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Properties of after-discharges from cortical electrical stimulation in focal epilepsies

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Abstract

Objective: This study sought to determine whether certain aspects of after-discharges (ADs) obtained during cortical functional mapping provide better correlations between stimulus site and that of spontaneous seizures. Secondly, we wished to determine the percentage of stimulations evoking ADs and, of these, the percentage which clearly involves more than one electrode position, potentially inaccurately localizing cortical function. Thirdly, we wished to quantify the incidences of the several AD morphologies described by Jasper [in: Epilepsy and the functional anatomy of the human brain, 1954, p. 183; p. 692] and to assess whether certain morphologies had a greater tendency to evolve in frequency or morphology.

Methods: In these 29 patients requiring invasive recordings to determine principal epileptogenic areas, only subdural strips were placed in 19 patients, only grids in 2 patients, and both in 8 patients. A median of 21 electrodes per patient was stimulated of a median of 63 electrodes placed, with the following parameters: biphasic, monopolar, 50 Hz, 0.3 ms pulse duration, 1.5–18 mA. Coverage involved the frontal and parietal lobes (9 patients), frontal parietal temporal lobes (8), frontal temporal (3), temporal (2), occipital (2) and occipital temporal (2) with other combinations in 3 additional patients. Classification of AD morphologies was determined by a pilot study using IFSECN definitions [Electroenceph clin Neurophysiol 1974;37:538] and descriptions by Jasper [in: Epilepsy and the functional anatomy of the human brain, 1954, p. 183] and Gloor [in: Advances in neurology, vol. 8, 1975; p. 59].

Results: Four hundred and two ADs (12%) were elicited by 3358 trains of electrical stimuli of which 260 (65%) clearly involved more than only the stimulated electrode position. Thus, 260 (8%) of 3358 stimulations evoked an AD that could mislocalize cortical function. The proportion of stimulating electrodes eliciting ADs ranged among patients from 4 to 83% (median 33%). Polyspike bursts and sequential spikes were the most common AD morphologies. Ten percent of ADs evolved in morphology, frequency or both. Evolution occurred more commonly (44%) with rhythmic waves than with other AD morphologies (7%). Neither evolving ADs, ADs producing clinical seizures, or ADs exceeding 10 s correlated topologically with spontaneous seizure origins.

Conclusions: Although occurring in a minority of cortical electrical stimuli, ADs may involve more than the stimulus site and therefore may inaccurately localize cortical function. Our material failed to disclose any consistent relationship between the site of stimulus eliciting ADs and that of spontaneously appearing seizures, even when certain aspects of such ADs were analysed.

Significance: These data illustrate the need for scrutiny of the post-stimulus electrocorticogram for ADs and particularly those involving extra stimulus sites. Whether varying the stimulus parameters prospectively will disclose a better seizure localizing value for ADs remains to be determined.

Keywords: Afterdischarge; Electrical stimulation; Focal epilepsy

1. Introduction

Consisting of repetitive epileptiform potentials, rhythmic waves or both that follow a precipitating stimulus; after-discharges (ADs) have become a potentially delusive by-product of cortical electrical stimulation since their initial portrayal by Adrian (1936) in that: (1) cortical stimulus sites eliciting them have not consistently correlated with those of spontaneous seizure origin (Jasper, 1954b; Wieser et al., 1979), and (2) by spreading to adjacent areas, they may elicit clinical manifestations not representative of the stimulus site. Paradoxically, 50 years before, Sir Victor Horsley (Horsley, 1886) first stimulated the human cortex to

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reproduce seizures clinically typical for the first patient to undergo surgical removal of an epileptogenic lesion. Several subsequent studies of the topological relationship between after-discharges and spontaneous seizures have yielded inconsistent results. Jasper (1954b) reported that the region whose stimulation produced the longest or the only after-discharge was the origin of spontaneous seizures in 75% of cases. Among 38 patients with single epileptogenic foci in the temporal, frontal or 'posterior' regions, Bernier et al. (1990) reported that concordances for site between spontaneous and electrically induced seizures from depth recordings were 88, 92 and 100%, respectively. However, Wieser et al. (1979) in a stereoelectroencephalographic study of 133 temporal lobe patients found only a 77% correlation. Asking whether certain aspects of after-discharges would provide better correlations, we studied: (1) those exceeding 10 s, (2) after-discharges whose morphology or frequency evolved, and (3) those leading to clinically typical or atypical seizures.

Regarding the second deceptive component, Walker (1949) found ADs in neighbouring gyri 2–3 cm from the stimulus point in 15% of cases. Therefore, to estimate the extent that after-discharges complicate functional mapping sessions we assessed the following proportions: (1) of stimuli evoking after-discharges, (2) of electrodes, when stimulated, were associated with after-discharges, and (3) of after-discharges initially and subsequently involving more than one electrode position.

Jasper (1954b) apparently first depicted the several after-discharge morphologies in humans which can be grouped as: (1) sequential spikes (Jasper types A, B, C, H, L), (2) sequential spikes with pauses (K, G), (3) spike-waves (D, F, J), and (4) rhythmic waves (E, I). We determined the proportion of each type in our material and their propensities to evolve as electrographic seizures. Our scalp EEG study of spontaneous seizures (Blume et al., 1984) revealed that clinical seizures evolve in morphology, frequency or both, and that rhythmic waves were the most common onset pattern. Will this association between rhythmic waves and evolution apply to electrically stimulated seizures?

Finally, although threshold for AD production and spontaneous seizure origin could not be systemically studied, data relative to this matter were obtained.

2. Method

Subdural strips, grids or both were placed to identify or further delineate epileptogenic areas. Ambiguity of non-invasive clinical and laboratory data arose when more than one epileptogenic area was implicated, a mesial or inferior surface epileptogenesis was suspected, or when a large cortical epileptogenic lesion was present. Median age of the 29 patients was 21 years (range 6–39 years). Coverage involved the frontal and parietal lobes in 9 patients; frontal-parietal-temporal lobes (8); frontal-temporal (3); temporal (2); occipital (2); occipital-temporal (2); frontal including Rolandic (1); occipital-parietal-temporal (1) and parietal-temporal (1). Electrodes were inserted over both hemispheres in 18/29, only left hemisphere in 8 and only right hemisphere in 3. Only strips were inserted in 19 patients, only grids in 2, and both in 8. A median of 63 (range 35–112) electrodes per patient was inserted of which a median of 21 (range 8–63) was stimulated to assess the following functions: language (23 patients); motor (22); somatosensory (14) and vision (5). Although functional mapping, not seizure evocation, was the purpose of this cortical stimulation, all epileptogenic foci were stimulated in 16 patients, 50% or more in 4, a minority in 4 and none in 4. No seizure origin was ever determined in one patient.

Subdural grid and strip electrodes were 38 mm^2 stainless steel disks imbedded in silastic at 10 mm intervals, designed and made in our laboratory. Subdural strips were placed through burrholes or by craniotomy if performed for grid placement. Stereo skull films disclosed electrode locations for most patients. In some, post-implantation computerized tomography images were superimposed upon pre-implantation magnetic resonance imaging to display anatomical relationships (Bihari et al., 1999). A Biomedical Monitoring Systems, Inc. (BMSI) telemetry system recorded clinical seizures and after-discharges (ADs). Usual settings were: HLF 70, LLF 1.0, sensitivity 50–70 μV/mm. Anti-epileptic medication was not altered for stimulus sessions, having been variously reduced for the investigative period.

To localize motor, somatosensory, and visual functions and language, 50 Hz biphasic monopolar stimuli with 0.3 ms pulse durations and 1.5–18 mA strengths were employed. At each electrode, stimulation was commenced at 1.5 mA with 1 mA increments for each subsequent stimulation until a functional alteration, an AD or 18 mA was obtained or attained. Calcarine and Rolandic cortices were stimulated for 3–4 s as more immediate responses were expected while association cortices received 5–6 s stimulus trains, all with at least 20 s intervals. Post-stimulus artefact lasted ~1.5–2 s. Stimuli were usually sequentially applied to adjacent electrodes. Recordings were referential to a subgaleal electrode to correspond to our electrocorticogram and subdural method, adopted because of easier identification of morphologies and of topography (Blume and Girvin, 1982). Seventeen patients underwent one stimulus session, 10 had two sessions, and 2 had more than two.

To identify and classify after-discharges, a pilot study was performed based on the following sources. (1) The definition of after-discharge by the International Federation of Societies For Electroencephalography and Clinical Neurophysiology (Chatrian et al., 1974) as "an EEG seizure pattern following repetitive electrical stimulation of a discrete area of the brain via cortical or intracerebral electrodes." (2) Its description by Gloor (1975) of "rhythmic, usually stereotyped activity which may assume a variety of forms: high frequency spikes, rhythmic sharp

waves, and others." Gloor further stipulated that the wave pattern should differ distinctly from pre-stimulation activity. (3) Morphologies depicted by Jasper (1954b) when the after-discharges attained "maximum amplitude and regularity." His 12 illustrations can be classified into (a) sequential spikes at various rates (Jasper types A, B, C, H, L), (b) sequential spikes with distinct ~300–500 ms pauses (K, G), (c) spike-waves (D, F, J), and (d) rhythmic waves (E, I). As we wished to examine AD evolution, they (ADs) were classified when a stable, i.e. persisting for 2 s, identifiable morphology appeared; this occurred within 2–3 s of onset. The following categories emerged from this pilot study. (1) Rhythmic waves, defined as sequential waves of approximately constant period (Chatrian et al., 1974). (2) Bursts of polyspikes (multiple spikes), occasionally repeating at 1–4 Hz; this phenomenon resembles polyspike-waves but lacks a prominent following delta wave; the bursts occupy ≤ 50% of the phenomenon. (3) Spike-waves at 1–4 Hz; this category resembles #2 except that spikes are usually single and the slow wave is equal to or higher than the spike. (4) Sequential spikes with or without pauses; i.e. more sustained than a burst (Chatrian et al., 1974) and resembling Niedermeyer's (1999) "Runs of Rapid Spikes"; this category differs from polyspike bursts as spikes occupy the majority of this phenomenon. As with any classification involving potentially continuous variables, phenomena occasionally straddled its borders.

The occurrence, morphology, evolution, duration and location of ADs were visually assessed by two authors (W.T.B. and D.J. or P.P.). Evolution of AD was defined as a progressive change in the AD morphology, as a twofold or greater increase in frequency of the same morphology, or both (Blume et al., 1984).

3. Results

3.1. Incidence, extent and durations of after-discharges (ADs)

Four hundred and two ADs (12%) were elicited by 3358 trains of electrical stimuli applied to these 29 patients, and always involved the stimulating electrode. One hundred and fifty-four ADs initially appeared at a single electrode; 131 at two adjacent electrodes; 70 at 3 contiguous electrodes and 47 at 4 contiguous positions. Of those initially at a single electrode, 142/154 (92%) remained principally there, while 12 spread to involve equally or principally other electrode positions. Thus 260/402 (65%) of ADs exhibited more than minimal involvement of > 1 electrode position, and thus at least 10 mm spread. Therefore, 260 of 3358 stimulations (8%) evoked an AD that could potentially have mislocalized cortical function, if not recognized. Stimulus artefact prevented distinction between initial involvement of multiple electrodes and prompt spread from a single site. Proportions of stimulated electrodes eliciting ADs varied widely among patients from 4 to 83% (median 33%). Omitting further stimulation at sites producing functional change likely accounted for this variability. The incidence of ADs was unaffected by a spontaneous electrographic or clinical seizure on the EEG during the stimulus session: 177 (13%) of 1350 stimulations in 12 patients with a spontaneous seizure during the session compared to 225 (11%) of 2008 stimulations in 17 patients without one. Average AD durations for individual patients ranged between 1–5 s in 3 patients, 6–10 s (14), 11–15 s (6), 16–20 s (3) and over 20 s (3).

3.2. Morphologies and electrographic evolution

Stimuli evoked a wide variety of AD morphologies, polyspike bursts and sequential spikes being the most common (Table 1 and Figs. 1–5). All morphologies assessed in the 10 patient pilot study encompassed those of the 29 patients. Borders between sets of categories were occasionally blurred such as: rhythmic waves and sequential spikes, sequential spikes with pauses and polyspike bursts, and polyspike bursts and spike-waves. Distinct inter-stimulus morphological variability occurred at the same or adjacent electrode positions in 18 (62%) patients. Thirty-nine (10%) of the 402 ADs evolved and therefore, 1% of all stimuli. Rhythmic waves evolved to constitute electrographic seizures more commonly than any other wave-form: 14 (44%) of 32 compared with 25 (7%) of the 370 with other morphologies ($P =< 0.0001$; Fisher's exact) (Table 1).

The presence of a spontaneously occurring electrographic or electrographic/clinical seizure during the stimulation session did not influence the percentage of evolving ADs: 20 (11%) of 177 stimuli with a spontaneous seizure as compared to 19 (8%) of 225 stimuli without this. Fifteen ADs in 14 patients led to clinical seizures of which 9 ADs exhibited evolution (Table 2). Of these 9, rhythmic waves were the initial morphology which evolved in 6 and sequential spikes were the morphology in 3. Non-evolving morphologies associated with a clinical seizure were principally spike-waves and sequential spikes (Table 2). Except in patient 5, stimulus sessions were aborted when a clinical seizure was evoked.

Table 1
Initial AD morphologies

Morphology type	ADs	ADs evolved
Rhythmic waves	32 (8%)	14 (44%)
Rhythmic waves + spikes	6 (1%)	0
Polyspike bursts	163 (41%)	12 (7%)
Spike-waves (1–3 Hz)	55 (14%)	1 (2%)
Sequential spikes (with and without pauses)	146 (36%)	12 (8%)
Total	402	39 (10%)

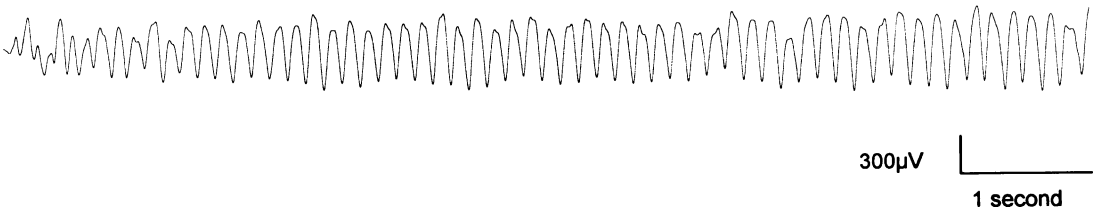

Fig. 1. Rhythmic waves. Sequential waves of approximately constant period. The junction between each wave in this example is apiculate (sharply contoured).

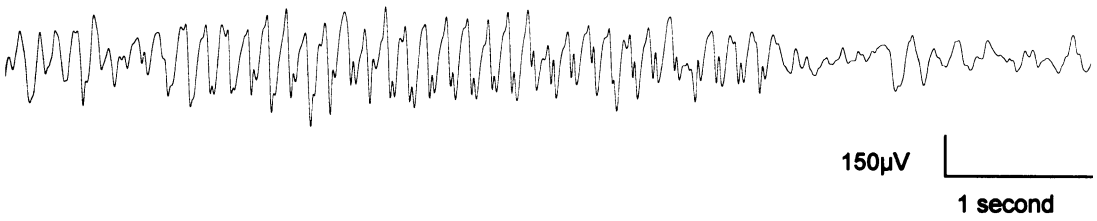

Fig. 2. Rhythmic waves evolving into spike waves.

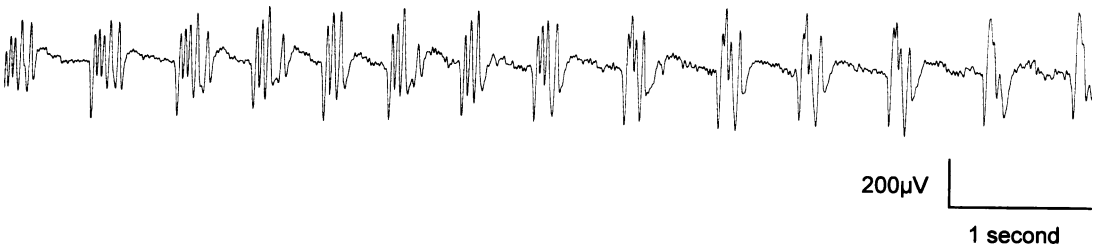

Fig. 3. Polyspike bursts. Although these resemble spike-wave discharges, the wave component appears minimally.

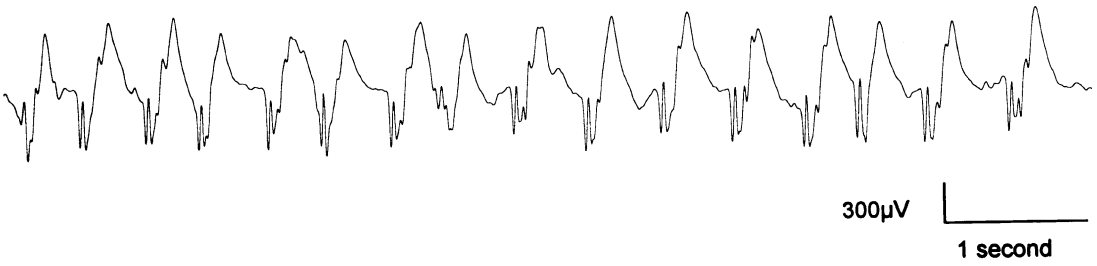

Fig. 4. Spike-waves. These resemble polyspike bursts but the subsequent slow wave is far more pronounced.

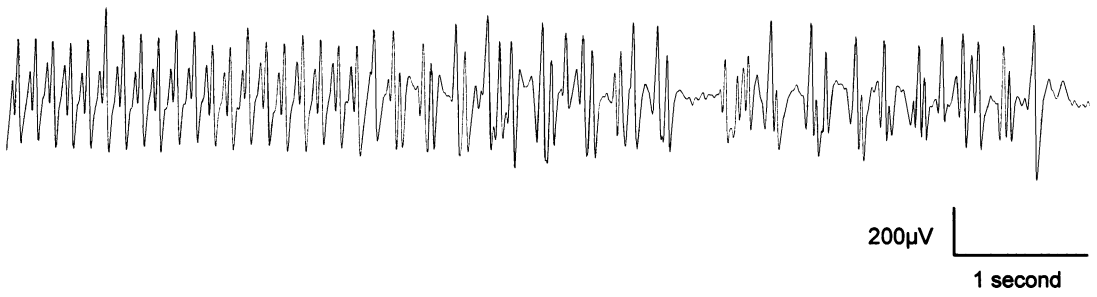

Fig. 5. Sequential spikes which appear initially without a pause and subsequently with a pause.

Table 2
Morphology and duration of after-discharges leading to clinical seizures

Patient no.	Type	Initial AD morphology	Evolution	AD duration (s)[a]
1	T	11 Hz polyspikes	Yes	300
2	T	8 Hz spikes	No	7
3	A	10–11 Hz spikes	Yes	27
5	T	2.5 Hz spike waves	No	10
5	T	2 Hz spike waves	No	5
6	A	14 Hz rhythmic waves	Yes	153
7	T	5 Hz rhythmic waves	Yes	12
9	A	20 Hz spikes with pauses and 2–3 Hz spikes	No	6
10	T	2 Hz spikes and polyspikes	Yes	48
12	T	20 Hz irregular waves and 10 Hz spikes with pauses	No	11
17	T	8 Hz rhythmic waves	Yes	12
19	T	20 Hz rhythmic waves	Yes	42
20	A	2–3 Hz spike-waves	No	38
23	A	8 Hz rhythmic waves	Yes	20
24	T	10 Hz semi-rhythmic waves	Yes	43

T, A: evoked seizure clinically typical, atypical.
[a] Prior to any clinical seizure.

3.3. After-discharges and spontaneous seizure origins

Neither evolving ADs, ADs producing clinical seizures, nor ADs exceeding 10 s consistently correlated topologically with spontaneous seizure origins (Table 3).

3.4. Thresholds

Stimulus thresholds producing ADs or highest stimulus strength failing to produce ADs did not vary significantly among regions when averaged among patients (Table 4). The considerable intra-region variability of threshold is reflected in the substantial standard deviations of values of Table 4.

Table 3
Origins of afterdischarge and spontaneous seizures: topographic correlation

	Congruent	Overlapping	Adjacent	Distant
Evolving ADs[a]	9 (23)	3 (8)	6 (15)	21 (54)
ADs with clinical seizures[b]	5 (36)	2 (14)	2 (14)	5 (36)
ADs ≥ 10 s	5 (19)	5 (19)	4 (15)	12 (46)

Congruent, majority of seizure onset positions same as majority of AD onsets, assessing all clinical seizures and AD onsets; Overlapping, minority of seizure and AD onset positions in common; Adjacent, at least some seizures begin adjacent to some AD onsets; Distant, no onset or adjacent positions in common. Numbers are ADs. Percentages calculated across in parentheses. Each patient had one ictal onset zone, with minimal interseizure variability.
[a] All 39 evolving ADs in 20 patients.
[b] One per patient (14).

4. Discussion

Four limitations of this study are recognized: (1) after-discharges occurred incidentally in stimulation for functional mapping; (2) subdural electrode coverage and stimulated electrodes, while both substantial, were chosen to ethically solve clinical questions and thus have limitations; (3) this study is retrospective; and (4) stimulus artefact often obscured immediate morphologies.

4.1. Morphology

Jasper (1954a,b), Creutzfeldt (1969) and Ajmone Marsan (1972) described the following AD morphological sequence as the most typical in both experimental and human conditions: low voltage asynchronous activity; a rhythmic discharge of oscillating waves or uninterrupted sequential spikes; sequential spikes or polyspike bursts interrupted by attenuation or slow waves and finally arrhythmic delta and attenuated activity. However, Jasper (1954b) found a variety of morphologies among samples from eleven patients taken when "the discharge reached maximum amplitude and regularity." As indicated above, Jasper's morphologies can be grouped into the following categories: (a) polyspike bursts, (b) sequential spikes with and without distinct pauses, (c) spike-waves, and (d) rhythmic waves. His morphologies also appeared in our study which found polyspike bursts and sequential spikes to occur most commonly (Table 1). In a minority of patients the ADs may have been categorized at an earlier point in our study, i.e. when a persistent, identifiable morphology appeared, as 10% of ADs evolved (Table 1). As definitions and illustrations indicate, such categories merge somewhat: rhythmic waves with sequential spikes, sequential spikes with pauses with polyspike bursts, and polyspike bursts with spike-waves. Jasper indicated that anatomical and physiological specificities of the cortical region, pathological condition of the cortex and stimulus parameters all can influence AD morphology. Such factors may underlie the variety of morphologies found in our study and others (Jasper, 1954b; Luciano et al., 1993), but AD morphologies varied notably within cortical areas in 18 of our 29 patients precluding any regional specificity as described by Jasper (1954b).

As stimulus artefact may occlude initial AD components, the variety of initially perceptible morphologies may also simply reflect varying rates of progression through the aforementioned sequence described by Jasper, Creutzfeldt and Ajmone Marsan.

4.1.1. Evolution

The following data from experimental work provide a possible explanation for the greater propensity of rhythmic waves to evolve than other AD morphologies.

Several authors (Gerin, 1960; Ajmone Marsan, 1961; Kandel and Spencer, 1961; Gloor et al., 1962; Sawa et al., 1963; Creutzfeldt, 1969; Sugaya et al., 1964; Sypert et al.,

Table 4
After-discharge thresholds (mA) by region

	Prefrontal	Premotor	Rolandic	Supplementary motor area	Temporal convexity	Parietal	Occipital
Lowest stimulus strength producing AD							
Sample size	5	3	10	10	7	11	7
Average	7.4	4.83	4.95	5.0	4.57	5.59	3.64
SD	5.378	1.258	4.045	2.427	4.659	4.116	2.231
Highest stimulus strength producing no AD							
Sample size	5	5	8	9	7	10	7
Average	11.2	7.8	7.125	8.67	9.71	10.55	7.71
SD	3.633	4.102	3.758	3.953	4.222	3.708	2.767

Sample size = number of patients. ANOVA for: (a) lowest strength: $P = 0.7680$, not significant; (b) highest strength: $P = 0.3357$, not significant. Inferior temporal region minimally stimulated in 3 patients only.

1970) have indicated that membrane depolarization precedes and presumably is indispensable for an AD to occur in experimental seizure activity induced by electrical stimulation. Extracellular-recorded 10–30 Hz rhythmic waves (fast runs) are associated with sustained membrane depolarization (Steriade et al., 1998). Such waves represent neuronal membrane oscillations associated with recurrent, transient further depolarizations (Spencer and Kandel, 1969; Li, 1955; Matsumoto and Ajmone Marsan, 1964a; Sawa et al., 1963; Ayala et al., 1970). Such oscillations have an approximately 10–20 Hz frequency range (Sypert et al., 1970; Sugaya et al., 1964). In this phase, neuronal and initial segment action potentials do not occur (Sugaya et al., 1964; Sypert et al., 1970; Kandel and Spencer, 1961; Creutzfeldt, 1969) although occurrence of more distally originating action potentials remains possible. In contrast, interrupted EEG spikes and specifically waves of spike-waves occur during the subsequent phase of membrane repolarization then hyperpolarization (Sugaya et al., 1964; Goldensohn and Purpura, 1963; Sypert et al., 1970; Kandel and Spencer, 1961; Kostopoulos et al., 1982). During this phase, recovery of action potential generation occurs (Sypert et al., 1970).

Sequential EEG spikes, associated with paroxysmal depolarization shifts and superimposed action potential bursts (Creutzfeldt, 1969), may elicit surround inhibition (Goldensohn and Purpura, 1963; Prince and Wilder, 1967; Matsumoto and Ajmone Marsan, 1964b; Li, 1959). In most cells of the inhibitory surround the IPSPs are preceded by brief excitatory postsynaptic potentials (EPSPs) (Prince, 1969). With decreased or lack of neuronal action potential discharges during the phase of membrane oscillatory activity (see above) surround inhibition may not develop then. In fact, inhibitory surround IPSP activity is inhibited during an ictal discharge (Prince and Wilder, 1967; Sawa et al., 1963). Therefore, one may postulate that the significantly higher (44%) proportion of rhythmic wave ADs evolving in morphology and frequency than any other AD feature may be consequent to a lack of effective inhibition at this stage.

4.2. After-discharges and epileptogenesis

4.2.1. Thresholds

Pinsky and Burns (1962), in one of the few systematic investigations of threshold stimulus parameters for AD production, studied ADs evoked by electrically stimulating unanaesthetized feline cortical slabs. They found the following thresholds: pulse duration, 0.3 ms (uniform value for all cortices); strength, 3.4 mA (mean); frequency, ~4 Hz;, number of stimuli, 40–80 (range), therefore 2–4 s at a rate of 20/s. Pinsky and Burns (1962) concluded that a minimum density of stimulated cortical neurones was required and that AD duration was independent of pulse duration, stimulus strength and number of stimuli once threshold values were exceeded. They concluded that an AD is produced by a stimulus that causes a critical number of neurones to discharge a critical number of times. This 'all or nothing,' as opposed to a graded, response suggested that local factors determine the subsequent evolution and duration of the AD. As ADs can be elicited by lower values of these parameters in certain experimental models of epilepsy (Walker and Johnson, 1948; Matsumoto and Ajmone Marsan, 1964a), one might presuppose that epileptogenic zones could be so identified in humans. However, lower neuronal density and other abnormalities in epileptogenic zones may raise the AD threshold (Cherlow et al., 1977). Additionally, a wide range of threshold stimulus strengths producing ADs (2 to > 15 mA) has been found in humans with epilepsy (Lesser et al., 1984). Importantly, Lesser's data reflect considerable overlap in AD threshold ranges among neocortical regions. Our data (Table 4) also disclose a generous range of stimulus strengths producing and not producing ADs among patients with ample inter-region overlaps. Moreover, the considerable inter-patient standard deviations for AD thresholds within each region and the fact that localization of function was our main purpose for stimulation both discouraged any attempts to localize epileptogenesis by AD thresholds.

4.2.2. Location

An electrically evoked long after-discharge in an area of principal interictal spikes and in association with a patient's habitual initial ictal symptoms has been said to serve as "confirmatory evidence of an already identified epileptogenic focus" (Luciano et al., 1993). Although Jasper (1954b) affirmed "a close correlation between a spike focus and the area of susceptibility to prolonged after-discharge" in a majority of patients, he cites cases of discrepancy where resection of the "spike focus" abolished seizures and others in whom removing the after-discharge area was ineffective. In our study the locations of ADs failed to reliably correlate with those of spontaneously occurring seizures when the ADs lasted longer than 10 s, evolved in frequency or morphology, or led to clinically typical seizures (Table 3). Our data therefore fail to confirm the seizure-localizing value of ADs exceeding 10 s (Luciano et al., 1993; Jasper, 1954b). Luciano et al.'s review cites several sources indicating that the morphological dissimilarity between spontaneous and electrically evoked clinically similar seizures diminishes the localizing value of the latter. Indeed, Gloor (1975) concluded "The localization of the areas from which after-discharges can be elicited are of doubtful validity in outlining the patient's epileptogenic region".

4.3. EEG monitoring during functional mapping

Our 12% incidence of AD in protocol-structured function localization sessions and the 65% of ADs involving > 1 electrode stress again that performance of electroencephalography is requisite: stimulation-induced ADs may distort functional maps as discharges may obviously or subtly spread (Luciano et al., 1993; Ajmone Marsan, 1972, 1973; Gloor, 1975; Lesser et al., 1984, 1991; Wyler, 1987).

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