	1	N	2 d		
Minkowski (1917; in French)	1	r, ns, ns	T(u)	1	Y	7.5 w	General behavior; reflexes	Functions in cortex are localized; previous studies used survival periods that were too short
	2	r, ns, ns	T(u)	1	Y	15 m		
Ogden and Franz (1917)	1–3	r, im, ♂	T(bs)	2	N	5–6 m	General behavior	Stimulated animal in order to enhance recovery
	4	r, im, ♂	T(u)	2	N	1 m		
Lashley (1924)	1	c, ns, ♂	T(b)	2	Y	35 d	General behavior; also quantifies manipulative abilities	Concludes motor cortex is not directly concerned with retention of complex learned activities
	2	c, ns, ♂	T(bs)	2	Y	7 m		
	3	r, ns, ♀	T(b)	2	Y	28 d		

Fulton and Keller (1932)	1(5) 2(11) 3(12) 4(13) 5(14) 6(15) 7(16)	man, im,♀ b, im,♂ b, im,♀ gi, im,♂ ch, im,♂ ch, im,♀ ch, im,♂	fo(u) hl(u) hl(bs) fo(bs) fo(bs) fo(bs) fo(bs)	2 2 2 2 2 2 2	Y Y N N N Y N	148 d 4 m 5 m 3 m 4 m 1 m 3 m	General behavior and reflexes, especially Babinski	Examines stages in evolution of cortical control over lower spinal centers in primates; Experiment 16 is also discussed in Kennard and Fulton (1933) as Experiment 1
Dusser de Barenne (1933)	grp1	gr, ns, ns	T(u)	ns	N	ns	General description; notes effect of illness on recovery	Functional relations of the motor cortex are flexible
Fulton and Dusser de Barenne (1933)	1(1) 2(4) 3(5) 4(6)	c, ns,♀ s, ns,♂ s, a,♀ s, a,♀	t(u) t(u) t(b) t(bs)	2 2 2 2	Y N Y N	20 d 18 d 29 d 4 d	Tail movements	Interested in tail area in monkeys with a prehensile tail
McKinley and Berkwitz (1933)	1(7) 2(12) 3(18) 4(19)	r, ns, ns r, ns, ns r, ns, ns r, ns, ns	T(b) T(u) T(b) T(b)	1 1 1 1	N N Y N	4 d 11 m 4 d 3 d	Degree, type and and location of rigidity compared to other lesions (e.g. decortication)	Very little descriptive data provided for these animals; presume simple extirpation procedure
Fulton and Kennard (1934)	1(1) 2(4) 6(8)	b, im,♀ c, im.♀ ch, im,♂	f♀hl(bs) f♀hl(u) f(bs)	2 2 2	N N N	30 d 5 d 6 m	General behavior and reflexes	Compared disturbances from motor cortex lesions to those of other frontal lobe areas

Table I. (*Continued.*)

Reference & yr	Experiment[a]	Sp, age, sex[b]	Lesion[c]	M[a]	H[e]	Obs per[f]	Types of observations	Comments
Jacobsen (1934)	1	r, a, ns	f(bs)	2	N	6 w	Quantitative measures of retention of manipulative abilities after preoperative training	Case 2 is the same as Fulton and Kennard's (1934) case 8; little residual deficits in monkeys from unilateral deficits
	2	ch, im, ♂	f(bs)	2	N	22 w		
Fulton and McCouch (1937)	1	ch, ns, ns	hl(u)	2	N	ns	Describes order of return of reflexes for primates across species	Provides details for 1 rhesus monkey; animals had subsequent spinal transections 8–117 days after cortical lesion
	2–3	r, ns, ns	hl(u)	2	Y	20 d		
	4–6	b, ns, ns	hl(u)	2	N	ns		
	7–8	p, ns, ns	hl(u)	2	N	ns		
	9	g, ns, ns	hl(u)	2	N	ns		
Hines (1937)	grp1	r, ns, ns	ant(u)	ns	N	2w+	Reflexes; muscle tone	Differing effects between posterior and anterior ablations
	grp2	r, ns, ns	pos(u)	ns	N	2m+		
	grp3	r, ns, ns	T(u)	ns	N	ns		
Green and Walker (1938)	1(56)	mo, ns, ns	fa(u)	2	N	14 d	Facial, tongue and swallowing movements	Details only provided for Exp3; interested in cortical representation of face area
	2(SP16)	b, ns, ns	fa(u)	2	N	5 m		
	3(SP19)	b, ns, ns	fa(u)	2	N	2 m		
	4(Exp3)	r, im, ns	fa(b)	2	N	56 d		
Zuckerman and Fulton (1941)	1	ga, a, ♂	T(u)	2	N	17 d	General motor behavior; reflexes	Interested in motor areas of these two prosimian primates
	2	po, a, ♀	hl(u)	2	N	20 d		

Hines (1943)	ns	ns, ns, ns	hl&f(b)	ns	ns	ns	Muscle tone and atrophy	Although a "review" paper some previously unpublished data are presented
Mettler (1944)	1(J115) 2(J141)	r, ns, ns r, ns, ns	T(b) pos(b)	ns ns	N N	5 d+ 3 m	Reflexes, muscle tone, general behavior	Case 2 previously had lateral column section at T4; concludes bilateral removals differ from unilateral or serial removals
Ades and Raab (1946)	1(M1) 2(M7)	r, a,♂ r, a,♂	T(bs) T(bs)	3 3	Y Y	12 d ns	General behavior with specific regard to compensation	Comments on bilateral compensation after unilateral lesion
Sperry (1947)	1 2 3	r, a, ns r, a, ns r, a, ns	mvi mvi mvi	v v v	na na na	3–4 d 3–4 d 9 d	General behavior	Case 3 had an earlier Area 4 lesion contralaterally; intent was to interfere with "horizontal" intracortical transmission
Denny-Brown and Botterell (1948)	1(FV) 2(FXIII) 3(FIV) 4(FIII) 5(FII)	r, a,♀ r, im,♀ r, a,♀ r, im,♀ r, a,♀	T(u) T(u) T(u) T(u) T(bs)	1 1 1 1 2	Y Y N Y Y	59 d 54 d 33 d 13 d 58 d	General behavior, reflexes, muscle tonus	Initial lesion in case 3 was incomplete and was expanded on day 32; case 9 spared forearm

Table I. (*Continued.*)

Reference & yr	Experiment[a]	Sp, age, sex[b]	Lesion[c]	M[a]	H[e]	Obs per[f]	Types of observations	Comments
	6 (FIX)	r, im, ♂	pos (u)	1	N	21 d		area; case 10 spared
	7 (FXVIIIL)	r, im, ♀	hl (u)	1	N	34 d		hind limb area;
	8 (FXXII)	ch.im, ♀	pos (u)	2	Y	125 d+		case 11 spared
	9 (FXIV)	r, im, ♂	T (u;inc)	1	Y	21 d		area 4 medial to
	10 (FXII)	r, a, ♀	T (u;inc)	1	Y	37 d		sup. pc gyrus; case
	11 (FXI)	r, a, ♀	T (u;inc)	1	Y	31 d		12 spared lateral
	12 (FVIII)	r, im, ♂	T (u;inc)	1	Y	26 d		anterior zone;
	13 (FXVI)	r, a, ♀	T (u;inc)	1	Y	10 d		case 13 spared
	14 (FXX)	r, im, ♂	ant (u)	1	Y	22 d		upper and lower
	15 (FXXI)	r, im, ♂	ant (u)	1	Y	27 d		anterior zone; concludes that motor function of Area 4 is less specific than commonly believed
Glees and Cole (1950)	1 (MCL1)	r, im, ns r, im, ns	th(u)	1	N	25 d	Description of hand/thumb movements; for some animals quantitative evaluation based on opening various test boxes	Brain removed and sectioned but no postop description of lesion provided except for Pansy; suggests that after lesions motor cortex acts in a less differentiated and more primitive manner
	2 (MCL2)	man, ns, ns r, im, ns	h(u)	1	N	14 d		
	3 (MCL4)	r, im, ♂	h,fa(u)	1	N	7 m		
	4 (MCL5)	r, im, ♂	th(u)	1	N	1 m		
	5 (Pan)	r, im, ♂	h(u)	1	N	5 d		
	6 (Pansy)	r, im, ♂	th(u)	1	Y	13 d		
	7 (Bimbo)		f(bs)	1	N	ns		
	8 (Giant)		f(bs)	1	N	2 d		
Travis (1952)[1]	1 (50–10)[1,2,4,5]	r,a, ♂	f,hl (bs)	3	Y	50 d	Reflexes, tonus and general behavior;	Almost no deficit in
Travis (1955a)[2]	2 (50–65)[1,2]	r,a,ns	h (u)	3	Y	4 w		animals with

Travis (1955b)[3] Travis and Woolsey (1956)[4] Hamuy (1956)[5]	3 (50–70)[1,2,5] 4 (50–74)[1,2,5] 5 (51–29)[3] 6 (51–30)[1,2] 7 (51–42)[1,2] 8 (51–44)[1] 9 (51–61)[1,2]	r,a,♂ r,a,♂ r,im,♂ r,a,♀ r,ns,ns r,a,♂ r,ns,ns	f(b) f,hl(bs) T(u) f(u) h(u) ant(b) h(u)	3 3 3 3 3 3 3	Y Y Y Y Y Y Y	5 m 10 w 1 m 4 w 4 w 8 m 6 w	Hamuy presents quantitative data on hand recovery using test boxes	unilateral hand lesions; showed that after ablations recovery dependent on numerous factors including sequence of operations, size of lesions, time for recovery, nursing care and exercise
Semmes and Chow (1955)	1(2) 2(5)	r,im,ns gr,im,ns	T(u) T(u)	3 3	Y Y	10 w 1 m	Reflexes and general behavior	Animals were subjected to subsequent contralateral lesions; interested in effects of pyramidal vs. extrapyramidal lesions
Liu and Chambers (1964)	1(m1) 2(m2)	r, ns, ns r, ns, ns	h(u) fo(u)	ns ns	Y Y	13 d 14 d	Behavior reported as similar to that described by Travis and Woolsey (1956)	Primarily interested in pattern and termination of corticospinal pathway

[a] Experiment: numbers and/or characters in parentheses refer to case numbers used in source material; ns = not stated; grp = group (number of animals within the groups are not detailed).

[b] Sp, age, sex = species, age, sex: r = rhesus macaque; ma = macaque (species not stated); mo = monkey (species not stated); l = lemur; c = capuchin; b = baboon; gr = green monkey; s = spider monkey; man = mangabey; p = patas (red) monkey; ch = chimpanzee; g = gorilla; gi = gibbon; ga = galago; po = potto; a = adult; im = immature; ns = not stated.

[c] Lesion: T = all or most of precental gyrus; hl = hind limb portion; f = forelimb; fa = face; fo = foot; t = tail; th = thumb; ant = anterior part of Area 4; pos = posterior part of Area 4; h = hand; t = thumb; u = unilateral; e = eyelid; he = head; ne = neck; b = bilateral; bs = serial bilateral removal; mvi = mutiple vertical incisions in precentral gyrus; inc = incomplete.

[d] M = method of lesion induction: 1 = extirpation by dissection; 2 = ablating cortex by cautery; 3 = suction; v = vertical incisions.

[e] H = Histological or photographic postmortem verification of lesion location: Y = yes; N = No; na = not applicable.

[f] Obs per = observation period: d = days; w = weeks, m = months; y = years.

Pertaining to the work of the 19th century Italian neuroanatomist, Luigi Luciani, Morabito, who recently published a biographical essay about him (Morabito, 2000), indicated to us (pers. comm.) that there was only one case involving primates in which a lesion was confined to the motor cortex (cf. Luciani and Seppilli, 1885). The remaining information presented about Luciani in the Table is derived from Morabito's essay and an English summary of Luciani's work (Luciani, 1884).

The following paragraphs explain aspects of the data presented in the Table.

Reference and Yr (year): In one cell in this column multiple citations are listed, i.e., the Travis grouping. In this case, differing aspects of identical animal experiments were reported in different articles; thus, we grouped all of the relevant citations together, listed all of the experimental animals in the second column, and used superscripts to designate the specific reports that provide details on each experiment.

Experiment: In certain reports (e.g., Leyton and Sherrington [1917]) all of the lesions were designed to be within the primary motor cortex and each animal was given a sequential number within the study. For these articles our "experiment" number corresponds to the number in the source article. In other studies, only some of the described cases involved lesions confined to the precentral gyrus (e.g., Fulton and Keller [1932]). In these situations we listed each relevant case numerically but placed in parentheses the associated case number used in the source article.

In some of the studies, animals were described as a group without specific statements detailing the number of animals used (e.g., Hines [1937]). In these cases we gave the animals a specific group number (e.g., grp1) to differentiate them from other animals discussed in the report. In all situations, our use of a "group" designation indicates that the number of animals studied was not disclosed.

Species, Age, and Sex: We compiled these data based on the information provided in the source article. For macaques we considered 4.0 kg and above to be adult. As indicated in the Table, some reports did not provide this information.

Lesion Area: For the purpose of compiling the bibliography we considered the precentral gyrus to be synonymous with the primary motor cortex or area 4 of Brodmann, although area 6 of Brodmann is essentially continuous with area 4. In most of the studies, stimulation procedures were used first to map out the designated area to be extirpated (e.g., hind limb area; stimulation procedures were especially necessary in the primate species in which a precentral gyrus is not discernable, e.g., lemurs). In the many reports by Fulton and colleagues the terms "hind limb" and "foot" areas appear to have been used interchangeably.

The Table indicates whether the lesion was unilateral, bilateral or bilateral and serial (i.e., a particular area was removed from both sides at different times). Also, if additional lesions were made in the same animal after the primary motor cortex lesion, we did not include this information in the Table.

Methods of Inducing Lesions: Since the earliest of these studies, only three methods have been used to make cerebral cortical lesions. The simplest method involves use of a spatula or similar tool to remove cortical tissue. The second uses an electrocautery to interrupt the arterial supply to the region of interest, which then may be removed. The third, subpial resection, involves aspiration by suction of cerebral cortical tissue after the pia has been incised in an avascular area and the tissue has been separated from the underlying white matter with a spatula. This technique has the advantage of preserving circulation through the pia to nearby regions of cortex and underlying white matter. Electrocautery was the most popular method during the late nineteenth and early twentieth centuries, especially among Fulton and his students. Nevertheless, as early as 1888, Horsley and Schäfer (1888) criticized the method. They stated, "We were led to adopt the knife instead of the galvanic cautery (which had been used by Professor FERRIER and YEO) on account of the greater facility with which the lesion can be limited exactly in depth and extent without risk of subsequent disintegration of the neighboring parts, while at the same time the bleeding is not markedly greater, and is usually readily stayed by gentle pressure," (p. 2). We consider this statement to be prescient, as lesions induced by cautery frequently include more cortical and subcortical tissue than can be appreciated at the time of operation. Subpial resection is generally considered to be the most accurate in matching planned and actual cortical ablations.

One study listed, Sperry (1947), was unique in that it was not exactly an ablation study; rather, vertical knife cuts were made in the cortex. It is included for the sake of completeness.

Histological or Photographic Verification: Our notation for this column was either Y (yes) or N (no). A "yes" response indicates one or both of the following conditions: 1) illustrations were provided that depicted the lesion postmortem; 2) the investigators stated that they had examined the brain postmortem and found lesions confined to the designated region and/or described the extent of the lesion.

Observation Period: These data are self-explanatory except for bilateral serial lesions in which the time listed is in all cases after the second lesion.

Types of Observations: This is self-explanatory.

Comments: This is self-explanatory.

RESULTS AND DISCUSSION

We hope that the information provided in Table I will assist contemporary primate functional neuroanatomists in utilizing valuable older studies. Although some contemporary investigators may believe that the "crude" methodology used before 1966 invalidates the work done during that period, we have previously demonstrated how contemporary researchers could have benefitted from examining some of the relevant earlier studies presented in the Table (Vilensky and Gilman, 2001). For example, Rouiller *et al.* (1998) reported for monkeys that reversible inactivation of the M1 hand area in the intact hemisphere did not affect the ipsilateral hand (contralateral to an earlier lesion). However, these authors indicated that they could not rule out the possibility that a larger lesion of M1 might have led to a reorganization of the intact hemisphere. It appears that, based on the larger lesions used in the early studies, there is good evidence that Rouiller *et al.*'s results could be extended to large M1 lesions as well. Thus Denny-Brown and Botterell (1948) stated, "The recovery of movement from an almost complete ablation of area 4 was not affected by ablation of area 4 from the opposite hemisphere (Experiment 5)," (p. 310). Similarly, for chimpanzees, Leyton and Sherrington (1917) stated, "Further, . . . the double arm area lesion showed clearly that the regaining of ability to use the limb could not be attributed to the arm area of one hemisphere taking over the functional powers of the arm area of the other hemisphere after the latter's ablation," (p. 207). Lashley (1924) also addressed this question and concluded that, "In no case has a recurrence of the motor symptoms produced by the first lesion been reported to follow the destruction of the corresponding area of the opposite hemisphere," (p. 8).

Another example is provided in recent work by Nudo *et al.* (1996) who demonstrated that retraining squirrel monkeys resulted in no functional loss of hand territory adjacent to an induced M1 infarct (as opposed to animals that did not receive the retraining). They concluded, ". . .after local damage to the motor cortex, rehabilitative training can shape subsequent reorganization in the adjacent intact cortex, and that the undamaged motor cortex may play an important role in motor recovery," (p. 1791). Thus, Nudo *et al.* demonstrated at least part of the neurophysiological mechanism for a concept with a long history, i.e., the importance of usage (retraining) of the affected structure. Specifically, Ogden and Franz (1917) reported that, after a precentral gyrus ablation, compulsory use of the affected limb and muscle/nerve stimulation greatly increased recovery in rhesus monkeys. Travis and Woolsey (1956) also recognized the importance of training after induced lesions in primates. They called it "assisted functioning" and believed it was critical to the animal's developing the ability to perform acts that initially it could not perform on its own.

A final example relates to the fact that, subsequent to inducing lesions, the experimental neurologists of the last century regularly evaluated the neurological status of the animals using techniques identical to those used on their patients (e.g., tendon reflexes, responses to pin pricks, examination of muscle tone, etc.). Such exams are no longer conducted, presumably because of animal care regulations that forbid such direct contact between the animals and their handlers. Nevertheless, the published results of these exams can contribute markedly to undestanding the behavior of lesioned animals. For example, Friel and Nudo (1998) described behaviorally and neurophysiologically the recovery of hand function in squirrel monkeys after small M1 infarcts. Among the measurements studied was the movement pattern the monkeys used to retrieve food pellets. The authors emphasized that three of the five animals modified their movement pattern after the infarct whereas two kept the same pattern. Of the three that changed, two had used a finger flexion paired with a wrist extension movement preoperatively, but none typically used this pattern postoperatively. The authors did not comment on this observation. It is reasonable that the absence of the finger flexion/wrist extension pattern postoperatively occurred because, as Denny-Brown and Botterell (1948) reported, M1 lesions result in spasticity with tonic contraction of the digital flexors, and extension of the wrist increases the flexor posture of the digits (see Fig. 64). This post-lesion synergistic activity would clearly interfere with an animal that was attempting to obtain pellets from wells using a finger flexion/wrist extension pattern. Similarly, after injection of muscimol into left motor cortex to inactivate it, Kubota (1996) reported maintenance of a hand in a posture similar to the one associated with radial nerve palsy in humans. The author hypothesized that the hand posture resulted from excessive inhibitory influences from the muscimol. However, this hand posture was typically observed after area 4 ablations in monkeys (cf. Fig. 64 in Denny-Brown and Botterell [1948]) and was, after neurological examination, associated with spasticity of the flexor musculature. Thus, Kubota's conclusion of increased extensor inhibition rather than increased flexor excitation appears unwarranted. Interestingly, the observed hand posture was initially described in 1917 (specifically referred to as a "wrist-drop") after a lesion of the arm area of a chimpanzee (Leyton and Sherrington, 1917).

Based on these examples, we believe it is justifiable to suggest to contemporary investigators of primate functional neuroanatomy that it is important to consult the early literature on this topic. The Table presented here should facilitate this consultation process by allowing them to quickly identify those studies that are particularly pertinent to their research projects (e.g., by species, limb segment involved, behavior analyzed, etc.).

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