Table 1. Summary of results for experimental and control subjects and significance of differences of changes from pretest to posttest. ($N = 14$ in each group)

Measure	Group means				t	p
	Experimental		Control			
	Pretest	Posttest	Pretest	Posttest		
Change in punitiveness indices:						
1. Shock intensity level	4.34	4.92	5.03	4.89	2.37	$<.025$
2. Level × duration	42.84	47.22	41.11	35.32	1.93	$<.05$

15 occasions. Since the experimenter had surreptitiously removed one of the electrodes from the strap, the confederate in fact received no shocks.

Immediately after the "trial" run, the experimental subjects and the confederate were shown the knife-fight scene, while the control subjects and the confederate were shown the painting scene.

The posttest series of trials on the learning task followed. Another program of 30 settings was handed to the subject and again he was required to punish the confederate 15 times during the run.

The mean intensity setting (in terms of shock levels numbered from 1 to 10) was calculated for each subject for the pretest and posttest sets of trials. The difference between these two means was used as one index of the effect of exposure to the audio-visual display. On the supposition that a lower shock level administered for a relatively long period of time might represent as high a degree of punitiveness as a higher level administered for a shorter period, a mean shock level × duration index was also calculated for both sets of trials, duration being recorded in 0.1-second units. The pretest-to-posttest change in this index formed the second behavioral measure of punitiveness.

Table 1 gives the group pretest and posttest means on both indices of punitiveness. Differences between group pretest means were not significant. ($p > .05$ in each case). Changes were in opposite directions, with experimental subjects showing a mean increase in punitiveness and control subjects a mean decrease. Tests based on individual pretest-to-posttest changes yielded significant group differences for both the shock level ($p < .025$) and the shock level × duration ($p < .05$) measures (one-tail tests). Comparable findings have since been obtained for both male adolescent and female adult subjects.

The results of this study indicate that exposure to audio-visual displays containing aggressive content can result in a significantly greater willingness in adults to inflict pain. It is of interest to note that they were obtained with a group of hospital attendants, whose training in a modern psychiatric hospital is definitely oriented toward the inhibition of aggression (*6*).

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Central Cholinolytic Action of Chlorpromazine

Abstract. Chlorpromazine was found to have an antagonistic action against the electroencephalographic (EEG) alerting produced in rabbits by cholinergic agents, particularly eserine salycilate and acetylcholine chloride. This cholinolytic action occurs at a prepontine, precollicular level where adrenergic agents fail to elicit EEG alerting. These results emphasize the importance of cholinergic mechanisms in EEG alerting.

Chlorpromazine is known to exert an adrenolytic action both peripherally on the terminations of the sympathetic nerves and centrally in the brain. Chlorpromazine's peripheral antiadrenaline activity was described by Courvoisier *et al.* (*1*) and was confirmed by Kopera and Armitage (*2*). Dell (*3*) and his co-workers, whose experiments became the prototype of subsequent investigations, demonstrated that the cortical pattern of EEG arousal, which normally accompanies the intravenous injection of adrenaline, was reduced by chlorpromazine. Bradley and Hance (*4*) made a further contribution by observing that the characteristic behavioral arousal and EEG alerting of amphetamine are jointly blocked by chlorpromazine. Anochin (*5*), in a series of studies conducted in the U.S.S.R., reported that chlorpromazine blocked adrenaline "mobilization" at the level of the brain stem reticular formation. Brodie *et al.* (*6*) have classified chlorpromazine as a central adrenergic blocking agent.

Most chemical agents evoke a multiplicity of responses from the body, and peripheral cholinolytic effects of chlorpromazine have been reported (*1, 2, 7*), including a diminution of salivation and pupillary dilation. In patients receiving chlorpromazine, such atropine-like characteristics as a fast heart, dilated pupil, and dry mouth have been observed. Quantitatively, however, chlorpromazine is a weaker cholinolytic agent than atropine.

The purpose of the present communication is to show that chlorpromazine also exerts a central cholinolytic action on the brain. Experiments from our laboratory indicate that chlorpromazine effectively antagonizes the EEG alerting produced by eserine, a cholinergic agent. Twenty-seven adult New Zealand male albino rabbits weighing approximately 3 kg each were employed in these preliminary experiments. Animals were tracheotomized under ether and local pontocaine anesthesia, curarized, and artificially respired prior to the administration of the drugs (*8*). Ten of the animals were studied under special procedures involving a prepontine, precollicular transection of the brain.

In the first series of experiments, rabbits were given 0.3 mg/kg of eserine salicylate by femoral vein. Within 3 to 5 minutes, the EEG pattern from the motor and limbic cortices, caudate nucleus, thalamus, hippocampus, amygdala, and reticular formation was one of sustained arousal. Chlorpromazine was then administered by femoral vein every 4 minutes in alternating 1 and 2 mg/kg injections at a concentration of 10 mg/ml until a complete reversal of the eserine arousal pattern had been obtained. Chlorpromazine typically effected this reversal at a total drug level of 6 to 8 mg/kg within 15 to 20 minutes. The normal period of

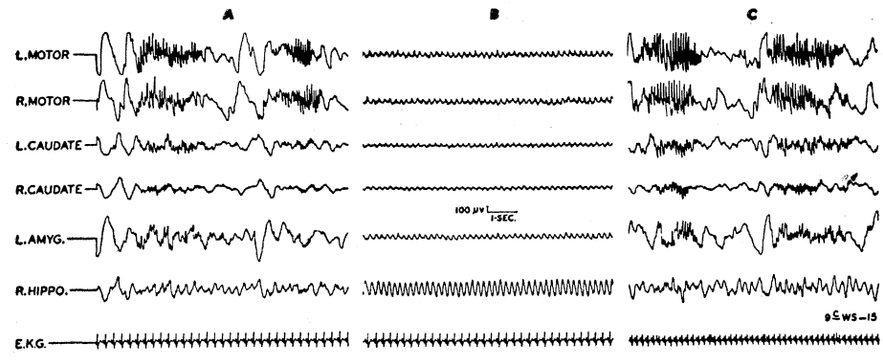

Fig. 1. Central cholinolytic action of chlorpromazine in rabbit with brain transected cephalad to mid-brain (prepontine, precollicular). (*A*) amphetamine (3 mg/kg) fails to alert EEG, (*B*) eserine induces EEG arousal (0.3 mg/kg) and (*C*) chlorpromazine reverses cholinergic alerting produced by eserine (5 mg/kg, 20 minutes post). Drugs were administered intravenously.

EEG arousal accompanying 0.3 mg/kg of eserine salicylate is a minimum of 45 minutes. Continuation of the chlorpromazine injections to a total drug level of 9 to 12 mg/kg produced an EEG pattern which was unresponsive to most sensory stimulation. The frequently reported chlorpromazine blockade of EEG alerting was thus established in the presence of a cholinergic agent.

Cholinergic antagonism was a different form when chlorpromazine was administered prior to eserine. Rabbits were first treated with 6 to 8 mg/kg of chlorpromazine. When the animals showed an absence of EEG alerting to sensory stimulation, 0.3 mg/kg of eserine salicylate was administered by femoral vein. Within 3 to 5 minutes, the typical EEG pattern of eserine-induced arousal occurred. At this juncture, chlorpromazine appeared not to be exerting a cholinolytic action on eserine but the eserine-induced EEG arousal was short-lived and a pre-eserine EEG pattern resembling that of a control animal was established without additional chlorpromazine. This re-establishment of resting rhythms occurred in one-third the time (12 minutes) normally required for the abatement of the eserine-induced arousal. Though the previous administration of chlorpromazine could not prevent the occurrence of the eserine-induced arousal, the duration of the eserine alerting was significantly curtailed.

Cholinolytic effects were also demonstrated in rabbits having a precollicular, prepontine transection of the mid-brain (Fig. 1). The transection was accomplished by fulguration after treatment with 3 g of urea. A dull spatula was then passed across the entire extent of the lesion to sever any remaining threads of neural tissue. The correctness of the plane of transection was confirmed at the end of each experiment. Seven animals transected in this manner and receiving spaced injections of ephedrine sulfate to maintain blood pressure (*4*) failed to produce EEG alerting both to peripheral sensory stimulation and to the administration of 3 mg/kg of amphetamine, an adrenergic drug. It is well known that amphetamine evokes EEG alerting when the transection is made below the level of the mid-brain reticular system whereas amphetamine alerting is lost when the transection is cephalad to the anterior border of the pons (prepontine) (*9*). Five of the transected animals showed typical eserine-induced EEG arousal patterns in response to 0.3 to 0.9 mg/kg of eserine salicylate. The remaining two animals of this group succumbed to eserine overdosage. Bipolar recordings were taken from the motor cortex, caudate nucleus, and hippocampus of each hemisphere. Pronounced seizure activity was often seen during this arousal. The EEG alerting was quickly reversed, however, by 2 to 4 mg/kg of chlorpromazine. Prior treatment with chlorpromazine could not be used satisfactorily because of an intensification in the fall of blood pressure produced by the transection. In some of the animals, the amplitudes had a tendency to remain slightly reduced, but spindle and slow wave activity returned as a sustained EEG pattern. Larger amounts of chlorpromazine only served to produce a marked fall in blood pressure and a corresponding flattening of the EEG tracing. Thus the effective dose of chlorpromazine is less in the transected animal than in the intact animal. Atropine also reversed eserine-induced arousal in these transected animals at a dosage of 2 mg/kg.

These experiments were repeated in 8 additional animals using acetylcholine chloride as the cholinergic agent. Our findings of acetylcholine arousal for the brain transected cephalad to the mid-brain parallel those reported by Rinaldi and Himwich (*10*) for transection caudad to the mid-brain. A 5 μg/kg intracarotid injection of acetylcholine chloride produced an immediate EEG arousal, symmetrical as to hemispheres, lasting 15 to 20 seconds. Higher dosages evoked somewhat longer durations. Prior treatment with chlorpromazine (7.0 mg/kg, intravenously) prevented the EEG activation normally accompanying 5 μg of acetylcholine chloride, but EEG arousal was still obtained at a dosage of 10 μg in these animals. The short action of acetylcholine chloride would not permit the use of chlorpromazine to reverse previously established acetylcholine chloride arousal. In the intact animal chlorpromazine successfully antagonized the EEG alerting even at a dose of 20 μg of acetylcholine.

It is clear from the experiments described that chlorpromazine has a central cholinolytic action in addition to the central adrenolytic action frequently cited in the literature. The action occurs at a precollicular, prepontine level where adrenergic agents fail to elicit EEG alerting. These findings offer additional evidence to support the conclusion of Rinaldi and Himwich (*10*) that the mesodiencephalic activating system is cholinergic in nature (*11*).

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Cosmogenic Carbon-14 in Meteorites and Terrestrial Ages of "Finds" and Craters

Abstract. Carbon-14 has been measured in several stone and iron meteorites. For "falls," the C^{14}/Al^{26} ratios in stones and the C^{14}/Cl^{36} ratios in irons are consistent with constant irradiation. The stone "finds" have radiocarbon ages of up to $\geq$ 21,000 years. The Henbury craters are apparently $\leq$ 7,000 and the Odessa craters $\geq$ 11,000 years old.

The study of stable and radioactive nuclides in meteorites from bombardment by cosmic radiation in space (*1*) has given valuable information on the relative intensity of cosmic radiation in the past (*2*) and on the histories of meteorites (*3*). The possible occurrence of C^{14} in meteorites was first mentioned by Bauer (*4*), and it has been recognized that its half life of some 5000 years would make it particularly useful for dating the falls of meteorite "finds" (*5*). Its observation in both a stone and an iron meteorite has recently been reported (*6*), and we present herewith the results of additional measurements. The results on "falls" supplement previous information on the past cosmic-ray intensity, and those on "finds" illustrate the utility of C^{14} for determination of terrestrial ages of meteorites.

The techniques are described elsewhere (*6, 7*). From stone meteorites (10 to 20 g), carbon is recovered as CO_2 by fusion with an oxidizing flux. Iron meteorites (200 to 400 g) are decomposed in a closed system by HNO_3, and the evolved CO_2 is absorbed in NaOH and precipitated as $BaCO_3$. The CO_2 is carefully purified with special care to remove radon. The pure CO_2 serves as the counting gas in proportional counters of various sizes, similar to those of Stoenner *et al.* (*8*). Conventional anticoincidence and shielding techniques are used to reduce backgrounds. Special alpha-particle monitoring circuitry indicates the absence of radon contamination. Counters with volumes (including dead-space) of 11 to 82 ml with CO_2 at about 76 cm-Hg have backgrounds from 0.20 to 0.45 count/min and counting yields of 44 to 65 percent as determined with National Bureau of Standards standard C^{14} and checked with biological carbon.

The results, in terms of meteorite specific C^{14} activity, are given in Table 1. Specimens differentiated by "I" and "II" in the table came from the same piece of meteorite; the agreement between such duplicates gives confidence in the techniques. The negative results (Potter, Sardis) demonstrate absence of appreciable contamination from terrestrial radiocarbon. Limits for inactive samples are calculated by adding two standard deviations (computed from counting statistics only) to the net counting rate, if positive, or to zero, if negative.

The production rate of a nuclide in a specimen should vary with the size of the meteoroid and the position of the specimen within it. It is therefore surprising that the range of variations observed in stone "falls" is so small. A mean value of the C^{14} activity level in stone "falls" is calculated as 63 $\pm$ 10 disintegrations per minute per kilogram (dpm/kg), the uncertainty index being the standard deviation characterizing the spread of the individual values. From the data of Rowe *et al.* (*9*), the mean Al^{26} content of stones is 52 $\pm$ 5 dpm/kg, giving a C^{14}/Al^{26} ratio of 1.2. Qualitative arguments (*6*) show that this is roughly consistent with expectations based on a constant cosmic-ray intensity.

For the calculation of the terrestrial ages of stone "finds," recorded in Table 1, the value 63 $\pm$ 10 dpm/kg was assumed for all at the time of fall. Simultaneous measurements of additional radionuclides should permit separate estimates for each. It is interesting that four of the six stone "finds" are fairly old (*10*).

For the iron "falls," Aroos (=Jardymlinsky) and Sikhote Alin, the Cl^{36} contents have been measured (*11*) to be 21.0 $\pm$ 0.4 and 15.0 $\pm$ 0.4 dpm/kg, respectively. In both, the ratio C^{14}/Cl^{36} is close to 0.10. Arnold *et al.* (*2*) give 0.13 as a theoretical production-rate ratio estimate. The closeness of these values makes it appear that the cosmic-ray intensity averaged over the mean lives of these two nuclides has been about the same. Carbon-14 integrates over a time span essentially missed by the radionuclides considered in the survey of Arnold *et al.* (*2*).

Table 1. Carbon-14 in Meteorites

Meteorite and place of fall or find	Year fell or found	Recovered mass (kg)	[Carbon-14] (dpm/kg)	Terrestrial age (years)
		Stone falls		
Bruderheim, Alberta, Canada	1960	303	63 ± 5	
Forest City, Iowa	1890	122	I: 47 ± 6 II: 51 ± 7 Av: 49 ± 5	
Harleton, Texas	1961	8.36	57 ± 5	
Holbrook, Arizona	1912	218	I: 63 ± 4 II: 58 ± 3 Av: 60 ± 3	
Kunashak, U.S.S.R.	1949	200	71 ± 5	
Modoc, Kansas	1905	211	59 ± 4	
New Concord, Ohio	1860	220	73 ± 5	
Richardton, N. Dakota	1918	100	78 ± 6(*6*)	
		Stone finds		
Achilles, Kansas	1924	16	I: 55 ± 11 II: 58 ± 11 Av: 56 ± 8	$\leq$5,200
Coldwater, Kansas	1924	11	39 ± 3	4,000 ± 1,400
Hugoton, Kansas	1927	350	36 ± 4	4,700 ± 1,600
Plainview, Texas	1917	700	61 ± 4	$\leq$3,500
Potter, Nebraska	1941	261	−0.2 ± 2.2 [$\leq$4.5]	$\geq$21,000
Selma, Alabama	1906	105	29 ± 2	6,500 ± 1,400
		Iron falls		
Aroos, U.S.S.R.	1959	150	1.80 ± 0.25	
Sikhote Alin, U.S.S.R.	1947	large	1.66 ± 0.33	
		Iron finds		
Henbury, Australia	1931	large	I: 1.20 ± 0.36(*6*) II: 1.44 ± 0.22 Av: 1.37 ± 0.18	$\leq$7,000
Odessa, Texas	1922	large	0.22 ± 0.10 [$\leq$0.42]	$\geq$11,000
Sardis, Georgia	1940	800	0.01 ± 0.13 [$\leq$0.28]	$\geq$16,000