

## Prognosis and Course of Bell's Palsy

### II. Electromyographic Study

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**Summary.** In 87 patients with ischemic palsy of the facial nerve, an electromyographic examination of five mimic muscles was made with a needle electrode. Denervation, voluntary activity and terminal latencies were determined in relation to the prognosis. The patients were divided into three groups according to the extent of their recovery. In the group with *complete recovery* (36 cases) terminal latencies were normal in half the patients. In the other half they are slightly above the level of double standard deviation (SD); in the first two weeks electrical activity is absent during voluntary motion in 10% of the muscles only. Denervation potentials are present in 38% of the patients in the first two months. In the groups with *incomplete recovery*, two subgroups can be distinguished with synkinesis. The first subgroup (11 cases) consists of patients with initially normal terminal latencies: a marked prolongation appears in the 1st—4th month. In the second month, a rich denervation activity occurs particularly in *M. frontalis* and *depressor labii inferioris*. Voluntary activity has been present since the onset; what is striking is a small deficiency in *M. orbicularis oculi*. This group can be distinguished from the first group in the second month. In the second subgroup (6 cases), terminal latencies have been markedly prolonged since the onset of palsy or electrical excitability of the nerve is absent. In the 2nd to 3rd month voluntary activity appears in the EMG record and there are rich denervation potentials. Classification is possible in the 5th month. In comparison with the following group, voluntary motion improves in this period and indirect muscular excitability is present, while denervation activity is absent. In the group with defective recovery (34 cases) with synkinesis, contracture, and insufficient muscular strength, markedly prolonged terminal latencies already appear in the second week after onset or the nerve is not excitable. The frontal branch generally preserves its electrical excitability. Terminal latencies become stabilized around the 12th month and are higher than in the normal state. Electrical activity in voluntary contraction appear in the 2nd to 4th month, most frequently in *M. orbicularis oris* and *M. frontalis*. Denervation potentials are richest in the 2nd and 3rd month, especially in *M. orbicularis oris* and *M. zygomaticus*, surviving the longest in *M. frontalis* until the 6th month. Classification is possible after examination in the 2nd week and the 5th month.

**Key words:** Facial Paralysis — Electromyography — Prognosis.

**Zusammenfassung.** Bei 87 Patienten mit einer ischämischen Facialisparese wurden elektromyographisch mittels einer Nadelelektrode je 5 Muskeln untersucht. Es wurden die Denervations- und Willküraktivität und die Werte der Terminallatenzen im Hinblick auf die Prognose der Paresen gewertet. Die Probanden wurden gemäß Folgeerscheinungen in 3 Gruppen aufgeschlüsselt.

In der Gruppe ohne Defekterscheinungen (36 Fälle) sind die Werte der Terminallatenzen bei der Hälfte der Patienten normal, bei der anderen Hälfte liegen sie gering über der Grenze der zweifachen SD (Standard deviation). In den ersten 2 Wochen fehlt die elektrische Aktivität bei der Willkürbewegung bloß in 10% der Muskeln. Denervationspotentiale sind in den ersten 2 Monaten bei 38% der Erkrankten zu beobachten. In der Gruppe mit leichten Folgeerscheinungen (Synkinesen) können wir zwei Untergruppen unterscheiden. In der ersten (11 Fälle) haben die Patienten am Anfang normale Terminallatenzen. Zu einer ausgeprägten Verlängerung kommt es im 1. bis 4. Monat. Im 2. Monat zeigt sich eine reiche Denervationsaktivität, insbesondere in den Mm. frontalis und depressor labii inferioris. Die Willküraktivität findet sich seit dem Beginn der Parese, auffällig ist der diskrete Befall des M. orbicularis oculi bei einigen Kranken. Eine Unterscheidung von der ersten Gruppe ist im 2. Monat möglich. In der zweiten Untergruppe (6 Fälle) sind die Terminallatenzen seit Paresebeginn sichtlich verlängert oder der Nerv ist elektrisch nicht reizbar. Im 2. bis 3. Monat kommt es zu einer Willküraktivität im EMG-Muster und es zeigen sich reiche Denervationspotentiale. Die Einreihung in dieser Krankengruppe ist im 5. Monat möglich. Gegenüber der nächsten Gruppe ist die Willkürmotorik zu dieser Zeit besser. Es zeigt sich eine indirekte Muskelreizbarkeit, die Denervationsaktivität fehlt. In der 3. Gruppe mit schwersten Folgen (34 Fälle) mit Synkinesen, Kontrakturen und Muskelkraftherabsetzung sind schon in der 2. Woche der Paresedauer die Terminallatenzen ausgeprägt verlängert oder der Nerv ist elektrisch unreizbar. Beim Frontalast ist am häufigsten die elektrische Reizbarkeit erhalten. Die Terminallatenzen stabilisieren sich gegen den 12. Monat und sind gegenüber der Norm erhöht. Die elektrische Aktivität bei der Willkürkontraktion erscheint im 2. bis 4. Monat, am häufigsten in den Mm. orbicularis oris und frontalis. Die Denervationspotentiale sind am reichsten im 2. und 3. Monat, vor allem in den Mm. orbicularis oris und zygomaticus. Am längsten überdauern sie im M. frontalis bis zu 6 Monaten. Die Einstufung in diese Gruppe ist gemäß Kontrolluntersuchung in der 2. Woche und im 5. Monat möglich.

*Schlüsselwörter:* Facialisparese — Elektromyographie — Prognose.

The first part of our paper on the prognosis and the course of ischemic palsy of the facial nerve dealt with the analysis of the clinical picture. Now we attempt to solve this problem with the assistance of electromyographic (EMG) and latency response examination. Prognosis of palsy has been investigated by a great number of authors, who used a simple EMG investigation [9, 11, 12, 24], a stimulatory EMG method [1, 4—6, 8, 22, 23] with special regard to appraisal of nerve conduction velocity and latency response. Other authors use a nerve excitability test (NET) as an orienting method [2, 3, 10, 13, 16, 17]. It is not easy to compare these reports as each examination period is different and the classification of the recovery is not uniform. Also very often facial nerve palsies of various origin are appraised.

### Material and Method

From the total number of 87 cases of ischemic palsy of the facial nerve, 86 cases were examined. Clinical features of this group were presented in the first part of our paper [19]. Examination was undertaken of insertion and denervation

activity, the patterns of voluntary contraction and the results of stimulatory examination. All patients were repeatedly examined; slight cases within six months, severe cases at least within one year in irregular intervals of 1 to 3 months. The patients were divided into three groups according to the degree of their recovery. Results of their clinical examination were presented in detail in our previous paper and are summarised in the conclusions.

A concentric facial needle electrode 13K51 (DISA) was used; the electrode was punctured into 5 muscles under examination: into pars orbitalis of lower eye-lid of *M. orbicularis oculi* (C), into the lateral portion of *M. frontalis* (F), into the upper portion of *M. zygomaticus* (Z), into the upper lip above the corner of the mouth of *M. orbicularis oris* (R), into the centre of *M. depressor labii inferioris* (Q) caudally beneath the corner of the mouth. The needle formed a right angle with the surface of the skin. Insertion activity was observed immediately after the insertion of the needle. The record was registered on the oscilloscope of a two-channel electromyograph DISA 14A20. Stimulation of *N. facialis* near tragus was performed in all patients where superficial stimulating bipolar electrode 13K62 DISA was applied and the place was stimulated by Ministim DISA impulse of right angle shape lasting 0.5 msec, the intensity being up to 350–400 V. The intensity of pulses was increased until maximum response was obtained; supramaximum stimulus was used. Summation potential latency was measured directly on the oscilloscope by means of a shifting shining point. The patients were examined when lying on their backs in a room where the temperature was 22–24°C.

21 control persons without metabolic disorders and without any symptoms of lesions of the peripheral nervous system were examined and latency times for each muscle were given (Table 1).

Table 1. Latency response in 21 healthy persons

Muscle	Number of persons	Average age	Mean latency value msec	Standard deviation (SD) msec	Upper level in twofold SD msec
<i>M. frontalis</i> (F)	21	37.29	4.26	0.38	5.02
<i>M. orbicularis oculi</i> (C)	21	34.38	2.85	0.31	3.47
<i>M. zygomaticus</i> (Z)	21	37.67	2.63	0.41	3.45
<i>M. orbicularis oris</i> (R)	21	35.81	3.43	0.47	4.37
<i>M. depressor labii inferioris</i> (Q)	21	35.81	3.02	0.40	3.82

## Results

Evaluation of denervation activity. Positive were interpreted as denervation activity sharp waves and prolonged insertion activity during at least 10 sec after the insertion of the needle into the muscle. To avoid wrong registration of nervous potentials the position of the needle tip was changed several times.

In Tables 2–4 present a survey of the incidence of denervation activity in each group of the patients and in each muscle. To correctly appraise the importance of the denervation activity with regard to the

Table 2. Incidence of denervation potentials in muscles examined in the first two months and in the course of 6—12 months

Group	Muscles examined	Within 2 months	Total sum in the course of 6—12 months
1st group	total	239	252
	from this denervation potentials present in	27 (11%)	27 (10%)
2nd group	total	122	224
	from this denervation potentials present in	53 (43%)	70 (31%)
3rd group	total	153	759
	from this denervation potentials present in	103 (67%)	158 (20%)
Total number of examined muscles			1235

Table 3. Incidence of denervation potentials in patients

Group	Number of examined patients in the first two months	From that the number of patients where denervation potentials were found	% of the patients with denervation potentials
1st	34	13	38%
2nd	16	16	100%
3rd	21	21	100%

prognosis of the disease, we evaluated its incidence within the first two months of the duration of palsy (Table 2). To determine the incidence of denervations in individual patients, we divided the patients into two groups. The first group contained those in whom denervation potentials were found at least once and those in whom no potentials were found at all (Table 3). On the adjoining Table 5 we can see the distribution of denervation activity in each muscle and group (according to Table 4).

Conclusion to the prognosis of palsy. If denervation potentials are revealed in the second week of the disease, it is not an ominous symptom as in other peripheral palsies. Such a patient can achieve complete recovery. If rich denervation activity is noticed in the first and second month and voluntary activity is preserved in at least three quarters of the muscles, prognosis can be relatively favourable and recovery can be incomplete and not always defective.

Table 4. Denervation activity found in patients of single groups throughout the course of palsy

Group	Number of examined muscles	Examination time									
		Week		Month							
		1.	2.	3.	1.	2.	3.	4.	6.	over 6	
1st	Total	48	103	25	24	39	13	—	—	—	
	With denervations	3 (6%)	13 (13%)	3 (12%)	4 (17%)	4 (10%)	—	—	—	—	
2nd	Total	6	24	30	15	47	37	22	43	—	
	With denervations	—	9 (37%)	12 (40%)	5 (33%)	27 (57%)	13 (35%)	3 (14%)	1 (2%)	—	
3rd	Total	5	10	23	30	85	55	61	127	363	
	With denervations	—	3 (30%)	14 (60%)	21 (70%)	65 (76%)	25 (45%)	13 (21%)	10 (8%)	7 (2%)	

Table 5. Denervation activity found in single muscles and groups throughout the course of palsy

Group		Time of examination																											
		1 week		2		3		1 month			2		3		4		6		over 6		total								
		I	II	III	I	II	III	I	II	III	I	II	III	I	II	III	I	II	III	I	II	III							
F	1	—	—	5	3	1	—	5	4	2	3	3	1	6	14	—	2	8	—	5	—	1	5	—	4	9	20	44	
C	—	—	—	—	1	—	—	1	—	—	—	1	—	1	9	—	1	5	—	1	2	—	1	—	—	1	—	4	20
Z	—	—	—	—	2	—	—	2	—	—	—	6	—	5	15	—	1	3	—	—	—	—	—	—	2	—	10	28	
R	1	—	—	5	2	1	2	3	3	—	1	6	1	7	15	—	4	5	—	1	3	—	4	—	—	—	9	18	37
Q	1	—	—	3	1	1	1	2	4	2	1	5	2	8	12	—	5	4	—	1	3	—	—	—	—	—	9	18	29
Total	3	—	—	13	9	3	3	12	14	4	5	21	4	27	65	—	13	25	—	3	13	—	1	10	—	7	27	70	158

F, M. frontalis; C, M. orbicularis oculi; Z, M. zygomaticus; R, M. orbicularis oris; Q, M. depressor labii inferioris. I, 1st group; II, 2nd group; III, 3rd group.

Conclusion with regard to the course of the disease. In Group 1 denervation potentials appear most often in the second week of the disease whereas they completely disappear in the second or third month. They do not appear in all muscles. We did not find them in *M. orbicularis oculi* and in *M. zygomaticus* (Table 5). In Group 2 denervation activity appears in all the patients in the first two months. It is very rich and lasts always for a long period after the needle insertion. Least frequently it was found in C, the most frequently in F, R, Q (Table 5). In this group it disappeared after the 4th month. In the Group 3 fibrillation potentials were also present in all the patients in the first two months. However, they were not so rich as in the foregoing group. Most often they were found in R and F (Table 5). They appeared for the longest period in F—as late as after the 6th month. From these results it follows that nervous branches in the trunk of the facial nerve leading to F and R bring about very rich denervation activity in ischemia, that is in such cases where no serious disorganisations of the inner structure of the nerve occur.

Evaluation of the voluntary activity in EMG. We determined 6 degrees of the intensity of the electrical curve in voluntary contraction: degree 0—isoelectric line, no voluntary activity, 1st degree—pattern of single motor unit potentials, 2nd degree—reduced mixed pattern, 3rd degree—mixed pattern, 4th degree—rich mixed pattern, 5th degree—interference pattern. We observed the period during which there was no voluntary activity in the EMG examination and we followed its revival in the course of palsy. Figs. 1—3 show both the percentage of muscles without any function and the percentage of muscles where the density of the curve is of the third or higher degree. The number of muscles was expressed in percentage to enable us to compare the unequal, absolute numbers of the muscles examined in each interval.

Group 1 (Fig. 1a and b) consisted of cases where clinical examination in the first week after onset revealed 20—50% muscles were without any function and the electromyographic examination only 10% muscle were without function. Thus we could far more precisely define the prognosis of palsy. Interference patterns were obtained in most patients in the course of the first and second month of the disease. In Group 2 (Fig. 2a and b) the percentage of non-functioning muscles decreased as against the clinical picture as shown by EMG examination. We did not manage to take advantage of this phenomenon for prognostic evaluation. In all muscles, restoration of voluntary activity was found in the third month or shortly afterwards. 10—30% of the muscles revealed a reduced mixed pattern. The first movement recorded in EMG was noticed in 14 selected patients. In 5 of them there was no response to the electrical stimulation of the nerve.

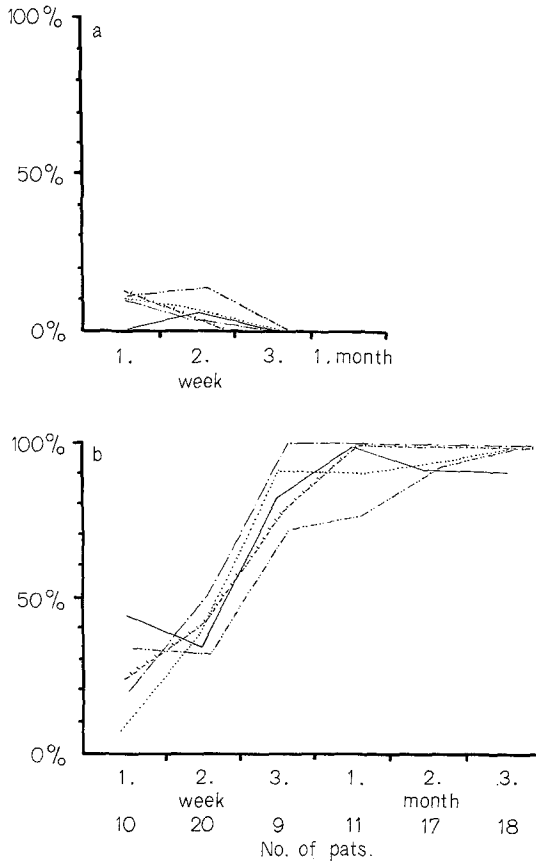


Fig.1. a 1st group. Loss of electrical activity in volitional contraction in the EMG examination in palsy improvement (in the course of palsy). Ordinate: % of the muscles without electrical activity in attempt at volitional contraction. Abscissa: time of examination. b 1st group. Return of electrical activity in EMG examination. Ordinate: % of the muscles which have during a voluntary contraction type of mixed or interference pattern. Abscissa: time of examination. Number of examined patients is given below the time data, this is valid for Fig.1a and b. M. frontalis — · — · —, M. orbicularis oculi — · — · —, M. zygomaticus —, M. orbicularis oris · · · · ·, M. depressor labii inferioris — | — | —

The first movement was found out in:

	1st	2nd	3rd week	1st	2nd	3rd month
Number of patients	1	4	3	3	1	2

78% of these patients revealed movement in one or more muscles in one month, 100% within three months.

In Group 3 (Fig.3a and b) we can see a noticeable lowering of the number of non-functioning muscles by about 30% in comparison with

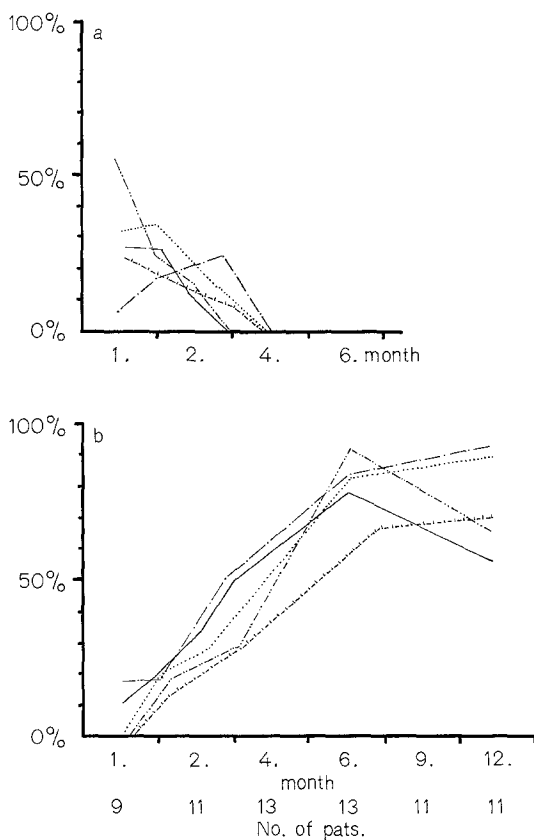


Fig. 2. a 2nd group. Loss of electrical activity during voluntary contraction in the EMG examination in the course of palsy. b 2nd group. Return of electrical activity in EMG examination. Legend is the same as in Fig. 1

clinical examination. However, no prognostic use of these data is possible. An increase of the percentage of non-functioning muscles in the second month is caused by a considerable increase of the patients with a severe form of this disease, who were at that time examined for the first time. Electrical activity in muscles is restored in the 3rd to 4th month. Since the 6th month nearly half of the muscles reveal a reduced mixed pattern. Only 5% of the muscles remain without any function up to the sixth month. We chose 23 patients with a severe form of palsy—in 14 of them the nerve was not irritable on stimulation—and we followed the restoration of electrical activity in the course of the disease.



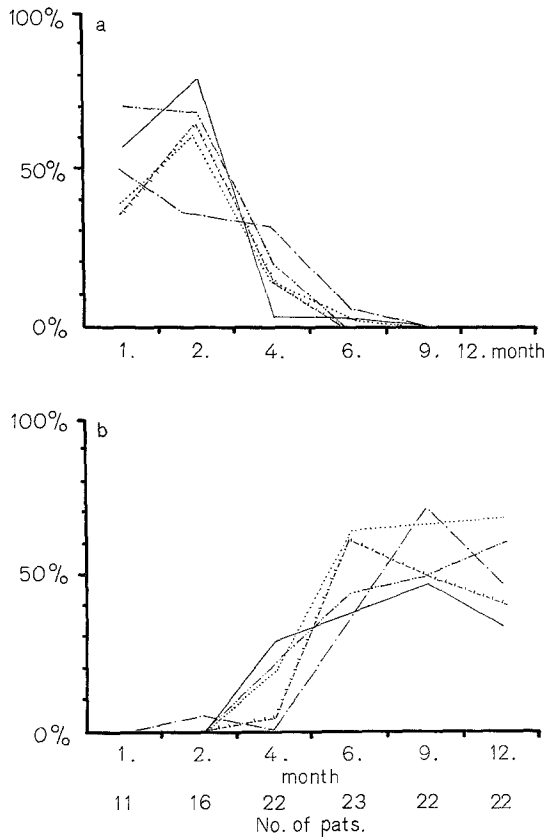


Fig. 3. a 3rd group. Loss of electrical activity in voluntary contraction in EMG examination. b 3rd group. Return of electrical activity in EMG examination. Legend is the same as in Fig. 1

First movement in:

	3rd week	1st	2nd	3rd	4th month
Number of patients	4	4	8	4	3

From 23 patients 70% revealed movement in one or more muscles during the 2nd month and 86% within the 3rd month. In 3rd to 4th month three or five muscles revealed motions.

Conclusion regarding palsy prognosis. Establishing voluntary activity in EMG is of prognostic importance only in benignant forms of palsy (Group 1) where already in the first week we find palsies in 90% of the muscles with electrical activity during attempts of voluntary contraction. Isolated interpretation of voluntary activity in the Group 2 and 3 can lead to a wrong conclusion.

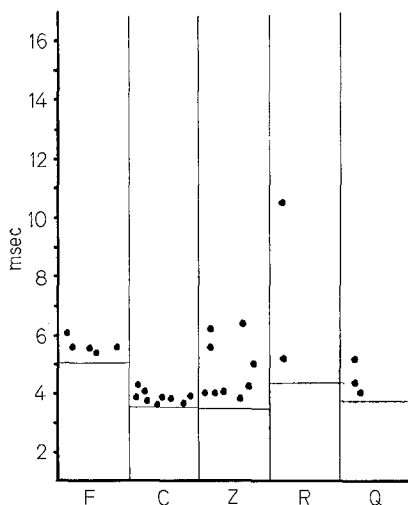


Fig. 4. 1st group. The highest measured values of latency of response in first 3 months of the palsy in 35 patients. Only values above the level of twofold standard deviation (SD) are recorded. M. frontalis—F, M. orbicularis oculi—C, M. zygomaticus—Z, M. orbicularis oris—R, M. depressor labii inferioris—Q. Level of twofold SD —. Ordinate: values of latency of response in msec. The muscle is not electrically excitable  $\infty$

Conclusion with regard to the course of palsy. In Group 1 of the patients, we find electrical activity in 90% of the muscles already in the first week of the disease. The contraction curve returns to normal in the 1st and 2nd month. In four fifths of the patients from the Group 2, voluntary activity in the EMG record was found in at least one muscle in the 1st month. In about the third month it appears in all muscles. The curve in the final state is rare in 30% of the muscles. In Group 3 restoration of voluntary activity in EMG examination is seen in two thirds of the patients during the 2nd month and in one third of the patients during the third and fourth month. In the final stage considerable rarefaction of the EMG curve can be seen in the voluntary innervation in approx. 50% of the muscles.

Results of latency time examinations. 86 patients were examined by the latency of response which has already been described. The results were classified according to each group of recovery (Figs. 4—10).

Group 1 of recovery. Values of increased terminal latencies in single muscles are recorded on the 4th graph as taken in the first three months. To make the arrangement clearer no values extending beyond twofold standard deviation (SD) have been recorded. In F, C and Q terminal latencies are only mildly increased and do not extend beyond the value

Table 6. Prolonged latencies of response in 17 patients of the first group in the first three months and in single muscles

		Examination						
		Time:	1.	2.	3. week	1.	2.	3. month
Number of muscles with prolonged latency of response	in F	1	3	1	1	2	0	
	in C	0	5	0	3	3	1	
	in Z	1	6	0	1	2	2	
	in R	0	1	0	1	1	0	
	in Q	0	0	0	1	1	1	
Total		2	15	1	7	9	4	
Number of all muscles where latency of response was stated	in F	10	22	7	5	8	2	
	in C	9	21	5	5	9	1	
	in Z	8	20	5	5	10	3	
	in R	10	21	8	5	11	4	
	in Q	8	20	5	5	6	2	
Total		45	104	30	25	44	12	
Prolonged latency of response appeared in % of all the examined muscles:		4%	14%	3%	28%	20%	33%	

of 5 msec. In Z and R higher values of latency times were found, reaching to over 5 msec in 4 patients: three times within a fortnight and once in the second month of the duration of palsy. On the whole a mild increase of latencies was found in 17 patients, that is in 50%. From Table 6 an obvious tendency to higher incidence of prolonged latency times was found in the first and third month, that is during the time when muscular weakness is returning to normal and the function of the nerve in the sense of muscular activity is also returning to a normal level. That is also the time when denervation potentials disappear and process of nervous fibres regeneration is being finished.

Conclusion regarding prognosis of palsy. If latency times are within the physiological range or if they are only slightly prolonged (in C, Z, R, Q lasting up to 5 msec and in F up to 6 msec), the prognosis is favourable. We can reach this conclusion in the second week of the palsy.

Conclusion regarding the course of palsy. In 50% of the patients, latency times are quite within the physiological range whose upper level is given by twofold SD. 50% of the patients have latency times slightly prolonged (in C, Z, R and Q lasting up to 5 msec and in F up to 6 msec). Prolongation can be of greater extent in Z and R in the second week as well as in the 1st and 2nd month because these muscles are the

most sensitive to an ischemic disturbance of N. facialis. Obviously in these branches the thickest nervous fibres are loosing the ability to transfer an electrical impulse by ischemic disturbance which causes a slight prolongation of the latencies. This mild prolongation does not favourable influence the course of palsy until the voluntary mobility is restored. The greatest percentage of muscles thus affected is found in the period of restitution of nervous activity, that is within the 1st and 2nd month.

Group 2 of recovery. This is a very unhomogeneous group of patients as far as the results of stimulatory EMG are concerned (see Figs. 5—7). It includes 17 cases. They can be divided into two subgroups. The first includes 11 patients with normal or nearly normal values of latency times in the first fortnight and with voluntary activity in most muscles preserved in clinical as well as EMG examinations. According to the above mentioned, criteria these patients should recover within 2 or 3 months. During a chek-up in the second month, however, latency times of several muscles, especially of M. orbicularis (R) and M. zygomaticus (Z) are found to be markedly prolonged, rich denervation activity appears and voluntary movement returns to normal much more slowly than in the Group 1 of the patients. The face assumes symmetry during movements in approximately the 5th month of the disease. Four patients

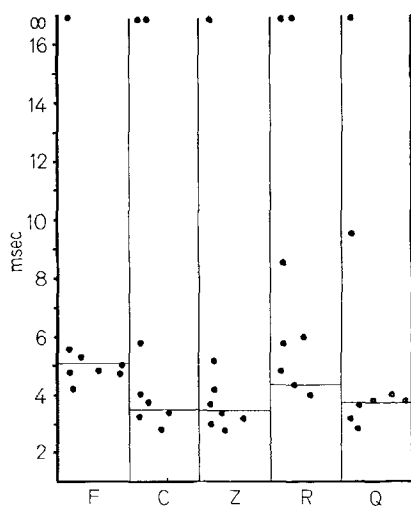


Fig. 5

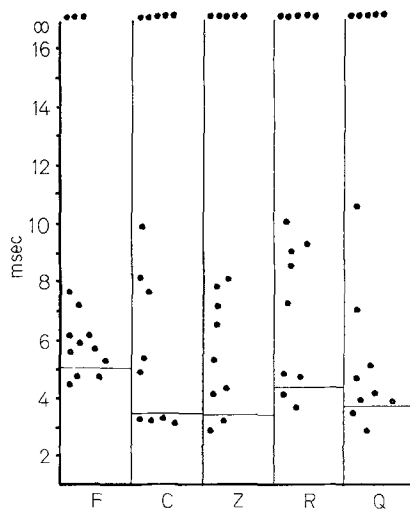


Fig. 6

Fig. 5. 2nd group. The highest values of latencies in the first 3 weeks in 8 patients. Legend is the same as in Fig. 4

Fig. 6. 2nd group. The highest values of latencies in the 1st to the 4th month in 14 patients. Legend is the same as in Fig. 4

suffered from transgeniculate lesion, two from suprachordal and five from infrachordal lesions. Muscular strength was restored in all patients. Marked synkinesis appeared in the 4th to 6th month. Prolonged latency times were found in the 1st to the 4th month in various muscles most often in Z and R. After this period they rapidly returned to normal level. The second subgroup contains 6 patients. We found a considerable prolongation of latency times in the first weeks and often also a complete loss of electrical excitability of the nerve. Still, the patients were recovering surprisingly well, even though the EMG findings corresponded with a heavy affection of nervous fibres. In the second and third month voluntary activity was always present at least in the EMG recording and often a rich denervation activity was noticed. Voluntary mobility returned to a normal level in the 7th month. Synkinesis appeared after the 4th month. Three patients suffered from transgeniculate lesion and three from infrageniculate lesion. In each subgroup one patient was decompressed. In both patients who underwent operation, an edem of the nerve was found in vertical section of the channel and during histological examination of the nerve sheath no inflammatory changes were found. In this group of patients the primary damage of the nerve is benignant. Inner structure of the nerve is generally preserved. The nerve recovers slowly but well. In the whole group we found prolonged

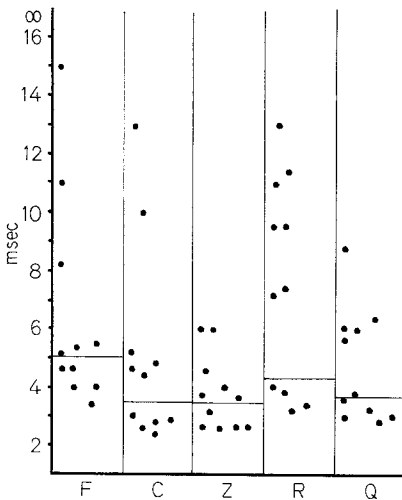


Fig. 7

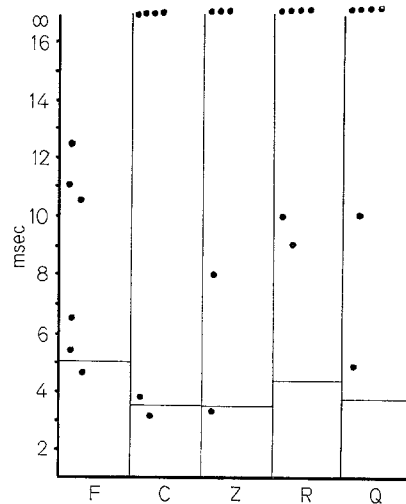


Fig. 8

Fig. 7. 2nd group. The highest values of latencies in the 5th to 12th month in 11 patients. Legend is the same as in Fig. 4

Fig. 8. 3rd group. The highest values of latencies in the first 3 weeks in 6 patients. Legend is the same as in Fig. 4

terminal latencies after 6 months in 8 muscles and 5 patients (in F 1  $\times$ , C 1  $\times$ , R 3  $\times$ , Q 3  $\times$ ). In that period the strength was symmetrical in most muscles. Four of these five patients belonged in the 2nd subgroup.

Conclusion with regard to prognosis. In the Group 2 of patients we found two sorts of results. The 1st subgroup contains such patients whose terminal latencies are nearly normal in the first fortnight, in the 2nd month they get conspicuously prolonged especially in M. zygomaticus and M. orbicularis oris and rich denervation activity appears. Voluntary movement has been partly preserved from the onset of the palsy but is progressing slowly. In the 2nd month we can differentiate this subgroup from the patients of Group 1.

Damage is more extensive in the 2nd subgroup. Terminal latencies are conspicuously prolonged from the very beginning and often the nerve is not electrically excitable. In the 2nd and 3rd month voluntary activity can be observed and simultaneously rich denervation potentials are seen in EMG recording. About the 7th month movement is symmetrical.

Conclusion with regard to the course of palsy. Terminal latencies are normal in the 1st subgroup from the beginning and they become prolonged especially in the 1st to 4th month, mainly in Z and R. After this time they quickly recover.

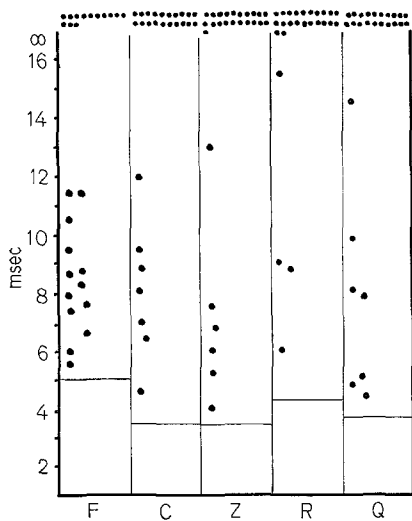


Fig. 9

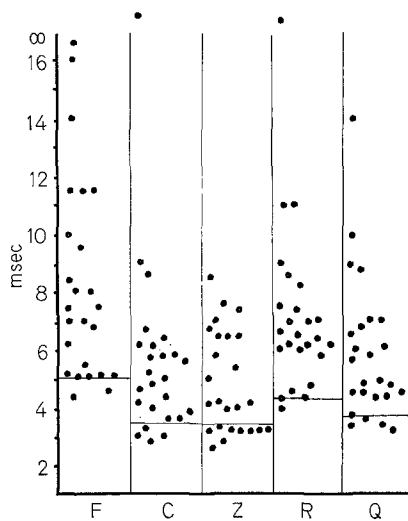


Fig. 10

Fig. 9. 3rd group. The highest latency values in the 1st to 6th month in 27 patients. Legend is the same as in Fig. 4

Fig. 10. 3rd group. The highest latency values in the 7th to 12th month in 25 patients

Terminal latencies are conspicuously prolonged in the 2nd subgroup or the nerve is not electrically excitable from the onset of palsy. If the nerve is not excitable at the onset of the disease, this quality is restored in the 4th month. Still, as late as in the 9th or 12th month prolonged latencies can be found. This fact provides evidence of a permanent lesion of the myelin sheath of the nervous fibres or the total destruction of the thickest nervous fibres. As in about 30% of the patients from the Group 2 the curve of voluntary contraction is rare, the destruction is more likely to take place. 30% patients correspond to the number of patients in the 2nd subgroup from the total number of patients in Group 2.

Group 3 of recovery. Figs. 8—10 present values of latency times measured in the first three weeks from 1st to 6th and from 7th to 12th months. The highest recorded value was taken only once in each patient although he was examined several times. This rule applies to the foregoing groups, too. Increased terminal latencies or a loss of electrical excitability is noticeable at the end of the first or second week. From Fig. 8 we can see that only sporadical muscles have normal values of latency times. In this sense the finding is more conspicuous than in the Group 2 of the patients. What strikes us about Fig. 8, and especially Fig. 9, is the fact that the loss of excitability of F is by about one third less frequent than in the other muscles. In the course of palsy, latencies are permanently prolonged in the period of the 1st to 6th month, if they appear at all. In this period we did not find any permanently normal value in any of the muscles (Fig. 9). After the 6th month, latency times are decreased even if their values fluctuate conspicuously during repeated examinations. When we observed the lowest latencies in 25 patients after the 6th month of the disease and examined 125 muscles, 17 muscles were found with markedly prolonged latencies: in F  $5\times$ , in C  $1\times$ , in Z  $2\times$ , in R  $7\times$  and in Q  $2\times$ . From this fact we deduce that latencies in F and R improve very slowly. As seen from Figs. 9 and 10, electrical excitability is restored after the 6th month.

Conclusion with regard to prognosis of palsy. Conspicuously increased latencies or loss of electrical excitability can be observed in most muscles, at the earliest, at the end of the 1st week, and is definitely established in the 2nd week. It is during the first three weeks that most often preserves its indirect muscular excitability.

Conclusion with regard to the course of palsy. Latency times are conspicuously prolonged from the 1st to the 6th month. After this time a tendency to lower latency times appears but the times still fluctuate from one examination to the next. However, they do not become quite normal even as late as about the 12th month. Most often they

are prolonged in R and F and in these cases the function of these muscles is very bad. Indirect muscular excitability is restored in most cases after the 6th month.

### Discussion

As far as the importance of the denervation activity is concerned, some authors agree with our findings which state that fibrillation potentials are not always a prognostically unfavourable symptom [1, 6, 12]. However, Kaeser and Pfaltz [11], Hiestand *et al.* [9] reached different conclusions. These authors consider the incidence of denervation at the end of the first month as an unfavourable symptom. Denervation activity is not always a manifestation of neuromuscular disorder. Mayer [15] found fibrillation potentials in hand muscles in 81% of healthy people when the arm was compressed for 30 min. This ischemic provocation moment can also occur in our patients of the Group 2 and leads towards a rich incidence of denervation activity. After all, fibrillation can occur also in central palsies [7]. Fibrillation potentials were already found in the 1st week of the course of palsy. In addition, Taverner [21] found them already during the 5th day although their commonest incidence is after a fortnight [14]. Establishing of voluntary activity only makes our information about the preserved neuromuscular connection more accurate. It has prognostic importance only in Group 1. The best improvement was found in M. orbicularis oculi. Kocher [12] found the best regeneration ability in M. frontalis.

Even in Group 1 latency times were prolonged in 50% of the patients. A more striking prolongation was recorded in M. zygomaticus and in M. orbicularis oris. Latency times of these muscles react most sensitively to the lesion of the facial nerve. This was also observed in the lesion of the facial nerve trunk in neurinome N. VIII. and in facial hemispasm where, when upon decompression, a mechanical obstacle in the channel was discovered [18, 20]. The analysis of the Group 2 of patients is of the greatest interest. This group contains such patients in whom a rapid improvement of palsy can be wrongly diagnosed (refer to Group 1) or, on the contrary, a very unfavourable prognosis can be made in the acute period (refer to Group 3). Results of latency time of this subgroup gives suggest that loss of electrical excitability of the nerve does not mean complete denervation but that this block can be reversible. Claes *et al.* [5] also share this view. From the total number of patients with symptoms of unfavourable prognosis in the first two weeks the number of patients included in the 2nd subgroup can show the delayed favourable influence of the treatment. It is interesting that both the 1st and the 2nd subgroups recover with practically the same results, with only one exception, namely that the restitution is slower



in the 2nd subgroup. Mechanism of nerve lesion are perhaps the same but the nerve lesion is more intensive.

In the Group 3 latency times are variously prolonged. This shows that remyelination of axons is not perfect and thus conduction velocity is lowered, too.

It is obvious that in ischemic palsy, examination of one or two muscles is not sufficient, as often stated in literature. It is necessary to examine at least four muscles: *M. frontalis*, *M. orbicularis oculi*, *M. zygomaticus*, *M. orbicularis oris*. Further it should be noted that prognosis and course of the palsy of the facial nerve of a different etiology, e.g. diabetic or traumatic, are subject to other laws.

### Conclusion

On the basis of the complete analysis of 87 cases of ischemic palsy of the facial nerve, prognosis and course of the disease were established. Patients are divided into three groups.

Group 1 contains those who have completely recovered. Group 2 contains patients with synkinesis with a symmetry of face, both during movement and at rest. Group 3 contains patients who suffer from asymmetry of face in motion and sometimes even at rest, with synkinesis and contracture. Each group is characterized clinically and electromyographically.

*Group 1* (36 patients). Values of latency times are normal in 50%; in the second half they are above the level of a twofold standard deviation. Examination is reliable after the first week of duration. These values may be higher in *M. zygomaticus* and in *M. orbicularis oris*. This increase survives until the 3rd month. In the first two weeks only 10% of the muscles are without electrical activity. Voluntary activity is found in all muscles in the 3rd week. Interference pattern is restored in the 2nd or 3rd month. 38% of the patients reveal denervation potentials in *M. frontalis*, *M. orbicularis oris* and *M. depressor labii inferioris* in the first two months. They disappear in the 2nd or 3rd month. Clinically obvious movement is preserved in 50% of the muscles in the 2nd week of palsy and is restored in all muscles during the 3rd week. *M. zygomaticus* and *M. depressor labii inferioris* are the most often affected while *M. orbicularis oculi et oris* are the least affected. Movement improves first in *M. frontalis* and in *M. orbicularis oculi*. The nasopalpebral reflex is not always recalled at that time. Bell's phenomenon is negative. Disturbance is infragenuate during the examination of topognosis.

*Group 2* is divided into two subgroups.

*1st Subgroup* (11 patients). Patients have normal or nearly normal latency times in the first two weeks. These are prolonged in first or

fourth month especially in *M. zygomaticus* and in *M. orbicularis oris*. Rich denervation activity appears in the 2nd month, esp. in *M. frontalis* and in *M. depressor labii inferioris*. Voluntary activity is present at the very beginning of the disease in EMG record and often also clinically. *M. frontalis* and *M. orbicularis oculi* are the most often preserved while *M. orbicularis oris*, *M. zygomaticus* and *M. depressor labii inferioris* are the most often affected. Muscular strength is restored slowly. Face is symmetric during movement in the 5th month.

*2nd Subgroup* (6 patients). Disturbance is more sensitive. Latency times are conspicuously prolonged since the beginning and often the nerve is not electrically excitable. In the 2nd or 3rd month voluntary activity is restored and simultaneously rich denervation potentials are present which disappear after the 4th month. Muscular movement is symmetric around the 7th month. Latency time appears about the 4th month. In examination of topognosis, lesion in transgeniculate level is fairly frequent.

*Group 3* (34 patients). As early as in the 2nd week latency times are conspicuously prolonged or the nerve is not electrically excitable. These findings deteriorate between the 1st and 6th month. Indirect muscular excitability is most often preserve in *M. frontalis* and is generally restored after the 6th month. Latency times are stabilized about the 12th month. Active movement in EMG recording is obvious after the 2nd to 4th month. During voluntary contraction approx. one half of the muscles has reduced interference patterns. Denervation activity is the richest in *M. orbicularis oris* and in *M. zygomaticus* in the 2nd and 3rd month. It survives the longest in *M. frontalis*. At the onset of palsy 80–100% of muscles are without any function, at clinical examination *M. zygomaticus* and *M. depressor labii inferioris* are affected most, while *M. orbicularis oris* is affected least. Movements clinically reappear between 6th and 9th month. Muscular strength is restored insufficiently, the best improvement being in *M. orbicularis oculi* at about 9th and 12th month.

Time table of examination. Classification into the first group is possible in the 2nd week of palsy. Classification into the 2nd group is possible after the first month when patients of the first group have already been separated. Classification into the 2nd subgroup is possible in the 5th month when patients of the third group have been separated.

### References

1. Buchthal, F.: Electromyography in paralysis of the facial nerve. *Arch. Otolaryng.* **81**, 463–469 (1965).
2. Campbell, E. D. R.: A simple prognostic test in facial palsy. *J. Laryng.* **77**, 462–466 (1963).

3. Campbell, E. D. R., Hickey, R. P., Nixon, K. H., Richardson, A. T.: Value of nerve-excitability measurements in prognosis of facial palsy. *Brit med. J.* **1962** *II*, 7—10.
4. Carbone, F.: Etude electromyographique de la paralysie de Bell. *J. neurol. Sci.* **7**, 219—228 (1968).
5. Claes, C., Jacobs, K., Smets, J.: L'electromyographie des paralysies faciales. *Acta neurol. belg.* **65**, 491—507 (1965).
6. Ernst, K.: Elektromyographische Untersuchungen bei peripherer Fazialisparese. *Psychiat. Neurol. med. Psychol. (Lpz.)* **19**, 139—141 (1967).
7. Goldcamp, O.: Electromyography and nerve conduction studies in 116 patients with hemiplegia. *Arch. phys. Med.* **48**, 59—63 (1967).
8. Granger, C. V.: Toward an earlier forecast of recovery in Bell's palsy. *Arch. phys. Med.* **48**, 273—278 (1966).
9. Hiestand, P., Kaeser, H., Kocher, R., Pfaltz, C. R.: EMG und Fazialisdekompression bei Bell'scher Lähmung. *Pract. otorhino-laryng. (Basel)* **31**, 144—153 (1969).
10. Jongkees, L. B. W.: Test for facial nerve function. *Arch. Otolaryng.* **89**, 127 to 130 (1969).
11. Kaeser, H. E., Pfaltz, C. R.: Elektromyographische Verlaufsuntersuchungen bei Fazialisparesen. *Confin. neurol. (Basel)* **23**, 256—269 (1963).
12. Kocher, R.: Die periphere Fazialisparese. *Schweiz. Arch. Neurol. Psychiat.* **99**, 247—269 (1967).
13. Laumans, E. P. J.: Nerve excitability test in facial paralysis. *Arch. Otolaryng.* **81**, 478—488 (1965).
14. Marinacci, A. A., von Hagen, K. O.: The prognosis in Bell's palsy. *Bull. Los Angeles neurol. Soc.* **36**, 83—92 (1971).
15. Mayer, K.: Klinik und Elektromyographie der Spontanaktivität des menschlichen Skelettmuskels. Berlin-Heidelberg-New York: Springer 1965.
16. Richardson, A. T.: Electrodiagnosis of facial palsies. *Ann. Otol. (St. Louis)* **72**, 569—580 (1963).
17. Saadé, B., Karam, F.: Simple electrodiagnostic test for Bell's palsy. *J. Amer. med. Ass.* **195**, 824—826 (1966).
18. Steidl, L.: Results of electromyographic examinations of the facial nerve in expansive process on the posterior cranial fossa (czek.). *Čas. Lék. čes.* **109**, 1146—1150 (1970).
19. Steidl, L.: Prognosis and course of Bell's palsy. Part 1: Clinical study. *Arch. Psychiat. Nervenkr. (in press)*.
20. Steidl, L., Černý, L.: Contribution to surgical treatment of facial hemispasm (czek.). *Čs. Neurol.* **32**, 140—145 (1969).
21. Taverner, D.: Bell's palsy. *Brain* **78**, 209—228 (1955).
22. Taverner, D.: Treatment of facial palsy. *Arch. Otolaryng.* **81**, 489—493 (1965).
23. Waylonis, G. W., Johnson, E. W.: Facial nerve conduction delay. *Arch. phys. Med.* **45**, 539—547 (1964).
24. Wigand, M. E.: Die Prognose der idiopathischen (Bellschen) Fazialisparese bei elektromyographischer Indikationsstellung zur Dekompressionsoperation. *Z. Laryng. Rhinol.* **46**, 439—451 (1967).

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