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Responses to hypovolaemic thirst in gerbils

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BY

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Perhaps some people naturally write well organized, coherent sequences of sentences. Unfortunately, I do not include myself in this group of people. It was necessary to have much assistance. Dr. Fred Kozub was invaluable in providing analytical rationale and an inexhaustible supply of red pencils for corrections. Dr. Ken Blick took many moments from a busy schedule to aid in the various statistical procedures. Special acknowledgement is given to my wife, Ruth Marie, who was not only helpful in data collection, typing, and animal maintenance, but also provided understanding and encouragement throughout the writing of the paper.

Abstract

In the present study, a series of experiments were conducted to examine how the gerbil responds to an extracellular challenge. Adult female gerbils, weighing from 50 gm. to 70 gm., were either exposed to a 10% (vol x wt) polyethelene glycol injection, or a vehicle control injection. Within each injection level, half of the animals had access to water after injection, while the other half had access to a .9% saline solution. There was a significant increase in fluid intake associated with injection dosages and animals drank significantly more when exposed to saline than when exposed to water. It was concluded that the gerbil responds to an extracellular insult in a similar manner as the rat. Hematocrit values, taken at 12 and 24 hours after injections, provide a supportive, interpretive corollary to the fluid intake data. Delayed access to fluids following injections resulted in an inconsistent fluid intake trend. It was postulated that an additional regulatory mechanism of a limited efficiency range was operating in conjunction with fluid intake to produce this inconsistency.

INTRODUCTION

The physiology of thirst and drinking has received a great deal of attention over the past twenty years. However, the roots of this investigation can be traced back at least as far as the eighteenth century. Dupuytren and Latta (cited in Blass, 1974) demonstrated that thirst could be assuaged by injections of water or isotonic solutions. These were important findings since they demonstrated that oropharyngeal factors could not singly explain the phenomenon of thirst. Subsequent research has clearly shown that although oropharyngeal factors such as taste, smell, and dryness of the pharynx, play a characteristic role in thirst and drinking, sensations from the oropharynx are not essential for the regulations achieved by food and water intake (Epstein, 1961). Today researchers believe that to a large extent the sensation of thirst is linked to volume deficits and/or concentration increases occurring in the cellular and extracellular fluid compartments of the organism (Blass, 1974). A review of the literature suggests that the physiological and neurological mechanisms underlying thirst associated with deficits in cellular and extracellular space are quite different and largely independent. Corbit (1968), demonstrated that, in the rat, cellular dehydration and hypovolaemia are both effective dispogens and their effects combine in an additive manner. Similar findings were arrived at by Almli et. al. (1975). These authors

demonstrated that a combination of polyethelene glycol (PG) and hypertonic NaCL injections (which produces extracellular and cellular deficits, respectively) produced drinking that did not differ in amount drunk from rats undergoing 24 hour deprivation (which causes deficits in both compartments). However, vascular measure revealed that the time course of vascular changes differ between the two treatments. With the water deprived rat, drinking results in osmotic dilution and volume increases. A similar effect is produced by the combined PG and NaCL treatment but the rate of osmotic dilution is much slower. This is due to the fact that the NaCL injection diffuses into the vasculature, adding ions, and allowing osmolarity levels to remain higher. The higher osmolarity associated with the combined treatment results in a larger volume of water being sequestered.

When extracellular depletion is produced without the addition of NaCL injections or stomach loads, an interesting phenomenon develops in the rat. Stricker (1969) has demonstrated that hypovolaemic rats discontinue drinking water prior to restoring their extracellular deficit. Stricker states, "Although this inhibition of drinking seems inappropriate for volume regulation, it is possible that it instead subserves osmoregulation by preventing further increases in body-fluid dilution."

Stricker validated this assumption by showing that hypovolaemic rats would drink to restoration of the volume deficit if an isotonic saline was offered in lieu of water.

Although research dealing with the physiology of thirst in the rat has been extensive, very little attention has been devoted to a comparative analysis using arid dwelling animals such as the gerbil. It is logical that arid dwelling animals should differ from the rat in their ability to maintain circulating plasma levels in spite of apparent water deprivation associated with hot, dry environments. Kutscher (1968) demonstrated that these differences do indeed exist. It was found that, following water-deprivation, there was statistically significant declines in mean plasma volumes in hamsters, rats, and guinea pigs, but not in the gerbil. Further differences between rats and gerbils were illuminated by Dunstone et. al. (1971). This study demonstrated that food-deprived gerbils lost significantly more body weight than did water-deprived gerbils. These results are in contrast to those obtained with rats which reveal no differences in the deprivation conditions. In addition, gerbils deprived of both food and water do not differ from gerbils deprived of food alone. Rats, however, lose

more weight in water-food deprivation conditions than in either deprivation condition alone.

There is an unfortunate scarcity of literature dealing with how the gerbil responds to specific dispogenic challenges. A notable exception is the work of Almli and Weiss (1974). These authors subcutaneously injected a 25% polyethelene glycol solution (20,000 MW) into the hindquarters of 54 male gerbils and 54 rats. There were three treatment groups; (a) animals allowed immediate access to water following PG injection, (b) animals allowed access to water with 6 hours of deprivation following PG injections and (c) untreated controls. Latency to drink and total volume of water ingested were measured. These authors demonstrated that both rats and gerbils initiated drinking following a similar time lapse, and all drank equivalent volumes of water. In addition, hemotocrits revealed that both species sustained equivalent hypovolaemia.

These findings suggest that the rat and gerbil respond in the same manner to extracellular insult. However, deficiencies in their design prevent the formation of a definitive statement. Unfortunately, only one dosage of polyethelene glycol was utilized, preventing generalization to other dosage levels. In addition, latency to drink does not tell us the total time sequence of drinking following extracellular insult.

Finally, the design does not fully investigate whether the gerbil undergoes voluntary dehydration in defense of concentration.

In the present study, a series of experiments will be conducted to examine how the gerbil responds to a pure extracellular challenge. The first experiment investigates whether the gerbil will react to hypovolemia induced by different dosages of polyethelene glycol and if he will increase drinking when offered saline in lieu of water. The second experiment is an interpretive corollary to the first, investigating whether hemotocrit values will be affected by the respective PG dosages. The third experiment will investigate drinking of the gerbils subjected to the same experimental manipulations as the first experiment, with the addition of delayed access to the drinking fluids.

Experiment I

Method

Animals

The animals used were adult female gerbils, weighing between 50 and 70 g., obtained from Tumblebrook Farms, Brant Lake, New York. The animals were housed under constant illumination in a temperature controlled room (23-26^{o}C) with ad lib access to food and water for approximately 2 weeks prior to the beginning of the experiment to allow for aclimitization to our laboratory conditions.

Apparatus and Procedure

A total of 108 animals were housed in individual cages and measurements of body weight and fluid intake were recorded every 24 hours for three days prior to treatment. After insuring that body weights did not fluctuate more than four grams and fluid intake was relatively invariate ($\pm$ 3 ml.), the animals were randomly assigned to six independent groups with 18 animals in each group. Subjects received the following 2% b.w. subcutaneous injections: Group 1, 30% PG (wt. x vol.) dissolved in a .9% NaCL vehicle; Group 2, 10% PG (wt. x vol.) in the saline vehicle; and Group 3, 0.9% NaCL. Animals were lightly etherized for approximately 20 sec. prior to injection time. Within each treatment level, half of the animals had access to water and the other half had free access to .9% saline solution. Fluid intake measurements were taken at 2 hour, 4 hour, 6 hour, 12 hour, 24 hour, and 48 hour periods following injections and for several days after the injection to observe the recovery from the PG.

Results and Discussion

Unfortunately, there were disproportionate losses in the six groups due to water bottle failure, illness, and questionable injections.[1] Fluid intake measures were converted to percent body weight and expressed as a fraction of the 24 hour intake immediately prior

to injection. Mean intakes and final N's are shown in Table I. Graphic illustration of the data appears in Figure I. Experimental data was analyzed with analysis of variance. A summary of results and significance levels is depicted in Table 2. As was predicted, there were significant differences associated with injection dosages. Significant differences were also found to be associated with type of fluid offered. However, both of these effects were dependent on time elapsed following injection. One would expect that differences in behavioral indices such as water intake would follow the developmental course of the physiological effects associated with polyethelene glycol injections. As time elapsed following injection, the animals increased their water intake in response to the induced dispogenic challenge. As can be seen from Figure I, the greatest increase in fluid intake occurs at the 24 hour data point, followed by a decrease to slightly depressed normal levels. Post hoc analysis of the data at 24 hours reveals that there were no significant differences among the experimental groups that had free access to water following injection. However, differences did exist among the groups that had access to saline. The 30% PG group differed significantly from both the 10% PG and .9% NaCL groups (p .011, Fisher probability test). There was no significant difference between the

TABLE 1

Mean Intakes Expressed as % b.w.
Prior 24 hour Intake
At 2, 4, 6, 12, 24, & 48 hours After Injection

Injection & Fluid

	Time After Injection (Hrs.)					
	2	4	6	12	24	48
30% H_2O N=11	.033	.168	.313	.562	1.15	.817
30% NaCL N=16	.028	.257	.442	1.09	1.78	.608
10% H_2O N=18	.026	.132	.225	.579	1.08	.773
10% NaCL N=11	.031	.151	.310	.662	1.01	.689
.9% H_2O N=10	.024	.092	.192	.363	.884	.988
.9% NaCL N=10	.023	.083	.144	.322	.904	.845

TABLE 2

Significance Table For Exp. 1

Treatments	F	Sig. Level
Injection Dosage	10.91	.05
Fluid Offered	4.48	.05
Time after Injection	332.8	.05
Injection x Fluid	2.30	N.S.
Injection x Time	14.84	.05
Fluid x Time	10.33	.05
Injection x Fluid x Time	3.37	.05

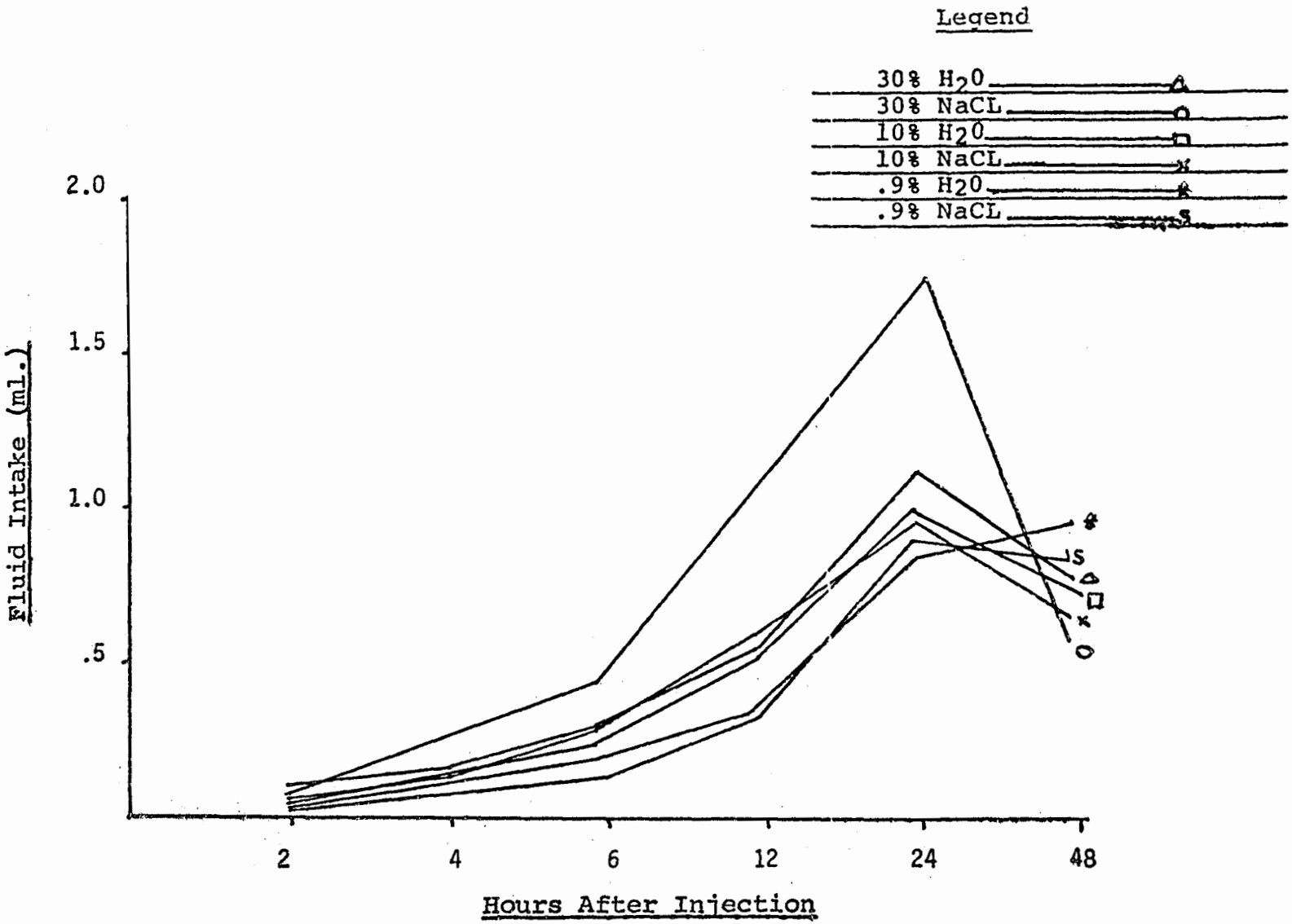

Figure 1.

Mean Intakes (%b.w./Prior 24 Hr. Intake)
at 2, 4, 6, 12, 24, & 48 Hrs. After Injections

10% PG and .9% NaCL groups. Comparing fluid access differences within separate injection levels, we find a similar pattern. The only significant difference between animals offered water to drink and those offered saline occurs in subjects that received the 30% PG injection ($p < .001$, Fisher probability test). This leads one to conclude that the .9% NaCL injections did not promote drinking in the gerbils; instead they slightly suppressed drinking through the introduction of additional isotonic fluid. The 10% PG injections increased drinking slightly but apparently alternative regulatory mechanisms are sufficient to compensate for this mild stressor. The 30% PG injection is a potent stressor and increases drinking substantially. Support for these conclusions are apparent in Figure I. At the point of maximum intake (24 hrs), the 10% PG groups are both above the prior 24 hour intake level and the .9% NaCL groups are both slightly below their prior level. The 30% PG group with access to water is above the 10% PG groups, although the separation is not statistically significant. This suggests that the gerbil is behaving like the rat and inhibits compensatory drinking in order to prevent excessive body-fluid dilution. Confirmation of this conclusion depends on the hematocrit values obtained in Experiment IA

Experiment IA

Method

Animals

Sixty gerbils were utilized and housed as in Experiment I.

Apparatus and Procedure

Animals were randomly distributed into six independent groups utilizing the same experimental design as in Experiment I. Heart puncture blood samples were withdrawn 12 and 24 hours following injection. The blood samples were withdrawn under light ether anesthesia using heperanized glass syringes and 25 ga. heperanized needles. The hemotocrit is highly correlated with plasma volume and therefore is an indirect measurement of hypovolaemia.

Results and Discussion

The experimental data was analyzed by analysis of variance. Mean hematocrit values are presented in Table 3. These values support the conclusions made from Experiment I. The .9% NaCL injections did not affect plasma volume and hematocrit values for these groups were close to the normal gerbil hematocrit value of 42. Hematocrit values for the 10% PG groups were slightly elevated but within normal limits at 12 and 24 hours. The hematocrit values for the 30% groups

TABLE 3

Mean Hematocrit Values
Taken at 12 & 24 hours After Injection

Injection & Fluid	Time After Injection: 12 hrs.	24 hrs.
30% H_2O	49.0	41.0
30% NaCL	44.6	39.8
10% H_2O	43.8	44.2
10% NaCL	45.7	42.0
.9% H_2O	42.0	42.2
.9% NaCL	41.4	42.0

TABLE 4

Significance Table for Exp. IA

Treatments	F	Sig. Level
Injections	2.72	N.S.
Fluid Offered	2.27	N.S.
Time After Injection	9.99	.05
Injection x Fluid	1.09	N.S.
Injection x Time	7.16	.05
Fluid x Time	0*	N.S.
Injection x Fluid x Time	2.2	N.S.

*Due to a slightly negative SS, F was rounded to 0.

were elevated at 12 hours (particularly the group having access to water) and had not only restored their deficits at 24 hours, but had overcompensated slightly. Analysis of variance confirms these observations. Treatment effects and significance levels are presented in Table 4. As one can see, there were significant differences between hematocrit values obtained at 12 hours and values obtained at 24 hours. However, these differences were dependent on injection level, specifically the 30% PG injection. Within this treatment level, 12 hours after injection the group having access to water had hematocrit values that differed significantly from the group that had access to saline ($p < .01$, Fisher probability test). The two groups did not differ at 24 hours, both having slightly depressed hematocrit values. The difference in hematocrit values taken at 12 hours compared to those obtained at 24 hours was significant for both groups ($p < .05$, Fisher probability test). The hematocrit values, in conjunction with the water intake data of Experiment I, verify that the gerbil does undergo voluntary dehydration, subserving regulatory mechanisms of tonicity. The 30% PG group with access to saline was able to take in ions in addition to fluid which enabled them to correct the extracellular deficit more efficiently than could the group with access to

water. The group with access to water had to inhibit fluid intake to prevent body fluid dilution. Hematocrit values also confirm that the .9% NaCL injections did not produce a dispogenic challenge and that the 10% PG injections were only a mild stressor.

Experiment II

Method

Animals

Sixty animals were obtained and housed as in Experiment I[2].

Apparatus and Procedure

The procedure followed in this experiment was identical to Experiment I, except the animals had 6, 12, and 24 hour delays before being allowed access to either water or saline. The three delay durations were run in three separate trials utilizing a repeated measures design. Each delay duration was separated by two or three weeks so animals would return to a consistent base rate of water intake.

Results and Discussion

Fluid intakes were converted to percent body weight and expressed as a fraction of prior 24 hour intake. The data were analyzed by analysis of variance. An analysis was conducted for each of the three delay

durations separately, as well as analysis of differences between the 6 hour delay and 12 hour delay trials at 24 hours after injections, and comparison of all three delay conditions at 48 hours after injection. To avoid confusion, results and discussion will be presented in the same order as the analysis.

Mean intakes and final N's for the six hour delay trial is presented in Table 5. These data appear in Figure 2. One can see that the pattern of drinking closely resembles that found in Experiment I. Twelve hours after injection, the treatment groups show differences, these differences reaching their greatest disparity at 24 hours after injection. The .9% NaCL groups have slightly suppressed intakes at 24 hours but return to normal at 48 hours. The 10% PG groups have slightly elevated intake at 24 hours but intake has dropped at 48 hours. The 30% groups have elevated intakes at 24 hours but drop to within normal limits at 48 hours. In addition, the 30% PG group with access to saline is substantially higher at 24 hours than the 30% PG group with access to water, further collaborating that the gerbil undergoes voluntary dehydration. Analysis of variance results for the six hour delay trial is presented in Table 6. As hypothesized, there were main effects associated with level of injection and type of fluid offered. Also, as one would expect, there was a

TABLE 5

Mean Intakes Expressed as % b.w.
Prior 24 hr. Intake for 6 Hour Delay

Injection & Fluid	Time After Injection 12 hrs.	24 hrs.	48 hrs.
30% H_2O N=6	.716	1.56	.948
30% NaCL N=8	1.36	2.20	.839
10% H_2O N=9	.649	1.21	.874
10% NaCL N=9	.843	1.69	1.43
.9% H_2O N=8	.149	.587	1.00
.9% NaCL N=7	.271	.766	1.43

TABLE 6

Significance Tables For 6 Hour Delay

Treatments	F	Sig. Level
Injections	18.38	.05
Fluid Offered	21.85	.05
Time After Injection	82.88	.05
Injection x Fluid	0*	N.S.
Injection x Time	39.86	.05
Fluid x Time	2.05	N.S.
Injection x Fluid x Time	4.38	.05

*Due to a slightly negative SS, F was rounded to 0.

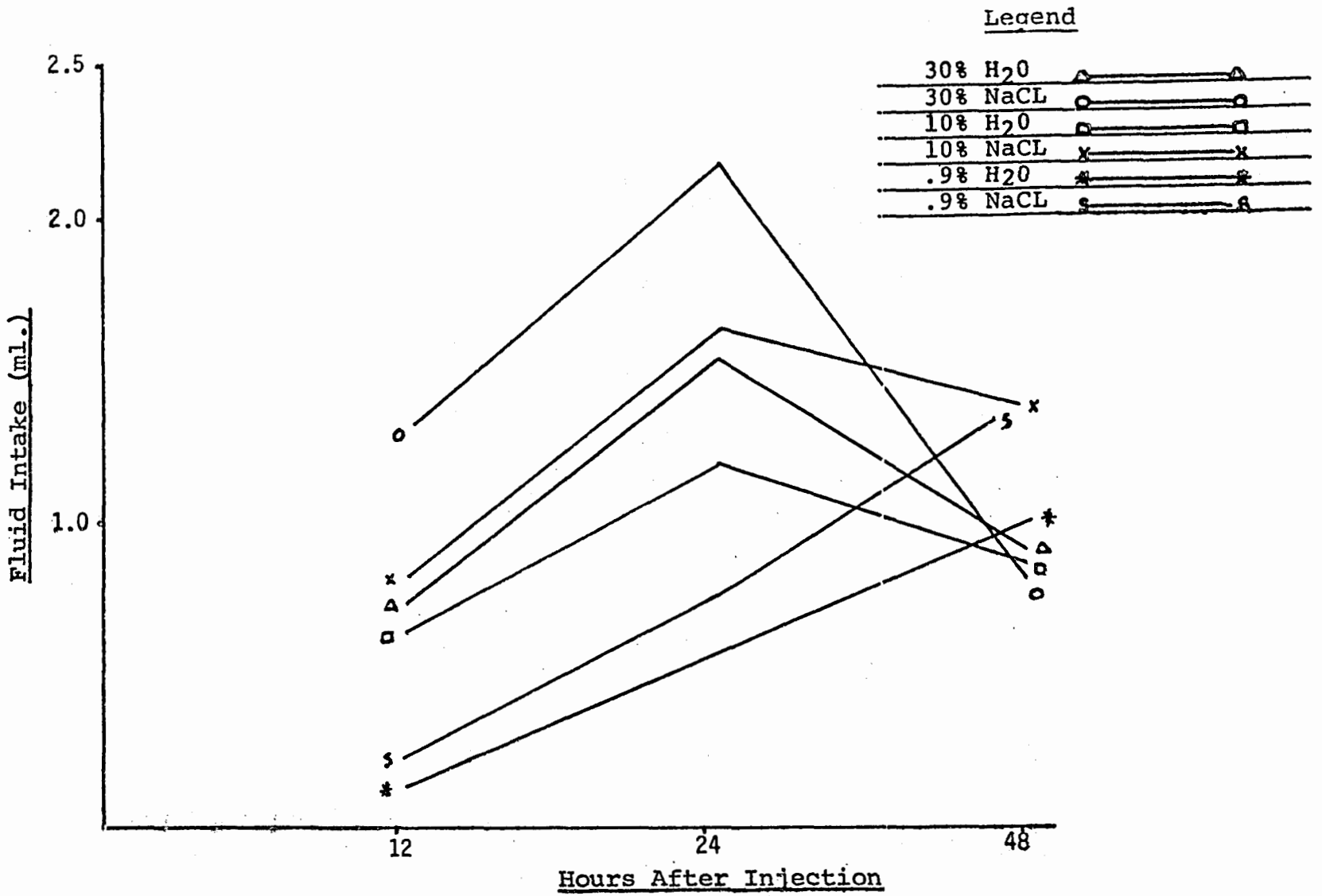

Figure 2.

Mean Intakes (%b.w./Prior 24 Hr. Intake)
at 12, 24, & 48 Hours After Injections

main effect associated with time after injection consistent with the hypovolemia produced by polyethelene glycol. There also were significant differences in how the injection levels developed over time. The .9% NaCL groups showed an increase to normal intake levels at 48 hours and PG injected groups showed elevated intakes at 24 hours followed by a decline in intake at 48 hours. One would conclude from these results that the six hour delay did not have an appreciable effect on the animals and they were responding primarily to stress associated with injection levels. Compared to the first experiment, the net effect of the 6 hour delay appeared to be a slight increase in fluid intake for the PG injected groups.

A slightly different intake pattern exists for the 12 hour delay trial. Mean intakes are presented in Table 7. These data appear in Figure 3. Surprisingly, although the 12 hour deprivation period should have an augmentive effect with the polyethelene glycol injections, the mean intakes are, in general, lower than those associated with the six hour delay trial. This would suggest that perhaps a threshold for an additional regulatory mechanism has been reached. With 12 hours of deprivation, the animal is now attending to an additional extracellular deficit, as well as increased tonicity, and cellular depletion. It is suggested

TABLE 7

Mean Intakes Expressed as % b.w.
Prior 24 Hour Intake For 12 Hour Delay

Injection & Fluid	Hours After Injection: 24 hrs.	48 hrs.
30% H_2O N=6	1.25	1.24
30% NaCL N=8	2.50	.641
10% H_2O N=9	1.17	.922
10% NaCL N=9	1.37	1.04
.9% H_2O N=8	.655	1.03
.9% NaCL N=7	.823	1.20

TABLE 8

Significance Table For 12 Hour Delay

Treatments	F	Sig. Level
Injection	11.59	.05
Fluid Offered	5.29	.05
Time After Injection	21.4	.05
Injection x Fluid	0*	N.S.
Injection x Time	24.5	.05
Fluid x Time	17.8	.05
Injection x Fluid x Time	0*	N.S.

*Due to a slightly negative SS, F was rounded to 0.

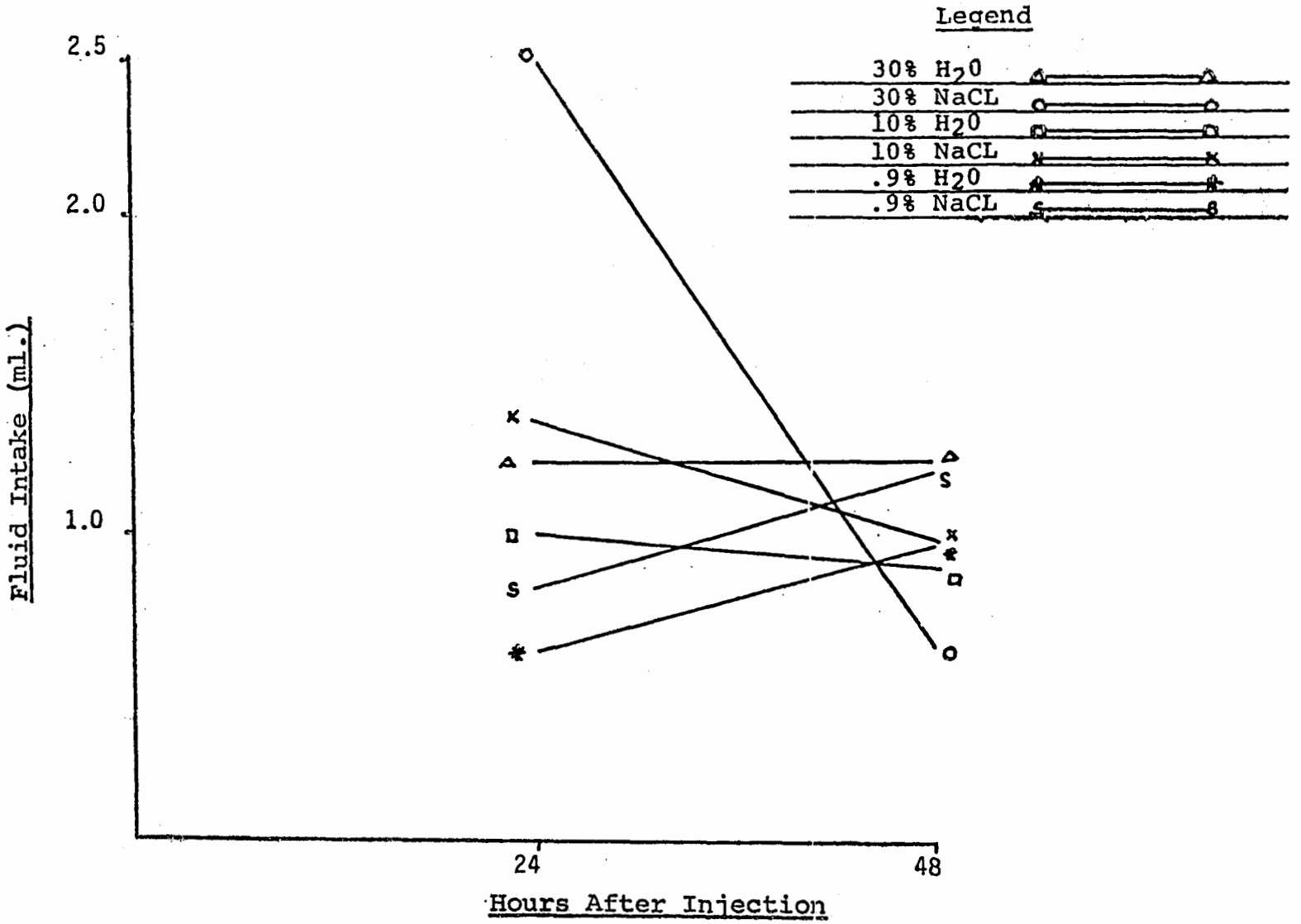

Figure 3.

Mean Intakes (% b.w./Prior 24 Hr. Intake)
at 24 & 48 Hrs. After Injections

that this places a sufficient burden on the intake mechanism, so that an additional mechanism such as mobilization of available body fluid comes into play to reduce the role demanded of water intake. In spite of this general suppression of intake, the treatment effects pattern for the 12 hour delay is similar to the pattern of the previous experiments. Treatment effects and significance levels are presented in Table 8. Again, injection levels and fluid offered effects are significant, as well as effects associated with the passage of time. However, the injection and fluid effects change over time as all groups are closely distributed within normal limits at the end of 48 hours.

The 24 hour delay trial produced some additional surprises. Mean intakes are presented in Table 9. As one can see, intake for all groups is substantially elevated compared to the previous trials. This would suggest that if the gerbil was mobilizing available body fluids to reduce its fluid deficit in the 12 hour delay trial, this practice must have a limited range of efficiency as it appears to have little dampening effect on animals undergoing 24 hour deprivation and polyethelene glycol induced hypovolaemia. The treatment effects pattern is also different for the 24 hour delay trial. Treatment effects and significance levels are presented in Table 10. The animals are still

TABLE 9

Mean Intakes Expressed as % b.w.
Prior 24 hour Intake for 24 hour Delay

Injection & Fluid	Hours After Injection: 48 hrs.
30% H_2O N=6	2.52
30% NaCL N=8	2.87
10% H_2O N=9	1.44
10% NaCL N=9	1.32
.9% H_2O N=8	1.09
.9% NaCL N=7	1.36

TABLE 10

Significance Table For 24 Hour Delay

Treatments	F	Sig. Level
Injections	42.91	.05
Fluid Offered	2.45	N.S.
Injections x Fluid	1.0	N. S.

attending to the polyethelene glycol induced hypovolaemia, but there are no longer significant differences associated with type of fluid offered. There is no evidence that the animals are undergoing voluntary dehydration in this trial. However, this is not too surprising. Although water deprivation causes a reduction in body fluid volume, it also produces increased fluid tonicity. This increased tonicity allows the animals to take in water without seriously taxing the osmoregulatory processes.

In order to more fully evaluate the effect of deprivation in conjunction with polyethelene glycol, two additional analyses of variance were performed. First, the six hour delay trial was compared with the 12 hour delay at 24 hours after injection. Also, the six hour delay, 12 hour delay and 24 hour delay were compared 48 hours after injection. Results of this analysis appears in Table 11 and 12 respectively. From Table 11, one can see that although there was a slight suppression of intake associated with the 12 hour delay trial, this trial did not differ significantly from the 6 hour delay trial. As expected, there were significant differences associated with injection and fluid offered effects. However, these effects primarily occurred within the 30% PG groups. The interaction is illustrated in Figure 4.

TABLE 11

Significance Table for 6 Hour vs. 12 Hour Delay at 24 Hours

Treatments	F	Sig. Level
Injections	177	.05
Fluid Offered	98.14	.05
Delay Trial	1.33	N.S.
Injection x Fluid	8.86	.05
Injections x Delay	1.27	N.S.
Fluid x Delay	0*	N.S.
Injection x Fluid x Delay	2.33	N.S.

TABLE 12

Significance Table For 6 vs. 12 vs. 24 hour Delay at 48 hours

Treatments	F	Sig. Level
Injections	14.2	.05
Fluid Offered	4.5	.05
Delay Trial	40.32	.05
Injection x Fluid	.77	N.S.
Injection x Delay	11.73	.05
Fluid x Delay	3.14	.05
Injection x Fluid x Delay	10.32	.05

*Due to a slightly negative SS, F was rounded to 0.

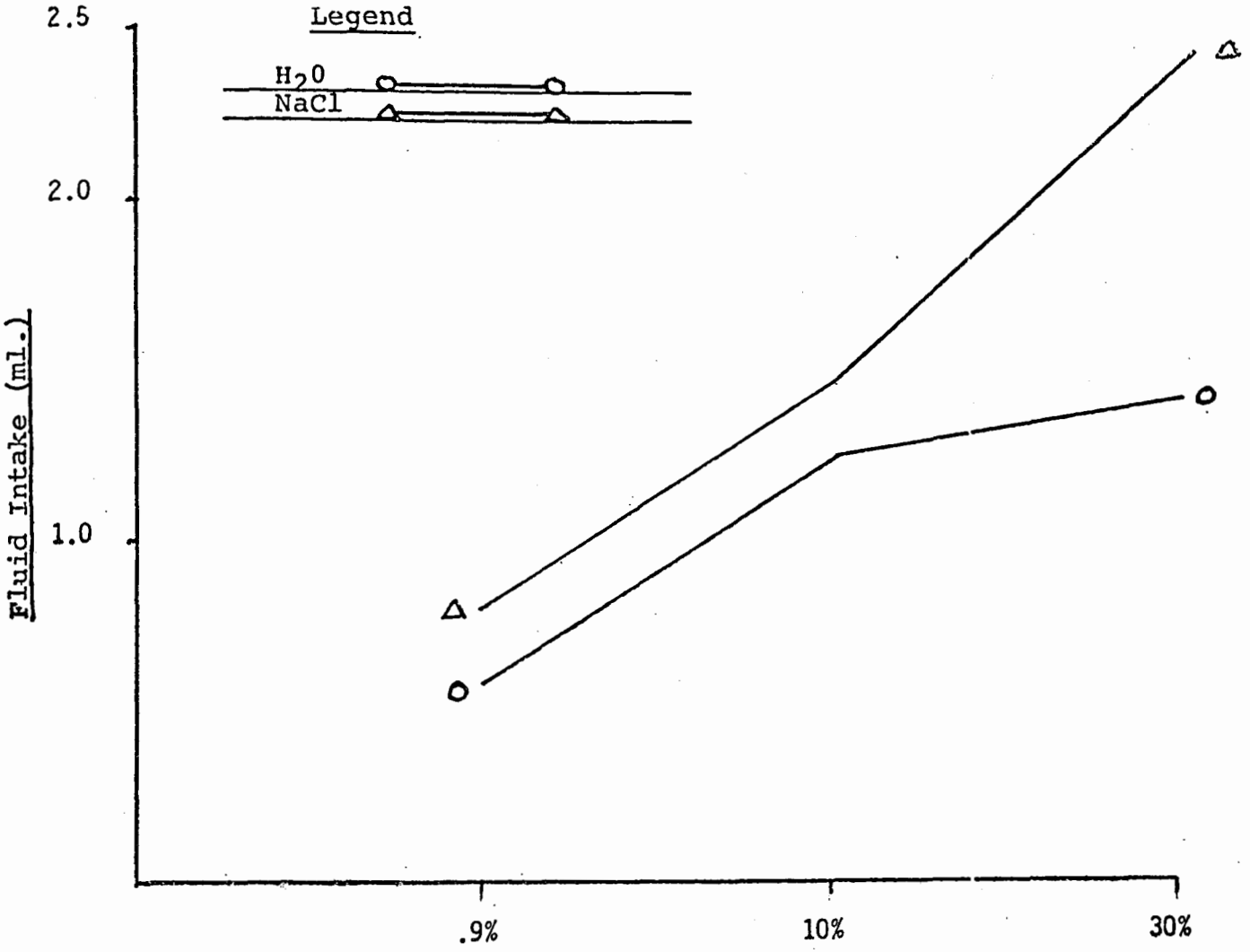

Figure 4

Injection x Fluid Interaction for
6 Hr. vs 12 Hr. Delay Analysis

A more complex picture arises when the 6 hour, 12 hour, and 24 hour delay trials are compared at 48 hours after injection. As usual, there are significant differences associated with injections and fluid offered. There were also significant differences in delay trials. Due to the fact that at 48 hours after injection, the 24 hour delay animals had only been drinking for 24 hours and were maximizing intake to correct their fluid volume while the six and 12 hour delay animals had corrected their deficit and intake had returned to normal levels, various interactions occurred. The injection x delay interaction resulted from the disproportionate increase of the 30% PG groups in the 24 hour delay trial, compared to the intake of these groups in the six and 12 hour delay trials. This interaction is illustrated in Figure 5. The significant fluid x delay interaction is consistent with the assumed presence of an additional regulatory measure, such as mobilization of available body fluids, operating in the 12 hour delay trial. Access to saline results in higher intakes than access to water in the six hour delay trial where the additional regulatory measure is not needed and in the 24 hour delay trial which evidently exceeds the range of efficiency for the regulatory mechanism. However, in the 12 hour delay trial, the additional regulatory measure shoulders the brunt of the compensatory burden, consequently resulting in

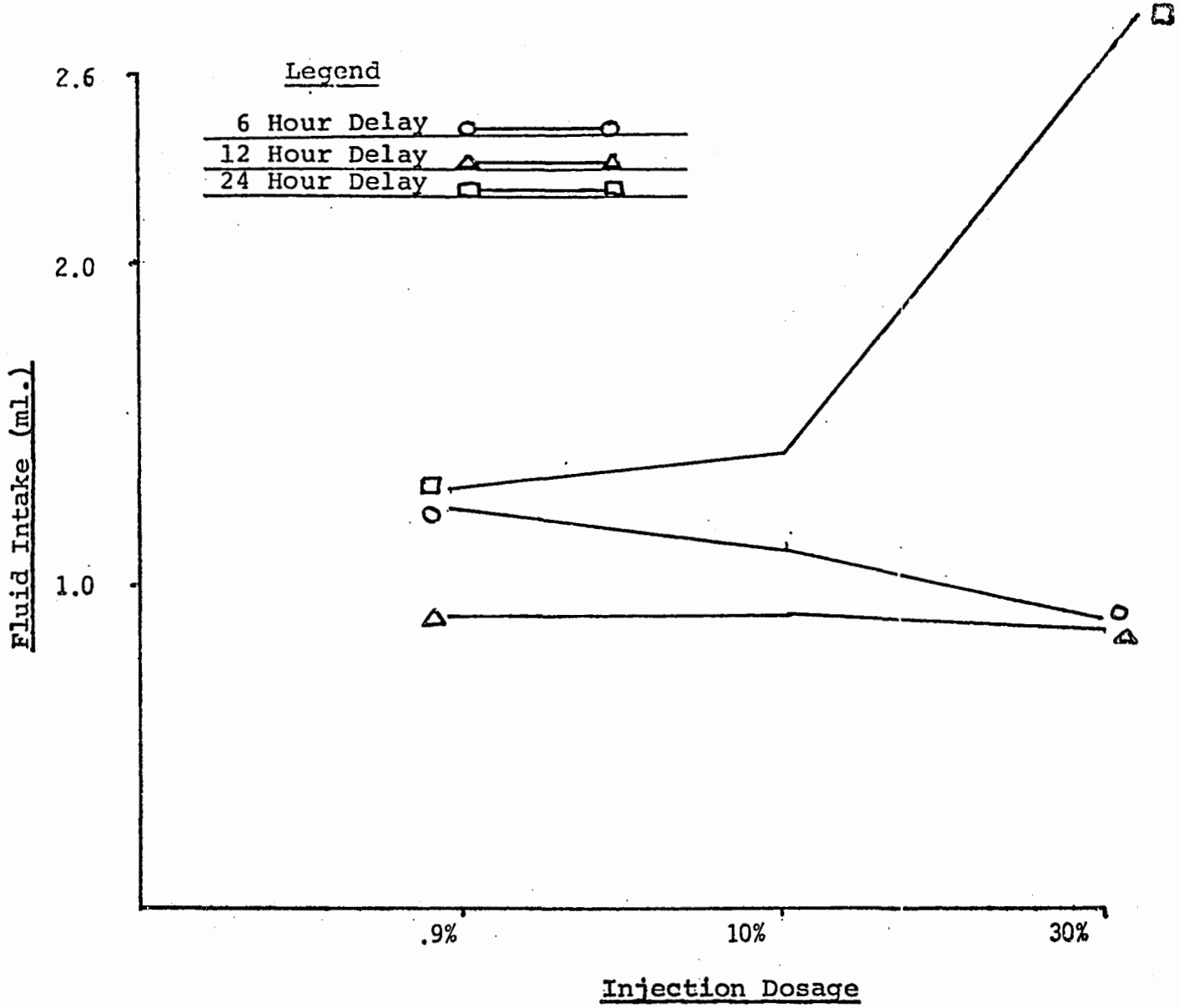

Figure 5

Injection x Delay Interaction
For 6 Hr. vs 12 Hr. vs 24 Hr. Delay Analysis

normal intakes for both access to saline and access to water conditions. The interaction is illustrated in Figure 6.

General Discussion

The net result of these experiments appears to be that the gerbil responds to an extracellular challenge much like the rat does. This finding is consistent with the conclusions of Almli and Weiss (1975). As was hypothesized, the experiments clearly show that water intake significantly increases following subcutaneous administration of polyethelene glycol. However, behavioral indices in conjunction with hematocrit values strongly suggest that 10% PG injections are only mild stressors while more puissant effects are associated with the 30% PG injections. It was also confirmed that the gerbil defends concentration and undergoes voluntary dehydration. In every experiment but the 24 hour delay trial, there were significant differences in fluid intake under access to saline conditions compared to access to water conditions. This single irregularity is explained by increased tonicity associated with 24 hour water deprivation. Under an increased tonicity state, animals with access to water can drink without excessively diluting their body fluid concentration. Hematocrit values verify that animals with access to saline can more efficiently

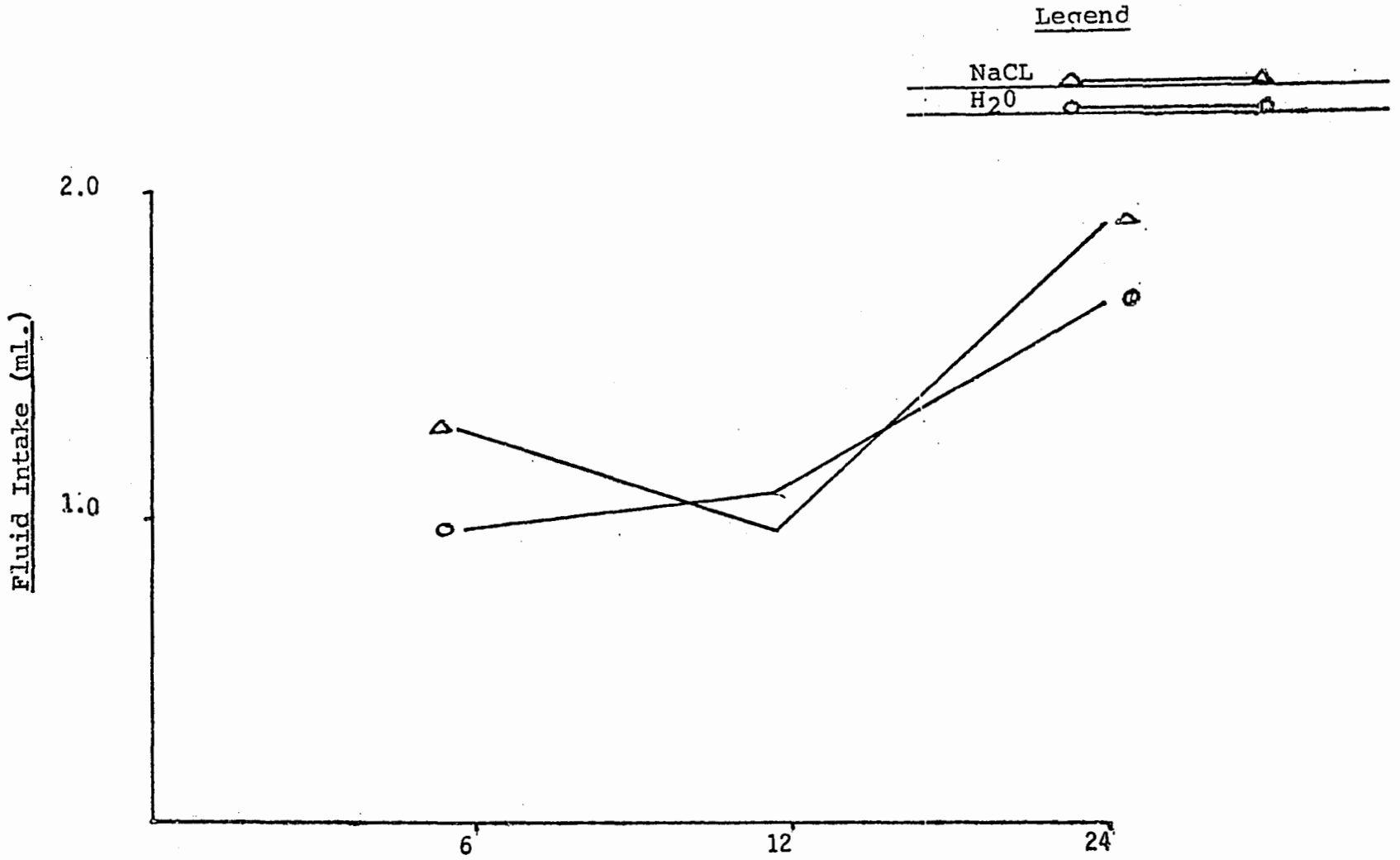

Figure 6

Fluid x Delay Interaction
For 6 Hr. vs 12 Hr. vs 24 Hr. Delay Analysis

compensate for an induced volume deficit than can animals with access to water, under equal initial states of hypovolaemia.

Intake data taken at different times following injection shows that fluid intake follows a developmental course that reaches a maximum peak around 24 hours after injection. Somewhat steeper intake slopes are obtained when water deprivation is added to the hyperoncotic colloids effect. However, with 12 hours of water deprivation, the opposite trend occurs, water intake slopes are slightly suppressed compared to intakes of the six hour delay trial. It is suggested that the animals are mobilizing available body fluids in addition to increasing fluid intake to meet the augmented volume deficit associated with the 12 hour delay trial. The efficiency range of this regulatory measure is evidently exceeded by the combined extracellular insult of the 24 hour delay trial. This explanation accounts for the lack of continuity from the 6 hour delay to the 12 and 24 hour delay trials. The significant fluid x delay interaction shown in Figure 6 is also consistent with this interpretation.

Although it seems clear that the gerbil responds to an extracellular challenge in a similar manner as the rat, further research is needed to clarify any distinctions that might exist. Kozub (1971, 1972) has

shown that intake mechanisms in the rat are affected by the age and sex of the animals. The possibility that the gerbil parallels these intraspecies differences needs to be explored. Also, since extravascular injections of hyperoncotic colloids are known to have some diuretic effect (Stricker, 1969), it would behoove us to measure urine concentration and output in later experiments.

Protein and osmolarity levels also yield useful information. Finally, further research should be conducted to see how the gerbil responds to cellular insults and combined cellular/extracellular insults.

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Notes

1. Due to the gerbil's constant gnawing on the wire cages, the calibrated water bottles were shaken and consequently lost fluid. This phenomenon was not identified immediately, and data from 32 animals were of necessity discarded. The severity of this loss was minimized in subsequent experiments by placing a rubber shock absorber between the cage and water bottle.
2. Water bottle failures were reduced in this experiment. However, the repeated measures design of this experiment increased the liklihood of data loss and data from 13 of the initial 60 animals were discarded.

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