EFFECT OF MESCALINE AND L.S.D. ON EVOKED RESPONSES ESPECIALLY OF THE OPTIC SYSTEM OF THE CAT [1]

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In 1898, Heffter reported his own experiences after ingestion of four different Peyotl alcaloids (Anhalonium Lewinii). He established that the previously described subjective disturbances evoked by this drug were due to mescaline (for references, see Heffter 1898; Beringer 1927; and Morselli 1950). These disturbances are recently vividly described in "The doors of perception" (Huxley 1954) and involve most spectacularly the visual system. Colors take on a new vividness, there are optical illusions and hallucinations with moving and colored patterns. More complex psychic alterations involve the sense of time and of reality, and initiative. Somatic disorders include nausea, vertigo, mydriasis, slowing of the pulse and ataxia (Heffter 1898; Dixon 1899). Changes in the EEG are confined to a decrease in amplitude (Walter 1938; Davis 1942).

In animals, reports deal mainly with autonomic dysfunction (Heffter 1898; Raymond-Hamet 1932; Grace 1934; Clerc, Paris and Janot 1936). In addition, somnolence and ataxia (Heffter 1898; Dixon 1899), catatonic states and convulsions (De Jong 1930) have been described. Hart and Marrazzi (1952, 1953, 1955) reported a diminution in the amplitude of cortical responses evoked by contralateral cortical stimulation after intravenous mescaline. It seemed, therefore, of interest to study the effect of mescaline systematically and topically applied on responses evoked by light stimuli. The findings were confirmed for other sensory systems and for direct cortical stimulation. In view of certain similarities between mescaline and lysergic acid diethylamid (L.S.D.) (Stoll 1947; Matefi 1952) on psychosensory phenomena, the effect of intravenous and local L.S.D. was studied.

MATERIAL AND METHODS

The experiments were performed on 21 cats anesthetized with intramuscular chloralose-urethane (1 and 3.5 per cent respectively) at a usual initial dose of 8 cc. per kg. Additional anesthesia was given as necessary to reduce spontaneous electrical activity. The head was fixed in a Horseley-Clarke headholder. The pupil was kept widely dilated with 2 per cent atropine sulphate solution and the body temperature was regulated.

Operative procedure and electrode placement. The cortex was exposed with appropriate hemostasis. For recording of visual responses, the electrode was usually placed on the mid portion of gyrus lateralis, sometimes as well in posterior and anterior placements, and occasionally on posterior gyrus suprasylvius. For recording of responses to sciatic and acoustic stimuli, the electrodes were placed on gyrus sigmoideus posterior and gyrus ectosylvius respectively, at the point of maximum response.

In 7 cats the lateral geniculate body was exposed through gyrus ectosylvius and hippocampus, care being taken not to damage gyrus suprasylvius and lateralis. The electrode was placed in the body of the geniculate usually through its supero-lateral aspect.

The retinal electrode was placed on the postero-lateral surface of the eye ball, at the point of maximum response, after resection of the masticatory and lateral extraoccular muscles.

Recording technique. Cerebral responses were recorded with concentric needle electrodes containing a platinum core with a

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recording area of 0.1 square mm. The recording depth was usually 0.5-0.8 mm., rarely 1.5-3 mm. Leading off took place between the core and the small area of the cannula (outer diameter 0.6 mm.) in contact with the tissue. The sign of the recorded potential depends on the position of the electrode relative to the activated cells and cannot be directly compared with unipolar recording. In the present measurements, maximal response amplitude was searched for independent of the sign. The electroretinogram was recorded with a silver placque, 3 mm. in diameter, and the reference electrode was placed on the stump of the ipsilateral temporal muscle. The animal was grounded at the headholder.

The amplifier was a 4 channel differentially coupled A-C amplifier with cathode follower input circuit and an input impedance of 100 megohms shunted by 60 $\mu\mu$ F. The lower limiting frequency was 1 c/sec., the upper limiting frequency 7000 c/sec. defined by 3 db. discrimination. The smallest measurable responses were approximately 10-20 μV. Single sweep responses were photographed on 35 mm. film from the screen of a double beam Dumont oscilloscope (type 279) at an appropriate sweep speed between 10 cm. to 3 m/sec. The sweep was triggered 1-2 msec. before onset of the sensory stimuli. The beginning and the end of each light flash or of electric or acoustic stimuli, led from the square wave generator, served as signal marking.

The light stimulation technique was very similar to that described (Lennox and Madsen 1955; Madsen and Lennox 1955). The light source was a Sylvania glow modulator tube (R 1130 B 2 white light). The light beam was focussed by two lenses at the cat's cornea where the light spot was 3 mm. in diameter. The color of the light could be altered by three interference filters each with a 20 mμ pass band and a maximum transmission at 445, 515 and 578 mμ. A fourth glass filter cut out light below 620 m μ.[1] The maximum intensity of white light was 754 μW. per square cm.; of green light 11,8 μW. per cm^2; of blue light 8.6 μW. per cm^2; of yellow light 79 μW. per cm^2; and of red light 638 μW. per cm^2. The intensity could be decreased by neutral filters in 12 steps over a 5 decade range. Threshold for white light could only be reached by diminishing the pulse width. It was approximately 0.002 μW. per square cm. with a 10 msec. flash. The rectangular light stimulus had a rising time of 0.8 msec. and a total duration of 33 msec., occasionally 2 or 10 msec. The stimulus frequency was one in 2 sec. Stimulation was carried out with the eye in complete darkness and after an initial 20 min. period of dark adaption.

Acoustic stimulation consisted of clicks at an intensity of five times the threshold of a just measurable cortical response. Sciatic stimulation was carried out between 2 electrodes placed across the sciatic nerve; direct cortical stimulation between two 0.1 mm. platinum wires, 0.5 mm. apart, placed in light contact with the cortex. Electrical stimuli consisted of single rectangular current pulses applied via a double-screened transformer (Buchthal, Guld and Rosenfalck 1955).

Substances. Mescaline chloride[2] was applied topically (filter paper) as a 5, 9, 20 and 33 per cent solution (by weight) in saline. The intravenous dose was 7 to 15 mg. per kg. *Strychnine sulphate* was applied topically as a 0.5 or 3.5 per cent solution in saline. *Lysergic acid diethylamid* (L.S.D. 25) was applied topically in a 0.001, 1, 5 and 9 per cent solution in saline and intravenously at a dosage of 20 to 40 μg. per kg.

Measurement of potentials. Each response was recorded 5 to 10 times, and the values for amplitude and latency represent a mean of these 5 to 10 measurements.

The *amplitude* of the *cortical potentials* was measured from the base line to the peak of the first deflection and from the maximum negative to the maximum positive peak.

Geniculate response amplitude was measured from the base line to the peak of the first positive, and from the peak of the first positive to the peak of the following negative deflection.

Amplitude of the *retinal potentials* was measured from the base line or from the peak

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[2] Mescaline was kindly supplied by Medicinalco, Copenhagen, and L. S. D. by Sandoz, Basel.

of the first positive to the peak of the following negative deflection.

Latency was measured for *cortical* potentials as the time interval between the onset of the light stimulus and the first departure from the base line. The *geniculate latency* was measured to the onset of the first positive deflection; *retinal latency* to the onset of the positive deflection.

plane. With increasing depth (above 0.5 mm.), the second phase of the potential tended to be more prominent (fig. 1 A_1, A_2). The peak to peak amplitude increased with increasing light intensity up to 400 μV. The latency varied between 18 msec. at maximum to 65 msec. at weak illumination.

The first effect of mescaline could be observed 1 to 5 min. after local application

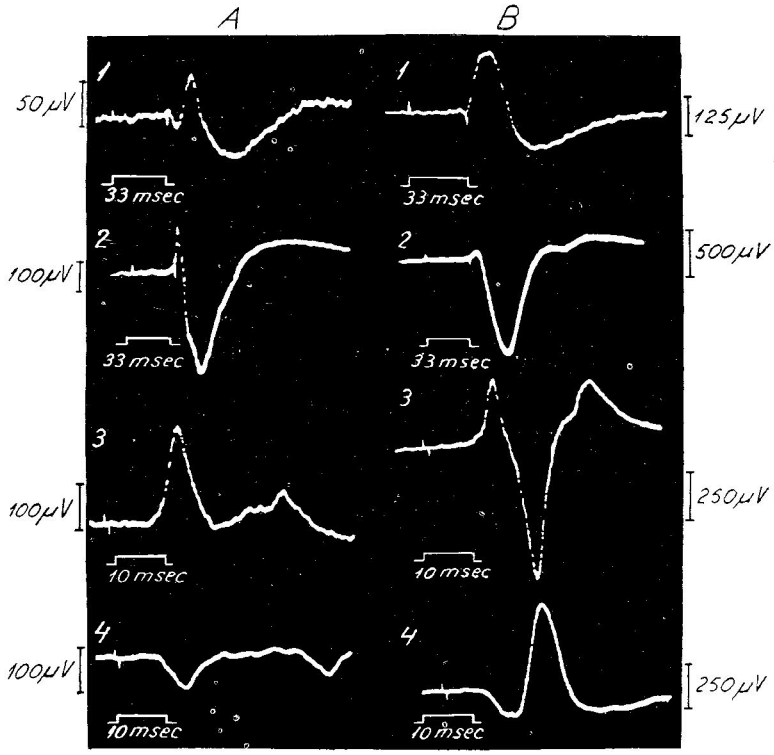


Fig. 1

Examples of cortical responses to white light flashes, intensity 67 μW. per cm². Column A before and column B after local application of 33 per cent mescaline. Flash duration 1 and 2, 33 msec.; 3 and 4, 1 msec. Time after application of mescaline 1, 7 min.; 2, 30 min., 3 and 4, 15 min.
Needle location 1, 2 and 3 mid portion gyrus lateralis, 1 and 2 at depth 1 mm, 3 at 0.5 mm; 4 posterior gyrus lateralis at 1 mm depth.
Upward deflection negative.

RESULTS

1. MESCALINE:

Effect of topically applied mescaline on evoked responses from optic cortex.

"On" response in untreated cortex. Four examples of responses evoked by white light stimuli are given in figure 1 A, corresponding essentially to the description by Marshall, Talbot and Ades (1943). There was no clear relationship between type of response and electrode placement in the anterior-posterior

(fig. 1 B). For a period of 20 to 45 min., the *response amplitude* increased progressively and then stayed at its maximum for 15 to 60 min. (fig. 2 and 3). When the filter paper with mescaline was removed after 15 min., the pre-mescaline values were reached within 2 hours. The percentage increase in amplitude over the pre-mescaline value tended to be higher with the highest mescaline concentrations. During the mescaline effect, a change in stimulus intensity had no effect on the

response amplitude. Even threshold stimuli evoked responses of the same amplitude as did stimuli of maximum intensity (fig. 4). At threshold, a response might not appear to each stimulus, but whenever a potential did appear it was of maximum amplitude. This obtained irrespective of the color of the flash. A threshold red light evoked a response of the same amplitude as a green light of maximum intensity.

The increase in response amplitude, up to 30 times the initial amplitude, was attributable to an enormous increase in a later component of the primary response. Sometimes this second component was visible in the pre-mescaline response (fig. 1 A_1, A_2). When the second component could not be distinguished in the pre-mescaline response, it was evoked by application of the drug (fig. 1 B_3 and B_4, fig. 3). The first phase of the response was usually increased about twofold in the early stages of mescalinization; later it might even be decreased in amplitude.

The *latency* of cortical responses increased with increasing time of mescaline action, up to 20 per cent one hour after application of the drug. The latency of the second phase of the response was disproportionately increased so that the time interval between the first and the second phase was greater after than before mescaline (fig. 1, 3, 4).

The amplitude of the off-response might be increased somewhat in early mescalinization; when the mescaline effect was fully developed, the "off" was lost in the large mescaline spike (fig. 4 A).

Acoustic and sciatic stimulation did not evoke responses in mescalinized optic cortex, nor did light stimuli evoke responses in mescalinized cortex outside electrophysiologically defined areas (Marshall, Talbot and Ades, 1943).

The degree and time course of the amplitude and latency increase was the same as at primary optic cortex when mescaline was applied to gyrus suprasylvius posterior.

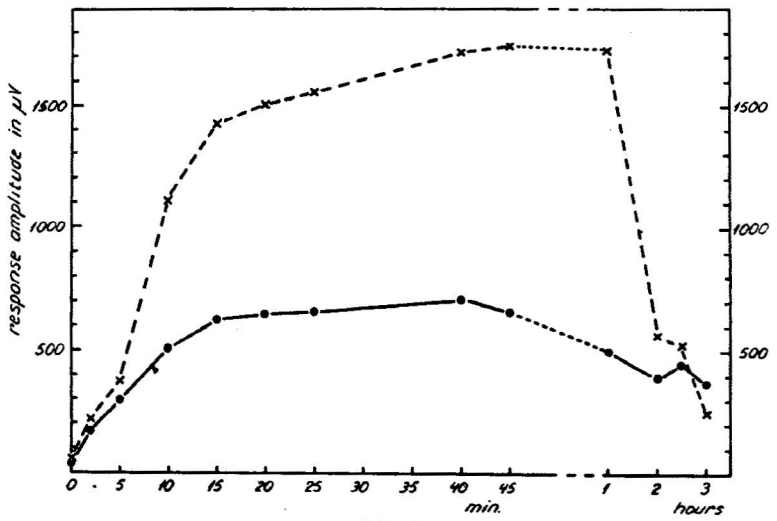


Fig. 2

Increase in amplitude of cortical responses to white light flashes 33 msec. duration, intensity 21 μW. per square cm as a function of time during local application of 33 per cent mescaline (cat 35).
x —— x total amplitude
● —— ● initial negative amplitude

Effect of topically applied mescaline on lateral geniculate responses evoked by light stimuli.

Before mescaline the main geniculate "on" response was positive and was frequently double (fig. 5 A), the second positive deflection varying somewhat in amplitude and position with light intensity and from cat to cat, but becoming increasingly prominent with increasing light intensity. At the highest intensities a third positive deflection was fre-

quently seen (fig. 5, 3). A negative phase of lower amplitude followed this positive complex; especially at high intensities an initial negative wave might be seen. The whole response was superimposed by small fast spike discharges with a maximum amplitude of 25-35 μV. and at intervals of 1-2 msec. characteristic for the spontaneous activity of the lateral geniculate (Dubner and Gerard 1939).

The maximum total amplitude of the evoked geniculate response was up to 200 μV.; the amplitude of the first positive phase up to 160 μV. The latency of the first positive phase was 15-20 msec. with stimuli of high intensity and 50-60 msec. with stimuli near threshold intensity.

Compared with cortex, mescaline acted considerably more slowly on the lateral geniculate, probably due to the fact that the drug had to diffuse through the encasing fibre layer. An enhancement of all components of the evoked response was observed 15 min. after application of a 20-33 per cent solution of the drug (fig. 5 C). A maximum enhancement of four times the original value was seen one hour after application of the drug.

The latency of the geniculate responses remained unaltered by mescaline.

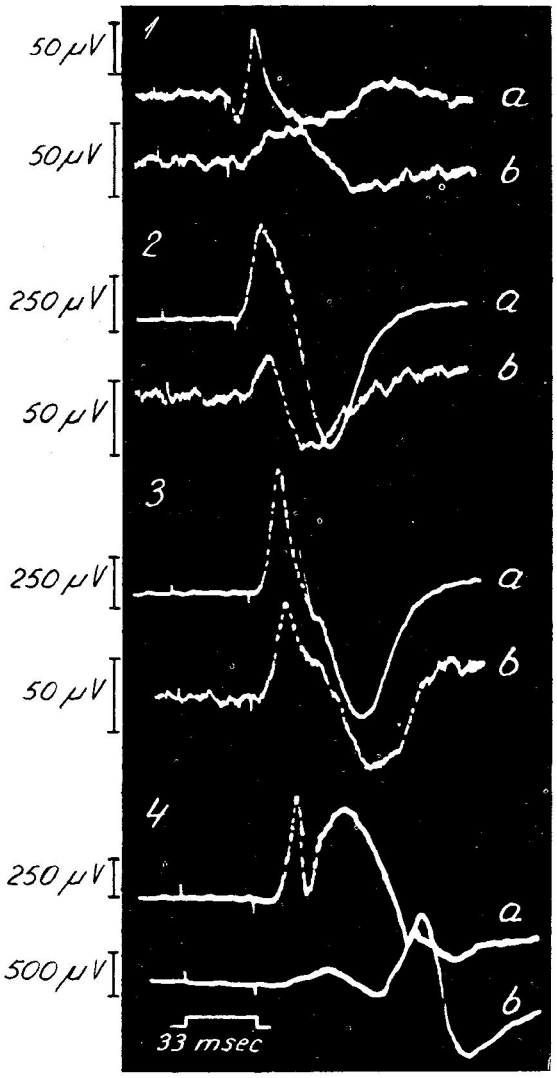


Fig. 3

Responses to light flash from gyrus lateralis (a) and posterior suprasylvian gyrus (b) before and at various time intervals during local 33 per cent mescaline at electrode (a). 1, before mescaline; 2, 25 min. after mescaline; 3, 35 min. after mescaline; 4, 120 min. after mescaline. Light flash white, intensity 220 μW. per square cm, duration 33 msec. Upward deflection negative.

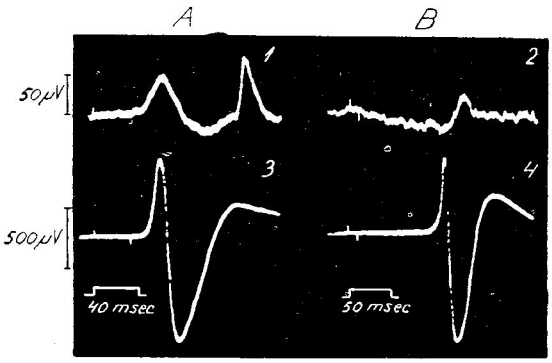


Fig. 4

Cortical responses to light flashes of large (A) and small (B) intensity before (1 and 2) and 30 min. after (3 and 4) local application of 33 per cent mescaline.

A. Light intensity 67 μW. per square cm, flash duration 33 msec.

B. Light intensity at threshold, 0.0124 μW. per square cm, flash duration 10 msec.

Upward deflection negative.

Other sensory systems.

As with light stimulation, local mescaline applied to somato-sensory cortex (fig. 6, $_{1a, b,}$ gyrus sigmoideus posterior) and acoustic cortex (fig. 6, $_{2a, b,}$ gyrus ectosylvius posterior) caused an up to 20 fold increase in amplitude of a second component of the response while the first component only in-

creased two to threefold. The latency showed no significant change.

As with light stimulation, the off-response to click was at first slightly increased and under full mescalinization was lost in the large mescaline spike.

Direct electrical stimulation of visual cortex.

In the untreated cortex, a response of simple wave form was evoked (fig. 6, 3a), surface positive and with increasing latency and decreasing amplitude according to the distance between stimulating and recording electrodes.

Action of mescaline on spontaneous cortical activity.

One hour after topical application large spontaneous spikes (up to 2 mV.) or potentials with two or more phases occurred simultaneously on 2 electrodes in mescalinized cortex resembling very much spontaneous strychnine spikes.

Effect of intravenous mescaline.

Autonomic effects. During and within the first 15 min. after intravenously injected mescaline at a rate of 10 mg. every 2 min.,

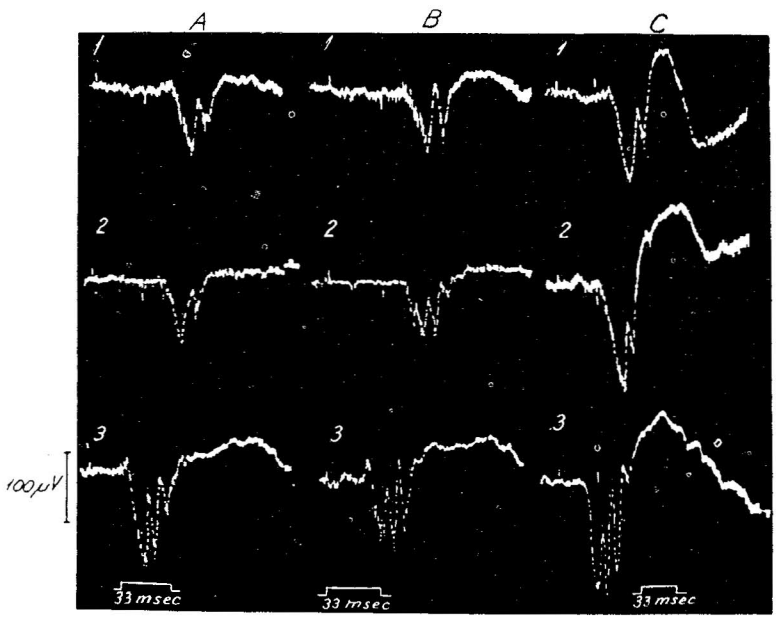


Fig. 5

Responses from lateral geniculate to white light flash before (A), after intravenous (B) and after local mescaline (C) at different light intensities. 1, 1.3 μW. per square cm; 2, 7.2 μW. per square cm; 3, 740 μW. per square cm; flash duration 33 msec.

B. 1, 5 min.; 2, 15 min.; 3, 30 min. after intravenous 9 mg. per kg. mescaline.

C. 20 min. after 20 per cent local mescaline.

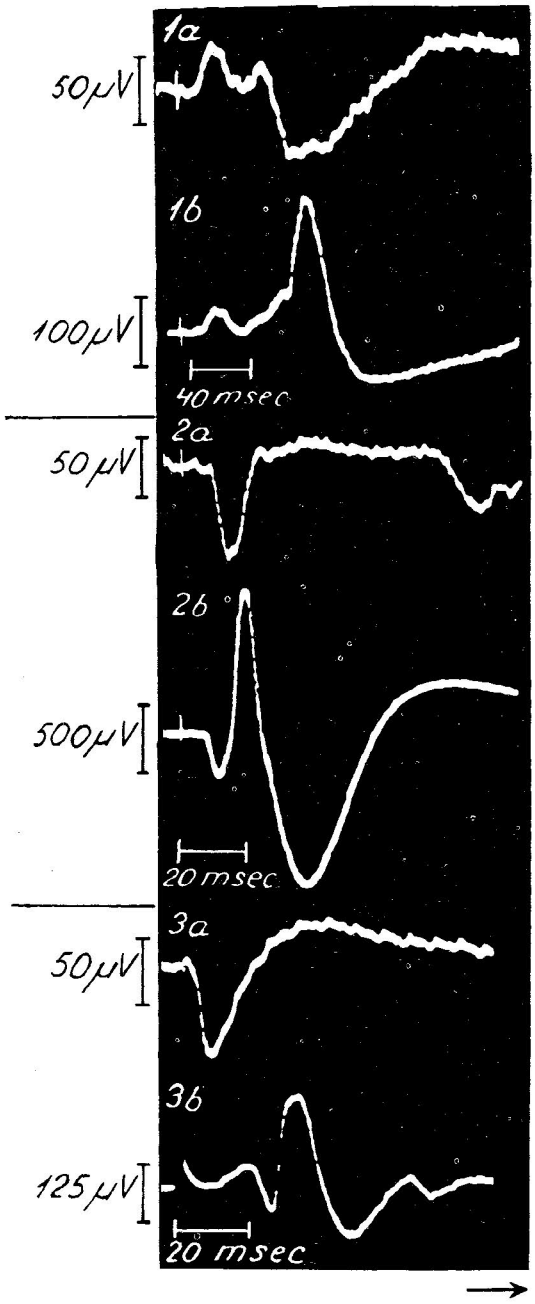
Upward deflection negative.

The effect of mescaline applied between the stimulating and the recording electrode is illustrated in fig. 6 3b. As with light stimulation, a high amplitude second component of the response appeared, and the latency of the first component was prolonged. Electrical excitability was *decreased* so that threshold was increased 2 to 3 times after mescaline.

defecation, urination and piloerection occurred regularly. The cardiac rhythm was irregular with periods of slow and of increased rate. Similarly there were periods of apnoea. These disturbances usually disappeared within 10 to 15 min.; one animal died, however, from respiratory failure immediately after injection of 27.7 mg. per kg.

Effect on potentials evoked in the visual system by light stimuli.

Retinal potentials. Before intravenous mescaline, the retinal potential had a main surface negative deflection, i.e. opposite in direction to the retinogram as recorded between an electrode on the cornea and a distant indifferent electrode (Granit 1955, p. 153). In 9 of 11 cats, the amplitude increased by an average of 20 per cent. The latency showed slight decreases rarely exceeding 10 per cent. It cannot be ruled out that the changes were due to more complete dark adaption.



Lateral geniculate. Immediately after mescaline the amplitude of the evoked responses was slightly diminished (not exceeding 20 per cent), but the different components of the response were somewhat more clearly distinguishable than before (fig. 5 B). The decrease in amplitude obtained in spite of the increase in amplitude of the ERG.

Cortical responses. The cortical evoked responses showed a diminution (15-20 per cent) in both positive and total amplitude (fig. 7). This diminution fluctuated irregularly between 0 and 20 per cent during the 2 hour observation period after drug administration. When slight latency changes occurred they were in the direction of a decrease by no more than 10 per cent. The amplitudes and latencies of responses to the four color stimuli relative to the retinal amplitude were

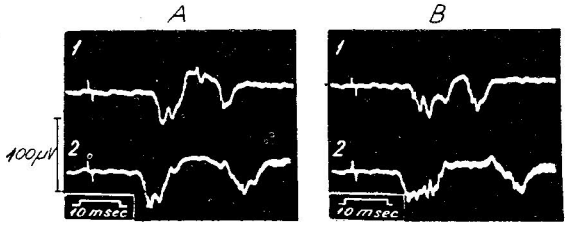


Fig. 7

Responses from optic cortex to white light flashes before (A) and after (B) intravenous mescaline, 12.5 mg. per kg.
Light intensity: 1, 7.2 μW per square cm; 2, 67 μW. per square cm; flash duration, 1 msec. Amplitude of electroretinogram: 1A, 130 μV.; 1B, 140 μV.; 2A, 354 μV.; 2B, 408 μV.

Fig. 6

Cortical responses evoked by sciatic (1a, b), acoustic (2a, b), and direct electrical (3a, b) stimulation. Before (a) and after (b) topically applied 20 per cent mescaline.
1a, b: sciatic stimulation, three times threshold, pulse duration 0.5 msec. 1b, time after mescaline 2 hours.
2a, b: click stimulus, five times threshold, 2b, time after mescaline 5 min.
3a, b: response recorded 2 mm from direct cortical stimulation. Stimulus intensity about threshold, pulse duration 0.5 msec. (a), 1 msec. (b). 3b, time after mescaline 1 hour.
Upward deflection negative.

the same after as before intravenous mescaline both at cortex and at geniculate.

2. TOPICALLY APPLIED STRYCHNINE:

The cortical potentials evoked by visual stimuli after application of mescaline appeared qualitatively similar to the effect of strychnine (fig. 8).

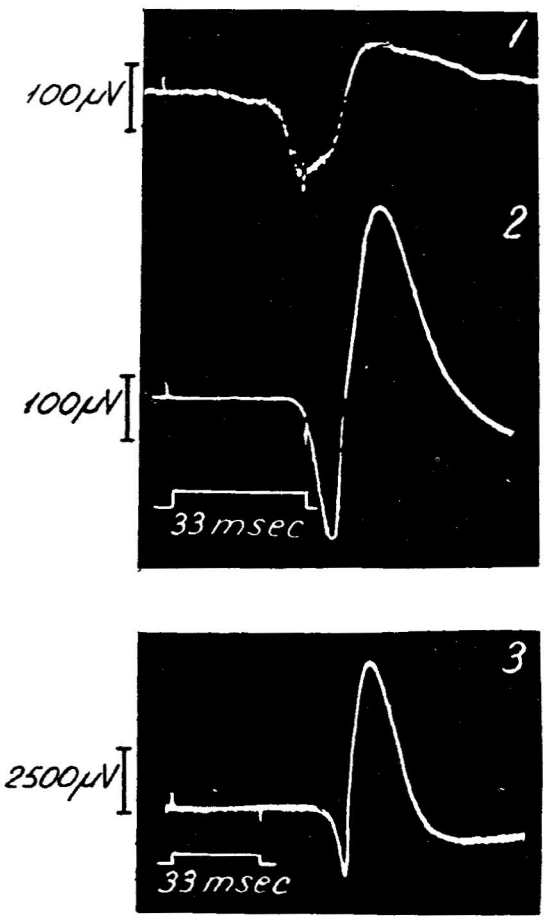


Fig. 8

Response from optic cortex to light flash before (1), and at 5 (2) and 20 min. (3) after local 3.5 per cent strychnine sulphate. White light, intensity 21 μW. per square cm, flash duration 33 msec. Upward deflection negative.

Both mescaline and strychnine caused an enhancement especially of the second component of the response, an increase in latency and in spontaneous spikes. The effects of strychnine were, however, more pronounced in that the amplitude of the evoked response was greater and the full effect developed more quickly. As with mescaline, the strychnine response was "all or none" not only for white but also for colored light.

3. LYSERGIC ACID DIETHYLAMID (L.S.D.):

Topical L.S.D. even in such large concentrations as 9 per cent and intravenous L.S.D. in dosages up to 40 μg. per kg., had no effect on evoked potentials comparable with that of mescaline. The latency of evoked responses to light flash and acoustic stimulation remained unchanged. The amplitude of the second component was questionably increased and the time interval between it and the first component was slightly prolonged.

DISCUSSION

The main result of the present study was the finding of the marked effect of topically applied mescaline on the amplitude and latency of evoked cortical responses. The amplitude increase involved all components of the response, but most spectacularly a component which occurred after the initial deflection. This second deflection was increased by as much as thirty time its value in untreated cortex, and might appear as a clear second component in a response which had only one component before mescaline. The increase in amplitude of a second response component occurred with all types of stimuli: light stimulus to the eye, electric stimulus to the sciatic nerve, click stimulus to the ear, and local electrical stimulation of the cortex. It was, therefore, due to an increase in a secondary central event, rather than in a later peripherally released discharge.

It was characteristic of the mescaline effect that a maximal response was evoked by every stimulus no matter what its intensity. It was also characteristic of the mescaline effect that responses were evoked only by adequate stimuli: no responses were evoked from visual cortex by other than visual stimuli, and visual stimuli evoked responses only from electrophysiologically defined optic receiving areas (Marshall, Talbot and Ades 1943). This characteristic of mescaline may have practical application, in that responses which were either very small or even not present before topical mescaline were so accentuated as to be clearly visible after topical mescaline.

In the present study, the effect of topical strychnine on evoked cortical responses was qualitatively identical to that of mescaline; quantitatively, the effect of strychnine was greater than with mescaline. The strychnine effect was the same as that described by Bishop and Clare (1952) with the exception that in the present experiments the response latency was markedly prolonged by strychnine. Both strychnine and mescaline caused spontaneous spiking. The fact that any stimulus caused a maximum response is very like the all or none evokation of strychnine spikes described by Chang (1951-1953).

Possibly the different effect of mescaline topically applied to the lateral geniculate was due to the different effective concentration of the drug. Diffusion through the overlying fibres to the body of the geniculate probably accounted in any case for the delayed action of the drug as compared with cortex. All phases of the primary response showed a modest increase in amplitude, as did the first component of the cortical response in the initial phases of drug action. At geniculate, there was not the clear cut enormous increase in amplitude of a later response component. In one cat there were large spikes, but they occurred at various time intervals after the primary response. The lack of an essentially and constantly increased later component may be an expression of the relative paucity of internuncials in the geniculate (Glees 1941) as compared with cortex.

The effects of locally applied mescaline are puzzling. In the case of strychnine, the effects of the drug applied topically are consonant with the systemic effects. In the case of mescaline, this was not so. Mescaline may have convulsant action in some species (De Jong 1930), but its main effect is the accentuation of visual sensation. Puzzling too is the very minor effect on evoked responses of intravenous mescaline, even in large doses. The slight decrease in amplitude was even less than that reported by Hart and Marrazzi (1952, 1953, 1955), and the appearance of the responses in many instances suggested desynchronization. Nor was there clear evidence of a peripheral effect to account for the enhancement of visual impressions reported clinically.

Finally, the autonomic changes observed in this study after intravenous mescaline are similar to those described by Heffter (1898), Dixon (1899), Raymond-Hamet (1932), Grace (1934), Clerc *et al.* (1936). It seems at least possible that these autonomic effects may account in part at least for the central effects of intravenous mescaline.

The lack of an effect with topical and intravenous lysergic acid diethylamid is in contrast with the findings of Marrazzi and Hart (1955) and with the clinical experience (Stoll, 1947) that L.S.D. is effective in much smaller doses than mescaline.

SUMMARY

Mescaline chloride (in solutions up to 33 per cent by weight) was applied topically to the cortex and lateral geniculate of the cat and its effect on evoked responses was studied. Responses were evoked by local cortical stimulation and from appropriate sensory receiving areas by flash stimuli to the eye, both of white and of four colors; click to the ear; and electrical stimulation to the sciatic nerve.

With all types of stimuli mescaline had the same marked effect on amplitude and latency of evoked cortical potentials. The effect appeared within 5 min., reached its maximum at about 30 min. and declined after 2 to 4 hours. The *amplitude* was increased up to 30 times and this was attributable to an increase in the amplitude of a second component of the response. The first component increased two- to threefold or might even be slightly decreased. The maximal response was obtained with stimuli of all intensities and in the visual system with light of all colors. The *latency* was prolonged to light flash and to direct cortical stimulation. The latency to sciatic and to acoustic stimulation was not changed.

Responses were evoked only by adequate stimuli; no responses were evoked from visual cortex by other than visual stimuli, and visual stimuli evoked responses only from known optic receiving areas.

When the cortical effect was fully develop-

ed, large (2 mV.) spontaneous spikes appeared.

At lateral geniculate, mescaline produced a two- to fourfold increase in all components of the response to light flash. Latency was not altered.

Local strychnine (3.5 per cent) produced qualitatively the same effect as mescaline on evoked responses to light flash but the effect was more marked in that the amplitude of the response was greater and the full effect developed more quickly. In other respects — increase in latency, increase in amplitude of a second component of the response, "all or none" response to every stimulus and evokation of spontaneous spikes — strychnine and mescaline acted identically on evoked responses to light flash.

Intravenous mescaline in doses up to 15 mg. per kg. produced immediate, temporary autonomic changes consisting of cardiac irregularity, periods of apnoea, piloerection, urination and defecation. There was a slight decrease in the amplitude and increase in the duration of cortical and geniculate potentials evoked by light flash.

Topical and intravenous lysergic acid diethylamid (up to 9 per cent solution by weight, or 40 μg. per kg.) produced no clear cut effect comparable to that of mescaline on evoked response amplitude. The time interval between the first and second components of the cortical response was prolonged.

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