STUDIES ON MESCALINE XI: BIOCHEMICAL FINDINGS DURING THE MESCALINE-INDUCED STATE WITH OBSERVATIONS ON THE BLOCKING ACTION OF DIFFERENT PSYCHOTROPIC DRUGS*

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The antagonism of one psychotropic drug for another may be studied according to a particular field of interest—clinical, pharmacological, neurophysiological, electro-encephalographic or psychological. For instance, the inhibiting effects of anti-Parkinsonian drugs on the extrapyramidal syndrome induced by psychotropic drugs can be considered from any one or any combination of these disciplines. Much basic work has so far been centered on the blocking action** of various compounds against drugs capable of inducing states of disordered behavior resembling the psychoses.[1] A comprehensive neurophysiological analysis of the problem has been made by Marrazzi, et al.,[2] whose studies with their "transcallosal monosynaptic" preparation have yielded results that seem to parallel clinical findings.

It has been shown that chlorpromazine directly antagonizes the action of mescaline in psychiatric patients, and potentiates the abnormal brain wave patterns of epileptics.[3, 4] Schwarz, et al.,[5] found that 25 mg. of intramuscular chlorpromazine blocked 400 mg. of oral mescaline, and in 15 minutes "the subject was essentially restored to his control condition." Hoch[6] reported that when chlorpromazine was given intravenously it was the most effective antagonist of mescaline, but he could not confirm the reported effectiveness of Frenquel.[7] The relevant neurophysiologic and biochemical data have been reviewed elsewhere.[8]

In a comparative clinical analysis of the antagonism of various phenothiazines to mescaline, it was found that chlorpromazine and trifluprομazine were most effective.[9] Proclorperazine and thiopropazate were only moderately effective. Promazine and promethazine were ineffective, although they produced transient

*These studies were carried out in the research division of Manhattan (N.Y.) State Hospital, and this report was presented at the symposium, "The Problem of Antagonists to Psychotropic Drugs," Collegium Internationale Neuro-Psychopharmacologicum, Basle, Switzerland, July 4-7, 1960.

**A blocking agent is defined here as a compound that reverses the effects of mescaline.

drowsiness. Diethazine aggravated the condition in five of six cases. When administered before the injection of mescaline, chlorpromazine and promethazine decreased the anxiety and symptoms, while in five of 13 cases diethazine aggravated the subsequent mescaline-induced state.[10] The hypothesis was advanced that "the blocking action . . . is not specific in its relationship to clinical activity," and that "an aliphatic two carbon-amine linkage seems to have some relevance to the problem of disordered states of human behavior."[9]

The biochemical effects of various psychotropic drugs on the mescaline-induced state in humans have not been the subject of any previous systematic study. Deniker[11] followed the biochemical changes during administration of mescaline and noted a hyperglycemia, fall in blood potassium, leucocytosis and eosinopenia. He did not attempt to alter the state once the drug was injected.

A preliminary study[9] suggested that the chemical formula influenced the clinical blocking effect; the halogenated straight chain phenothiazines (chlorpromazine, trifluprομazine) produced sedation with anti-psychotic effects.* The halogenated piperazine phenthiazine (i.e., thiopropazate) was devoid of sedative action, and showed only moderate to minimal antipsychotic activity. The nonhalogenated phenothiazines (promazine) had no antipsychotic effect. The two carbon diethylamide side chain phenothiazine (diethazine) aggravated the mescaline-induced state. There was no evidence available to indicate whether or not these findings were reflected by any parallel biochemical changes. If such changes existed, a better understanding might be provided for the mechanism of action of the various drugs.

The aim of this paper will be to examine some of the biochemical parameters possibly influenced by mescaline, and to determine what effect is produced by a series of compounds capable of antagonizing its action. This is an exploratory survey type of study designed to yield a general comparative picture of the biochemical effects of mescaline and their modification by several different drugs.

*An analogy is made here between the apparent relationship of the endogenous psychoses and the mescaline-induced state. Acknowledgment is made of the unsettled nature of this subject.[12]

Materials and Methods

The Setting

The studies were carried out in the research division ward of Manhattan State Hospital, a 3,000-bed psychiatric hospital of the New York State Department of Mental Hygiene in New York City. There are 60 acute and chronic psychotic female patients in the ward, which operates under the principles of a therapeutic community. The major treatment is chemotherapy, with psychotherapy and work therapy as the major ancillary aids. The patients are aware of, and take part in, the research activities. They were selected at random for this study; the only contraindication being some physiological abnormality (i.e., cardiovascular disease). One male patient from another ward was studied.

Twenty-six trials were carried out with 22 female and one male patient. Their ages ranged from 23-53 years, with a mean of 35.

The major diagnosis was schizophrenia. Eight different drugs were used (Table 1).* There were six sets of controls (Tables 2, 2A).**

Patients were selected at random, were without overt symptoms and were relatively quiescent. All medication was stopped at least 72 to 96 hours before the study. A fasting control venous blood sample (10 cc. each of clotted and oxalated blood) was drawn 24 hours before the test and repeated one-half hour before the injection of mescaline on the day of the test. The patient then received 500 mg. of mescaline sulfate dissolved in 20 cc. of physiologic saline, injected intravenously in three to five minutes. The patient was told to "talk about anything you feel or think about."

Blood was drawn at one-half hour and one hour after the injection of mescaline. The blocking agent was given intramuscularly at this point. Blood was drawn again one hour after the injection of the blocking agent, and in 24 and 48 hours. These times were decided upon arbitrarily. Even though it became obvious as the study progressed that determinations two, three and four hours after mescaline would be interesting and informative, the time schedule was kept unchanged for comparative purposes.

*The tables (No.'s 1 to 19) are grouped together at the end of this paper under the heading of "Statistical Analysis," which was performed by David N. Teller, M.S., senior research scientist (biochemistry). Pp. 34-47.

**The groups will be known by the names of the pharmaceuticals administered in the rest of the paper: as "saline and chlorpromazine" group, "saline and trifluperazine," "saline and saline," "saline and haloperidol," "no injections," "mescaline only," etc.

Controls

The preparation, methodology and time schedule for venipuncture were the same for all subjects. There were 24 female patients drawn randomly from the same ward population who served as controls. They received 20 cc. of physiological saline injected in the same manner in all cases. The blocking agents (chlorpromazine, trifluperazine or saline) were administered one hour afterward intramuscularly. Four patients were kept in bed under the same standard conditions and did not receive any injections; blood was drawn according to the foregoing schedule. Three patients received mescaline only, and blood was drawn one-half, one, two, three, 24 and 48 hours afterward; a fourth patient required chlorpromazine to terminate the study two and one-half hours afterward.

Biochemical Determinations

The following determinations were done for the mescaline and control groups: Total amino acids, total cholesterol, blood glucose, leucocyte, differential count, eosinophils and sedimentation rate, cephalin cholesterol flocculation, bilirubin, and thymol turbidity. The amino acids were done according to the method of Troll and Cannan.[13] Cholesterol was determined by the method of Zlatkis, Zak, and Boyle.[14] The standard cephalin cholesterol flocculation was done by Hanger's method.

Results

Clinical

The clinical picture induced by mescaline was similar to that described elsewhere.[15] The halogenated phenothiazines were the most effective blocking agents; the nonhalogenated were ineffective. The piperazine derivatives were only moderately, or not at all, effective. Haloperidol had no action. The mescaline-induced state was aggravated in three cases with diethazine, and was unchanged in two.

Biochemical Results

Amino Acids: *Mescaline + Blocking Agents*— (Tables 3 and 4)*

The mean value of the mescaline group for serum free amino nitrogen (amino acids), 24 hours before the day of the experiment

*Values refer to combined determinations for all groups receiving blocking agents.

was 3.21μM (ESE=0.883 μM).* This was 3.16 μM (ESE=0.238 μM) one-half hour before the test was done. The 24 patients in the control group showed a mean of 3.29 μM (ESE=0.703 μM) 24 hours before the study, and 3.21 μM (ESE=0.442 μM) one-half hour before the intravenous injection of saline.

One-Half Hour After Injection of Mescaline

The amino acid level fell to 2.70 μM [(ESE=0.509 μM) and P=>.001], while the control saline group fell to a mean of 3.08 μM [(ESE=0.395 μM) and P=>.20]. The 4 per cent decrease in the controls was significantly less than the 14.6 per cent decrease in the mescaline group. On the basis of percentages, P=>.20 < .40 for the controls and P=>.02 <.025 for the mescaline group. The standard error at one-half hour postmescaline was significantly larger than at one-half hour premescaline.

One Hour After Injection of Mescaline

The experimental group showed little change [mean=2.69 μM (ESE=0.330 μM) and P=>.001]. The same held true for the controls [mean=3.04 μM (ESE=0.635 μM) and P=>.25].

One Hour After Blocking Agents

The mescaline group amino acid levels dropped slightly with a mean of 2.61 μM (ESE=0.410 μM and P=>.001). The control group showed a mean of 3.22 μM (ESE=0.145 μM and P=>.80). *These values were significantly different on the basis of mean differences and variance due to difference between estimated standard errors.*

The amino acid fall after diethazine (five patients) was particularly interesting. The premescaline mean value was 2.94 μM. It was 2.64 μM one hour afterward, hardly changing even one hour after the injection of diethazine. At 24 hours, the mean was 2.46 μM (P=>.001), and at 48 hours, it was 2.22 μM (P=>.01).

The fall after injection of trifluprom azine (four patients) and chlorpromazine (three patients) was most significant at this time (P=>.001). The mean values for trifluprom azine fell from 2.98 μM one-half hour before mescaline to 2.37 μM at this time. The mean for the chlorpromazine group was 3.26 μM premescaline, falling to 2.73 μM one hour after the blocking agent.

The estimated standard error with trifluprom azine was extremely small (0.092 μM); it was larger with chlorpromazine (0.363

*ESE=Estimated standard error.

μM). It was largest with pipamazine. Thioperazine and trifluperazine were intermediate. Promethazine showed similar changes.

The amino acid values for pipamazine were already rising when the drug was injected. In general, the blocking agents did not reverse the fall of amino acids; as a matter of fact, with diethazine the fall continued even past 24 hours.

24 and 48 Hours After Mescaline

Most of the values had returned to, or were near, the base line levels with the exception of diethazine by 24 to 48 hours after mescaline injection.

Blood Glucose: (Tables 5, 6)

One-Half Hour After Mescaline

The mean value of blood glucose rose from 88.9 mg.% premescaline to 97.7 mg.% at the one-half hour period [(ESE=16.31 mg.%) and P=>.05]. The highest value was 146.5 mg.% (patient A.R.), and the lowest 65.6 mg.% (Table 5).

One Hour After Mescaline

The mean value had risen to 107.6 mg.% [(ESE=22.5 mg.%) and P=>.001]. The highest level attained was 183.5 mg.% (patient A.R.), and the low was 82.9 mg.%. Thioperazine showed the most significant rise at this period.

One Hour After Blocking Agents

The mean value (combined averages) had only fallen slightly to 103.2 mg.% and P=>.01 at this time. Trifluperazine, triflupromazine, diethazine and chlorpromazine caused a reversal of the upward trend, and the values had now approached or were approaching the premescaline base line. The findings with thioperazine, promethazine and haloperidol were equivocal.

24 Hours After Mescaline

The mean value was 94.3 mg% (P=>.10) as compared with one-half hour premescaline of 88.9 mg.%. None of the blocking agents showed any particular trends.

48 Hours After Mescaline

The mean value was 90 mg.% with a range of 81.0 mg.% to 135 mg.%.

Cholesterol: (Table 7)

The cholesterol showed no unusual variations throughout the study. Some patients had fairly sharp shifts, but these were not considered to be statistically significant.

Leucocytes: (Table 8)

The leucocytes rose slowly in a variable fashion, and at one hour after mescaline had made a slight dip. Following the blocking agent, there was another abrupt rise. At 24 hours after, there were still some leucocyte counts 3,500 to 8,050 above the control samples. In addition, there was a fairly marked scatter at one hour after the blocking agents (range=4,500 to 18,650 with a mean of 10,217), as well as 24 hours afterward. The highest values observed were in the diethazine group, with four of five patients showing a significant rise, continuing up to and including 24 hours.

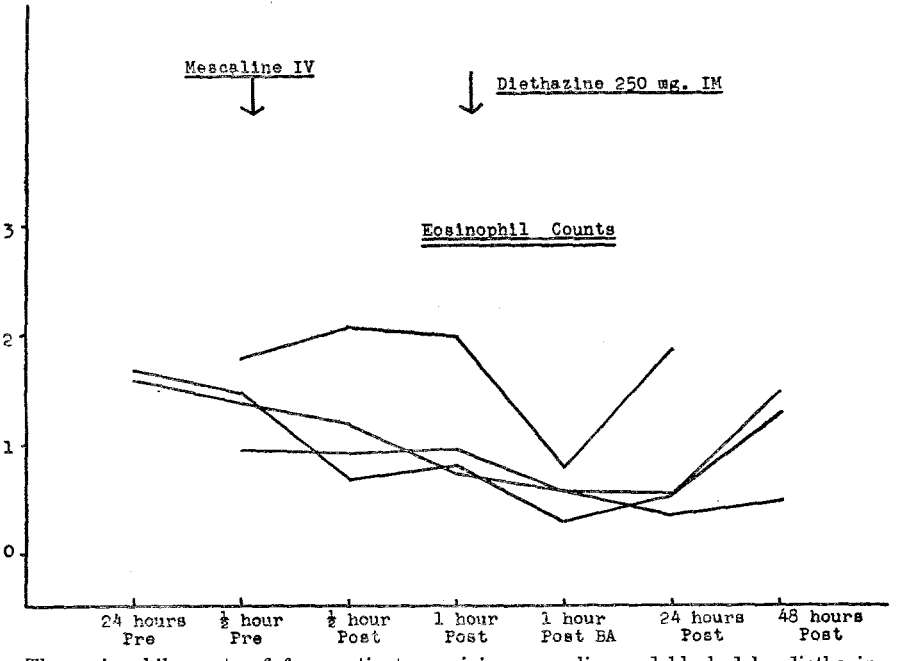
The differential count and sedimentation rate showed no marked consistent variations.

Eosinophils: (Tables 9-11)

The eosinophils fell during the mescaline period. The mean one-half hour before mescaline was 1.21. One-half hour postmescaline it was 1.00. It was 0.81 one hour after injection (P=>.005). Zero determinations were noted in several cases.

One Hour After Blocking Agents

The mean for all groups was 0.69 (P=>.001). The fall was



The eosinophil counts of four patients receiving mescaline and blocked by diethazine.

particularly evident in the diethazine group (see the figure), where the mean fell from 1.02 at one-half hour after mescaline to 0.04 at this time.

24 *Hours After Mescaline*

The mean value for all groups was 0.85 and P=>.005. The mean for diethazine was 0.8.

48 *Hours After Mescaline*

The mean had returned to a value of 1.10.

Hepatic Function

The bilirubin and thymol turbidity (Table 12) were not particularly different during the mescaline period. The cephalin cholesterol flocculation changed in one case from 1+ to 3+ in 24 hours. There were some other variations that were considered within the normal range.

THE CONTROLS (Tables 13-19)

a) Saline + { Chlorpromazine / Trifluperazine

b) No injections

Amino Acids

The fall in amino acids in those patients receiving saline instead of mescaline was smaller in magnitude than that observed with mescaline (see the foregoing) and not significant (P min.=>.10) (Table 18). The mean value for all groups one-half hour before beginning the study was 3.21 μM (ESE=0.442 μM). This fell at one-half hour after saline to 3.08 μM (ESE=3.95 μM and P=>.20). At one hour it was 3.04 μM (ESE=0.635 μM and P=>.25). At one hour after the blocking agents (or two hours after beginning the experiment), the mean was 3.22 μM (ESE=0.145 μM and P=>.80). Twenty-four hours after the experiment was begun, the mean value was 3.29 μM.

Saline-Saline Group

Those patients receiving intramuscular saline as a block showed a rise in amino acids between the time of its injection and one hour after, continuing until 24 hours, after which there was a fall to levels slightly below the base line (Table 13).

Saline-Chlorpromazine Group

There was a slight fall until one hour after injection, with a rebound to the pretest figures at 24 to 48 hours (Table 14).

Saline-Trifluperazine Group

The amino acids were already rising one-half hour after the injection of saline intravenously. When the trifluperazine was given intramuscularly, the rise continued above the base line. Yet the final levels and those during the entire test were not significantly outside the range of the subjects' initial values (Table 15).

No Injection Group

There was a progressive fall until the time when the blocking agent would have been administered—that is, two hours after the start of the study. Then the levels rose slowly, so that, at 24 to 48 hours, they had returned approximately to the pretest figures, but were still slightly inferior (Table 16).

The mean amino acid level two hours after the injection of saline for those who received the blocking agents was 3.24 μM with an estimated standard error of 0.085 μM. For those who received no blocking agents, it was 3.21 μM with an estimated standard error of 0.563 μM. It was 3.22 μM for all of the controls (ESE=0.145 μM). There is, thus, in the control group a significant difference between the estimated standard errors of those receiving a blocking agent and those not receiving one (F=7.683).

In the saline-chlorpromazine group and the saline-trifluperazine group, the amino acid values did not fall significantly outside the range of the base lines obtained. The saline-saline group showed unusual fluctuations (Table 15), while the "no injection" group showed a steady fall to the two-hour level with a rise thereafter.

The Mescaline Only Group

This group showed fairly wide oscillations in all parameters tested; this was most marked with the patient requiring chlorpromazine to terminate the mescaline-induced state two and one-half hours after the onset (Table 17). There is a suggestion that the amino acid levels begin to return to the base line two hours after the mescaline injection. However, the number of cases is too small and the scatter too large to allow any conclusions.

Glucose

The combined averages for all controls (Table 19) showed a mean premescaline value of 88.80 mg.% which fell to 82.9 mg.% one hour post-block ($P=>.20$). In general, there were some slight variations in serum glucose in the different groups, without significant changes.

The serum cholesterol showed some slight fluctuations in the various groups that were not significant.

The leucocyte count varied widely in the saline-trifluperazine and saline-chlorpromazine groups, as well as in the "mescaline only" group, but was relatively stable in the saline-saline and no injection groups.

There are no significant changes in eosinophil counts.

The cephalin cholesterol flocculation, bilirubin and thymol turbidity did not show any significant variations where they were done.

Haloperidol had no effect on any of the parameters tested in the one case where it was used as a blocking agent.

Discussion

The clinical picture induced by mescaline in the chronic and semi-chronic patients is less florid than with the acutely ill patients whose onset of illness is of relatively short duration.[16] In addition, the repetitive withdrawal of blood by venipuncture interposes a whole series of psychodynamic factors which are beyond the scope of this study and could conceivably influence the patients' responses.[17] The results must be considered, therefore, within the framework of the patient population with whom this work was done. Whether similar results would be obtained with acute, first attack schizophrenic patients, or with normal volunteers, is a matter for further study.

The experimental design was not intended to, and could not, yield definite conclusions regarding the biochemical activity of each blocking agent, in view of the small sample in each group. Yet the combined averages gave data that were significant in comparison to the controls. Obviously to obtain specific comparative information about one drug against another, one would have to study its action in at least 10 or 12 patients.

It could conceivably be argued that each patient should have served as her own control with a saline injection before the mes-

caline study. On the other hand, the choice of another group from the same patient population gives a wider distribution of data for analysis. In addition, the patients did not exhibit the same quantitative amino acid and glucose values from one time to another (Patients T.S., N.A. and M.C.). In any case, another more concentrated study is planned, in which the first method will be used.*

From a biochemical point of view, it must be emphasized that the amino acid determinations were really measures of free serum amino nitrogen. The filtrate contained various peptides and polypeptides as well. The results suggest a need for further intensive study in this area to delineate the particular compounds undergoing change.

A thorough statistical analysis was necessary (see p. 33 ff.) since it had to be shown, beyond a doubt, that the changes induced by mescaline did not occur purely by chance and that the blocking drugs had a real effect upon the biochemical patterns. The tables show some ranges to be extremely wide; this was often due to one figure being well above or below the mean. It was felt, however, that the statistical analysis should include all data *as collected.*

The data indicate that the changes in amino acids result from the action of mescaline. However, it must be recalled that they occurred to a slight degree in the controls as well. But in the latter case, the changes were without significance (Table 18). At one hour after mescaline, the average fall in amino acids was two and three-quarter times greater from the base line, as compared with the controls for the same period, and was sharper with mescaline, beginning at the time of injection. The statistical studies support the hypothesis that mescaline exaggerates this fall. The amplitude of change in the control amino acids was insufficient to be significant as determined by an analysis of the means, and was probably due to the fasting state. The amino acid levels with mescaline, observed at one-half hour and one hour postmescaline and one hour postblock, may well represent the low levels attainable under these conditions, since the further net changes between all were no greater than an additional 0.09 μM.

The changes in amino acids occurring with intravenous saline or no injections showed a progressively marked scatter with an

*Studies on mescaline XIII: the effect of prior administration of various psychotropic drugs on different biochemical parameters—N. Y. Acad. Sci. In press.

estimated standard error of 0.395 μM at one-half hour and 0.635 μM at one hour. In the mescaline group, the estimated standard error at one-half hour after injection was 0.509 μM, and at one hour it was 0.330 μM. It is difficult to explain this phenomenon. With mescaline, one could suppose that the variable clinical state at one-half hour is mirrored by the large deviation from the mean. At one hour, the clinical picture is fairly stabilized, and there is little oscillation in the amino acid values as reflected by the small estimated standard error. Yet this leaves the result in the first group still unexplained.

Chlorpromazine and trifluperazine were chosen for the controls, since the former is a straight chain, halogenated phenothiazine and the latter a piperazine phenothiazine. When they were injected after saline, the combined estimated standard errors were significantly smaller (0.085 μM for 11 patients), than with no blocking agent (0.653 μM for 8 patients). This could suggest that both drugs inhibit any oscillations in amino acids. One could then ask the question, "Is this related in any way to the clinical activity of phenothiazines of which these are fair representatives?" When the foregoing drugs were used to block mescaline, no significant changes were noted in the variance of the amino acids in this group as compared to the estimated standard errors of their base lines, one-half hour before mescaline, or the estimated standard errors of the entire group, one hour postblock.

One is faced with a curious paradox, since chlorpromazine and trifluperazine, when injected after mescaline, did not reverse the fall of amino acids, but when given after saline produced an abrupt upswing with reduction of the estimated standard error. The latter findings suggest that these psychotropic drugs do effect the amino acid metabolism, and general aspects of this problem would bear further study. It is possible that larger doses could have produced a similar effect with mescaline. But it is known clinically that 100 mg. of chlorpromazine do not block the mescaline-induced state with any more effect than do 50 mg. It can be reasoned further that the structure and composition of the amino acids formed after intravenous mescaline are not the same as with the controls and, hence, will not respond to the drugs in an analogous manner. It should be noted that Block[18] has postulated that mescaline is "incorporated into the protein of the liver."

This could conceivably prevent the direct biochemical action of the psychotropic drugs.

The blocking action of thioperazine, trifluperazine, triflupromazine, promethazine and chlorpromazine on mescaline is not immediate, since the fall in amino acids continues at least one hour past their intramuscular injection. The upsweep takes place some time afterward, and may be due in part to the nutritional state, since the patients ate shortly thereafter. It would be important to know what happens in the second, third and fourth hours after the injection of a blocking agent. The inability of the blocking agents to reverse the amino acid fall after mescaline (even though the mescaline-induced state was blocked completely with chlorpromazine and triflupromazine) suggests either (a) that the drug is acting on a parameter that we have not measured or (b) that the amino acids are reflecting a more primary process which produces the clinical state after mescaline. In this case, the total amino acids mirror other changes that must be looked for with more refined techniques.

The blood glucose shows significant elevations in the mescaline group. This increase is similar to the finding with LSD of Mayer-Gross, et al.,[19] who postulated that "the carbohydrate metabolism in the presence of LSD is interrupted and cannot pass beyond the stage of hexosemonophosphate." The amino acid findings in this study suggest that the problem is probably much more complex. The fall in the controls was undoubtedly due to the effect of fasting. In this group, a single dose of chlorpromazine had a delayed effect, since at 24 hours the mean blood glucose was 105.0 mg.% ($P=>.10$), and at 48 hours it was 113.5 mg.% ($P=>.05$) (Table 13).

The amino acid and blood glucose patterns show inverse relationships under mescaline. But at one hour after the blocking agents, in general, the amino acids were continuing to fall while the blood glucose rise was reversed. The discordance in their relationship bears further study.

The liver chemistries yielded little information to suggest parenchymatous involvement in the drug-induced state, i.e., absence of changes in cephalin cholesterol flocculation or thymol turbidity. European workers have suggested that mescaline produces variations in the different indices of liver function. Richter[20] has re-

viewed the data regarding "liver function," and questions the value of the tests reported by Georgi.[21]

Since mescaline is considered by some to induce a "shock state," it is surprising that no greater variations were observed in cholesterol. It is possible, of course, that variations were present in its fractions which were not tested. It is also conceivable that serum cholesterol levels will only show variations after a much longer period of time following a given single or multiple stress.[22]

The data confirm Deniker's observations of the "mescaline shock." The increases in blood glucose, leucocytosis, and eosinopenia are similar to his findings. The present data, which seem to suggest that with mescaline alone the various parameters begin to return to their base lines after two or three hours, are in accordance with his observations.

The data do not clearly indicate any specific action of one phenothiazine over another with regard to the parameters studied. Chemical structure does not influence the biochemical findings as had been supposed originally While clinically the halogenated straight chain phenothiazines block the mescaline-induced state at once, the amino acid, blood glucose, white blood count and eosinophils did not reflect this change; the same held true for the others where the clinical symptoms were partially or not at all influenced.

Diethazine was the only compound which showed a continuous fall of amino acids and eosinophils up to the 24- or 48-hour period, as well as a leucocytosis. The highest white count was noted in this group (Patient T.S.—Table 11). The biochemical data are in accordance with the clinical observations of an aggravation of the mescaline-induced state by diethazine. This lends further support to a previous hypothesis regarding the two carbon amine linkage in abnormal states of human behavior.[9] There was no relationship, however, between the severity of the mescaline-induced state and the effects of diethazine.

Is there any correlation between frequency of changes in the various biochemical parameters and the types of clinical reactions to mescaline? Of the 26 trials, 11 were considered to have shown a strong reaction, 14 a moderate response, and two only minimal response. Using the fall in amino acids, increased blood glucose, the fall in eosinophils, and leucocytosis as indicators, there appears to be a positive correlation between the intensity of clinical

reaction and positive biochemical changes. However, the evidence is not complete and the problem is being considered further.

In general, analysis of the experiments suggests the following hypotheses: (1) The fall of amino acids is, perhaps, the result of, or due to, increased deamination, giving rise to increased blood glucose. This would explain the drop in amino acids and elevated levels of glucose. The time sequence is appropriate to this hypothesis. (2) Glucose increases, possibly as a measure of increased mescaline glucuronide conjugation, an indication of metabolic detoxification. (3) The fall in amino acids and rise in glucose are the results of activity in two separate biochemical systems. (4) The rise in glucose might be associated with the reported activity of mescaline inhibiting glucose oxidation. (5) The composition of the amino acid pool is qualitatively changed by mescaline; hence, the blocking agents are ineffective. The opposite results after saline are suggestive. (6) In view of the negative correlation of clinical state, blocking effects and biochemical changes, it may be that the parameters studied are reflecting other, probably more primary processes, which can be producing the observed effects.

The study, while producing much interesting information, has also raised puzzling questions. At what point in time does the reversal of the changes in amino acids, glucose, eosinophils and leucocytes take place? What is the nature of the amino acid changes, and are there qualitative changes in their composition? Are they influenced by the blocking agents? What is the relationship between the fall in amino acids and rise in blood glucose? What happens if patients are pre-treated with psychotropic drugs before mescaline? Will the foregoing biochemical changes be inhibited?

The potential psychotomimetic properties of diethazine deserve further study. These are some of the questions that it is proposed to look into in the future.

In all probability, the reactions observed represent part of a total body response to a stress—in this case chemical. It is conceivable that analogous events are taking place where the stressing agent is psychological or otherwise.

Summary and Conclusions

1. There is a fall in total amino acids and eosinophils, as well as a rise in blood glucose and leucocytes, within one hour after

the intravenous injection of mescaline sulfate. The changes of the first three are statistically significant. Because of the large variation between patients, reliability of the leucocyte averages was low. The total serum cholesterol, cephalin cholesterol flocculation, thymol turbidity and bilirubin did not appreciably change during this time.

There was a slight fall in total amino acids with the control group, but these results were not significant.

2. The intramuscular injection of various psychotropic drugs did not influence the continous fall of amino acids or eosinophils. The leucocytes continued to rise. The blood glucose began to revert to its base line. By 24-48 hours, most of the base line levels had been attained.

3. The changes in total amino acids, eosinophils and leucocytes were still present 24 hours after the injection of diethazine. This compound aggravates the mescaline-induced state in many patients.

4. Chlorpromazine and trifluperazine caused a sharp upswing in amino acids with patients receiving saline instead of mescaline.

5. There was no relationship between chemical structure of the blocking agents and biochemical activity.

6. There appears to be a relationship between clinical blocking activity of a drug and biochemical changes.

7. The theoretical aspects of these findings have been considered with particular reference to the problem of amino acids.

Appendix: Statistical Analysis

Performed by DAVID N. TELLER, M.S., Senior Research Scientist (Biochemistry)

For sample groups with N less than 30, the estimated standard error or ESE was used instead of the standard deviation. Calculation of significance of difference between two means M_1 and M_2 was developed from three formulae for the derivation of t.

The statistical analysis of the data was complicated by the possibility of inherent pairing of results by patients over the experimental period. However, calculation of correlation coefficients, while numerically indicating the pairing of values in certain instances, shows no definite completeness of pairing. This is further shown by the significant differences between certain estimated standard errors and the fact that many $N_1 \neq N_2$. If paired

data analyses were to be used all the low P values would be much lower.

The symbol $P>.01$ refers to the upper limit of P for a t value which is between $P=.01$ and $P=.005$. Thus, where $P>.01$ is written, t is actually larger than $t=P\leqq.01$, which is a more common expression. For large values of P ($P>.20$, $P>.70$, etc.), the t value is in the class $P=.20$ to .10, or $P=.70$ to .60, respectively. Borderline P values (P between .025 and .02) are given as $P<.05>.025$, etc.

Table 1. Blocking Agents Used

	Patients
Diethazine	5
Thioperazine	4
Trifluprομazine	4
Trifluperazine	4
Chlorpromazine	3
Pipamazine	3
Haloperidol	2
Promethazine	1

Table 2. Controls

	Patients
Saline + Chlorpromazine	7
Saline + Trifluperazine	4
Saline + Saline	4
Saline + Haloperidol	1
No injections	4
Mescaline only	4

Table 2A. Blocking Agents—Dose Used

	Mg.
Diethazine	250
Thioperazine	2
Trifluprομazine	50
Trifluperazine	2
Chlorpromazine	50
Pipamazine	20
Haloperidol	3-5
Promethazine	50

Table 3. Mescaline Group. Amino Acids (μM/ml.)

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Diethazine				
M.C.	4.01	2.82	2.42	2.46	2.58	1.66	2.32
M.C.	—	3.62	2.51	2.83	3.01	3.41	—
F.Y.	2.79	2.71	2.53	2.57	2.32	2.39	2.19
T.S.	—	2.61	2.61	2.61	2.42	3.15	2.26
N.A.	—	2.96	2.73	2.72	2.85	1.68	2.12
			Trifluprom azine				
M.D.	2.01	2.50	2.29	2.36	2.31	3.02	—
T.S.	—	3.00	2.90	2.60	2.30	3.20	—
T.S.	2.96	3.10	2.92	2.88	2.36	2.71	2.78
C.V.	3.24	3.30	3.20	2.70	2.50	3.06	—
			Promethazine				
L.C.	3.05	2.70	2.35	2.35	2.32	—	2.99
			Chlorpromazine				
M.L.	3.90	3.40	2.70	—	2.40	4.00	3.90
H.D.	—	3.06	2.62	2.78	2.68	2.91	3.18
Y.C.	3.37	3.32	3.42	3.22	3.12	2.82	2.49
			Haloperidol				
M.V.	3.55	3.66	3.37	2.63	2.84	2.97	3.17
H.G.	—	2.70	2.57	2.43	2.17	2.47	1.31
			Pipamazine				
G.R.	4.50	2.90	1.70	—	2.60	4.30	—
A.R.	3.31	3.09	2.50	2.43	2.42	3.39	3.29
K.P.	3.50	3.80	3.00	3.30	4.00	3.90	—
			Thioperazine				
M.C.	2.40	5.10	3.80	3.05	3.10	3.40	—
J.U.	—	3.20	2.80	2.60	2.50	2.90	—
H.G.	3.06	3.32	3.12	3.32	3.02	2.18	2.26
W.H.	3.12	2.98	1.97	2.04	2.04	1.31	3.57
			Trifluperazine				
A.H.	3.39	2.88	2.64	2.44	2.64	—	3.78
G.H.	3.27	2.87	1.76	2.65	2.25	2.76	3.31
G.D.	2.53	2.91	2.31	2.41	2.41	3.00	—
F.C.	2.58	3.57	3.52	3.17	2.62	2.88	3.57

Table 4. Statistical Analysis. Mescaline + Blocking Agents (Combined Values) Amino Acids (μM/ml.)

Time	N*	M**	High	Low	ESE	P
24 Hrs. Pre	19	3.21	4.50	2.01	0.883	$>.80$
½ Hour Pre	26	3.16	5.10	2.50	0.238	—
½ Hour Post	26	2.70	3.80	1.76	0.509	$>.001$
1 Hour Post	24	2.69	3.32	2.04	0.330	$>.005$
1 Hour Post Block ..	26	2.61	4.00	2.04	0.410	$\leqq.001$
24 Hour Post	24	2.72	4.30	1.31	0.868	$>.10$
48 Hours Post	17	2.85	3.90	1.31	0.711	$>.40$

*N=Number **M=Mean

Table 5. Mescaline Group. Blood Glucose (mg.%)

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Diethazine				
M.C.	—	88.1	93.1	94.1	89.0	89.0	—
M.C.	95.8	98.2	96.3	119.0	—	95.2	102.4
F.Y.	74.2	75.1	80.0	82.9	71.8	101.5	106.0
T.S.	—	89.0	102.7	106.1	98.0	85.6	87.2
N.A.	—	82.2	84.4	94.8	97.1	66.0	104.2
			Trifluprom azine				
M.D.	84.3	86.4	95.4	96.0	102.0	83.0	—
T.S.	—	83.2	105.4	92.5	98.6	86.0	—
T.S.	139.5	84.3	84.7	86.4	59.0	89.2	87.3
C.V.	80.0	90.0	107.0	115.0	110.5	134.0	—
			Promethazine				
L.C.	110.5	81.4	81.4	116.0	109.0	—	92.8
			Chlorpromazine				
M.L.	83.0	105.0	105.0	95.0	95.0	95.0	81.0
H.D.	—	71.1	65.6	88.0	94.0	96.2	—
Y.C.	112.0	91.5	117.5	120.0	133.0	85.9	92.2
			Haloperidol				
M.V.	—	84.6	88.0	89.7	87.6	104.8	95.1
H.G.	—	105.4	108.5	115.5	118.6	114.8	—
			Pipamazine				
G.R.	83.0	82.0	95.0	—	44.0	92.0	—
A.R.	98.1	103.8	146.5	183.5	143.5	93.0	96.8
K.P.	68.1	75.3	103.4	110.4	86.4	93.5	85.2
			Thioperazine				
M.C.	77.5	117.2	97.8	113.9	117.3	108.5	—
J.U.	—	76.8	97.0	111.5	105.5	100.3	—
H.G.	105.0	85.5	83.0	108.0	159.0	80.0	82.7
W.H.	122.0	97.0	108.4	93.6	—	92.0	98.3
			Trifluperazine				
A.H.	95.4	93.0	108.0	106.8	105.8	—	94.4
G.H.	77.0	100.0	118.0	158.0	118.0	102.0	135.0
G.D.	76.8	85.9	74.0	85.9	80.0	78.6	—
F.C.	97.0	80.0	95.0	108.4	95.0	98.0	—

Table 6. Statistical Analysis. Mescaline + Blocking Agents (Combined Values) Blood Glucose (mg.%)

Time	N	M	High	Low	ESE	P
24 Hours Pre	18	93.3	139.5	68.1	18.86	>.40
½ Hour Pre	26	88.9	117.2	71.1	12.87	—
½ Hour Post	26	97.7	146.5	65.6	16.31	>.05
1 Hour Post	25	107.6	183.5	82.9	22.5	>.001
1 Hour Post Block ..	23	103.2	143.5	44.0	23.3	>.01
24 Hours Post	24	94.3	134.0	66.0	13.41	>.10
48 Hours Post	15	90.0	135.0	81.0	13.19	>.80

Table 7. Mescaline Group Total Cholesterol (mg.%)

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Diethazine				
M.C.	—	231	254	256	280	229	—
M.C.	277	228	268	263	286	247	235
F.Y.	333	281	278	272	256	268	256
T.S.	—	290	300	260	302	311	280
N.A.	—	327	344	352	346	434	207
			Trifluprom azine				
M.D.	247	171	238	228	257	183	—
T.S.	—	302	353	342	342	286	—
T.S.	372	275	290	282	279	339	334
C.V.	198	195	198	169	180	201	—
			Promethazine				
L.C.	270	272	214	242	210	—	248
			Chlorpromazine				
M.L.	322	331	328	327	314	328	331
H.D.	—	229	222	210	217	261	280
Y.C.	209	209	233	227	215	204	214
			Haloperidol				
M.V.	296	343	340	265	288	348	330
H.G.	—	306	346	335	335	265	345
			Pipamazine				
G.R.	258	234	245	—	245	203	—
A.R.	276	286	284	313	289	285	295
K.P.	191	—	197	190	195	194	—
			Thioperazine				
M.C.	244	177	248	245	235	168	—
J.U.	—	286	264	252	286	234	—
H.G.	—	143	149	135	149	140	127
W.H.	264	277	290	311	290	264	271
			Trifluperazine				
A.H.	232	230	222	229	229	—	221
G.H.	292	318	327	318	327	308	292
G.D.	249	239	232	213	236	225	210
F.C.	382	294	299	244	311	267	—

Table 8. Mescaline Group
Leucocytes (per cu. mm.)

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Diethazine				
M.C.	—	9,800	10,650	10,100	14,200	9,400	—
M.C.	10,150	10,550	12,200	11,350	16,552	18,200	10,100
F.Y.	6,700	—	7,100	8,800	15,150	15,300	6,850
T.S.	—	11,750	11,800	8,850	11,450	18,900	14,200
N.A.	—	5,450	6,950	6,500	4,050	7,600	7,550
			Trifluprom azine				
M.D.	4,700	6,950	6,200	5,850	5,950	—	—
T.S.	—	7,600	7,475	7,400	4,425	9,000	—
T.S.	—	12,500	9,800	11,500	9,200	13,350	10,950
C.V.	10,100	8,400	8,500	8,900	8,400	9,300	—
			Promethazine				
L.C.	7,250	8,000	9,700	8,500	9,850	—	7,800
			Chlorpromazine				
M.L.	8,650	8,800	8,750	—	10,100	8,800	7,850
H.D.	—	8,450	6,550	5,625	8,300	7,300	4,050
Y.C.	5,000	4,800	5,500	5,000	6,450	4,150	4,600
			Haloperidol				
M.V.	10,550	10,150	12,850	11,300	14,400	13,350	10,950
H.G.	—	16,600	12,900	11,900	12,600	11,650	11,850
			Pipamazine				
G.R.	7,770	8,120	5,820	4,900	6,400	—	—
A.R.	6,600	—	7,850	7,900	10,950	7,850	6,950
K.P.	5,650	5,350	5,400	5,850	5,650	5,300	—
			Thioperazine				
M.C.	9,800	9,000	11,250	11,300	18,650	9,600	—
J.U.	—	16,650	18,350	14,250	12,800	12,800	—
H.G.	4,600	3,900	5,350	6,000	5,500	4,050	4,300
W.H. ...	—	6,250	6,700	7,000	—	7,400	7,000
			Trifluperazine				
A.A.	9,400	9,250	10,250	10,450	10,650	13,450	10,300
G.H.	8,950	10,400	10,600	10,700	11,200	12,900	14,850
G.D.	8,500	8,000	9,200	9,750	12,300	12,000	—
F.C.	8,000	8,000	8,500	8,430	10,250	10,000	—

Table 9. Mescaline Group
Eosinophil Count (per 100 leucocytes)

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Diethazine				
M.C.	—	1.8	2.1	2.0	0.8	1.9	—
M.C.	1.7	1.5	0.7	0.8	0.3	0.5	1.5
F.Y.	1.6	—	1.2	0.7	0.5	0.5	1.3
T.S.	—	0.9	0.9	0.9	0.5	0.3	0.4
N.A.			Not done				
			Triflupromazine				
M.D.			Not done				
T.S.	—	1.4	1.2	0.6	1.0	1.0	—
T.S.	—	1.0	1.1	0.9	0.9	0.9	1.0
C.V.			Not done				
			Promethazine				
L.C.	—	2.5	0.6	0.4	0.5	—	1.7
			Chlorpromazine				
M.L.	0.2	0.5	0.5	—	0.0	0.7	0.9
H.D.	—	0.7	0.6	0.0	0.0	0.0	1.7
Y.C.	—	1.0	2.0	1.0	2.0	1.0	1.0
			Haloperidol				
M.V.	2.3	1.7	0.5	0.6	0.3	1.3	2.2
H.G.			Not done				
			Pipamazine				
G.R.	3.1	1.3	1.1	—	1.8	1.9	—
A.R.	0.5	—	0.3	0.0	0.0	0.3	0.3
K.P.	1.4	1.4	1.1	1.2	1.2	1.2	1.4
			Thioperazine				
M.C.	2.1	1.8	1.1	1.1	0.4	0.8	—
J.U.	—	0.7	0.3	0.3	0.3	0.5	—
H.G.	—	2.0	2.0	1.0	2.0	1.0	—
W.H.	—	1.0	1.0	1.0	Clotted	1.0	1.0
			Trifluperazine				
A.H.	0.9	0.8	0.6	0.5	0.7	0.7	0.9
G.H.	0.0	0.0	0.0	0.0	0.0	0.0	0.0
G.D.	1.0	1.0	1.0	1.0	1.0	1.0	—
F.C.	1.0	1.0	2.0	2.0	2.0	1.0	—

Table 10. Statistical Analysis. Mescaline + Blocking Agents (Combined Values)
Eosinophils*

Time	N	M	ESE	P
24 Hours Pre	12	1.32	0.90	>.70
½ Hour Pre	20	1.21	0.57	—
½ Hour Post	22	1.00	0.59	>.20
1 Hour Post	20	0.81	0.55	>.005
1 Hour Post Block ...	21	0.69	0.64	>.001
24 Hours Post	21	0.85	0.50	>.005
48 Hours Post	14	1.10	0.59	>.50

*High-low range not given because zero findings in each group do not give accurate range. See Table 9.

Table 11. Statistical Analysis. Controls (Combined Values)
Eosinophils

Time	N	M	ESE	P
24 Hours Pre	14	1.46	0.97	>.80
½ Hour Pre	14	1.40	0.79	—
½ Hour Post	13	1.62	0.89	>.40
1 Hour Post	13	1.63	0.75	>.30
1 Hour Post Block ...	14	1.75	0.97	>.20
24 Hours Post	15	1.39	0.73	>.90
48 Hours Post	9	1.54	0.85	>.70

Table 12. Mescaline Group
Thymol Turbidity (MacClagan units)

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Diethazine				
M.C.	—	0.9	0.9	0.9	0.9	0.9	—
M.C.	0.6	0.7	0.7	0.9	0.7	0.6	0.6
F.Y.	1.5	1.3	1.5	1.3	1.0	1.5	1.3
T.S.	—	0.6	0.6	0.6	0.5	0.4	0.4
N.A.	—	0.7	0.6	0.6	0.7	0.7	0.7
			Trifluprom azine				
M.D.	1.3	0.9	1.0	0.9	1.0	1.2	—
T.S.	—	0.4	0.4	0.5	0.5	0.5	—
T.S.	0.6	0.5	0.5	0.5	0.5	0.6	0.9
C.V.	1.2	1.0	1.0	0.7	0.6	1.2	—
			Promethazine				
L.C.	1.8	1.2	1.2	1.2	1.2	—	1.2
			Chlorpromazine				
M.L.	2.5	2.3	2.0	2.1	2.0	2.0	2.0
H.D.	—	1.2	1.2	1.0	1.0	1.3	—
Y.C.	0.7	1.0	1.0	1.2	1.0	1.0	1.0
			Haloperidol				
M.V.	1.0	1.0	1.0	1.0	0.9	0.7	1.0
H.G.	—	1.2	1.2	1.0	1.0	0.9	1.0
			Pipamazine				
G.R.	2.1	2.3	2.3	—	2.3	2.3	2.0
A.R.	2.8	3.1	3.7	3.3	3.5	2.1	2.1
K.P.	2.3	2.3	2.3	2.3	2.3	2.2	2.2
			Thioperazine				
M.C.	0.7	0.7	1.3	1.2	1.0	1.6	—
J.U.	—	1.2	1.5	1.3	1.3	1.3	—
H.G.	0.7	1.5	1.2	1.0	1.0	0.7	0
W.H.	1.5	1.5	1.5	1.5	1.5	1.5	1.3
			Trifluperazine				
A.H.	1.3	1.3	1.3	1.3	1.3	1.3	1.3
G.H.	2.0	2.0	1.8	1.8	1.8	2.0	1.5
G.D.	1.8	1.0	1.0	1.0	0.9	0.1	—
F.C.	2.6	2.5	2.5	2.3	2.3	2.3	—

Table 13. Control Group
Saline + Saline

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Amino Acids (μM/ml.)				
M.H. ...	4.465	2.880	3.740	3.190	3.860	4.880	2.580
M.H. ...	3.620	3.620	3.260	3.400	4.095	4.600	2.982
R.R.	3.860	3.980	2.580	3.190	3.860	3.740	2.670
L.G.	4.600	2.982	2.880	2.580	3.190	4.880	3.295
			Total Cholesterol (mg.%)				
M.H. ...	312	311	266	309	293	309	—
M.H. ...	293	246	253	231	311	233	—
R.R.	291	233	224	230	264	238	302
L.G.	312	311	266	309	293	309	—
			Blood Glucose (mg.%)				
M.H. ...	109.2	85.4	82.7	67.0	88.1	129.5	80.0
M.H. ...	116.2	74.6	100.0	112.4	85.2	59.6	82.7
R.R.	126.0	112.4	133.5	153.0	103.2	96.9	61.9
L.G.	93.8	100.0	103.2	61.9	45.8	129.5	82.7
			W.B.C.				
M.H. ...	5,600	5,500	5,750	5,900	6,150	6,300	5,350
M.H. ...	7,000	6,825	7,000	7,250	7,300	7,350	6,300
R.R.	7,400	7,000	7,150	7,150	7,650	7,050	6,250
L.G.	9,200	9,650	9,650	10,050	10,150	11,000	9,850
			Eosinophils				
M.H. ...	1.4	1.1	1.3	1.3	1.4	1.0	1.4
M.H. ...	1.0	1.0	1.0	1.1	1.4	1.1	1.1
R.R.	1.6	1.6	1.6	1.8	1.6	1.4	1.2
L.G.	2.8	2.4	2.5	2.8	2.6	2.4	2.0

Table 14. Control Group
Saline + Chlorpromazine

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Amino Acids (μM/ml.)				
C.L.	3.690	3.690	3.475	3.582	3.380	3.796	3.654
W.C. ...	3.475	2.999	3.284	2.999	3.189	3.690	3.118
L.L.	3.094	3.475	3.189	3.094	2.725	3.534	3.062
H.F.	1.735	3.375	3.470	3.275	3.275	3.790	3.570
L.L.	2.544	2.675	3.470	3.675	3.570	2.080	4.010
D.M. ...	—	4.120	3.275	3.375	3.375	1.895	3.470
C.F.	2.710	3.570	3.570	3.375	3.180	2.283	3.470
			Total Cholesterol (mg.%)				
C.L.	322	308	308	343	357	325	394
W.C.	313	286	273	307	304	311	303
L.L.	278	286	286	293	303	290	261
H.F. ...	358	340	288	270	266	293	410
L.L.	188	259	255	241	258	300	311
D.M.	—	331	365	364	292	326	393
C.F.	240	300	244	247	251	280	321
			Blood Glucose (mg.%)				
C.L.	90.0	114.5	90.0	51.5	59.2	107.5	110.0
W.C. ...	48.5	128.5	100.5	83.5	93.5	93.5	115.0
L.L.	68.5	59.5	54.0	68.5	68.5	97.0	71.0
H.F. ...	80.0	113.0	111.0	109.0	105.0	83.5	125.5
L.L.	75.7	105.0	—	—	—	147.0	—
D.M. ...	—	—	—	121.5	109.0	101.5	125.5
C.F.	133.8	113.0	121.5	121.5	125.5	105.0	121.5
			W.B.C.				
C.L.	5,150	5,700	5,600	6,100	6,150	5,200	6,050
W.C. ...	13,500	9,000	9,650	8,250	8,400	8,050	7,700
L.L.	7,800	6,200	6,400	6,950	7,050	7,800	6,700
H.F.	8,750	9,000	9,900	10,050	10,000	8,050	8,200
L.L.	6,400	5,750	—	—	—	5,550	5,750
D.M.	—	—	—	8,300	8,000	6,950	6,650
C.F.	6,250	6,500	6,875	—	7,100	7,500	7,600
			Eosinophils				
C.L.	3.3	1.3	1.1	1.3	1.3	1.0	1.0
W.C. ...	0.0	0.0	0.2	0.9	0.8	0.7	0.2
L.L.	1.3	3.0	3.1	3.4	3.5	3.0	2.8
H.F.	1.4	2.0	2.6	2.1	2.0	0.8	2.8
L.L.	1.0	1.7	—	—	—	2.0	—
D.M. ...	—	—	—	1.1	3.9	1.8	—
C.F.	3.0	0.8	2.7	—	0.7	2.1	1.4

Table 15. Control Group
Saline + Trifluperazine

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Amino Acids (μM/ml.)				
J.S.	2.827	2.878	2.437	2.878	3.265	2.878	2.934
A.B.	2.700	2.613	2.262	2.613	3.265	2.525	2.616
N.A.	3.468	3.065	2.878	2.878	2.789	2.700	2.716
B.T.	—	2.525	3.065	2.878	3.567	3.265	3.104
			Total Cholesterol (mg.%)				
J.S.	265	288	284	265	265	325	272
A.B.	205	193	194	230	308	237	230
N.A.	344	295	304	320	312	317	299
B.T.	—	233	295	363	304	338	348
			Blood Glucose (mg.%)				
J.S.	57.0	57.0	80.0	74.2	70.0	68.5	71.3
A.B.	71.4	72.8	77.1	74.2	77.1	77.1	81.0
N.A.	65.6	80.0	89.6	80.0	77.1	77.1	77.1
B.T.	—	59.4	74.2	74.2	74.2	89.6	80.3
			W.B.C.				
J.S.	5,000	5,000	4,900	4,910	5,000	5,850	6,100
A.B.	7,400	7,550	7,100	8,000	7,200	9,600	7,450
N.A.	7,000	5,250	7,150	6,900	6,000	5,950	6,300
B.T.	—	8,200	8,300	8,200	8,200	8,550	8,200
			Eosinophils				
			Not done				

Table 16. Control Group
No Injections

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Amino Acids (μM/ml.)				
A.B.	3.070	3.172	2.891	2.538	2.367	2.794	2.891
M.E.	3.562	3.070	2.977	2.623	2.623	3.367	2.708
H.H.	3.465	3.172	2.891	2.977	2.977	2.708	2.538
V.M.	2.977	3.172	2.977	2.708	2.708	3.070	2.981
			Total Cholesterol (mg.%)				
A.B.	303	380	—	345	323	320	312
M.E.	231	326	293	293	294	281	286
H.H.	283	283	301	281	294	299	286
V.M. ...	354	296	320	297	320	367	367
			Blood Glucose (mg.%)				
A.B.	86.2	63.5	92.4	92.4	92.4	52.9	48.0
M.E. ...	112.0	98.7	86.2	98.7	92.4	98.7	133.8
H.H.	98.7	101.8	80.0	92.4	98.7	105.3	86.2
V.M. ...	63.5	119.1	92.4	83.1	95.5	98.7	129.8
			W.B.C.				
A.B.	7,200	8,700	8,900	8,950	8,850	7,100	—
M.E. ...	5,500	5,750	6,050	6,950	6,850	5,800	—
H.H. ...	6,600	7,650	7,750	8,150	8,200	8,050	—
V.M. ...	7,200	7,450	7,500	7,600	7,550	6,500	—
			Eosinophils				
A.B.	0.7	1.9	1.8	1.8	1.9	1.2	—
M.E. ...	1.3	1.3	1.3	1.4	1.3	1.2	—
H.H. ...	1.3	1.2	1.3	1.2	1.1	0.8	—
V.M. ...	0.3	0.3	0.5	1.0	1.0	0.3	—

Table 17. Control Group
Mescaline Only

Name	24 Hrs. Before	Pre-Mesc.	½ Hr. After	1 Hr. After	1 Hr. After B.A.	24 Hrs. After	48 Hrs. After
			Amino Acids (μM/ml.)				
M.C.	2.904	3.142	3.094	2.820	2.856	2.642	3.480	3.033
M.L.	3.237	3.480	3.480	3.540	2.550	3.030	3.120	—
J.P.	3.480	2.720	2.350	2.360	2.370	—	—	—
D.McG. ..	3.120	3.870	3.270	2.970	2.880	*	3.550	4.060
			Total Cholesterol (mg.%)				
M.C.	235	206	197	279	266	261	282	256
M.L.	233	403	433	405	423	248	314	—
J.P.	216	209	210	210	210	—	—	—
D.McG. ..	312	225	273	314	313	*	242	380
			Blood Glucose (mg.%)				
M.C.	114.5	90.0	122.0	134.0	97.0	118.5	93.8	96.5
M.L.	111.0	105.5	108.5	89.4	95.5	96.5	99.2	—
J.P.	97.2	93.4	85.7	70.7	83.1	—	—	—
D.McG. ..	—	105.0	162.0	158.0	107.0	*	110.0	111.0
			W.B.C.				
M.C.	13,450	13,800	17,950	19,050	19,000	12,550	12,900	11,400
M.L.	8,350	8,400	8,750	8,900	8,850	8,850	9,500	—
J.P.	9,900	9,950	9,350	10,350	9,750	—	—	—
D.McG. ..	—	6,650	7,550	7,800	7,450	*	6,750	6,150
			Eosinophils				
M.C.	0.5	1.0	1.3	1.5	1.5	1.7	1.1	1.0
M.L.	1.4	1.0	1.1	1.1	1.3	0.9	1.0	—
J.P.	1.0	1.0	0.0	2.0	2.0	—	—	—
D.McG. ..	—	2.6	1.2	1.0	2.5	*	1.4	1.9

*Chlorpromazine, 50 mg. I.M. at 2½ hour period

Table 18. Statistical Analysis Controls (Combined Values)
Amino Acids (μM/ml.)

Time	N	M	High	Low	ESE	P
24 Hours Pre	17	3.29	4.60	1.74	.703	>.20
½ Hour Pre	19	3.21	4.12	2.53	.442	—
½ Hour Post	19	3.08	3.48	2.26	.395	>.20
1 Hour Post	19	3.04	3.68	2.54	.635	>.25
1 Hour Post Block ...	19	3.22	4.10	2.38	.145	>.80
24 Hours Post	19	3.29	4.88	1.90	.877	>.70
48 Hours Post	19	3.07	4.01	2.54	.414	>.30

Table 19. Statistical Analysis Controls (Combined Values)
Blood Glucose (mg.%)

Time	N	M	High	Low	ESE	P*
24 Hours Pre	17	88.0	116.0	48.5	25.08	—
½ Hour Pre	18	88.8	119.0	57.0	22.09	—
½ Hour Post	16	90.4	133.5	54.0	17.70	—
1 Hour Post	16	86.0	112.0	51.5	24.31	—
1 Hour Post Block ...	16	82.9	105.0	45.8	16.50	—
24 Hours Post	19	95.7	129.5	52.9	23.63	—
48 Hours Post	19	95.2	133.8	48.0	26.47	—

*P values are all greater than .20

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