A METHOD FOR REAL TIME EVALUATION OF PHYSIOLOGICAL STRESS DUE TO OZONE EXPOSURE

by

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Finally, this work is dedicated to the memory of my father, , whose influence on my life is continually felt and appreciated.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. INTRODUCTION

In the early 1950's, work by Dr. A. J. Haagen-Smit suggested a link between automobile exhausts and the smog typical of the Los Angeles region. Man's activities were thus linked to this form of photochemical air pollution, of which ozone is a prime constituent.

Since then, the results of research into the adverse effects on public health and welfare induced by ozone have led to the implimentation of automobile emission standards in an effort to reduce tropospheric levels of ozone.

In order to insure and verify the effectiveness of these emission standards, experimentation to determine and analyze detrimental effects of ozone has continued. Towards that end, this thesis was conceived.

Much of the work performed with laboratory animals in this area has dealt with effects resulting from exposures at levels of concentration greater than 1 part per million by volume (ppm), a concentration which is rarely exceeded in the lower atmosphere. Also, experiments have often been structured so that examination of the test subject was made after exposure as opposed to continuous monitoring during exposure.

The purpose of this work was to develop a method for real time evaluation of physiological stress due to near ambient concentrations of ozone. The parameter investigated

was skin temperature. For the purposes of this study, a rabbit was chosen because of the large exposed skin area in its ears.

Specific objectives included: development of non-destructive temperature measurement techniques, i.e. methods which would not influence rabbit metabolism; the design and construction of a test chamber that would allow skin temperature observations to be made during exposure periods; and the use of low levels of ozone (near 1.0 ppm) in order to more accurately relate results to ambient conditions.

To fulfill the first objective, a real-time thermographic camera was incorporated. This device translates radiation in the middle infrared wavelength band emitted by an object into equivalent electronic video signals. Projection of these signals onto a color monitor provides a series of color contours, each color representing a 1^{o} C increment. Thus, temperature patterns may be obtained without coming into contact with the object under investigation.

The experimental phase of this study consisted of three test exposures, each of approximately 45 minutes in duration, made on a single rabbit. Prior to administering ozone, a 10 minute period of acclimatization in the exposure chamber was allowed, with the rabbit in a restraining cage sealed within the test chamber. For approximately 5 minutes preceding the introduction of ozone, a series of photographs

of the thermal contours in the rabbit's left ear was taken in order to establish a background or pre-ozone stress temperature pattern. Ozone was then introduced and allowed to rise to a concentration nearing 1.0 ppm. The total time of ozone introduction during each test was approximately 20 minutes, followed by a tapering off period of about 10 minutes in which ozone was flushed from the chamber. Photographs of the color monitor were taken every 2 to 3 minutes during the entire period to provide a permanent record of the thermal contours in the rabbit's ear before and after exposure.

The results of each test, a further elaboration of methods and materials, and a discussion of related literature are included in this text.

II. LITERATURE SURVEY

INTRODUCTION

This chapter is divided into three basic sections. The first deals with ozone toxicology and presents material on the biochemical mechanism of ozone toxicity in animals, the tolerance inducing characteristics of ozone, and adverse effects noted in various test animals due to a wide range of administered dosages. Part two discusses ozone uptake in the respiratory tract. Relevant physical and chemical properties of ozone and mucus are mentioned, as well as other parameters which pertain to uptake models. The final section concerns the use of thermography in the field of medicine. Typical applications include early diagnosis of breast cancer and peripheral vascular disease.

OZONE TOXICOLOGY

Biochemistry

The exact biochemical mechanism by which ozone exerts its toxicity is not known. Coffin and Stokinger (28) list the following postulated mechanisms: (1) more or less nonspecific stress, with the release of histamine, (reported by Dixon and Mountain to occur immediately after exposure and persist for as long as 11 days in mice and 17 days in rats) (29); (2) oxidation of sulfur bearing compounds or their precursors (indicated by Fairchild et al., who showed that the addition of sulfhydryl compounds was protective) (30);

(3) oxidation of polyunsaturated lipid mainly contained in cell membranes (based on the reactivity of ozone with ethylene groups of unsaturated fatty acids); (4) formation of toxic compounds (such as ozonides and peroxides) through reaction with polyunsaturated lipid; (5) formation of free radicals (which plays a vital role in ozone's mutagenic characteristics), and (6) injury induced by a neurohumoral mechanism.

Tolerance

In considering the toxicology of ozone, it is important to note its tolerance inducing nature. That is, the adverse effects noted from the second of two identical dosages are less severe than those resulting from the first. Tolerance was found to be initiated at 0.3 ppm and reached a maximum protection at 3-4 ppm for the initial dose by Stokinger & Scheel (31). Within 30 minutes signs of tolerance were noted which reached a maximum approximately 24 hours later (32, 33). These studies have shown a persistence of 14 weeks or longer (34).

Possible tolerance development modes as reported by Stokinger & Coffin include (28): (1) protective chemical mobilization by the sensitizing dose; this would possibly include histamine antagonists or the introduction of a specific enzyme; (2) physical thickening of the alveolar walls as the result of edema; and (3) development of

cellular resistance. None of these mechanisms, however, adequately explain tolerance phenomena.

The development of a tolerance to ozone makes the characterization of a dose-response relationship more difficult.

Adverse Effects

In the early stages of this project, a literature search was conducted to determine the scope of reported adverse health effects in animals such as mice, dogs, rabbits and humans due to ozone exposure. Table 1 is a compilation of these effects covering a dosage range of from 0.08 ppm to 51.7 ppm concentration, and an exposure time of from 30 minutes to 18 months. Entries in Table 1 are arranged in order of increasing ozone concentration.

In addition to the effects listed, evidence has been gathered which depicts ozone as a possible mutagen. Chromosomal abnormalities were reported by Fetner in the root tips of Vicia fava (22) and an increase in the mutation rate of E. coli after exposure to ozone of only 0.05 ppm for 5 minutes was noted by Hamelin and Chung (23, 24). Other studies which indicate ozone's mutagenic role include those done by Zelac et al. (25, 26), in which Chinese hamsters exposed to 0.2 ppm ozone for 5 hours showed an increase in the number of chromosomal breaks in their circulating lymphocytes. This investigation, by comparing

similar effects induced by X irradiation, concluded that ozone exposure was more likely than X irradiation to produce chromosomal breaks at the respective concentrations and intensities commonly found in industrial environments. Human circulating lymphocytes were exposed to ozone at 0.5 ppm for 6 - 10 hours by Mertz et al. (27). They found that the number of minor chromosomal abnormalities significantly increased.

These studies are but a few that have been conducted to link ozone and mutagenisis. Still, the evidence is not conclusive.

Table 1

REPORTED ADVERSE HEALTH EFFECTS FROM OZONE

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
0.08	3 h	Increased mortality from pulmonary infection with Streptococcus	Mouse	35
0.1	7 h/day, 5 days/week, 3 weeks	Increased incidence of neonatal mortality in litters of exposed parents (related effects and reduced fertility noted in synthetic oxidant smog)	Mouse	36, 37, 38, 39, 40
	7 days	Increased succinate-dependent lung mitochondrial oxygen consumption in rats on diet relatively low in vitamin E; no significant effect in vitamin E-replete rats at this concentration of ozone	Rat	41
0.13-1.5	90-day continuous	Severe toxic signs with deaths in all species except dog at concentrations>0.3 ppm	Rats, dogs, mice, monkeys, guinea, pigs	42

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
0.20	30 minutes	Increased sphering of red blood cells when irradiated	Rabbits, rats, mice	43
0.2	3 h	Degenerative changes in Type I alveolar cells; later replaced by Type II cells	Rat	44
	Continuous for 28-32 days	16% increase in lung volumes; over-distention at high lung volumes, suggesting some change in elasticity; no change in respiratory frequency, tail length, or external appearance	Young rat	45
	6 hr. or continuous for 7 days	Decreased voluntary runaway activity during exposure; no-effect concentration not reported; reduced gross motor activity	Mouse	46
0.20	5 hrs/day/ 3 weeks	Structural changes in heart myocardial fibers	Mice	47
	7 h/day, 5 days/week, 3 weeks	Increased incidence of blepharophimosis and jaw abnormalities in neonates	Mouse	48

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
	7 days	Increased succinate-dependent lung mitochondrial oxygen consumption	Rat	49
	Continuous for 8 days	Dose-related increased activity of lung glutathione peroxidase and glutathione reductase	Rat	50
	5 h	Lymphocyte chromosomal breaks	Hamster	51, 52
0.2 - 0.25	0.5 - 2 h	Increased red-cell spherocytosis after in vitro radiation	Mouse, rat, rabbit, man	53
0.25	4 - 6 h	Morphologic changes in medium-sized airways	Cat	54
0.26, 0.5, 1.0	4 - 6 h	Changes in dynamic compliance resistance at 1.0 and 0.5 ppm	Cats	55
0.25 - 0.5	6 h	Threshold for lung edema formation with Ialbumin test	Rat	56

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
	3 h	Decreased lysozyme, acid phosphatase, and B-glucuronidase activity in alveolar macrophages (appears to be linearly related to doses up to 1 ppm)	Rabbit	57, 58
0.26 - 0.5	4 - 6 h	Increased lung flow resistance in 2 animals at 0.26 ppm; effect in all at 0.5 ppm	Cat	59
0.3 - 4.4	4 h	Tolerance to acute lung edema begins at 0.3 ppm	Mice	60
0.34	2 h	30% increase in frequency of breathing; 20% decrease in tidal volume	Guinea pig	61
0.37 - 0.5	2 h	Decreased red-cell acetylcholinesterase and increased osmotic fragility (no effect at 0.25 ppm)	Man	62
0.4	6 h/day, 5 days/week/ 10 months	Pulmonary arterial wall thickening	Rabbits	63

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
0.4 - 0.7	4 h	Conjugated diene bonds, suggesting lung lipid peroxidation	Mouse	64
0.5	2 - 6 h	Swelling and sloughing of alveolar type I cells	Rats	65
0.5	6 - 10 h	Minor chromosomal abnormalities	Man	66
	3 h	Inhibition of intracellular hydrolytic enzymes of alveolar macrophages; increased fraction of polymorphonuclear leukocytes	Rabbit	67
	165 min	Alterations in blood, including red-cell membrane and enzyme changes and increased serum vitamin E and lipid peroxides	Man	68
	6 h	Decreased lung DNA synthesis	Mouse	69
0.5	6 - 10 h	Damage to type I with proliferation type II cells	Rats	70
0.5	8 h/day/7 days	Changes in lung macrophage osmotic fragility	Rabbits	71

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
0.5 - 1	1 h	Decreased electric response of specific areas of brain with evoked-response technique	Rat	72
0.54 - 0.88	Continuous for up to 3 weeks	Morphologic changes in distal and respiratory bronchioles, alveolar ducts, and associated alveoli	Young rat	73
0.6	Not applic.	Avoidance of cage ventilated with ozone	Mouse	74
	93 days	No change in behavior or chronaxial muscle ratios	Rat	75
0.68	4 h	Decreased rate of bacterial killing in lungs in vivo	Mouse	76
	2 h	No significant increase in respiratory flow resistance	Guinea pig	77
0.7 - 0.8	Continuous for 7 days	Increased acid phosphatase in specific lung areas determined histochemically	Rat	78

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
	Continuous for 5-7 days	Increased activity of lysosomal hydrolases in whole-lung homogenates	Rat	79
0.75	3 h	Decreased benzopyrene hydroxylase in lung and tracheobronchial mucosa	Hamster	80
	4 - 8 h	Histologic changes in parathyroid glands	Rat	81
0.8	Continuous for 7 days	Histochemically determined alteration in several lung enzyme activities	Rat	82
	Continuous for 8 days	Increased activity of lung and plasma lysozyme (no effect at 0.2 and 0.5 ppm)	Rat	83
	7 days	Increased activity of lung pentose shunt and glycolytic enzymes; decreased lactic dehydrogenase	Rat	84

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
0.84	4 h/day, 5 days/week, 2 weeks	Increased susceptibility to respiratory infection with Klebsiella pneumoniae	Mouse, Hamster	85
0.85	4 h	Heinz bodies in circulating red cells; further exposure led to decrease in Heinz body formation	Mouse	86, 87
0.9	Continuous	Lungs 38% heavier than those of normals; 50% dead in 3 weeks	Young rat	88
1	1 h	Chemical changes in ground substance and lung protein	Rabbit	89
	4 h	Engorged blood vessels and excess leukocytes in lung capillaries	Mouse	90
	90 min	Decreased lung cytochrome P-450	Rabbit	91
	1 h	Formation of carbonyl compounds and alterations of hyaluronic acid in lung	Rabbit	92

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
1.0 - 7.0	3 h	Decrease in numbers of pulmonary alveolar mascrophages	Rabbits	93
	8 - 24h/day for 18 months	Alteration in catechol-O-methyltransferase and monoamine oxidase of brain tissue	Dog	94
	3 h/day for 2 - 3 successive days	Prolongation of phenobarbital sleeping time (no effect after 1 or 4 - 7 days)	Mouse	95
	Continuous for up to 18 months	Bronchitis; bronchiolitis; emphysematous and fibrotic changs; acceleration of lung-tumor development	Mouse	96
	Continuous for 1 week	Decreased voluntary running activity	Rat	97
1.0	6 h	60% increase in mortality as a result of exercise for 15 minutes/hour	Rats	98
1.0	8.2 - 18.5 days continuous	Acceleration of vitamin E deficiency	Rats	99

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
1.3	Continuous for 18 months	Thickening of terminal and respiratory bronchioles; barely noticeable at 1 ppm; at 3 ppm, formation of peribronchiolar collars with resulting narrowing of small airways	Dog	100
1.08	2 h	47% increase in respiratory flow resistance	Guinea pig	101
1.3	3 h	Increased susceptibility to Klebsiella pneumoniae	Mouse, Hamster	102
2.0	3 h	Increased lung weight, decreased tidal volume, decreased minute ventilation	Rats	103
2.3	8 h/day, 5 days/week, 6 weeks	Mildly toxic signs and no deaths	Monkeys, dogs, mice, rats, guinea pigs	42
3.0	8 h daily, 18 months	Squamous metaplasia of bronchiolar epithelium	Dogs	104

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
3.0	2 h	Change in neutrophil-lymphocyte ratio	Rats	105
3.1	20 h	Increased liver weight; increased liver alkaline phosphatase	Rats	106
3.2	4 h	Gross pulmonary edema	Mice	103
4.0	4 h	Decreased mortality with age; young 50% mortality, old 10% mortality	Mice	107
4.5	2 h every 3rd day, 75 days	Severe epithelial changes in lung in tumor resistant strain	Mice	108
5.0	2 h	Increased lung compliance, increased susceptibility to histamine	Guinea pigs	109
5.0	3 h	Decreased activity of bacteriocidal lysozyme	Mice, rabbits	110
5.0 - 6.0	90 minutes	Production of hydrogen peroxide in erythrocytes	Rats, mice	111

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
6.0	4 h	Gross pulmonary edema, increased lung serotonin	Rats	112
6.0	4 h	Decreased brain serotonin	Rats	113
8.0	4 h	Decrease in erythrocyte acetylcholinesterase activity	Mice	114
8.0 (Avg)	3 - 5 h	Protection versus lethal effects by various enzyme-inducing agents	Rats	115
8.0 - 45.0	1 h/week up to 49 weeks	Damage to epithelium of the lower tracea and bronchioles; fibrosis	Rabbits	103
10.4	4 h	Vitamin E deficiency increases lethality of ozone	Rats	116
10.0 - 30.0	8 h/day/18 mos.	Decrease in brain catecholamine metabolism	Dogs	117
15.0	30 minutes	Decreased tidal volume, decreased oxygen consumption	Rabbits	103
15.0	426 minutes	Protection from acute lethal effects by paminobenzoic acid	Rats	118

Table 1 (continued)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
21.0	3 h	50% mortality	Mice	119
21.8	3 h	50% mortality	Rats	119
34.5	3 h	50% mortality	Cats	119
36.0	3 h	50% mortality	Rabbits	119
51.7	3 h	50% mortality	Guinea Pigs	119

OZONE UPTAKE IN THE RESPIRATORY TRACT

To better understand effects of ozone (O_3) at a given concentration and exposure duration, it is necessary to consider the uptake mechanism or mode of entry. Currently, there is no single model which accurately depicts the uptake of non-inert gases, such as ozone which react with body tissues and fluids (1). So many uptake variables exist that the likelihood of duplicating an exact dose response under the same circumstances is negligible. What follows is a description of the major factors controlling ozone uptake in an effort to relay the complexity of modeling efforts.

It is important to note that in addition to the factors mentioned, variables such as age and health are also of paramount importance.

Controlling Factors

Research on primates by Dungworth *et al.* (2) indicates that ozone is absorbed along the entire respiratory tract, with the most severe damage occurring in the bronchioles as concentration increases from 0.2 to 0.8 ppm. Work done on rabbits (3) indicates that the underlying cells in large airways are not completely protected from ozone by the mucus layer. Acute exposures at 0.25, 0.5, and 9 ppm lead to patches of desquamated ciliated cells often found at bifurcations. Yokoyama and Frank's (4) experiments on dogs support this by showing that 27-70% of inhaled ozone is

removed before reaching the first bifurcation. By comparison, nearly 100% of sulfur dioxide is removed in the same region. Still other influences may alter the site of action or response.

Certain particulates, for example, may absorb ozone and result in its removal from the air stream at varying locations. Some gases, such as SO_2, react synergistically with O_3 to produce detrimental responses at concentrations lower than either could produce alone.

Relevant properties of ozone and mucus

One of the reasons why an effective model of uptake does not exist is the lack of information concerning the solubility of ozone in mucus. In Air Quality Criteria for Photochemical Oxidants (5), an ozone solubility of 0.494 ml/100 m. of water at 0° C and 760 mmHg is given. Along with solubility, the diffusivity of ozone in mucus, tissue and water also should be known in order to trace ozone's biological lifetime. A National Academy of Sciences report (6), indicates that these diffusivity values are unknown. They report, however, that the diffusivity of oxygen in water, which is 2.5×10^{-5} cm^2/sec, may be used as a value for the diffusivity of ozone in water and mucus layers. Another relevant chemical property is the reaction rate of O_3 in water, mucus, and tissue. The latter two are not known, but Alder and Hill (7) and Hoigne' and Bader (8)

have determined the rate of ozone decomposition in aqueous solutions. These values may be used as an approximation (6).

Respiratory tract morphology

Several mathmatical airway models of the human respiratory tract have been developed. Of these, Weibel's model "A" (9), which has 16 generations of conducting airways in the tracheobronchial region, is widely used. Table 2 shows the numbers, diameters, lengths and time averaged velocities of the conducting airways for an adult with a long volume of 4,800 cm^3 at 75% inflation (10). This model is also characterized by seven partially or completely alveolated generations.

Modeling work by Phalen et al. (11) compared the airway morphology of the dog, rat, hamster and human. This investigation revealed a higher degree of symmetry with respect to diameter ratios and branching angles in the human tracheobronchial tree. The structure of the bronchial tree was found to vary from species to species, from lobe to lobe within a given lung, and from one lung depth to another.

The underlying cells of the mucus layer may either be a point where the toxic gases accumulate or serve as a diffusion route for the gases to the blood. Altshuler et al. (12) have developed a model for calculating tissue doses which considers this possibility. In their model,

Table 2

AIRFLOW ANALYSIS OF WEIBEL'S MODEL A

Number of Conducting Airways	Diameter (cm)	Length (cm)	Time-averaged Velocity (cm/s)
1	1.800	12.00	88.60
2	1.220	4.76	96.60
4	0.830	1.90	105.60
8	0.560	0.76	112.50
16	0.450	1.27	90.70
32	0.350	1.07	72.30
64	0.280	0.90	56.80
128	0.230	0.76	44.10
256	0.186	0.64	32.40
512	0.154	0.54	23.50
1,024	0.130	0.46	16.80
2,048	0.109	0.39	11.50
4,096	0.095	0.33	7.81
8,192	0.082	0.27	5.06
16,384	0.074	0.23	3.24
32,768	0.066	0.20	1.99
65,536	0.060	0.17	1.25

(Taken from Ozone and Other Photochemical Oxidants) (6)

the basement membrane is covered with three discrete layers: an inner layer of variable thickness containing the basal, goblet, and ciliated cells; a middle layer 7 mm thick which contains a waterlike or serous fluid; and an outer 7 mm layer composed of viscous mucus. The National Academy of Sciences reports, however, that airways smaller than 1 mm in diameter do not show separate mucus and serous fluid layers (13).

Respiratory flow

Several factors contribute to the complexity of flow patterns in the respiratory system. One factor is the effect of the larynx. Olson et al. (14) have determined that flow patterns typical of smooth bifurcating tubes do not occur until the lobar bronchi are reached. These flow patterns are characterized by secondary motions and high shear rates along the inside wall. Turbulent eddies produced by flow separation below the larynx were found to exist as far down as the sublobar bronchi with 200 ml/s flow in the trachea. The influence of the larynx on flow patterns is an ever changing one due to the variable orifice nature of the glottis.

The corrugated walls of the trachea also contribute to turbulence. Inspiration brings a jet of air into the trachea directed against its back wall. Here, a turbulent flow pattern is enhanced and may cause the turbulence to

reach a fully developed state by the end of the trachea for a Reynolds number greater than 3000 (15). (Reynolds numbers refer to a turbulence index that considers such parameters as viscosity and flow channel diameter). Owen's (16) model indicates that turbulence will gradually decay in any branch in which the Reynolds number is less than 3000. Assuming a Reynolds number of 1865, his model predicts that turbulence will have approximately 50% of its initial intensity upon entering the third generation of bronchi.

The heartbeat is another contributor to the respiratory flow pattern. West (17) detected flow oscillations in the segmental bronchi due to the heart's beating action during periods of breath-holding or during pauses between inspiration and expiration. The observed peak oscillatory flow rate was 0.5 l/min, corresponding to 20% of the peak flow rate during quiet breathing.

Another complication with the flows typical of the respiratory tract is the mixing of residual and tidal air by convection and diffusion. Altshuler et al. (18) worked with 0.4 mm particles and a tidal volume of 500 ml and found that 11 to 27% of air in a new breath mixes with residual air. Baker et al. (19), working with Weibels symmetric model, used a time varying flow with longitudinal diffusion and concluded that convection plays a much smaller role in mixing than does molecular diffusion. There is, however, no general agreement on the relative roles of

diffusion and convection.

Nasal airflow

The nose, because of the complex flow patterns induced by its structure and because of the large surface area of the nasal mucus, is an effective humidifier and pollutant scrubber. An estimate of the amount of gaseous pollutant uptake and scrubbing efficiency can be obtained by Proctor & Swift's nasal-passage model (20). This model was developed by studying the flow of water through a clear plastic model of the nasal passage walls. In their work, the flow was steady and a Reynolds number was chosen equal to that for air in the human nose. By their model, a flow rate of 0.4 l/s corresponding to quiet inhalation, gave a maximum value of 10-12 m/s for nasal entrance velocity. This is quite large when compared to the peak velocity in the bronchial tree of 2 m/s during quiet breathing. The influences of the nasal passage should not be overlooked in tracing the fate of inhaled oxidants.

Mucus flow

The beating action of the cilia within the stationary serous layer causes the outer mucus layer to move up the respiratory tract. Thus, deposited particles and absorbed gases along the tracheobronchial tree are partially cleared by this flow, where the degree of removal is determined by the rate of mucus flow. Bronchial openings tend to retard

flow by presenting obstructions and are, therefore, areas of mucus accumulation.

Inhaled irritants

The presence of inhaled irritants may affect both air and mucus flows, thus affecting the fate of other pollutants. Acute exposures to ozone, for example, may be expected to change the flow patterns due to a constriction of airways. Also, edema may result in altering the absorptive properties of airways and affect their aerodynamical properties as well (21).

MEDICAL THERMOGRAPHY APPLICATIONS

Although the use of real time thermographic techniques has found a wide range of uses in business and industry, it is the use of thermovision in the field of diagnostic medicine that is of relevant importance to this study. A brief investigation into prior applications is beneficial to the investigator in lieu of sufficient previous experience in thermographic camera operations.

Thermovision as a tool to diagnose early breast cancer has been the subject of numerous investigations. The temperature difference often found between healthy and cancerous tissues makes thermography an ideal tool in evaluating this type cancer. Nomura et al., report the results of a 91 case study in which the skin temperature over

diseased breasts was noted by thermographic techniques (122). In 16 of 20 cancer cases, 80%, the skin temperature over cancerous regions was at least 1° C higher than corresponding noncancerous regions of the opposite breast. Only 6 of 67 benign cases, excluding inflammations, showed temperature differences of this magnitude. Thus, the 1.0° C difference appeared to separate malignant from nonmalignant cases.

Patients in the aforementioned test were examined while prone on their backs with hands placed at their necks. Fifteen to 20 minutes prior to testing, the upper halves of their bodies were exposed and allowed to equilibrate. In some cases, a wet towel was placed on the patient's chest in order to cool it as evenly as possible. Temperature in the draft free examining room ranged from 20-25° C.

The effectiveness of rheumatoid arthritis treatment has been investigated thermographically in several hospitals (123). Through thermography, the degree of swelling, activity and localization of the rheumatic inflammation was checked. In cases where small changes in activity and the development of the inflammation was difficult to follow conventionally, thermal pictures were found to be especially useful. In order to better compare thermograms taken over a wide time interval, a constant temperature reference source was placed in the field of view during each test. The resulting diagnostic efficiency lead to the recommendation

that thermography be used as a routine diagnostic method for patients suffering from rheumatoid arthritis (124, 125).

The study of peripheral vascular disease has also been done thermographically. Mishima et al. report that in cases with chronic arterial occlusion, functional changes of the cutaneous circulation were revealed thermographically (126). In studying a vasopastic condition thermography again proved beneficial. Here, digital circulation is a strong function of environmental temperature. A normal flow is typical of summer temperatures, but winter's cold induces a greatly diminished flood flow so that discoloration of digits occurs. Thermographic techniques were capable of detailing the temperature differences resulting from such vasoconstriction (126).

Other medical applications of thermography include its use in: obstetrics and gynecology for placental localization and early detection of pregnancy (127, 128); orthopedics for diagnosis and evaluation of herniated discs, fractures, sprains and contusions (129); dermatology to help determine the degree and depth of burns and frostbite injuries (130); urology for detection and evaluation of testicular lesions and renal malignancies (131); and in surgery to aid the evaluation of various acute and chronic thoracic and abdominal inflammatory processes (132, 133).

In conclusion, thermography has found many valuable roles in the field of diagnostic medicine. Its applicability to

this study is strengthened by reports of successful detection of minute skin temperature changes, particularly due to vascular anomalies.

III. EQUIPMENT

Introduction

In order to fulfill the second objective, design and construction of a test chamber that would allow skin temperature observations to be made during exposure periods, numerous factors had to be considered. Among these were: size requirements; provisions for access to the chamber's interior during experimentation without terminating exposure; incorporation of a "window" which was transparent in the middle infrared region for thermographic work; and a mechanism for adequately mixing ozone and ambient air in the chamber to prevent concentration gradients.

The following pages provide a detailed description of chamber construction as well as a description of all pertinent equipment. Figures 1 and 2 give a summary of equipment in flow diagram fashion.

Exposure Chamber

Experimentation was conducted in a specially designed exposure chamber of plywood construction, see Figure 3. Its dimensions were chosen to accomodate the size of a fully opened rabbit restrainer for future work with more than one specimen.

On the left and right sides of the main chamber body, as viewed from the front, much smaller diffusion enhancement plates are located. It was concluded in preliminary design

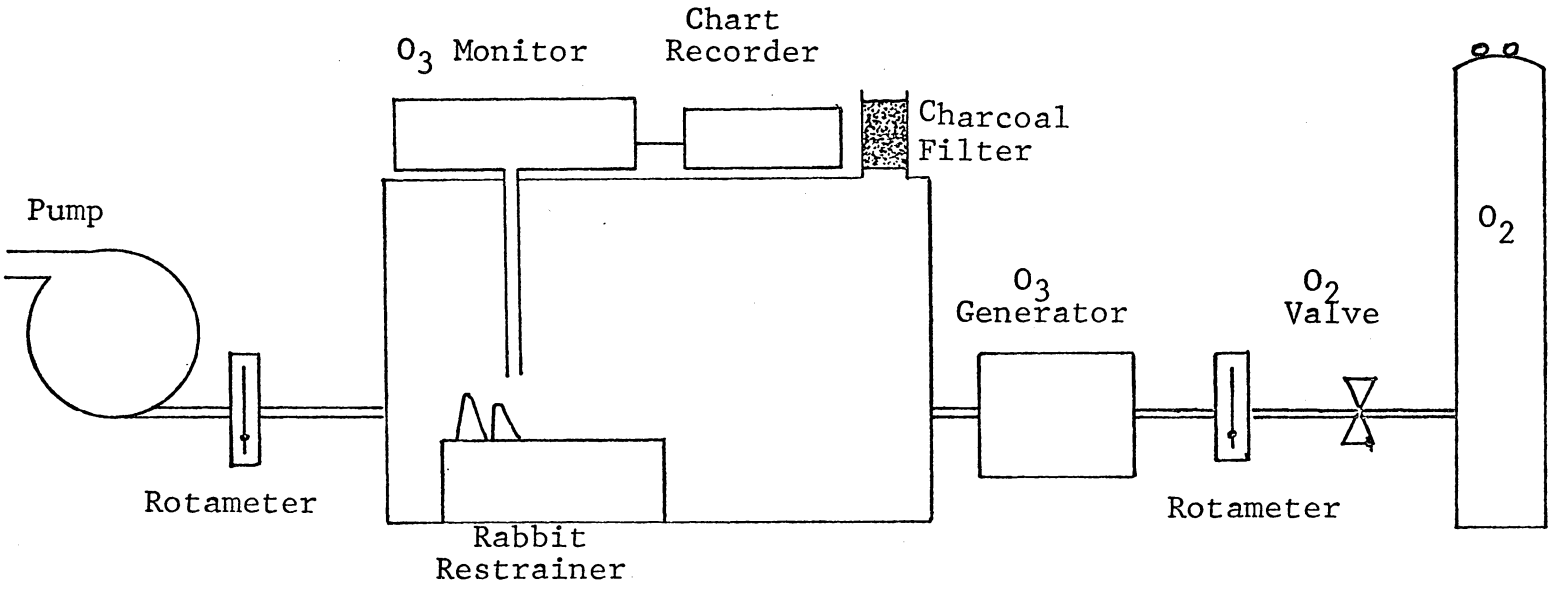


Figure 1. Schematic of Exposure Chamber Equipment

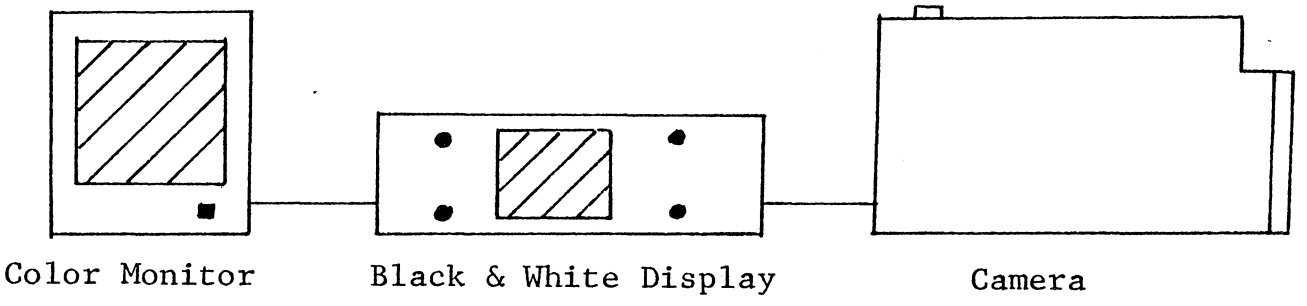


Figure 2. Schematic of Thermographic Camera System

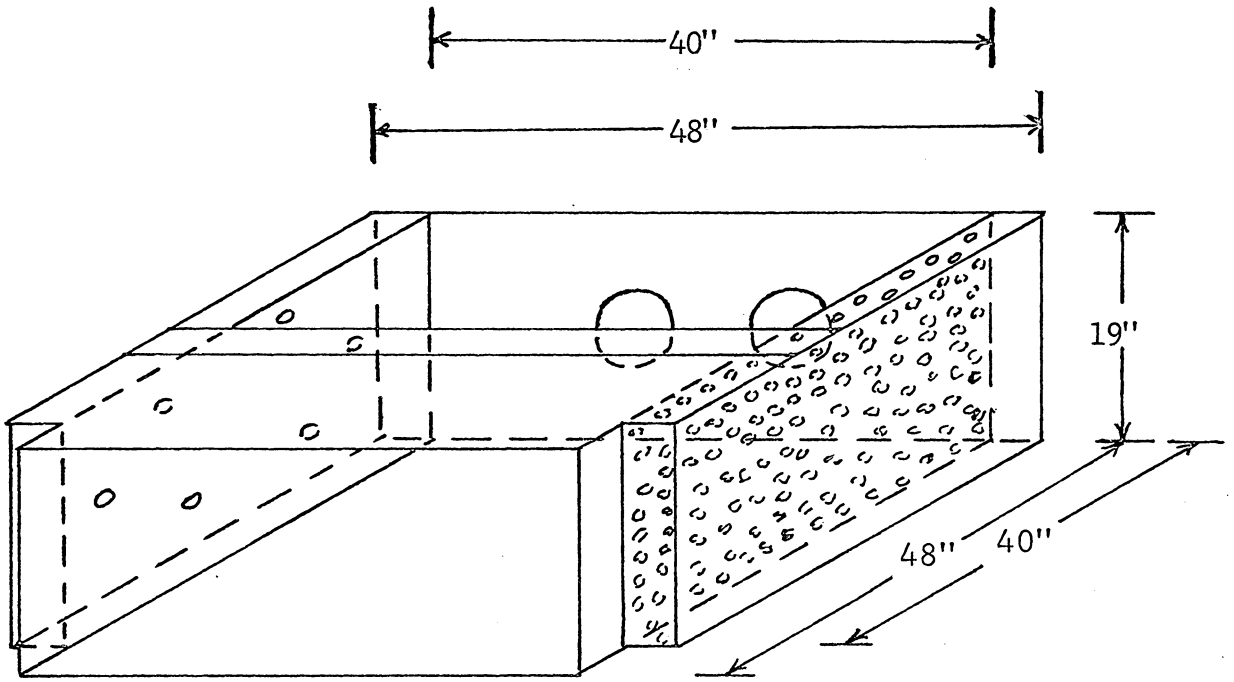


Figure 3. Experimentation Chamber

stages that the suction from a single circulation pump might create concentration gradients. One measure taken to reduce this effect was the placement of a plate the length and height of the chamber with six 1" diameter holes, 4" in front of the circulation pump intake.

A second plate with dimensions corresponding to the chamber length and height was placed on the right side of the chamber, 4" in front of the point of entering ozone, thus forming an enclosed pre-mix chamber on that side of the main chamber. Into the plate, approximately 50 uniformly distributed 1/4" holes were drilled. Ambient air, upon entering this right side chamber through one of two activated charcoal filers, was thus allowed to mix with the ozone in this chamber before entering the main chamber through the diffusion plate. These two features serve to reduce the occurrence of concentration gradients in the main chamber.

In order to have access to the rabbit during experimentation, a "glove box" feature was designed and placed along the rear wall of the chamber, see Figure 3. Each glove was custom constructed of heavy duty plastic with heat sealed seams to ensure an air tight nature. The seams were reenforced with black electrical tape as a precaution against leakage.

The open end of each glove was wrapped around a pyrex ring 6" in diameter and 2" wide, and secured with black electrical tape. Each glove-ring assembly was then mounted

into the chamber wall at a relative center point separation corresponding to the investigator's shoulder width. To allow a means of viewing inside the chamber while using the access gloves, a plexiglass window was placed along the back half of the chamber top. Structural support between the plexiglass and plywood portions of the top was provided for by a 4" wide strip of plywood, extending the entire chamber width, which was positioned so that 2" overlapped the plexiglass and 2" the plywood. The supporting strip was then screwed into the respective top components and the resulting seams, as were all chamber seams, caulked with Acrylic caulking material.

The front of the main chamber was left open to provide for installation of a window which was transparent to infrared radiation. This window was made of Reynolds Film #916 which is greater than 99% transparent in the IR range (134). A layer of this film across the main chamber's front, therefore, provided not only a means for sealing the chamber but also allowed observations of skin temperature changes to be made with the thermographic camera during exposure periods. To prevent puncture of this delicate film, the plywood edges of the main chamber's entrance were covered with black electrical tape. It was found that the smooth surface of the tape had superior adhesive properties with the plastic film. A final external strip of 1" wide masking tape served to provide an air-tight seal.

Ozone Generation

Ozone was generated by a water cooled ultraviolet lamp manufactured by the Virginia Polytechnic Institute and State University glass shop. Bottled oxygen supplied by Airco Corporation, whose purity exceeded 99.5%, was used as feed for the ultraviolet lamp. Some oxygen atoms, upon bombardment by the ultraviolet radiation are ionized. These ions, when combined with molecular diatomic oxygen, produce ozone.

The power supply for the ultraviolet lamp was a model SCT-4 ozone generator manufactured by Ultraviolet Products, Inc.

Air Flow Monitoring and Control

Air Circulation

Ambient air was drawn via the charcoal filters through the chamber by a Model 5KC48PG370T, 3/4 H.P. Gast vacuum pump. The flow rate was 28 l/min., which corresponded to approximately 0.2 air changes per hour. Figure 4 shows the volume displacement with time for the pump as determined by a Precision Instrument Wet Test Meter.

Monitoring and Control of Flow

Flow from the chamber was monitored by a rotameter manufactured by Brooks Instrument, Division of Emerson Electronic Company, Type 1355-01A1AAA, serial 7104-60093/2. This instrument was placed between the chamber and the Gast pump, see Figure 1.

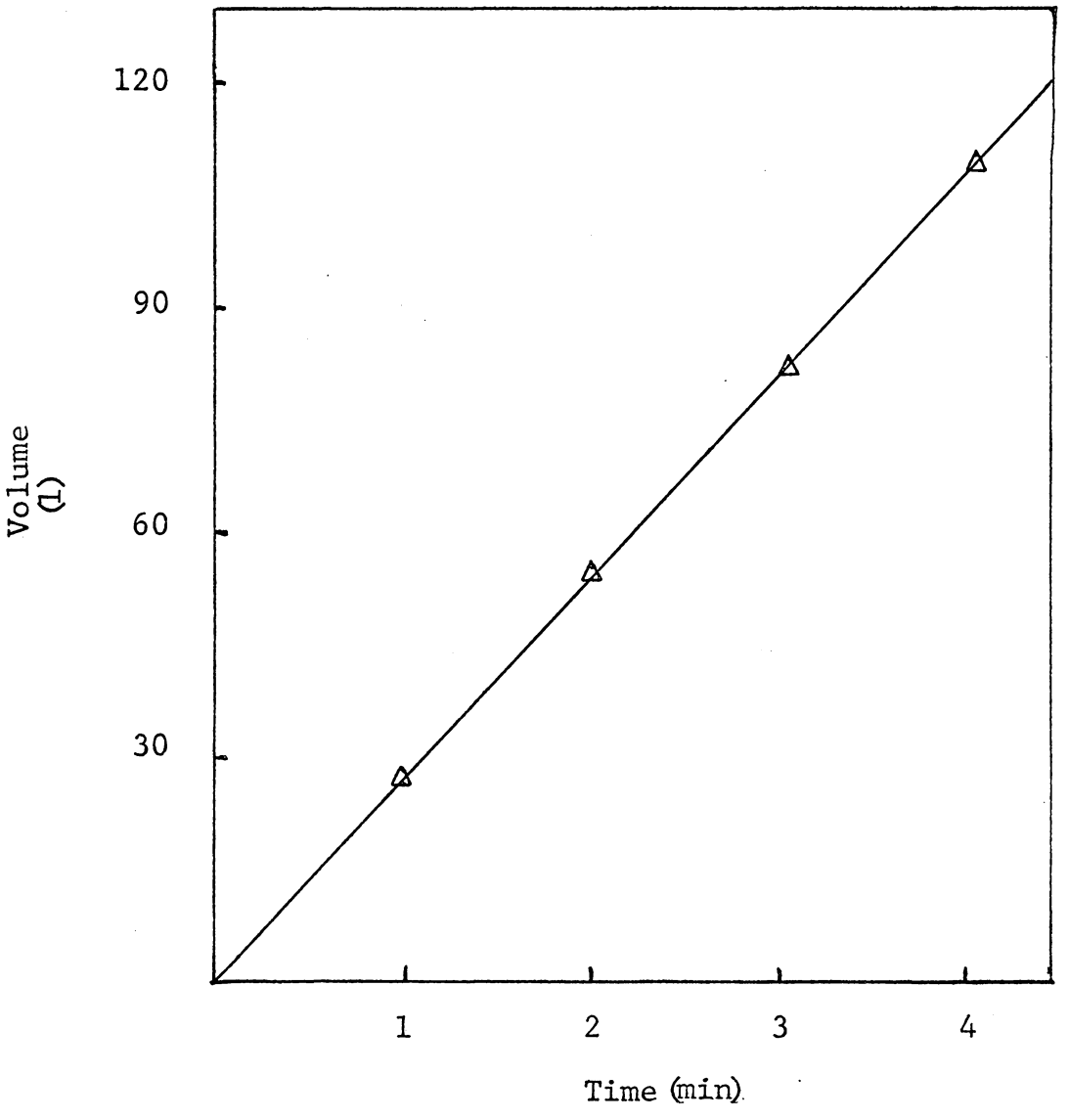


Figure 4. Circulation Pump Volume Displacement With Time

Since the rotameter was not in line with the exhaust flowing from the chamber, it was not measuring this flow. Because of the same source, i.e. the pump, was responsible for both the flow through the rotameter and the flow through the chamber, the observed rotameter flow was used as a monitor of fluctuations in chamber flow. A power surge in the pump would alter flow through the chamber and the rotameter as well. Thus, a change in rotameter flow would indicate a change in chamber flow.

Bottled oxygen flowing into the ozone generator was controlled both by an Oxweld oxygen regulator, serial C60 and by a Brooks Instrument Rotameter, Type 1365-01A1AAA, serial 7104-60093/1. Figure 5 shows the results of a calibration of the rotameter via a Precision Instrument Wet Test Meter and Little Giant vacuum pump.

Ozone Monitoring and Data Recording

During experimentation, a continuous sample of approximately 3 LPM of chamber air, taken from a location approximately 5" from the rabbit's nose, was analyzed by a Dasibi Environmental Corporation Ozone Monitor, model 1003 AAS, serial 2070. This unit is self-contained and capable of detecting ozone in the range of 0.002 to 20.000 ppm with an accuracy of $\pm$ 1% or 1 digit, whichever is greater. In measuring ozone concentration, the Dasibi takes advantage of the ultraviolet absorption characteristics of ozone. By

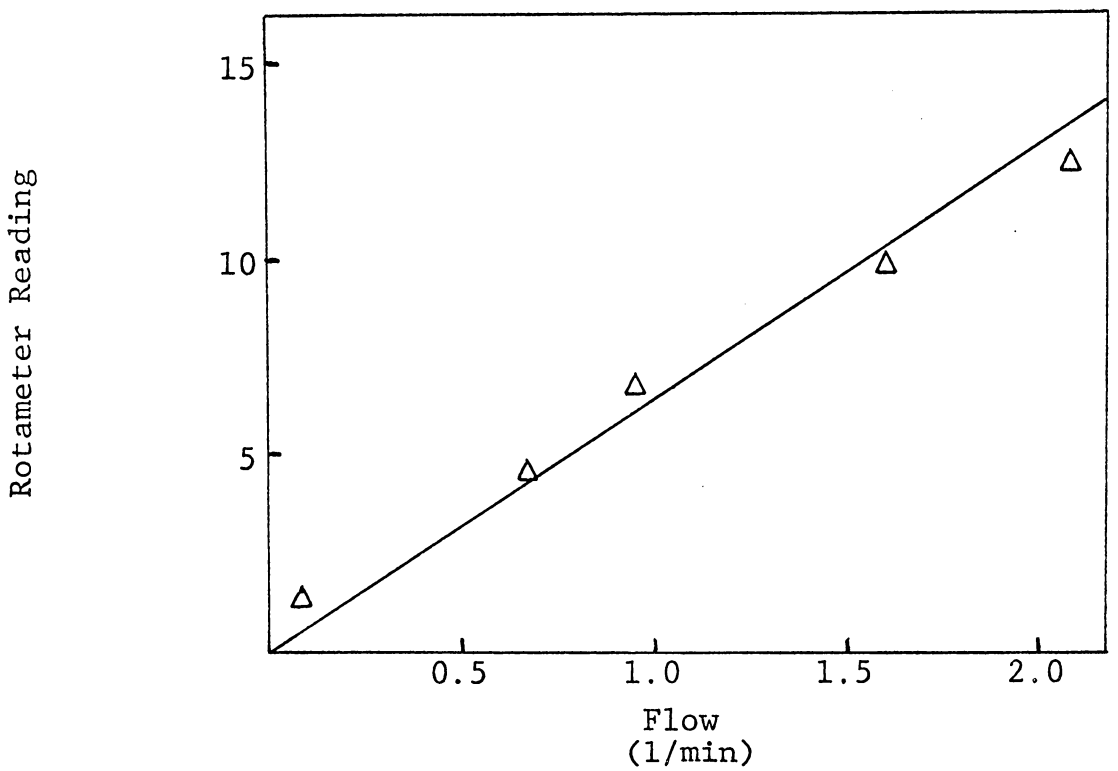


Figure 5. Calibration of the Oxygen Feed Rotameter Via Wet Test Meter

passing an air sample over a known distance through an ultraviolet light of known wavelength and intensity, the degree of attenuation will be a function of ozone concentration. Comparing the attenuation with a known standard leads to a numerical assignment of ozone concentration value.

Every 15 - 25 seconds, in the single cycle the completion of a measurement cycle resulted in an electrical output which was recorded on a strip chart recorder. The particular instrument used was Model 255, serial 13906, manufactured by Linear Instruments Corporation. The pen response of this instrument is greater than 20 in./sec. During operations, the recorder full scale input was 1 volt and the chart speed was 0.5 in./min.

Calibration of the Dasibi was done by the Virginia State Air Pollution Control Board's Monitoring Division using a gas phase titration reference. These results are given in Figure 6.

Thermographic Camera

In order to detect any significant changes of the temperature pattern in the rabbit's ear, a real time thermographic camera was used, Figure 2. The AGA Corporation System 680 camera, serial 4078, converts radiation in the middle infrared wavelength band (2-5.6 um) given off by an object into equivalent electronic video signals which are amplified and transferred to a black and white display unit,

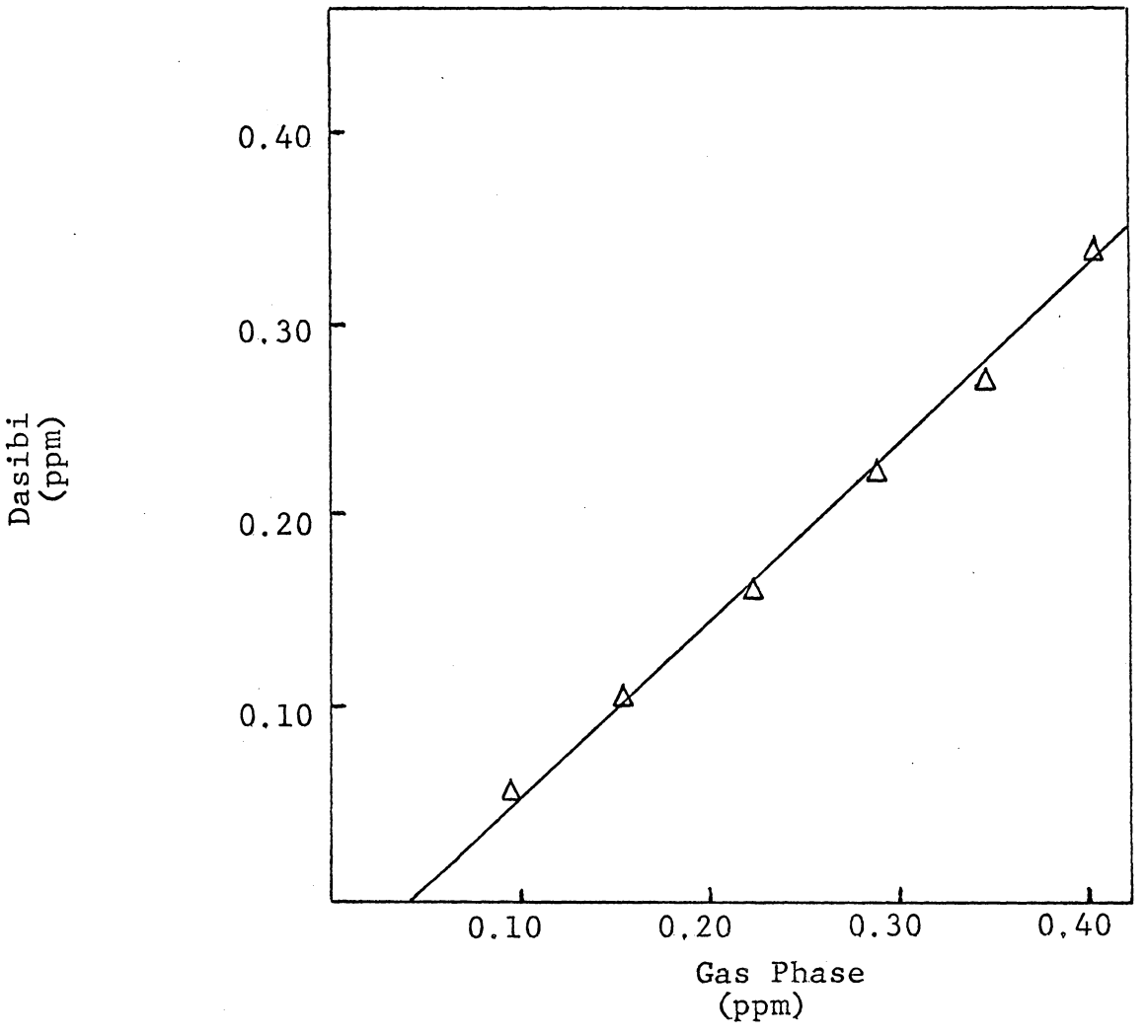


Figure 6. Dasibi Calibration Via Gas Phase Titration (135)

serial 705847. A color monitor, serial 049, then divides the signal into 10 increments and assigns a particular color to each division. The temperature range of each division is predetermined by the operator and varies from .2° C to as great as 100° C. In this experiment, a scanning range of 10° C was used, thus each color represents 1° C. A color scale, along the bottom of the color display, indicates the coolest temperature's assigned color on the far left and the warmest temperature's on the far right.

The System 680 incorporates an indium antimonide (InSb) photovoltaic cell in the detector assembly. Sensitivity of this cell is maximized when cooled to cryogenic temperatures. Thus, it has been mounted within the double walls of a Dewar vessel which is filled with liquid nitrogen (-196° C) prior to camera use. In order for the infrared beam to enter the detector housing, the outer wall of the Dewar vessel contains a sapphire window, transparent to infrared radiation.

This system, then, provides the operator with a continuous output of temperature patterns without physical contact with the target (rabbit ears).

Photography

A Canon Pellix 35 mm camera, serial 158006, equipped with 100ASA Kodacolor II film, was used to record thermograms from the color monitor's display screen. A shutter speed of 1/8 seconds was utilized throughout the data recording process.

The hands of a non-functional 24-minute clock (located in the field of view) were manually advanced after each picture. In such a manner, each photograph was assigned a particular number for identification purposes.

Rabbit

The particular animal used in this experiment was a white rabbit, approximately 3 years old, and in good health. Prior to use in this test, it had been cared for by the Virginia Polytechnic Institute and State University Veternary Science Department. No previous experimentation had been conducted on it.

IV. METHODOLOGY

The same basic format was followed in each of three exposure tests. The Dasibi ozone monitor in each case was allowed to warm up for at least 40 minutes prior to test initiation. At the end of this time the rabbit was secured in the restraining cage and his left ear propped in a vertical position by wedging a rolled sheet of plastic through the case and behind the ear. Rabbit and restrainer were then placed in the chamber and the Reynolds Film door was secured in place. At this time, the circulation pump was turned on and set at a flow of 28 l/min. Approximately ten minutes have at this point elapsed since the rabbit was first placed in the restrainer. During the next 5 minutes several pictures were taken from the color monitor's display screen in order to establish a pre-exposure temperature pattern. Following was a period of from 12 to 17 minutes in which ozone was introduced into the chamber via teflon tubing. The oxygen feeding the ozone generator was flowing at a rate of nearly 0.1 l/min. Since the efficiency of the ozone generator was not known, an exact ozone flow rate was unavailable.

During this exposure period one picture was taken approximately every 2 minutes. Approximately 5 minutes were then allowed for the ozone concentration to decrease to levels corresponding to pre-test conditions. During this

tapering period, data were continually gathered by photographing the color monitor's display screen approximately every 2 minutes. Upon test completion the rabbit was returned to his housing, located a few meters from the test chamber.

The following pages contain: a minute-by-minute account of events occurring before, during and after each of the three ozone exposures; a photographic record of thermal contours of the rabbit's ear made during each testing period; and a summary table which provides information pertaining to the time each photograph was taken, the temperature decrease noted between each picture and the original background print, the concentration present at the time of each photograph and the page on which each picture is found.

As a comparative aid, the first background picture of each test was placed beside each succeeding photograph. This enables one to readily observe differences in thermal patterns occurring since the beginning of a test exposure.

It is important to note that the temperature differences referred to in the Summary Table are subject to the author's own interpretation of the data. They should, therefore, be considered only as a general statement of developing trends.

Rabbit Ozonation Experiment #1

Time	Comment	Room Temperature (° C)	Ozone Concentration (ppm)
12:00 (noon)	Dasibi turned on;		
1:00	Rabbit placed into restrainer and chamber; Reynolds Film sealed; circulation pump on;	26.5	
1:12	1st picture;	26.5	0.08
1:14	2nd picture;	26.5	0.09
1:16	3rd picture;	26.5	0.06
1:18	Ozone generator turned on; Oxygen feed set at 1.7 on rotameter 1;		
1:20	4th picture;	26.5	0.47
1:22	5th picture;	26.5	0.70
1:24	6th picture;	26.5	0.77
1:26	7th picture;	26.5	0.64
1:28	8th picture;	26.5	0.61
1:30	9th picture; ozone generator off; ozone feed off;	26.5	0.76
1:33	10th picture;	26.5	0.25
1:35	11th picture;	26.5	0.27
1:37	12th picture;	26.5	0.22
1:39	13th picture;	26.5	0.16
1:41	14th picture; rabbit returned to habitat	26.5	0.18

The photographic record of this test can be found on pages 52 - 64.

Rabbit Ozonation Experiment #2

This test was initiated approximately 3 hours and 50 minutes after termination of experiment #1. During this time, the Dasibi ozone monitor was continually operating.

Time	Comment	Room Temperature (° C)	Ozone Concentration (ppm)
5:30 p.m.	Rabbit secure in restrainer and placed in chamber; Reynolds Film sealed; circulation pump on;	27	
5:42	1st picture;	27	0.15
5:44	2nd picture; ozone generator on;	27	0.16
5:46	3rd picture;	27	0.14
5:48	4th picture;	27	0.15
5:50	5th picture; ozone feed introduced;	27	0.21
5:52	6th picture;	27	0.23
5:54	7th picture;	27	0.35
5:56	8th picture;	27	0.80
5:58	9th picture;	27	0.75
6:00	10th picture;	27	0.83
6:02	11th picture;	27	0.80
6:04	12th picture;	27	0.91
6:06	13th picture;	27	0.99
6:07	Ozone generator off; ozone feed off;		
6:08	14th picture;	27	0.65
6:10	15th picture;	27	0.35
6:12	16th picture;	27	0.27
6:14	17th picture;	27	0.23
6:16	18th picture;	27	0.21

Rabbit Ozonation Experiment #2 (continued)

Time	Comment	Room Temperature (° C)	Ozone Concentration (ppm)
6:18 p.m.	19th picture;	27	0.19
6:20	Rabbit returned to habitat		

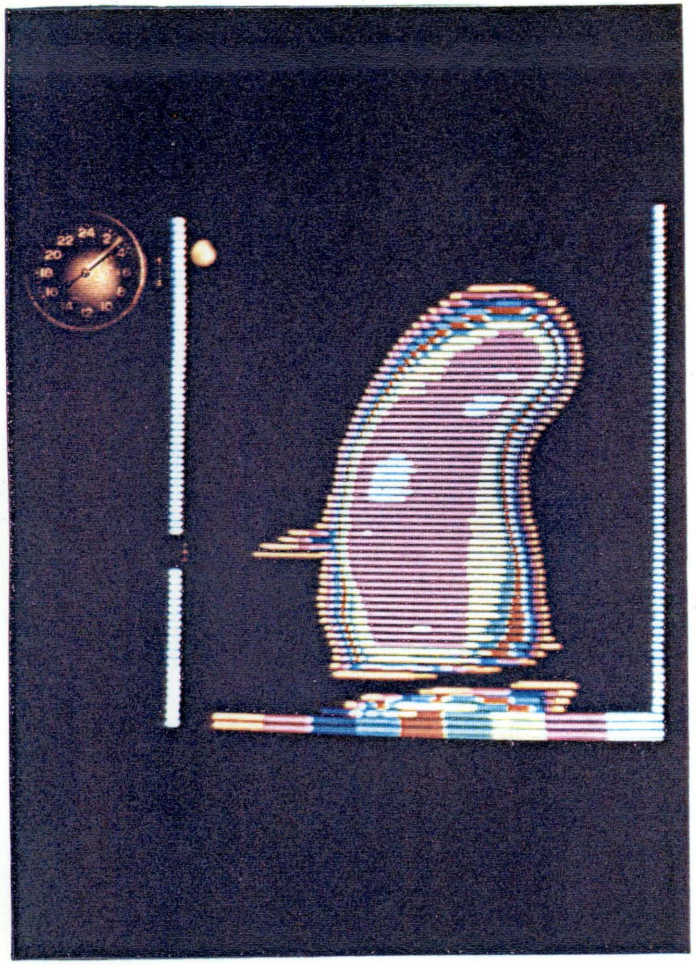
The photographic record of this test can be found on pages 65 - 82.

Rabbit Ozonation Experiment #3

This test was conducted the morning following experiments 1 and 2.

Time	Comment	Room Temperature (° C)	Ozone Concentration (ppm)
7:00 a.m.	Dasibi turned on;		
7:45	Rabbit placed in restrainer and chamber; Reynolds Film secured in place; pump on;		
7:55	1st picture;	24	0.11
7:57	2nd picture;	24	0.11
7:58	Ozone generator on; ozone feed on;	24	
7:59	3rd picture;	24	0.27
8:01	4th picture;	24	0.51
8:03	5th picture;	24	0.75
8:06	6th picture;	24	0.72
8:08	7th picture;	24	0.77
8:10	8th picture;	24	0.77
8:12	9th picture;	24	0.72
8:13	Noticed ozone feed had drifted down to 1.5; therefore brought back up to 1.7;		
8:14	10th picture;	24	0.23
8:17	11th picture;	24	1.06
8:18	Oxygen off; ozone feed off;		
8:19	12th picture;	24	0.98
8:21	13th picture;	24	0.49
8:24	14th picture;	24	0.35
8:25	Ear brace removed and restrainer loosened via glove box.		

The photographic record of this test can be found on pages 83 - 95.



Time: $t = t_o$

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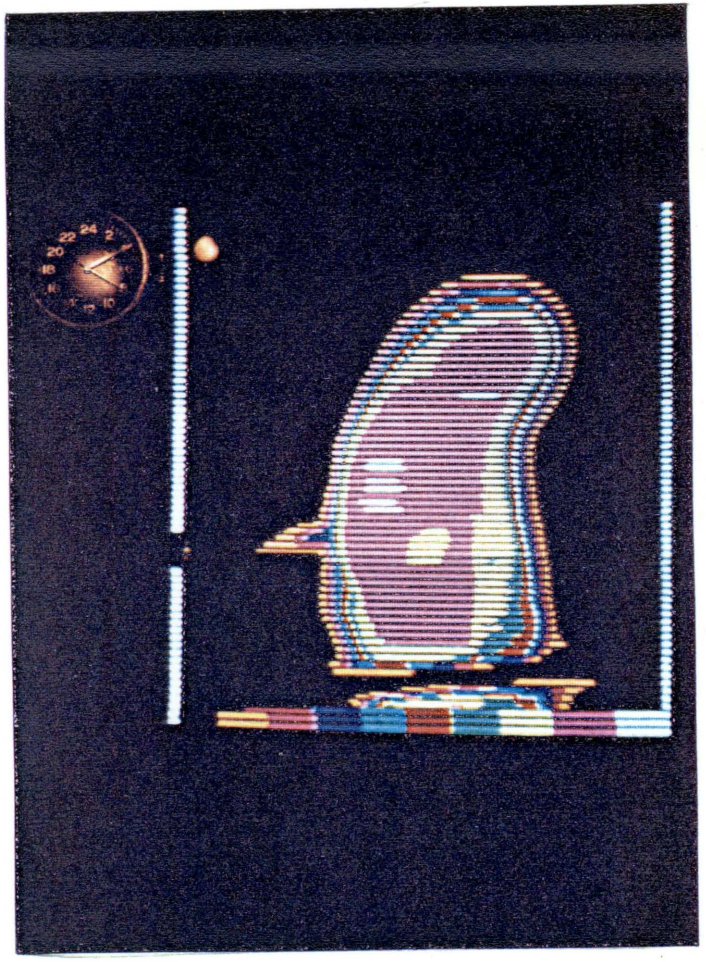

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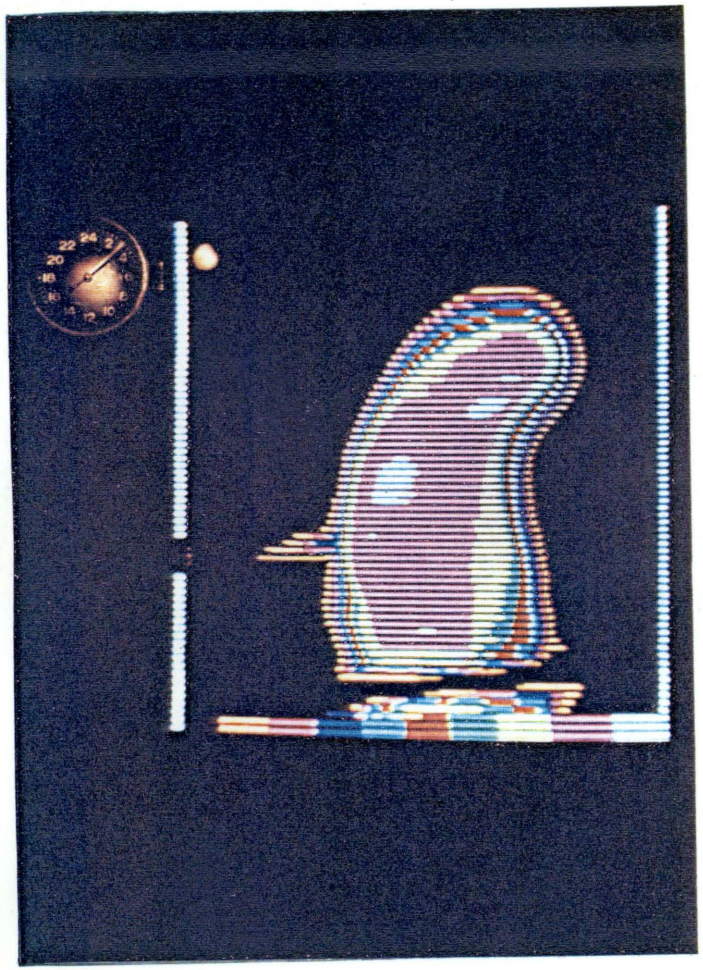

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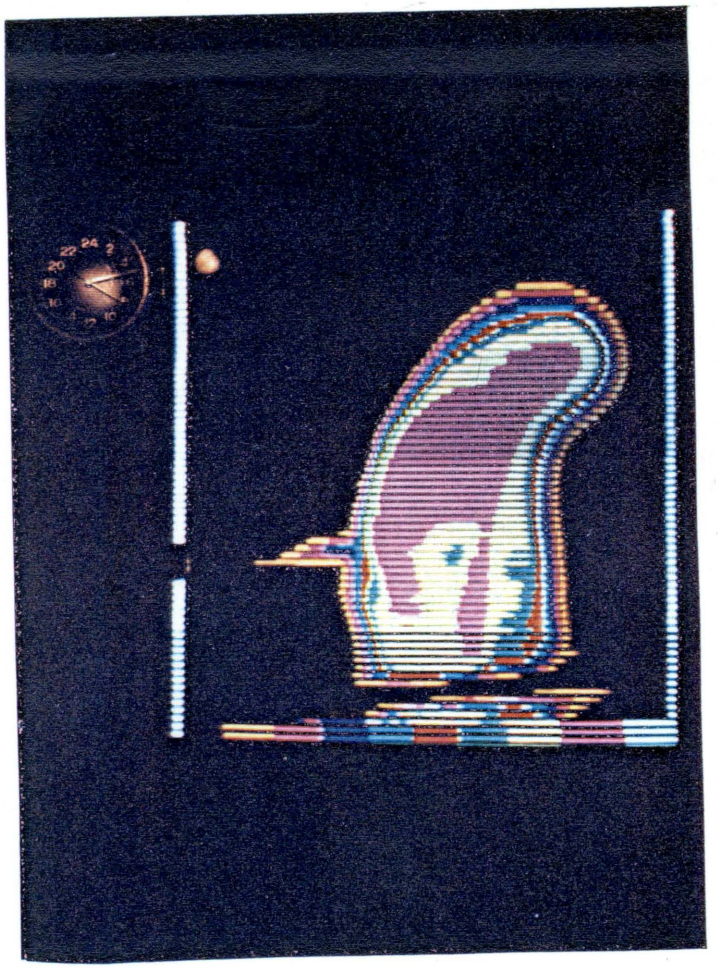

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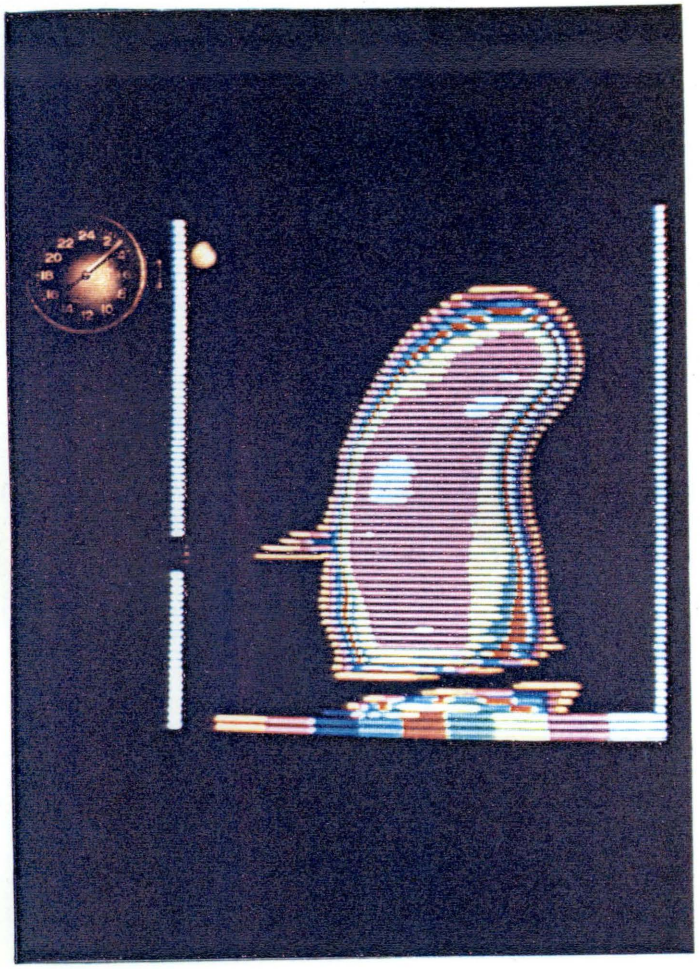
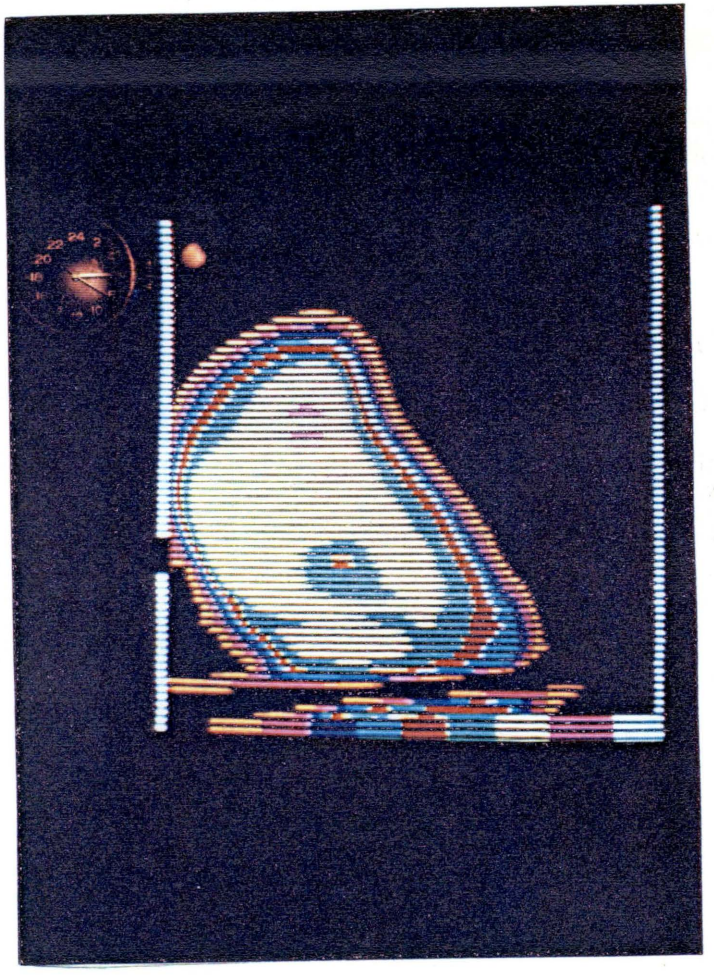


Time: $t = t_o$

Experiment 1



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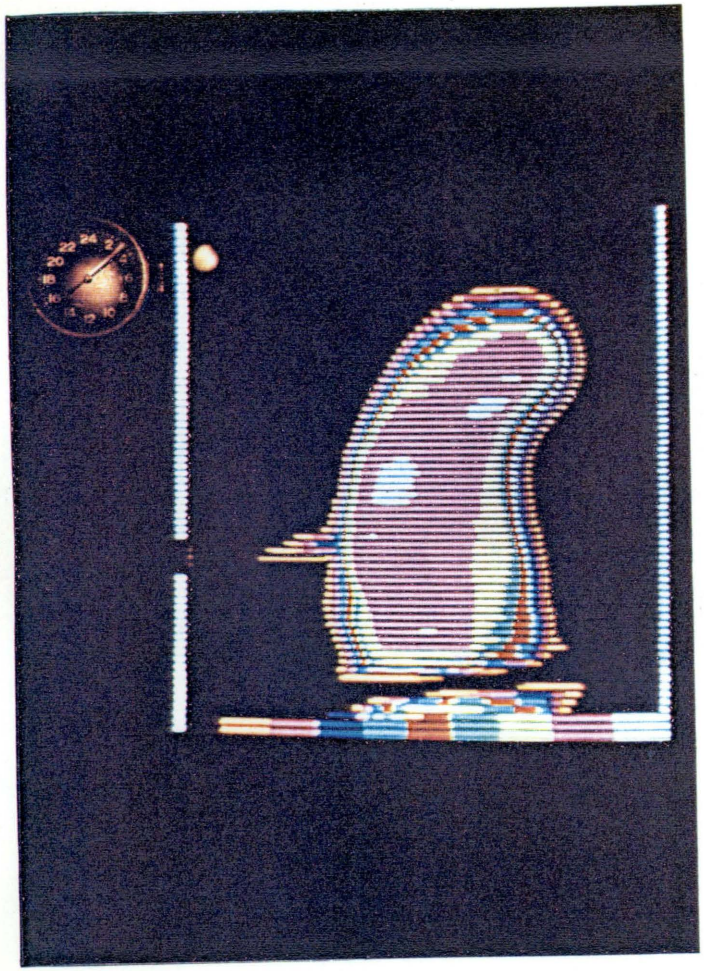

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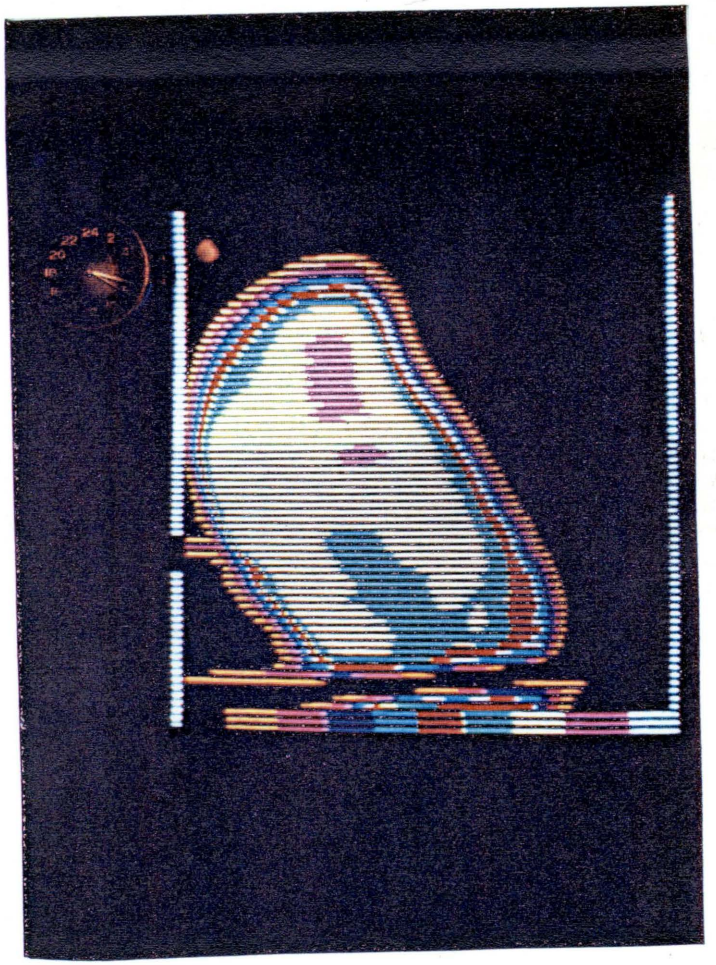
Experiment 1



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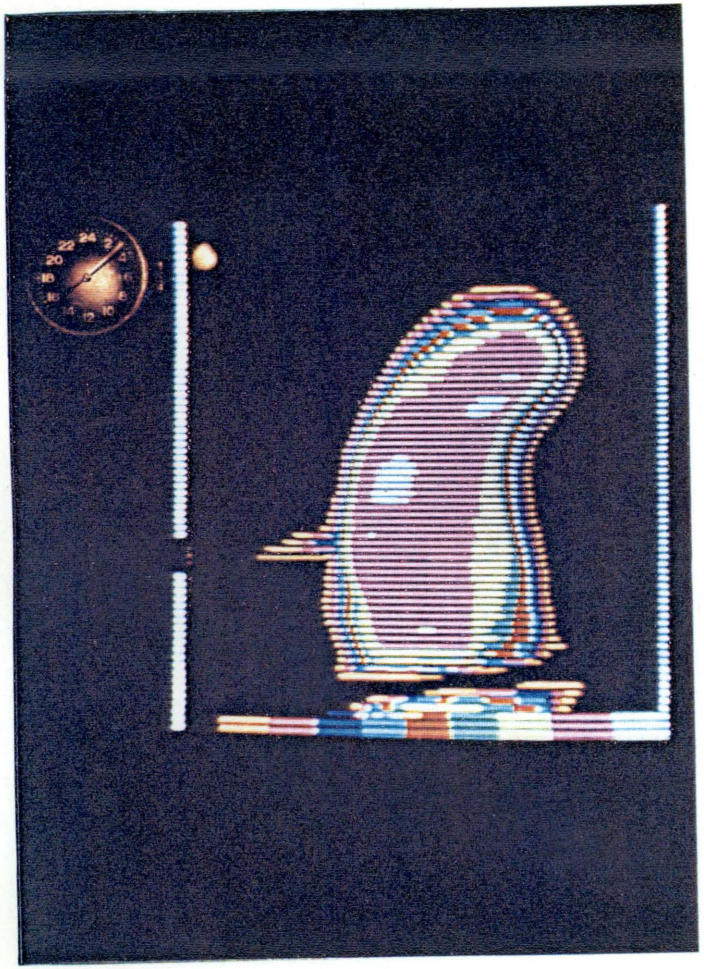

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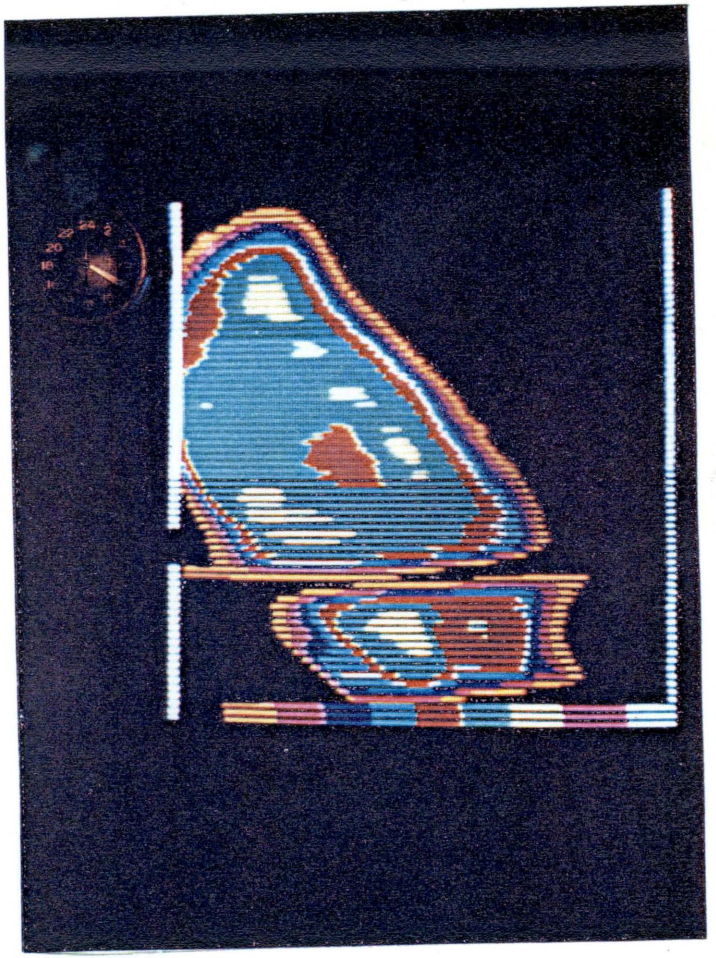

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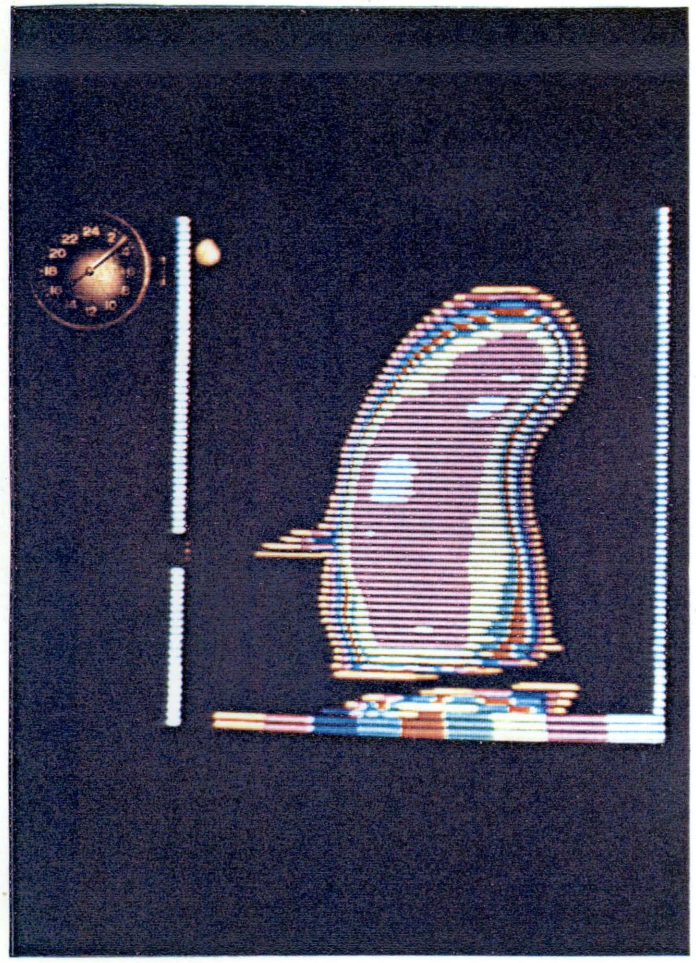

Time: $t = t_o$

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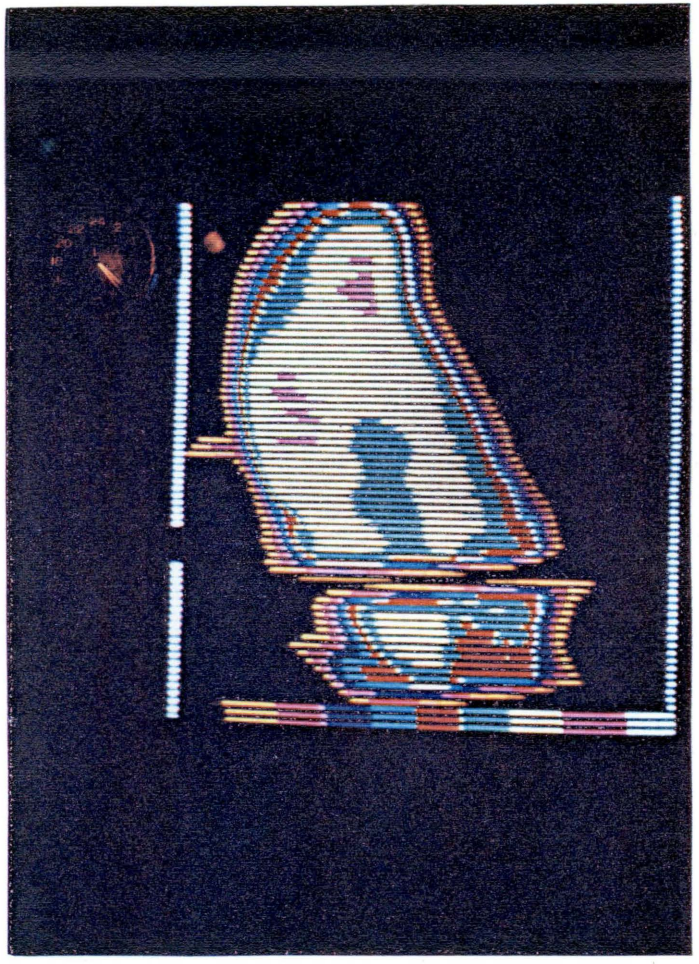

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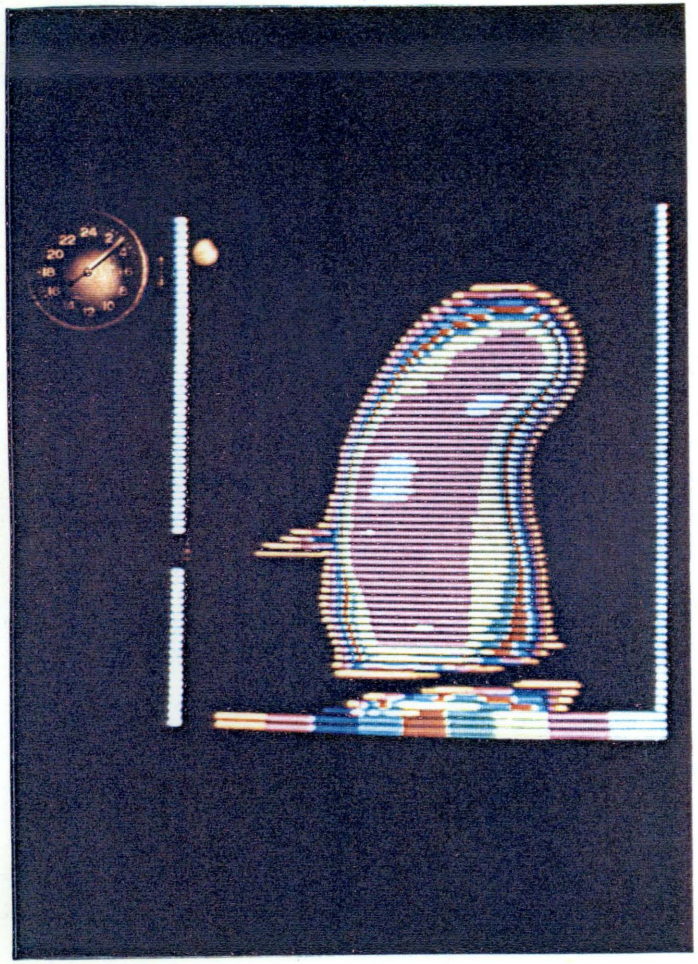

Time: $t = t_o$

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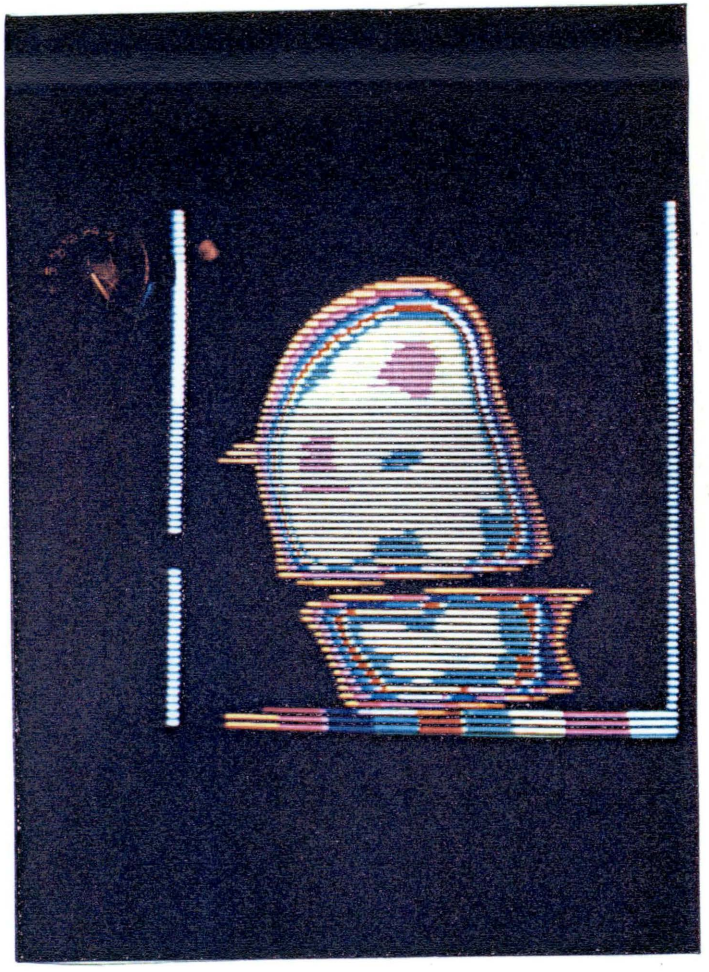

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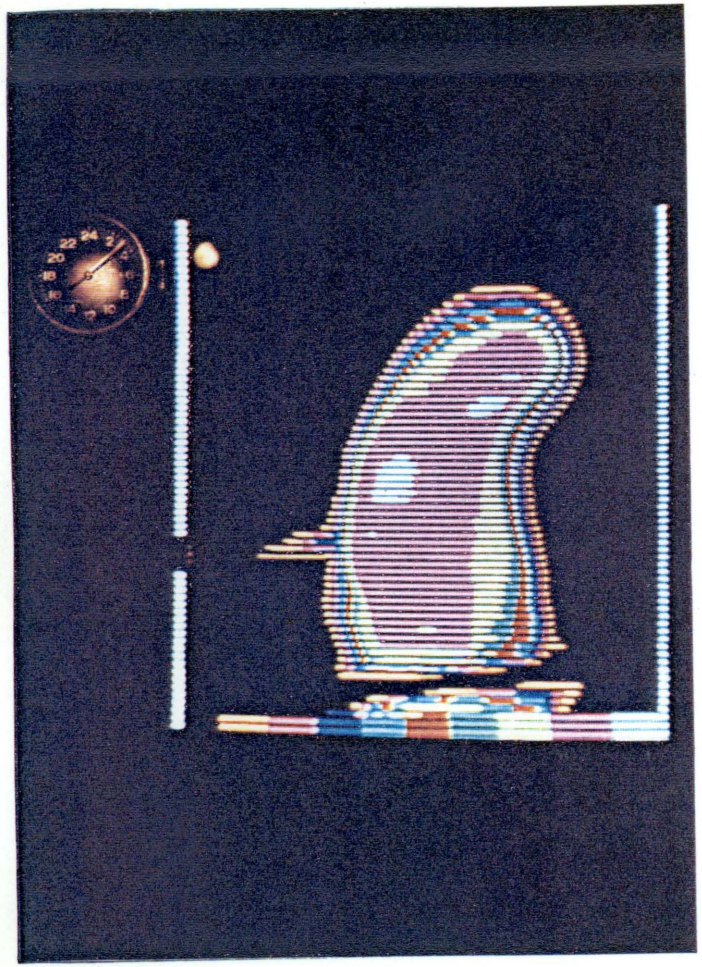
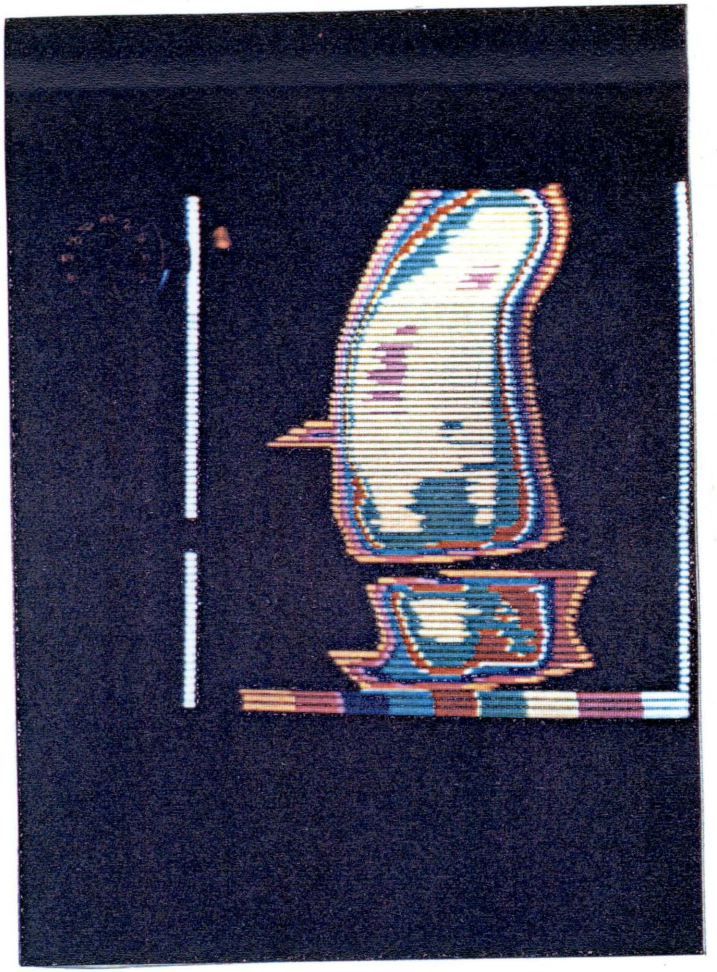


Time: $t = t_o$

Experiment 1



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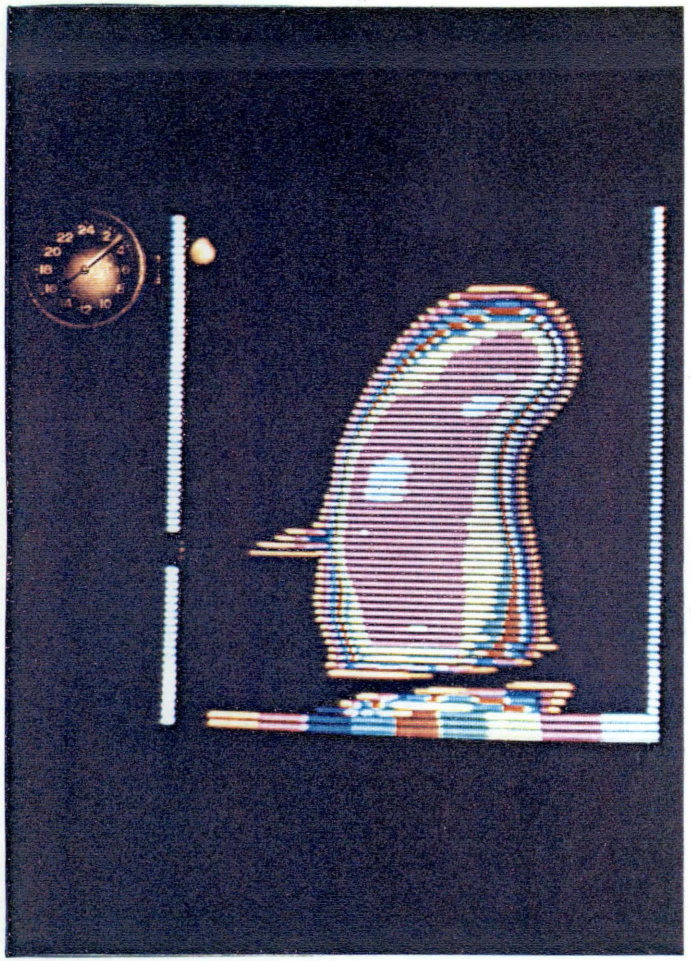

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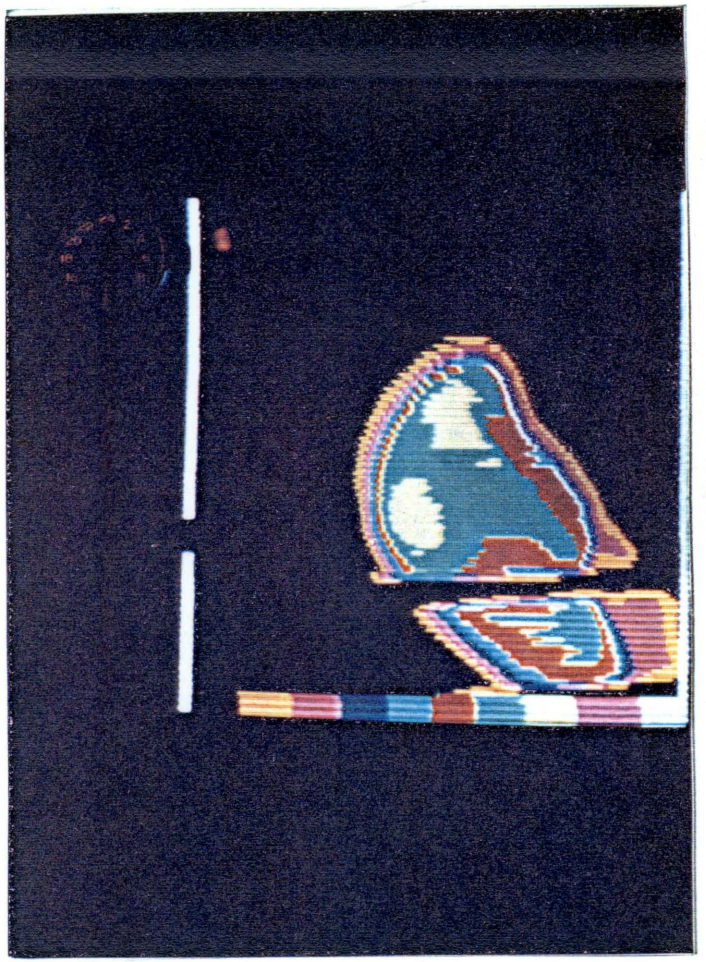

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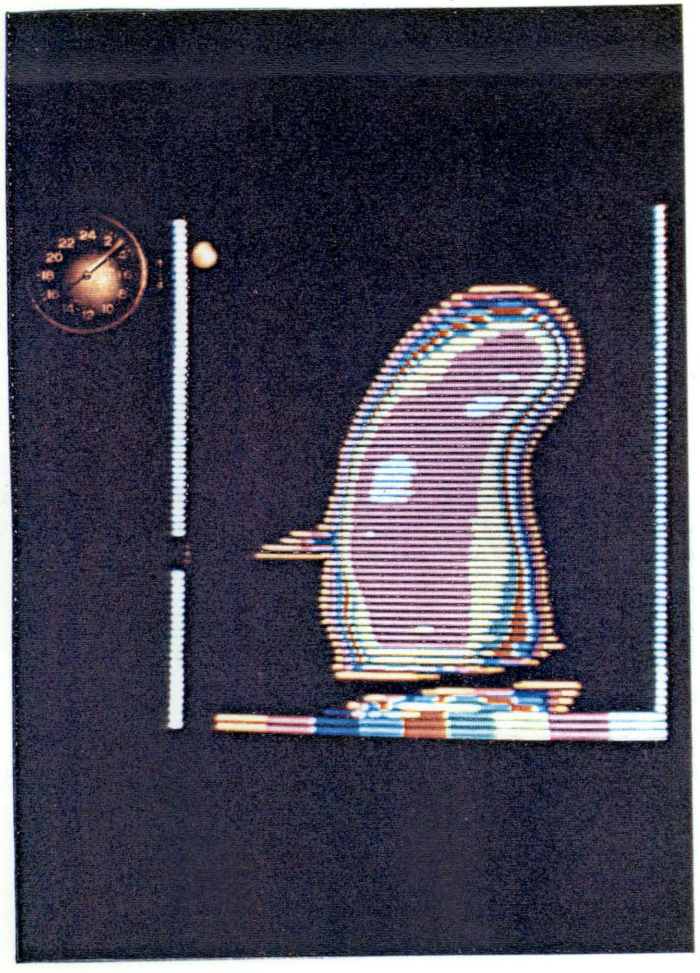
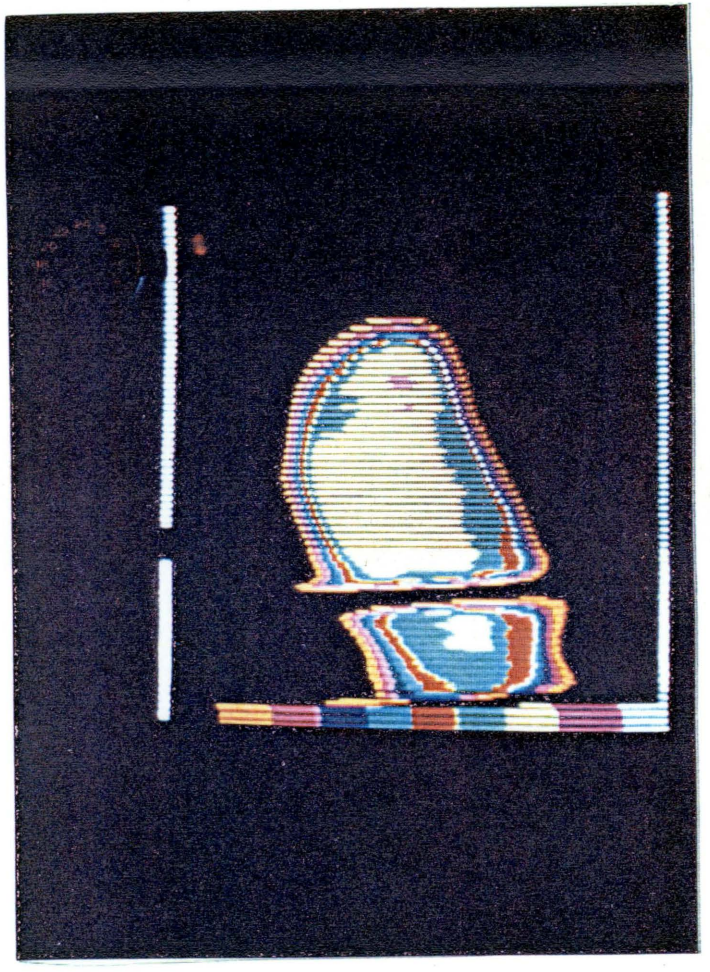


Time: $t = t_o$

Experiment 1



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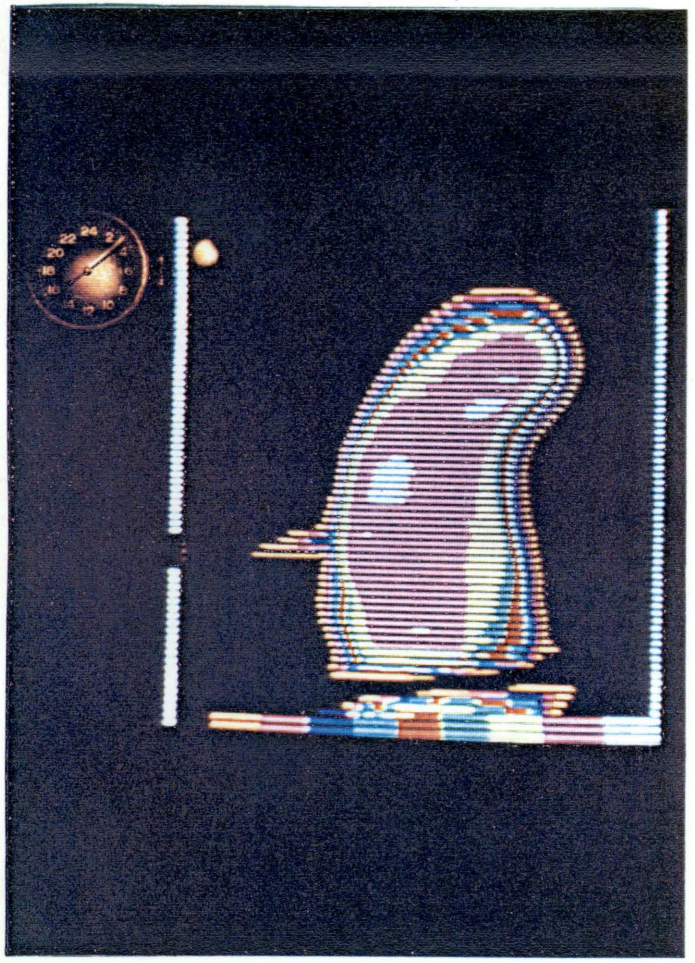
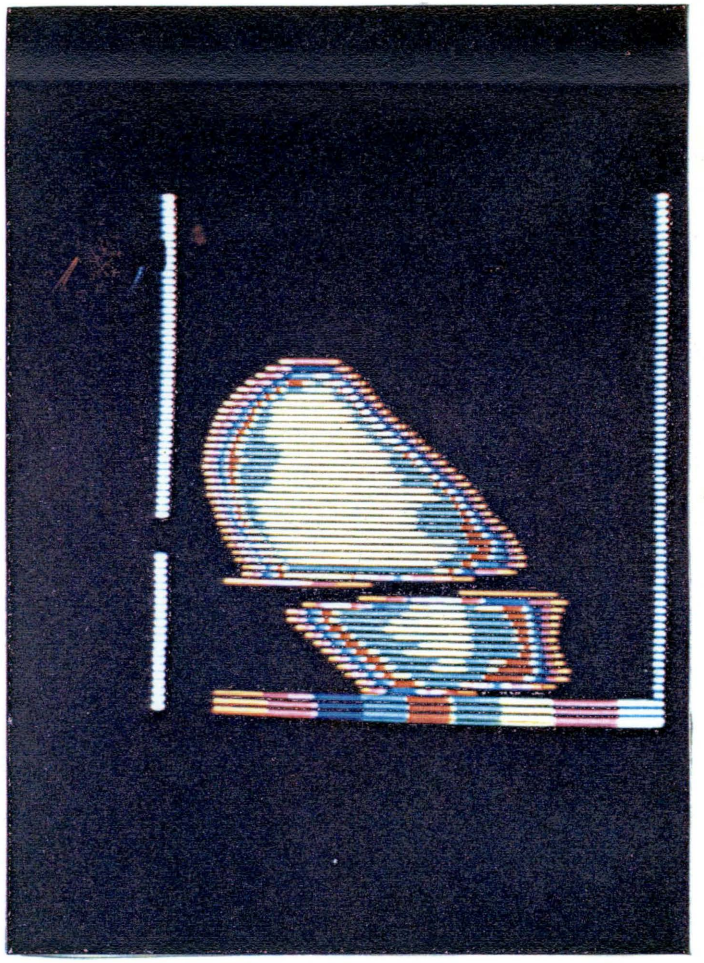


Time: $t = t_o$

Experiment 1



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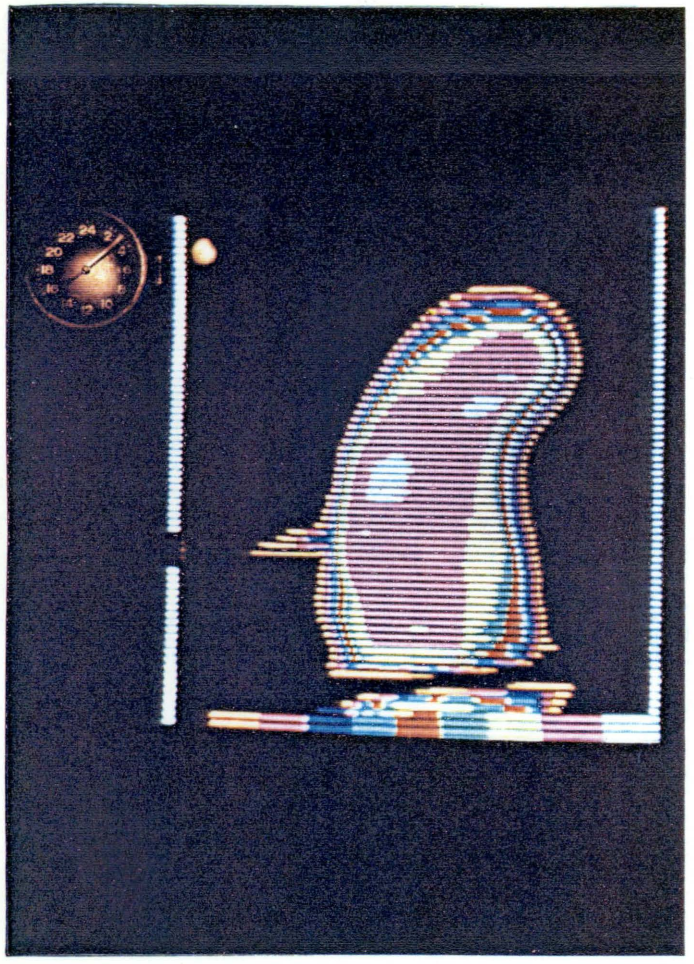

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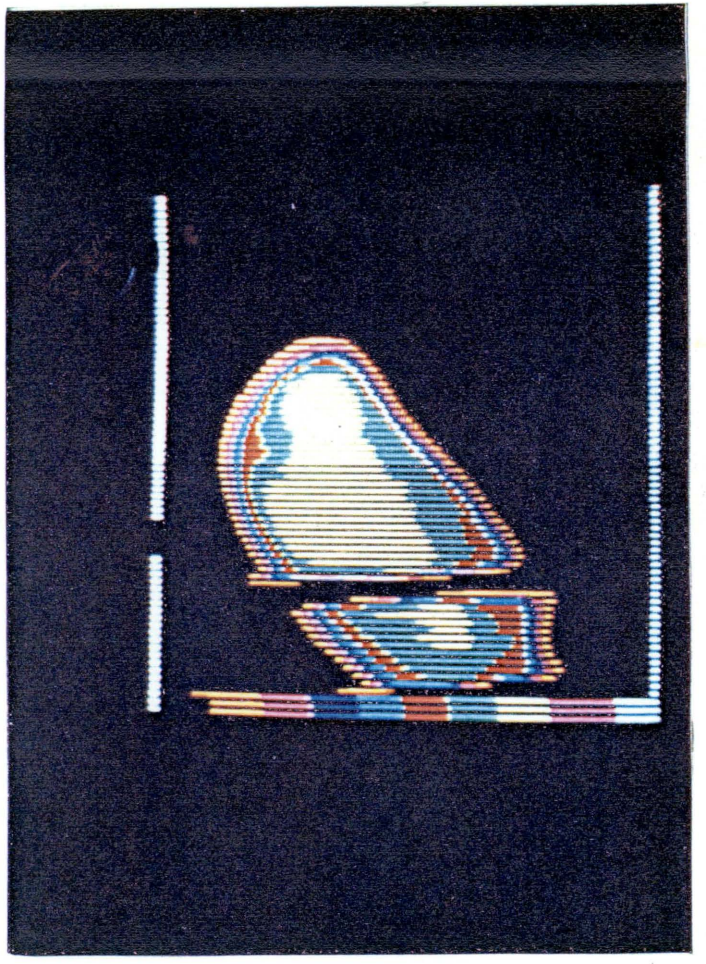
Experiment 1



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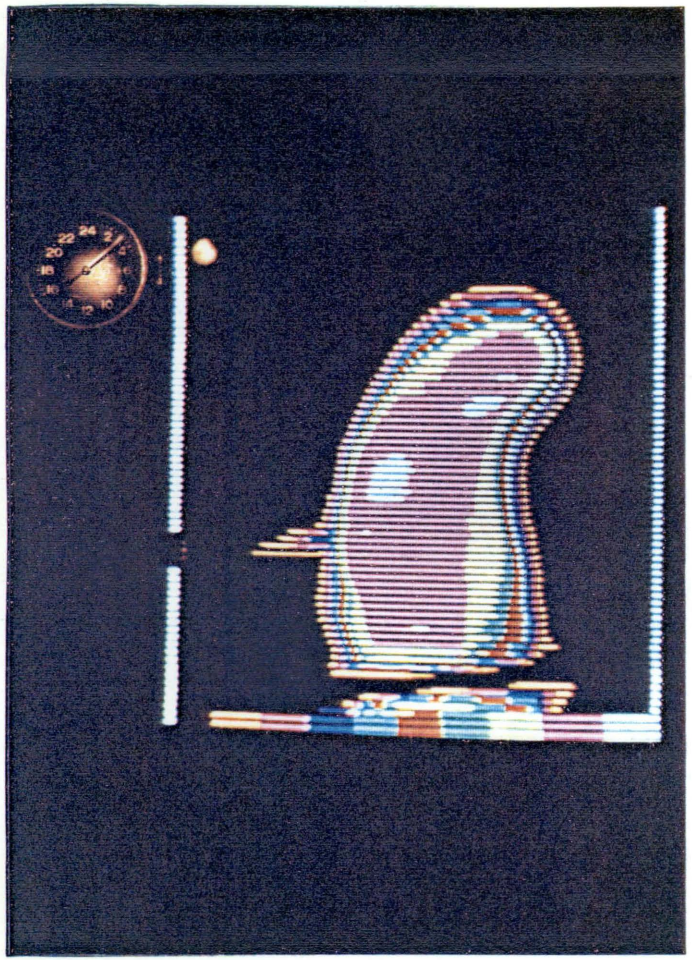

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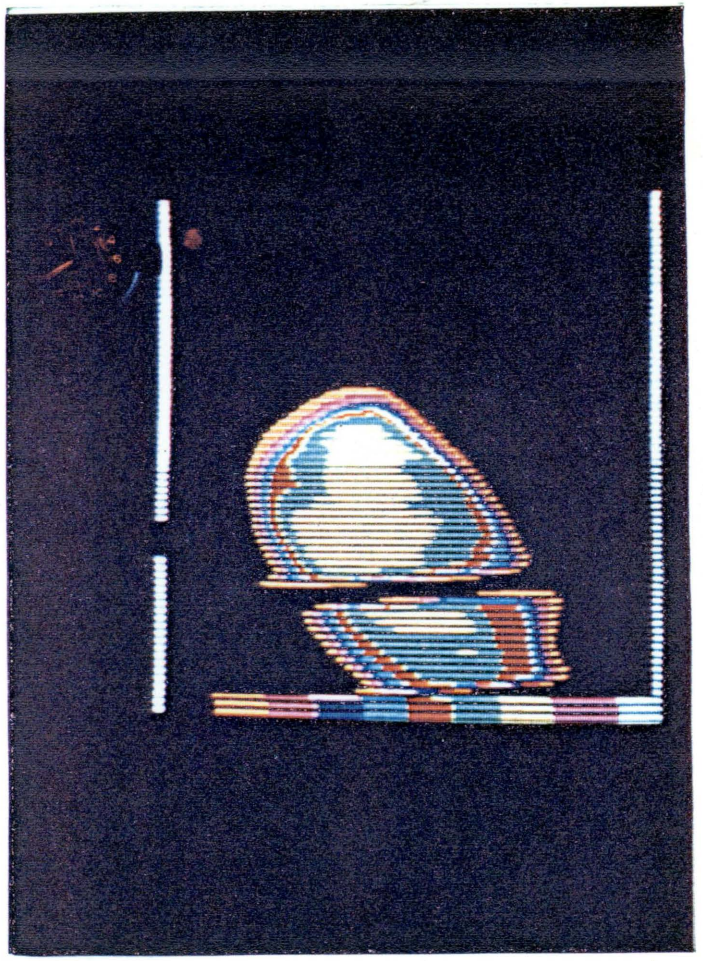
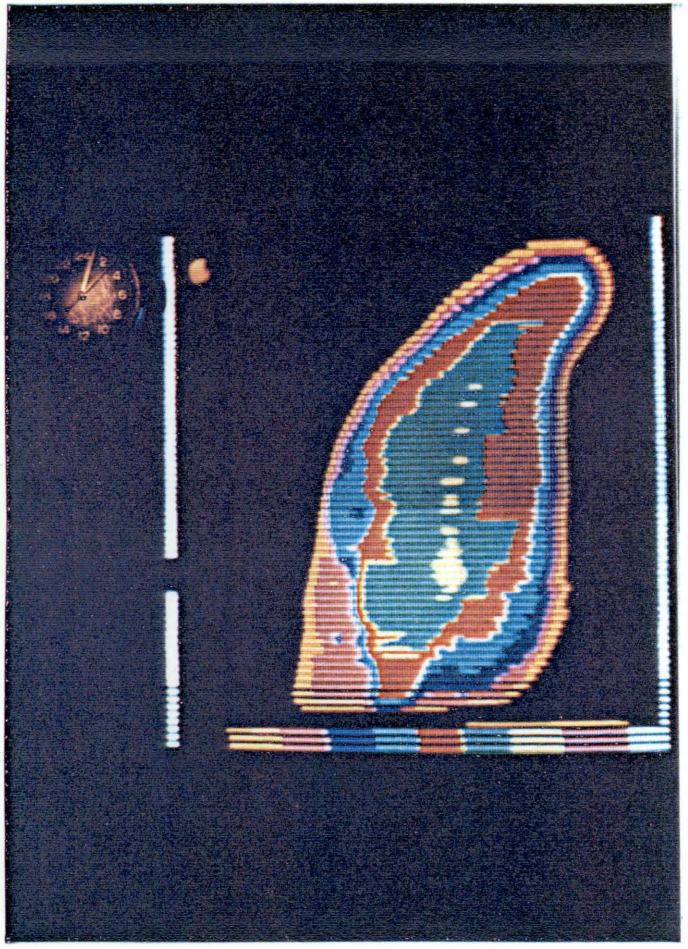
Experiment 1

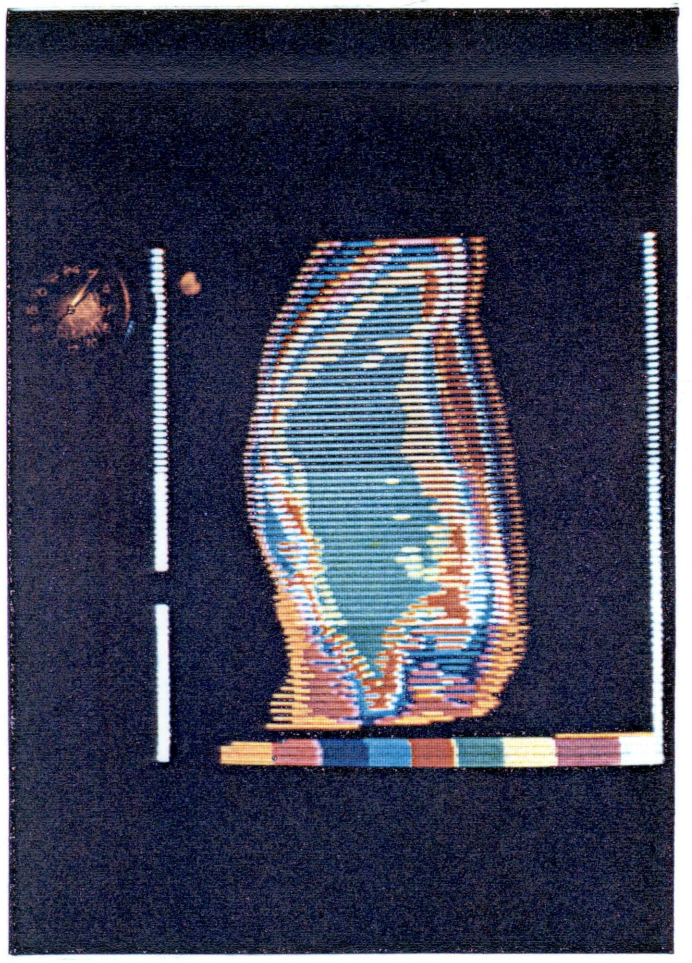


Time: $t = t_o + 29$ min.

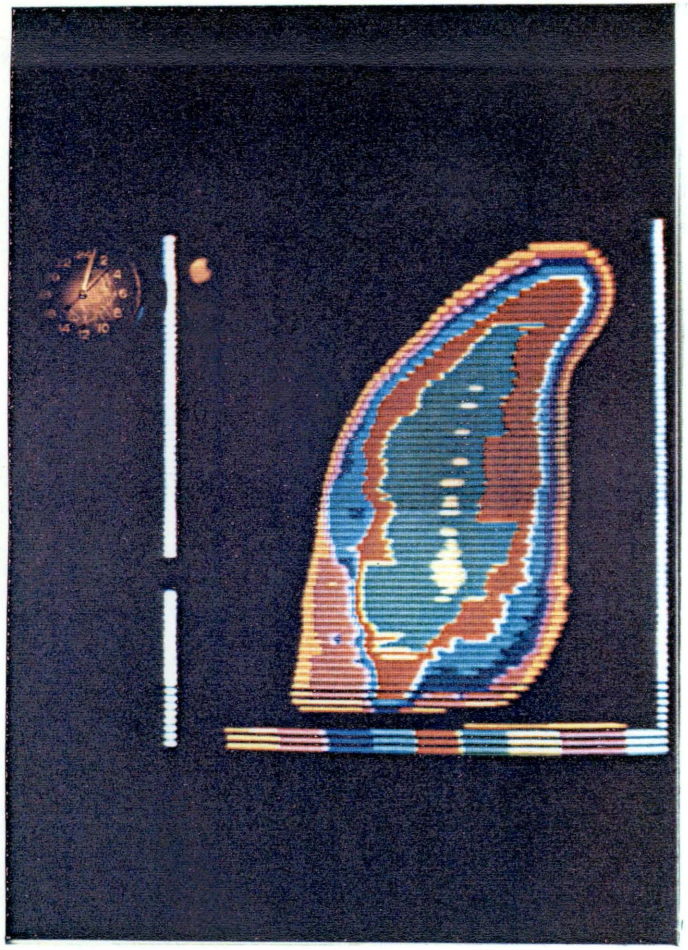


Time: $t = t_o$

Experiment 2

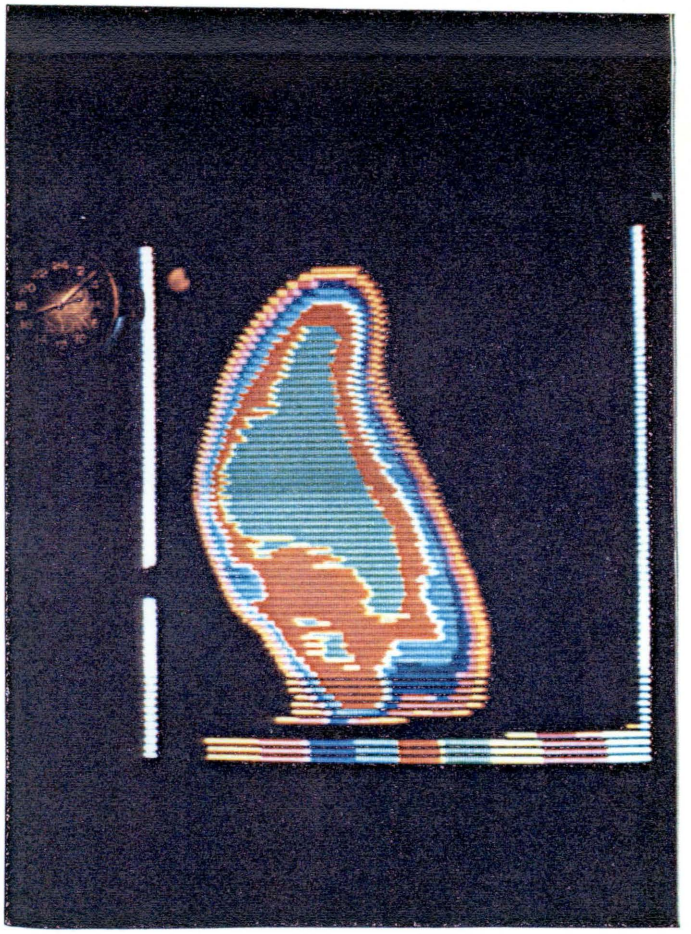


Time: $t = t_o + 2$ min.

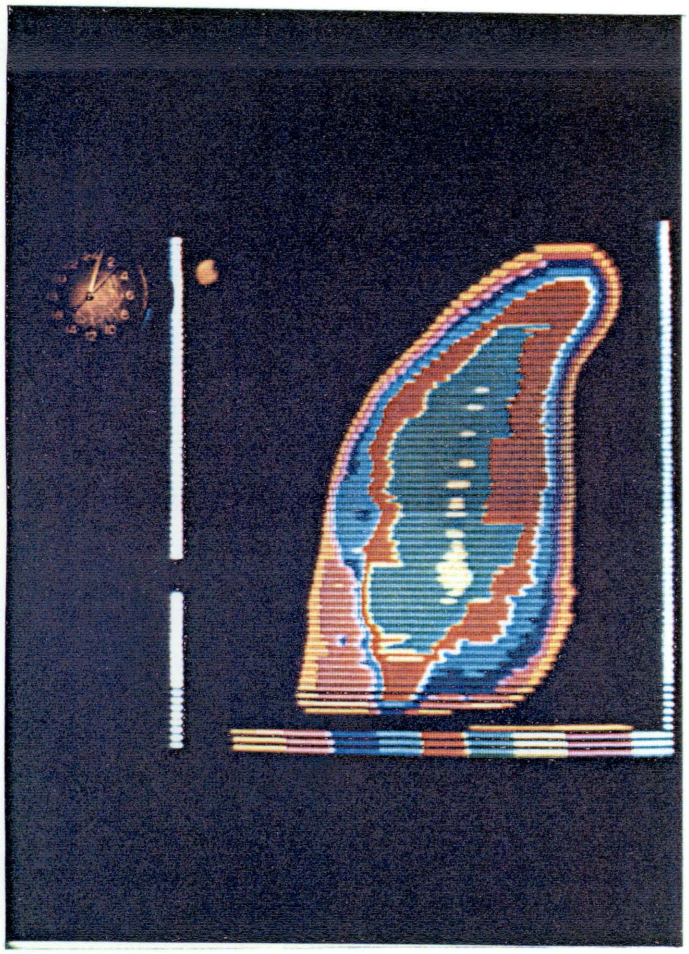


Time: $t = t_o$

Experiment 2

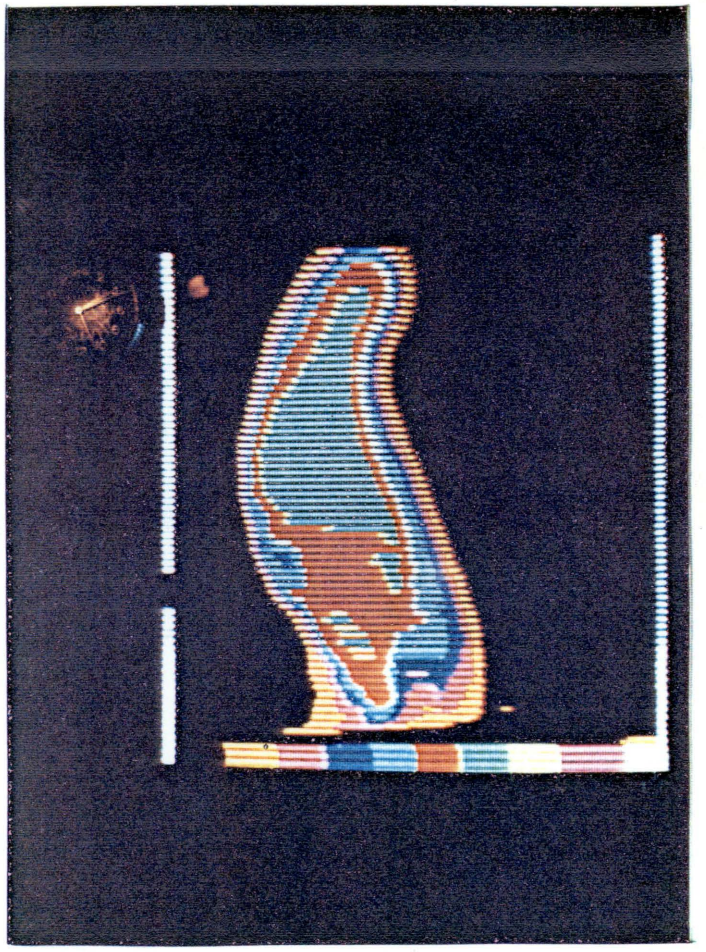


Time: $t = t_o + 4$ min.

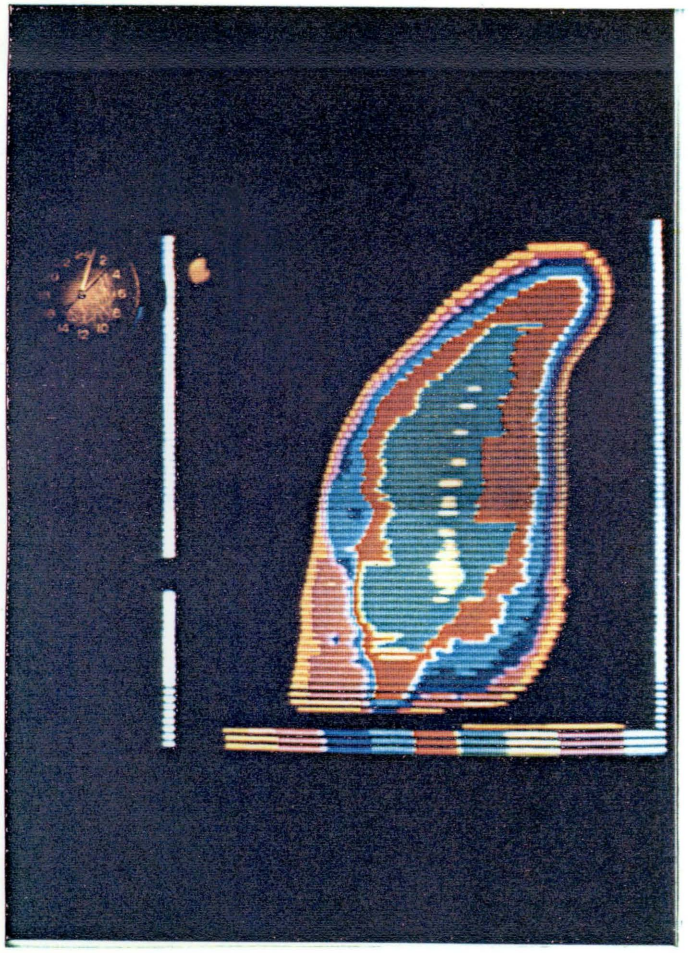


Time: $t = t_o$

Experiment 2

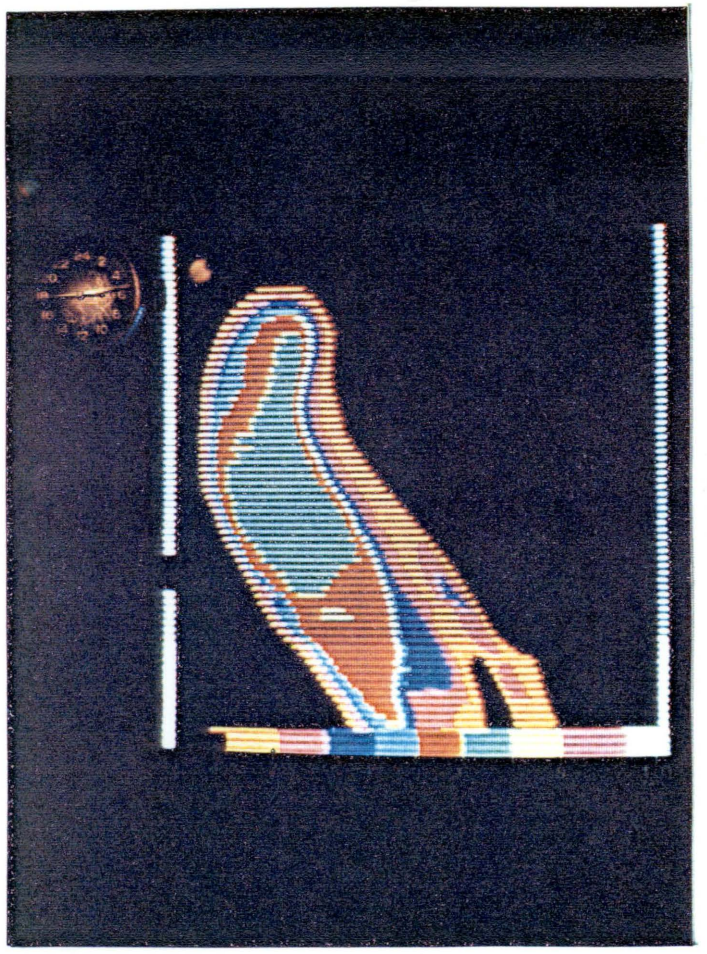


Time: $t = t_o + 6$ min.

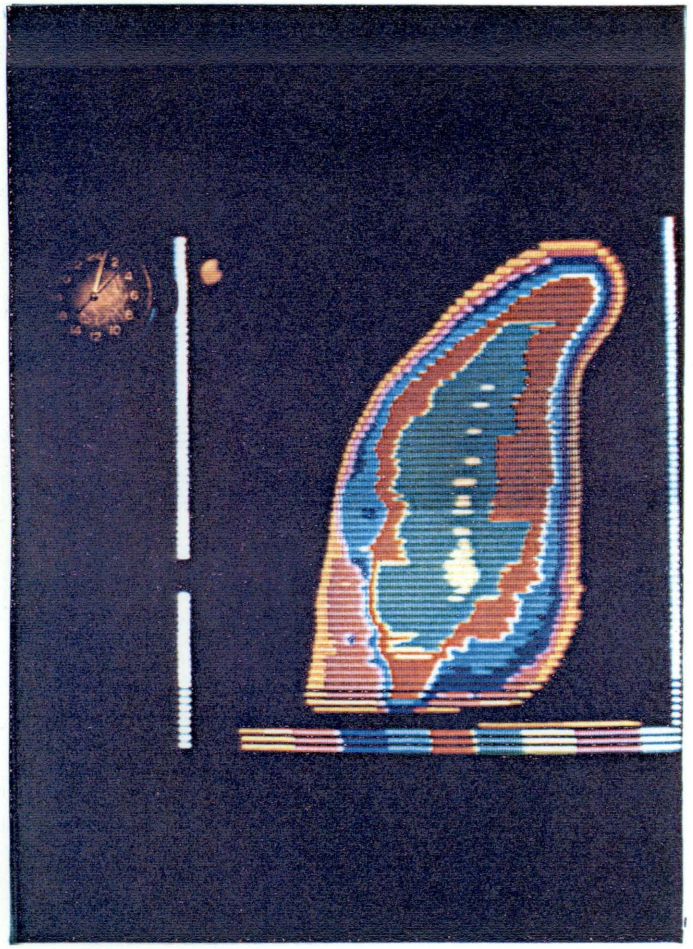


Time: $t = t_o$

Experiment 2

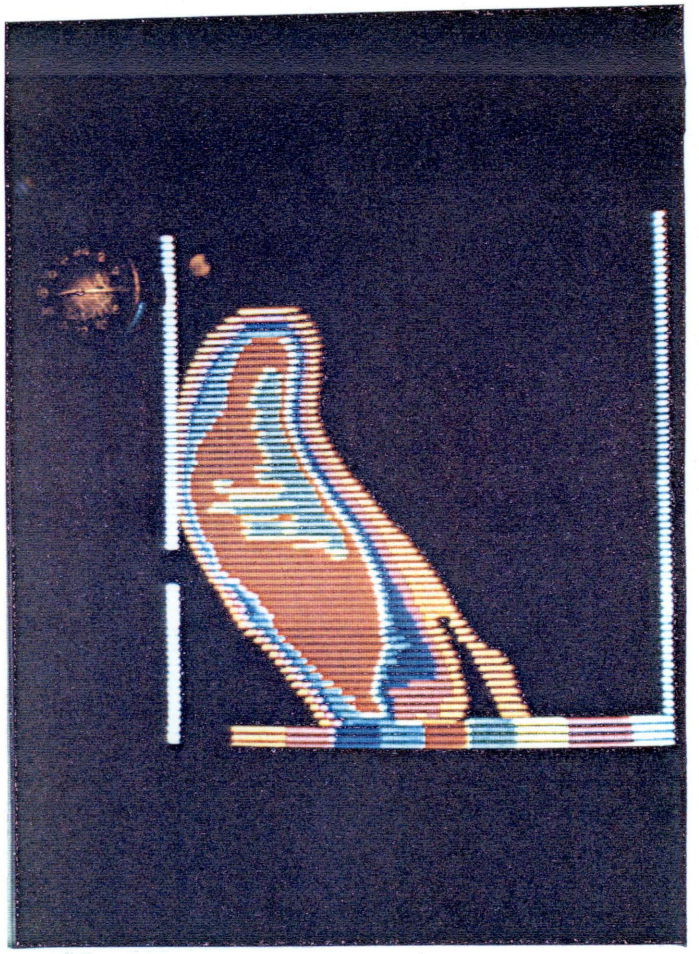


Time: $t = t_o + 8$ min.

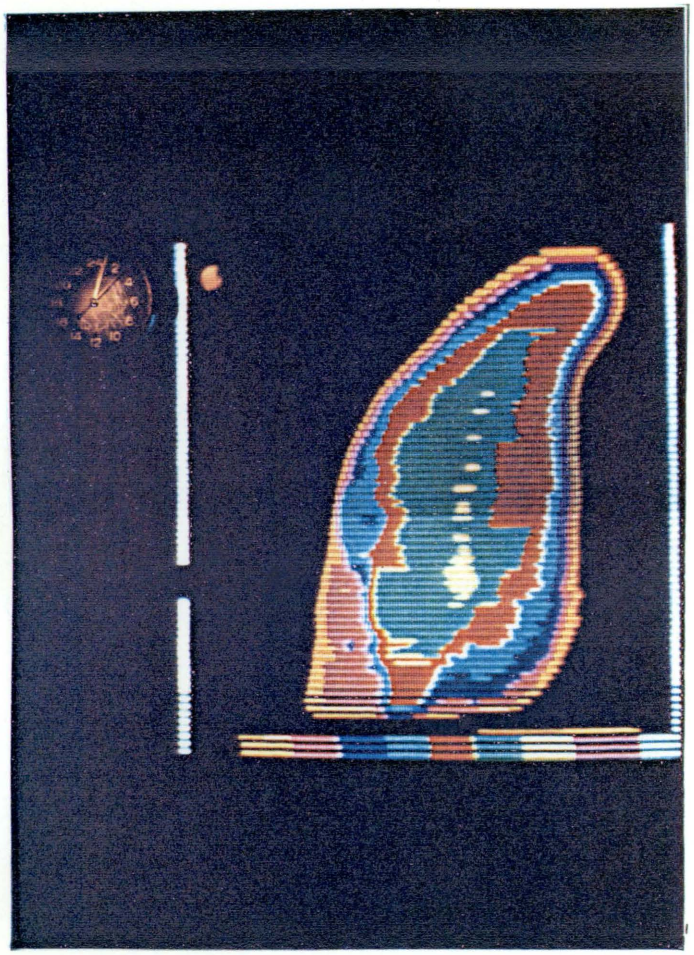


Time: $t = t_o$

Experiment 2

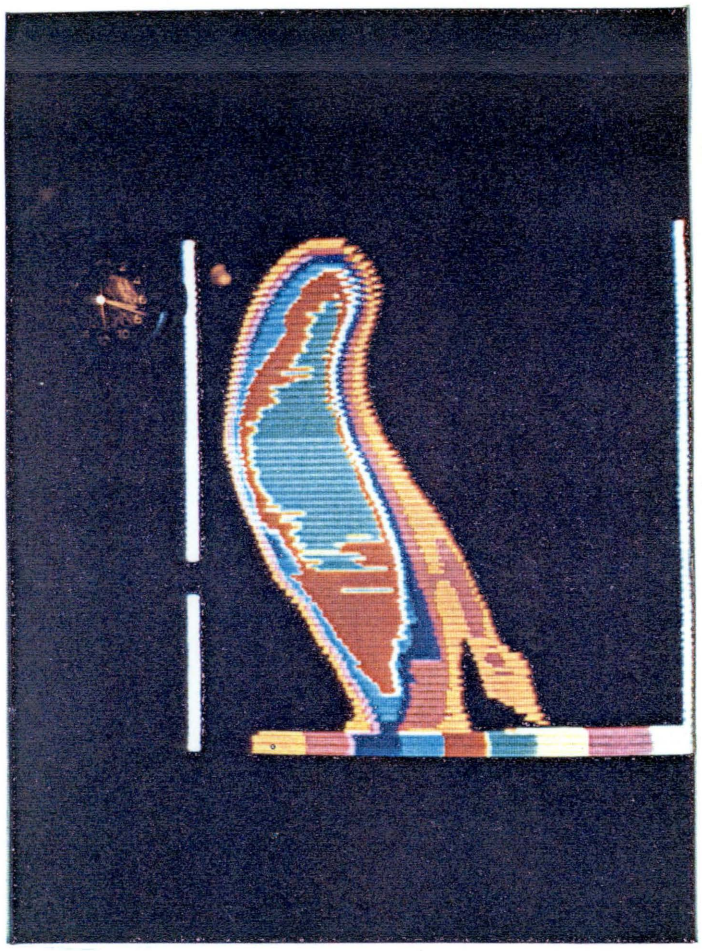


Time: $t = t_o + 10$ min.

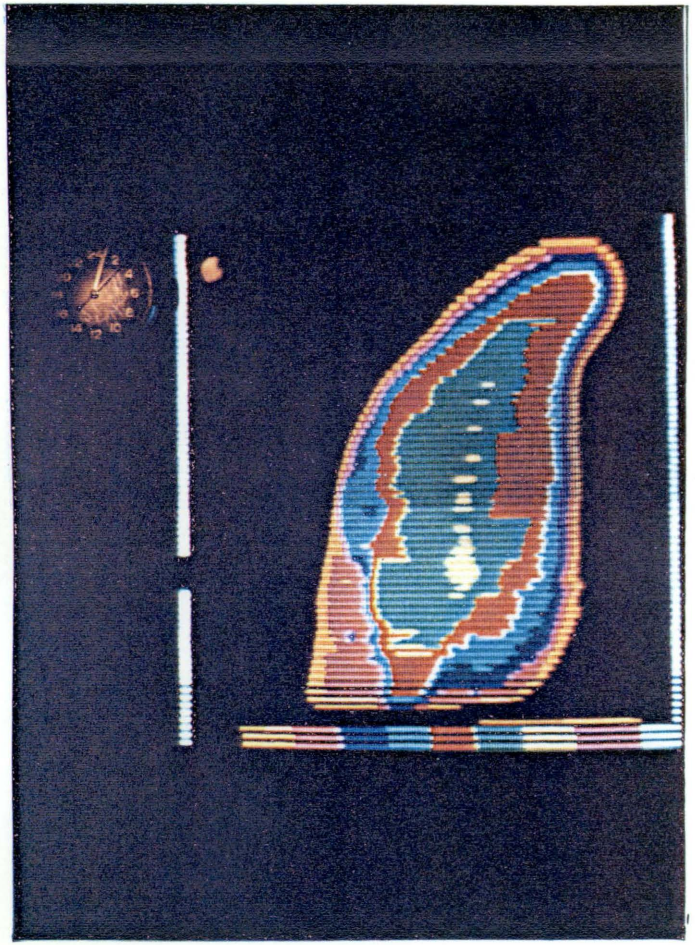


Time: $t = t_o$

Experiment 2

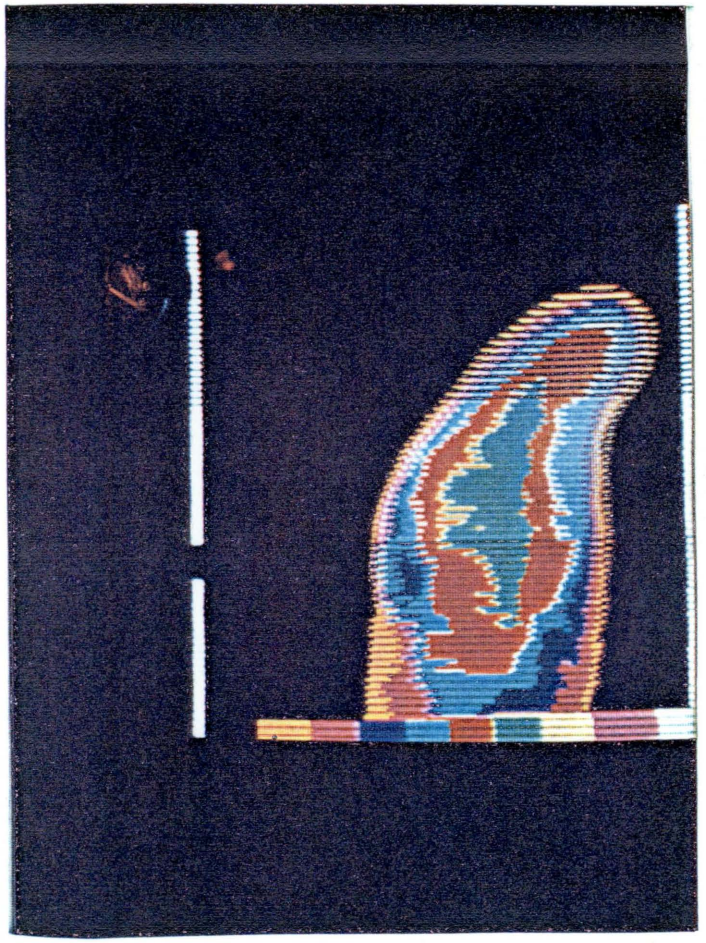


Time: $t = t_o + 12$ min.

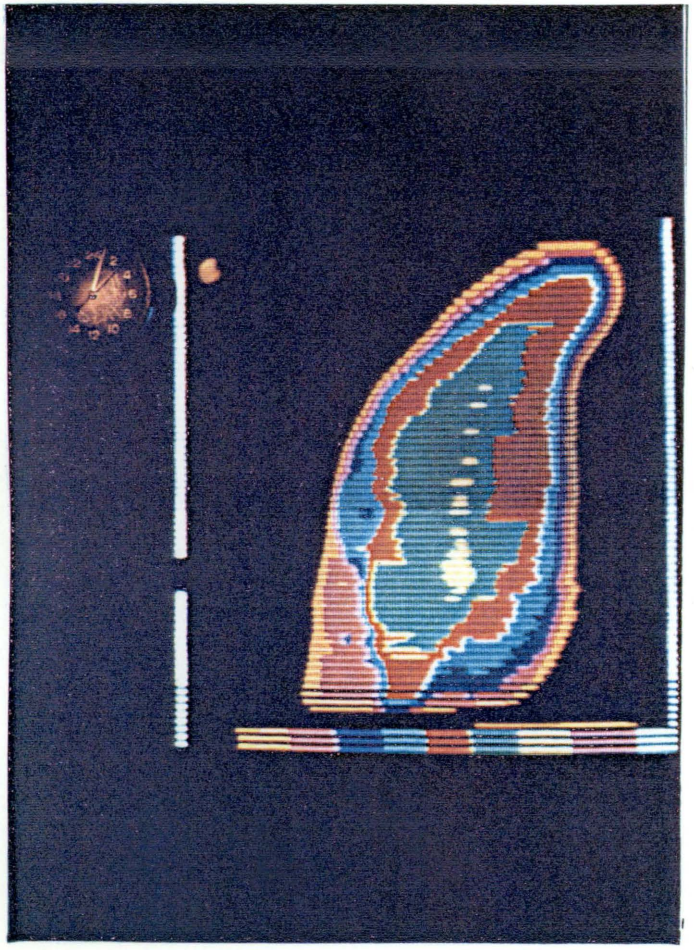


Time: $t = t_o$

Experiment 2

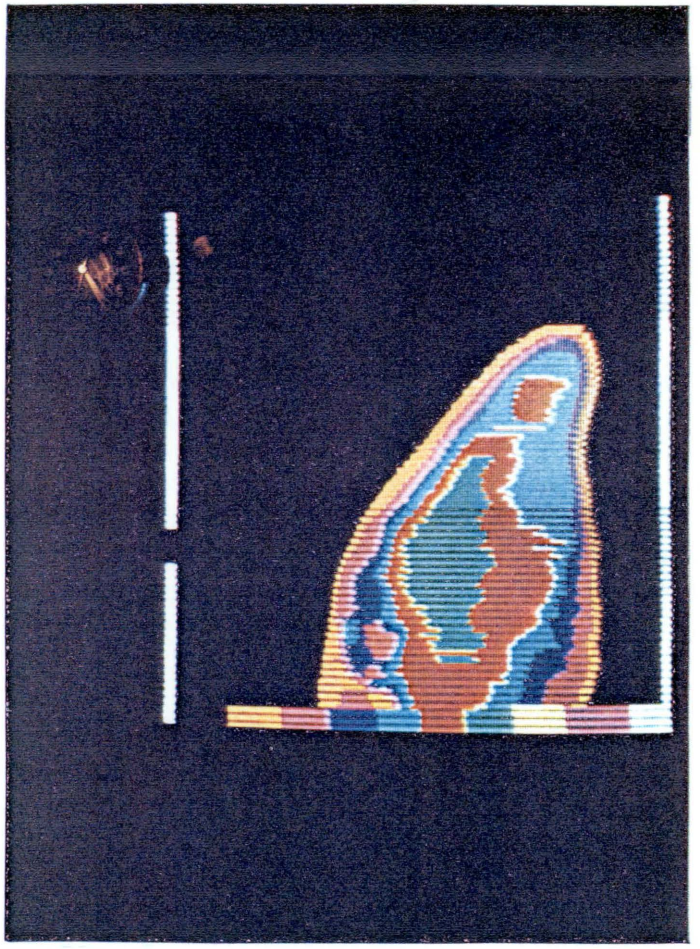


Time: $t = t_o + 14$ min.

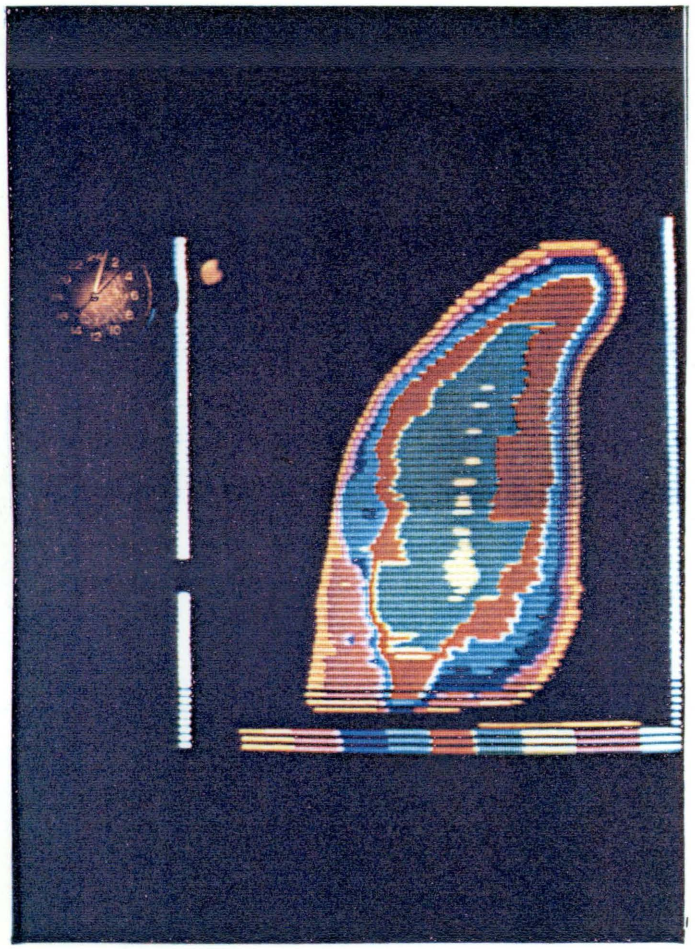


Time: $t = t_o$

Experiment 2

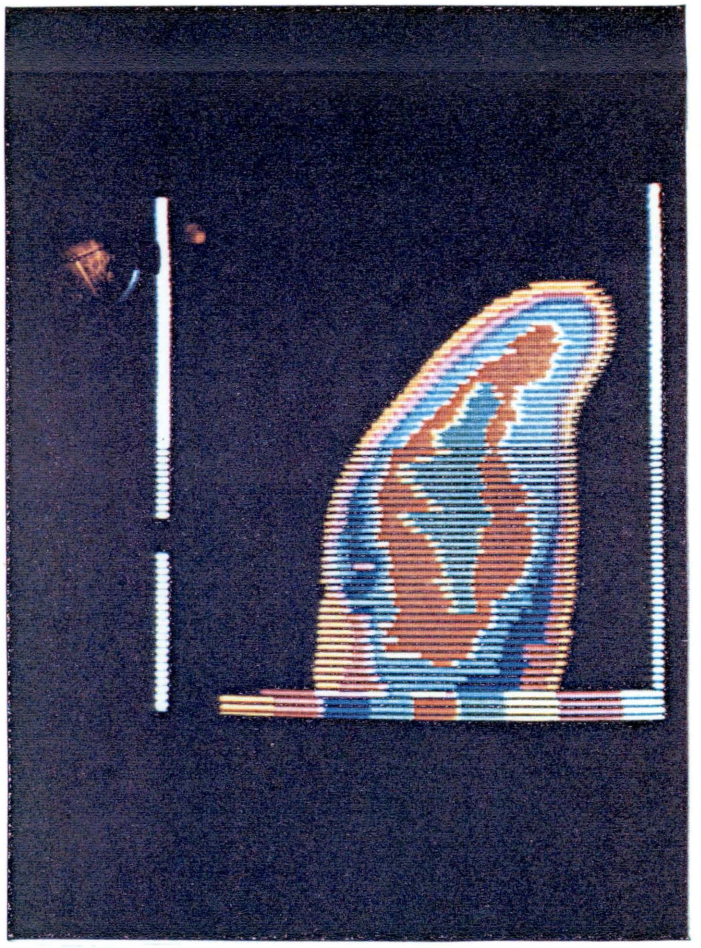


Time: $t = t_o + 16$ min.

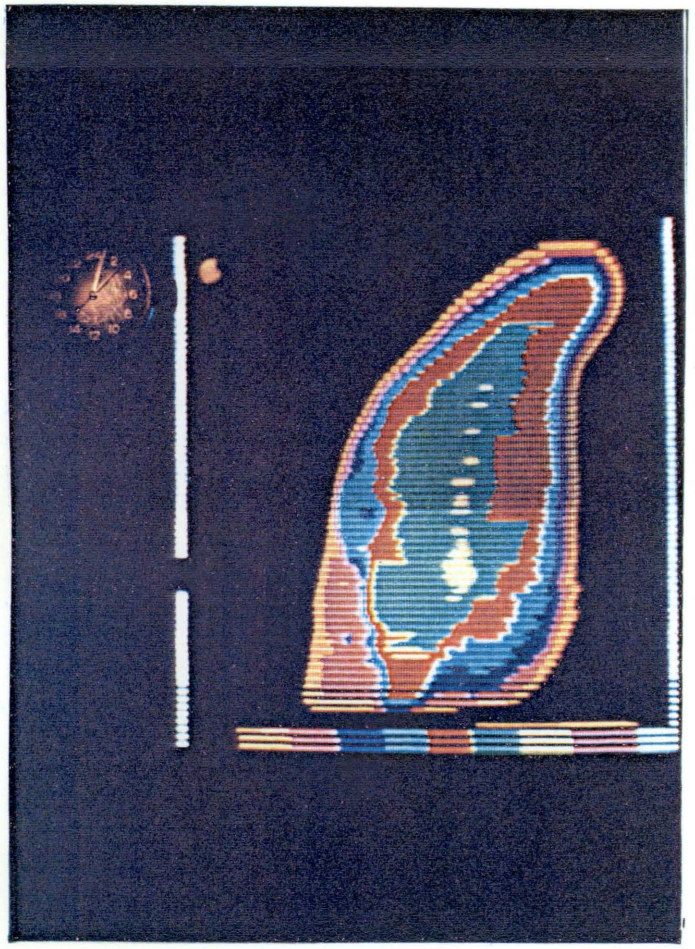


Time: $t = t_o$

Experiment 2

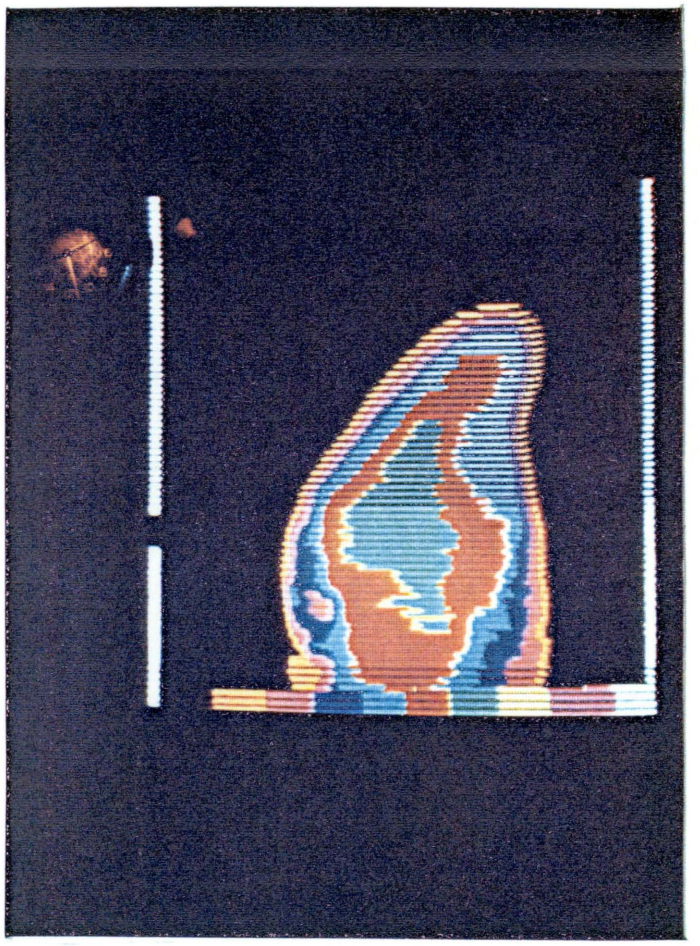


Time: $t = t_o + 18$ min.

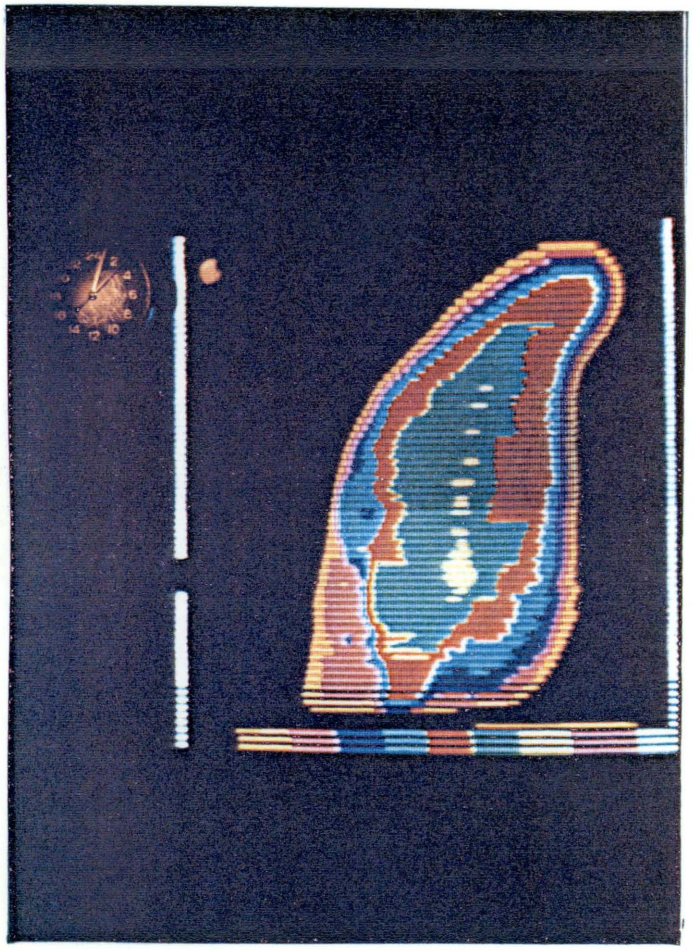


Time: $t = t_o$

Experiment 2

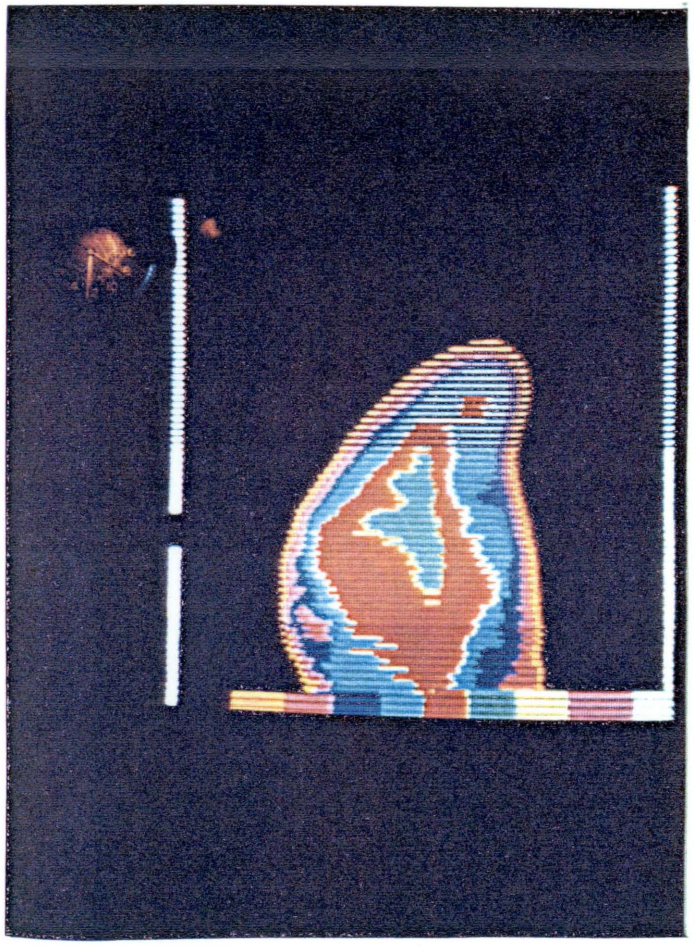


Time: $t = t_o + 20$ min.

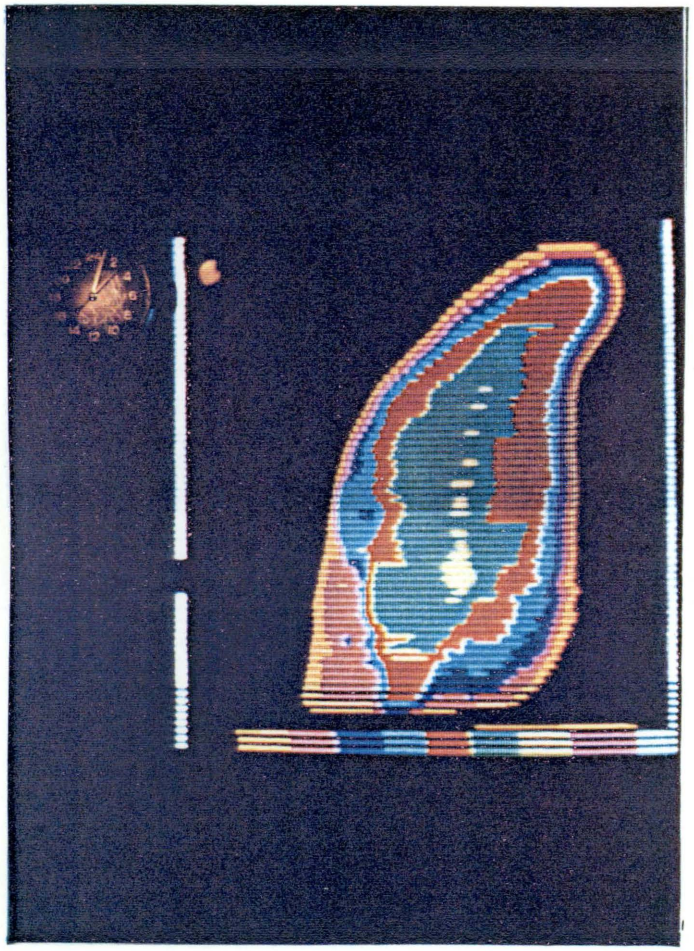


Time: $t = t_o$

Experiment 2

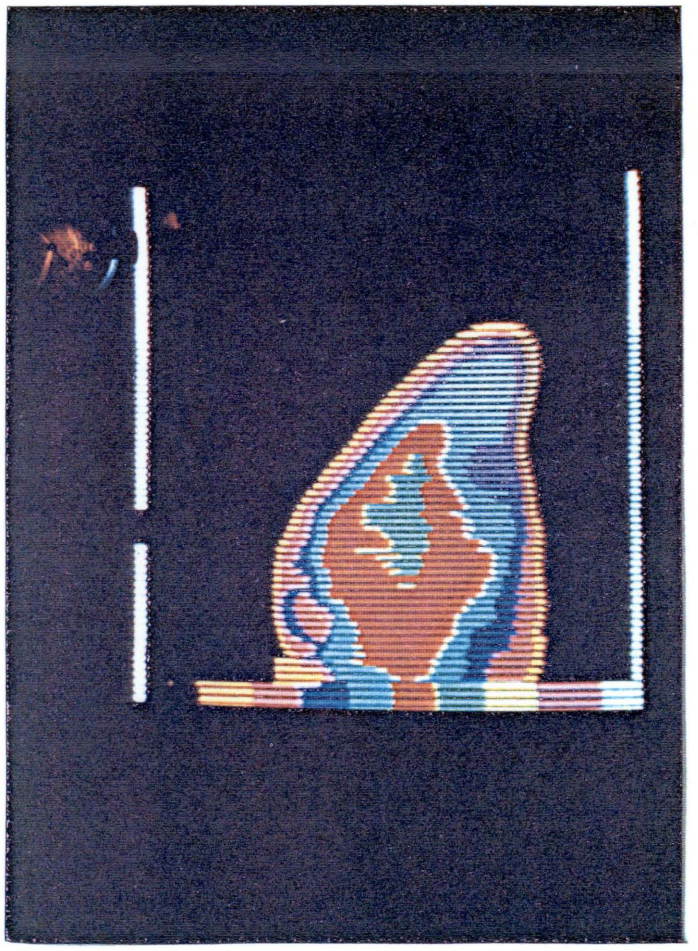


Time: $t = t_o + 22$ min.

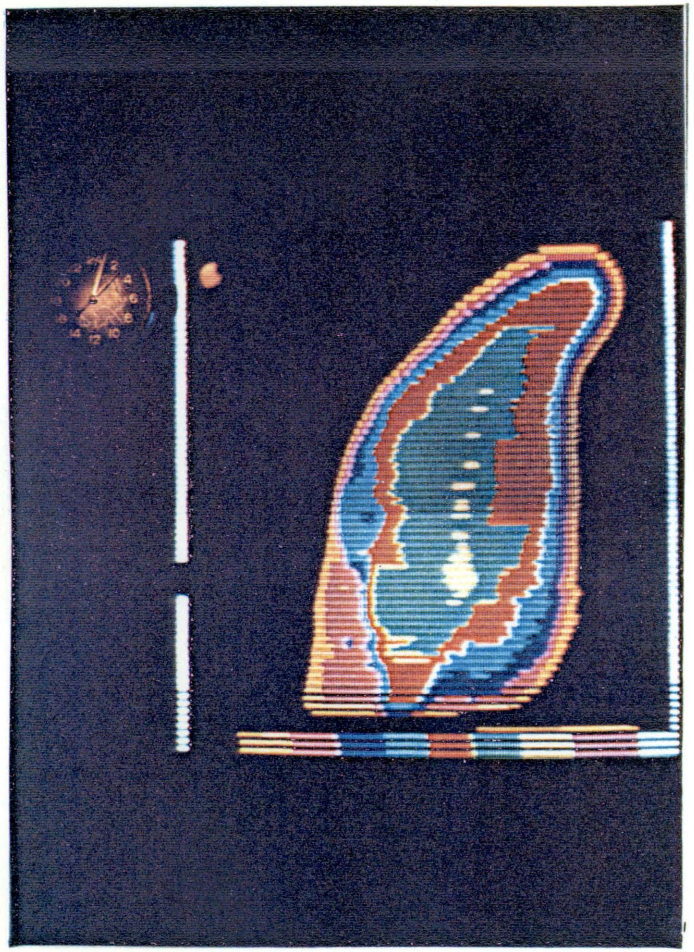


Time: $t = t_o$

Experiment 2

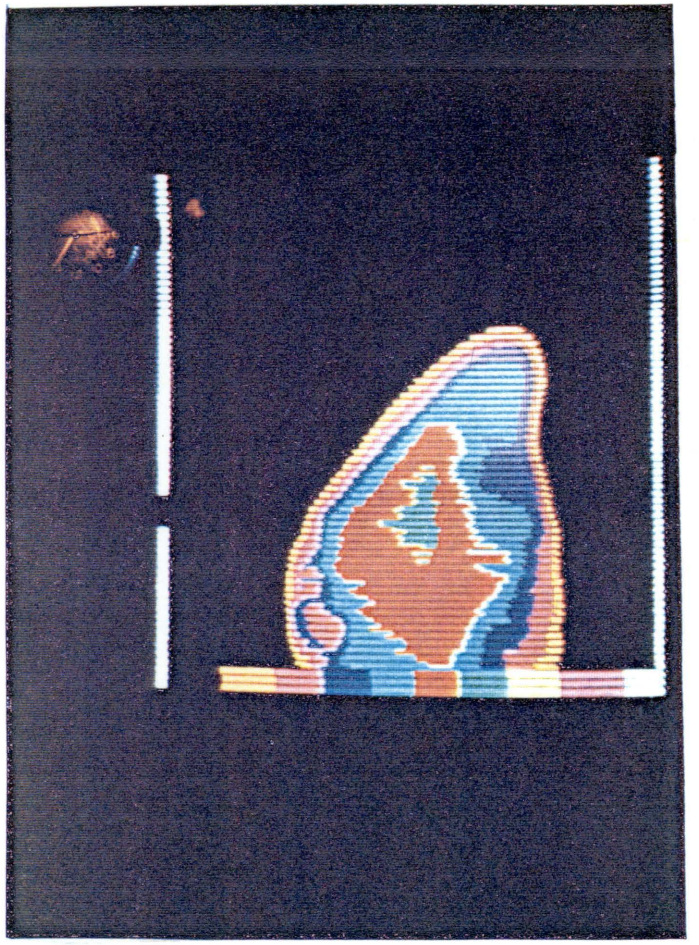


Time: $t = t_o + 24$ min.

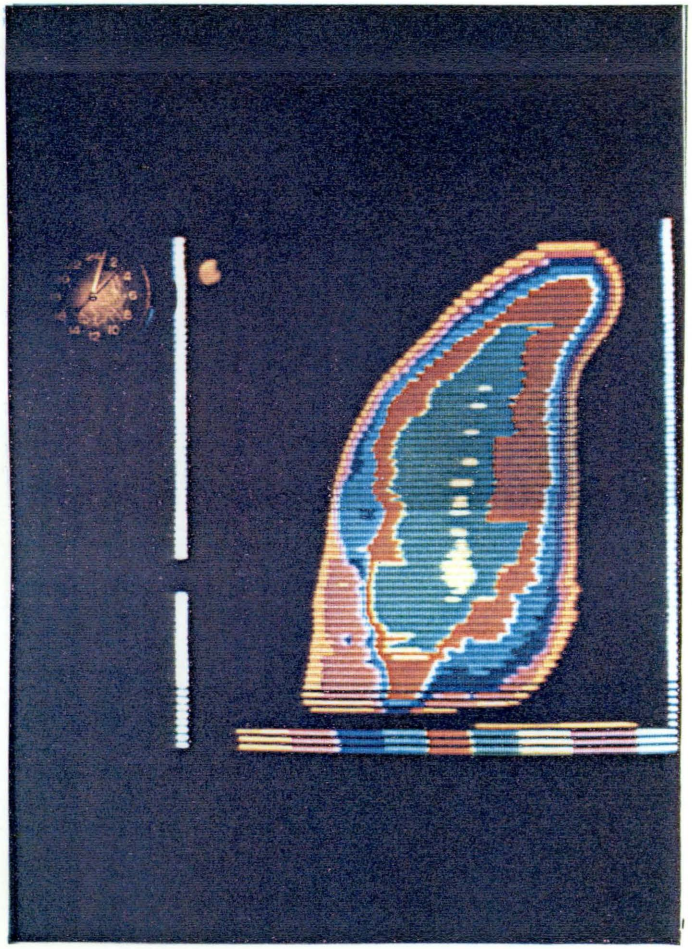


Time: $t = t_o$

Experiment 2

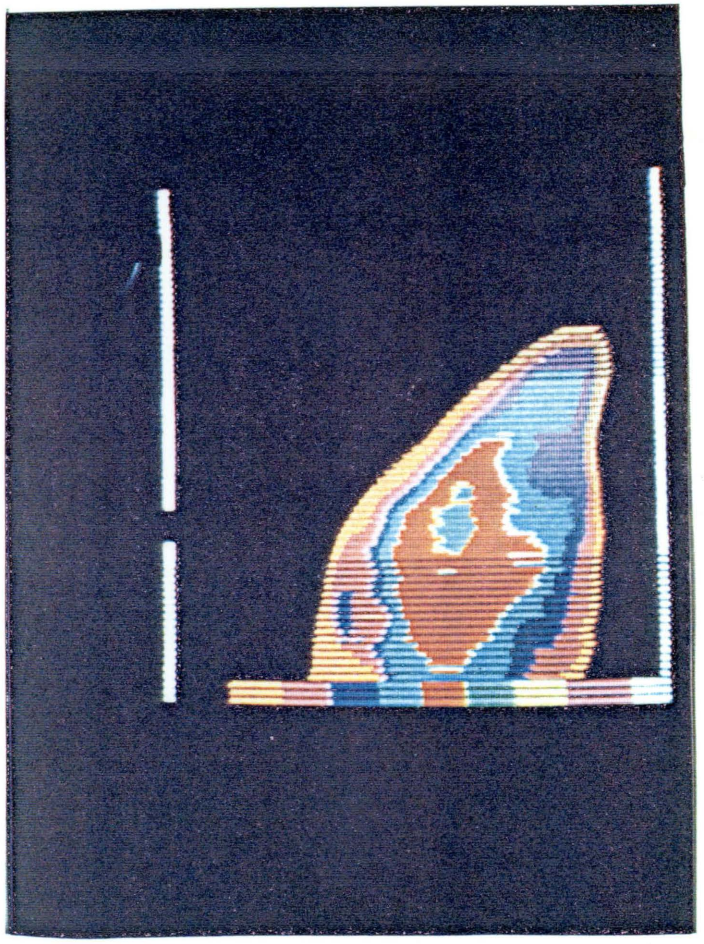


Time: $t = t_o + 26$ min.

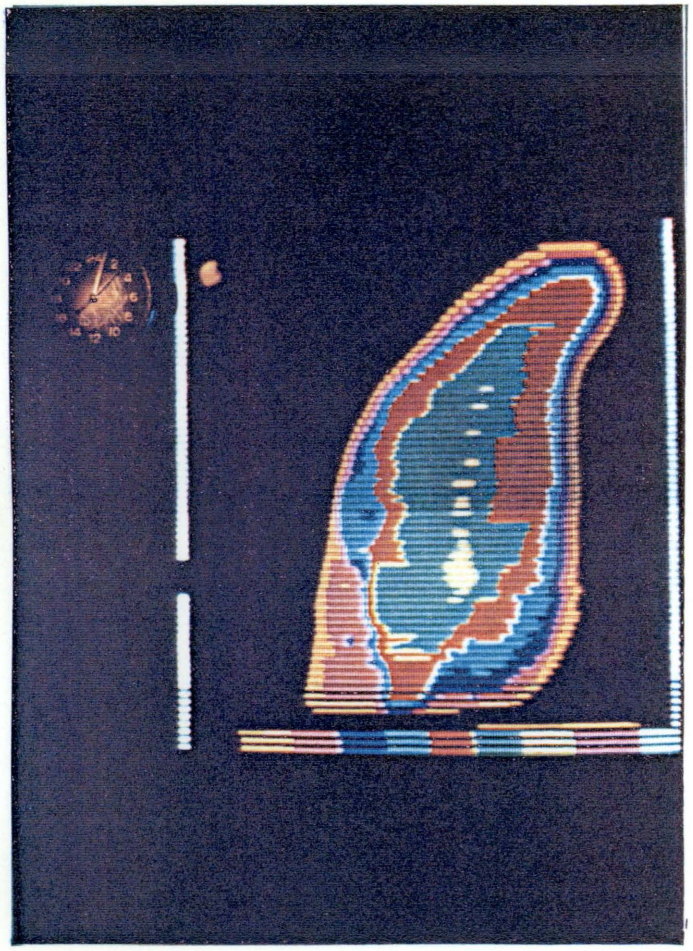


Time: $t = t_o$

Experiment 2

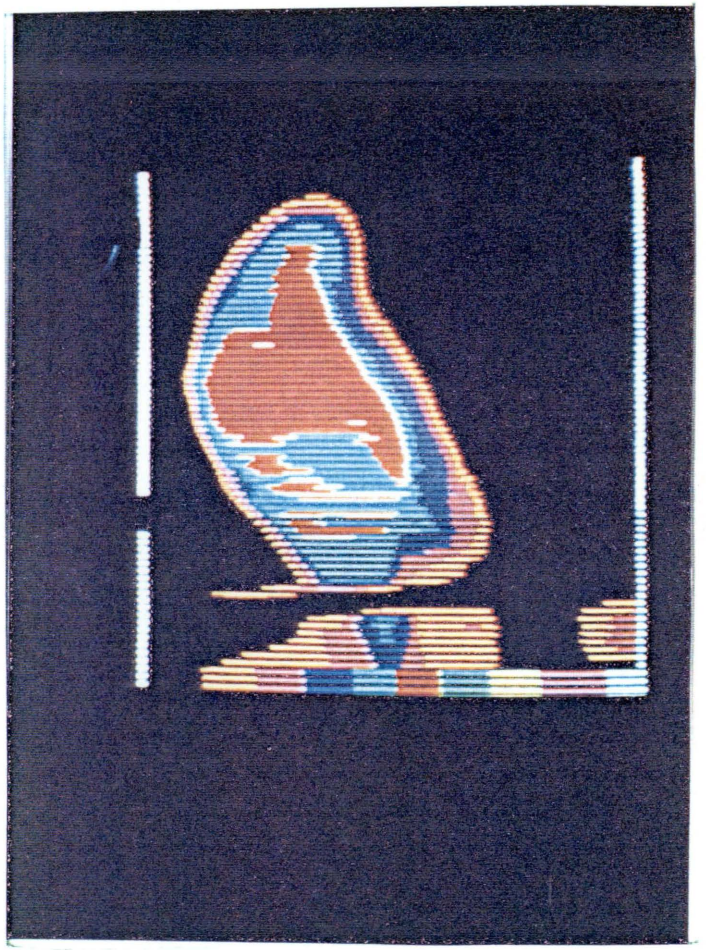


Time: $t = t_o + 28$ min.

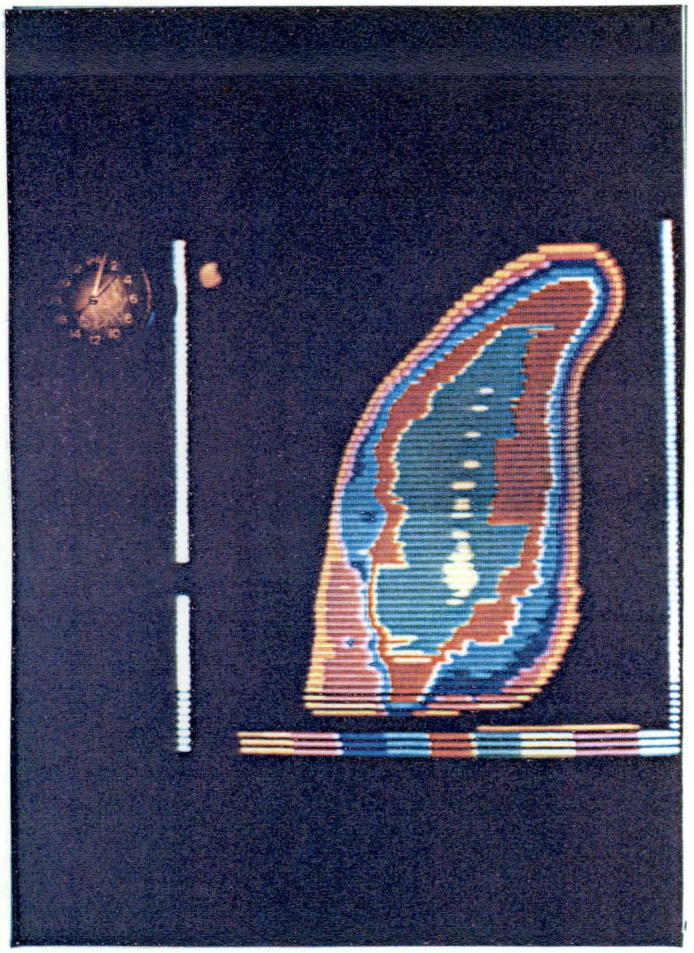


Time: $t = t_o$

Experiment 2

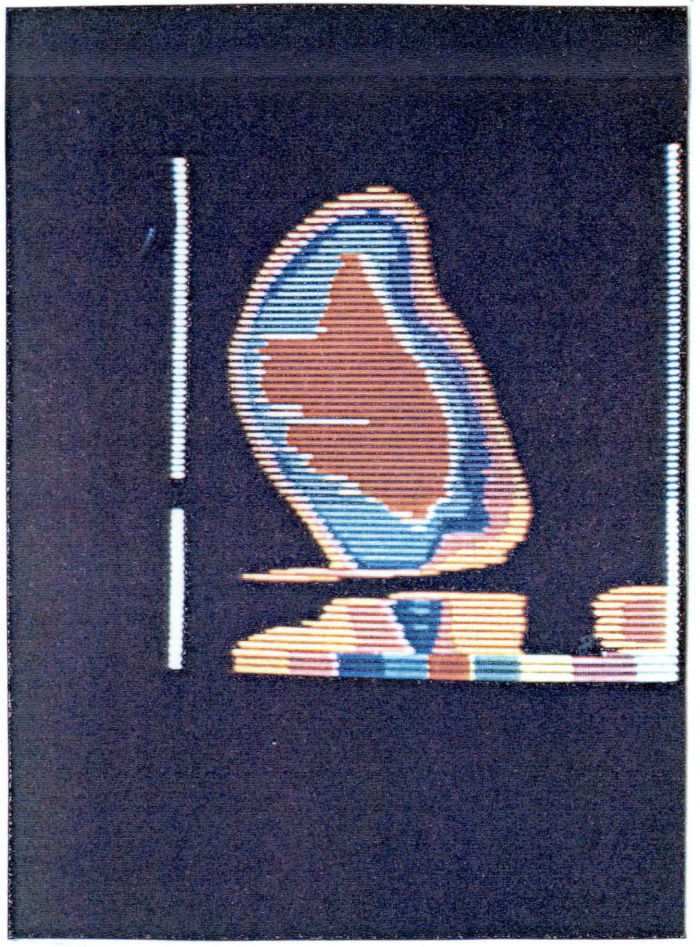


Time: $t = t_o + 30$ min.

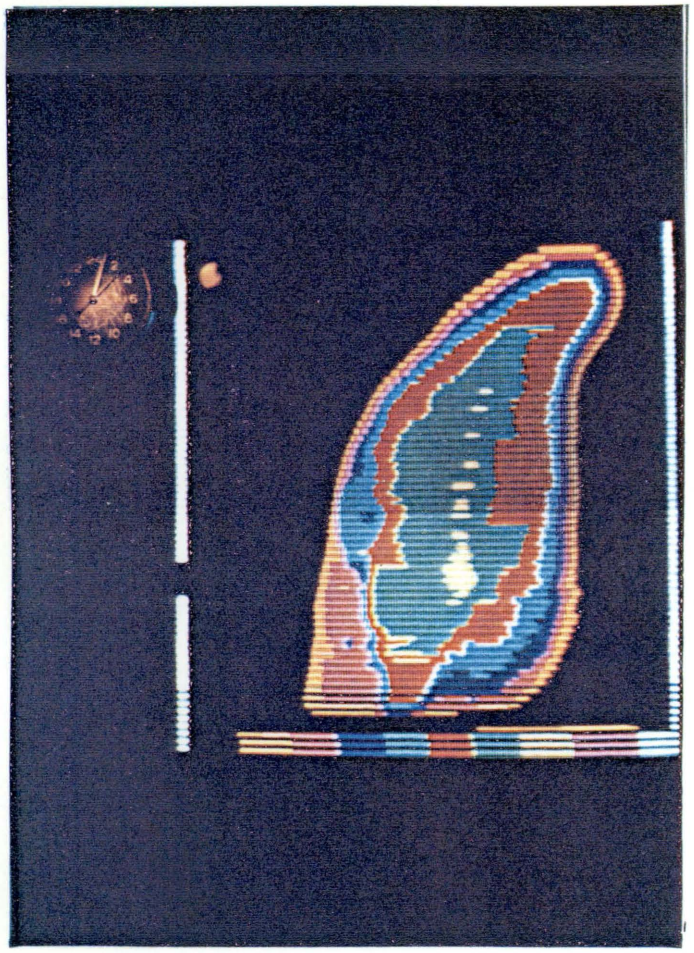


Time: $t = t_o$

Experiment 2

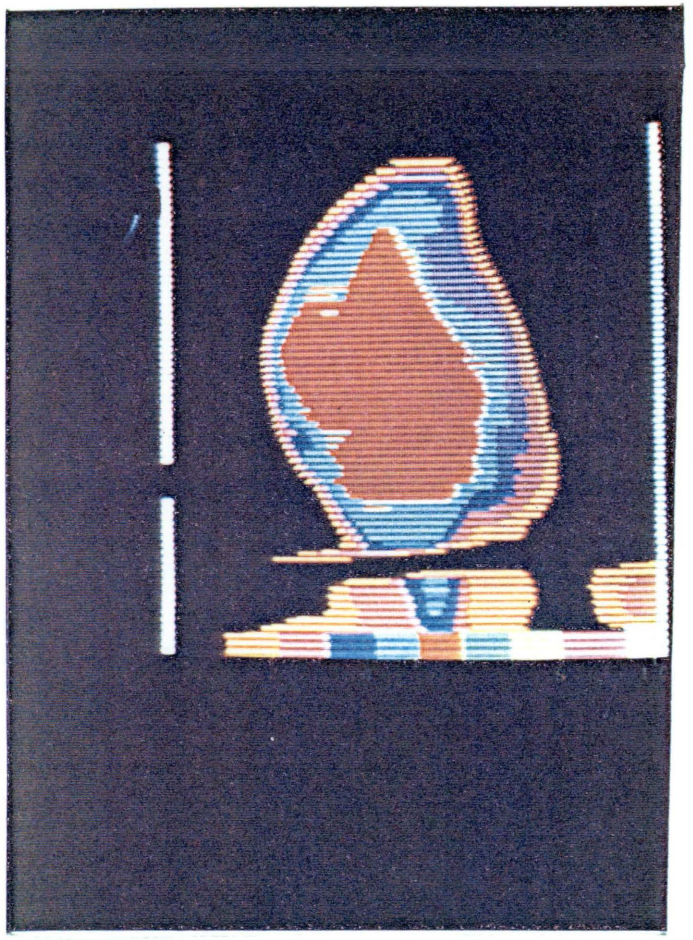


Time: $t = t_o + 32$ min.

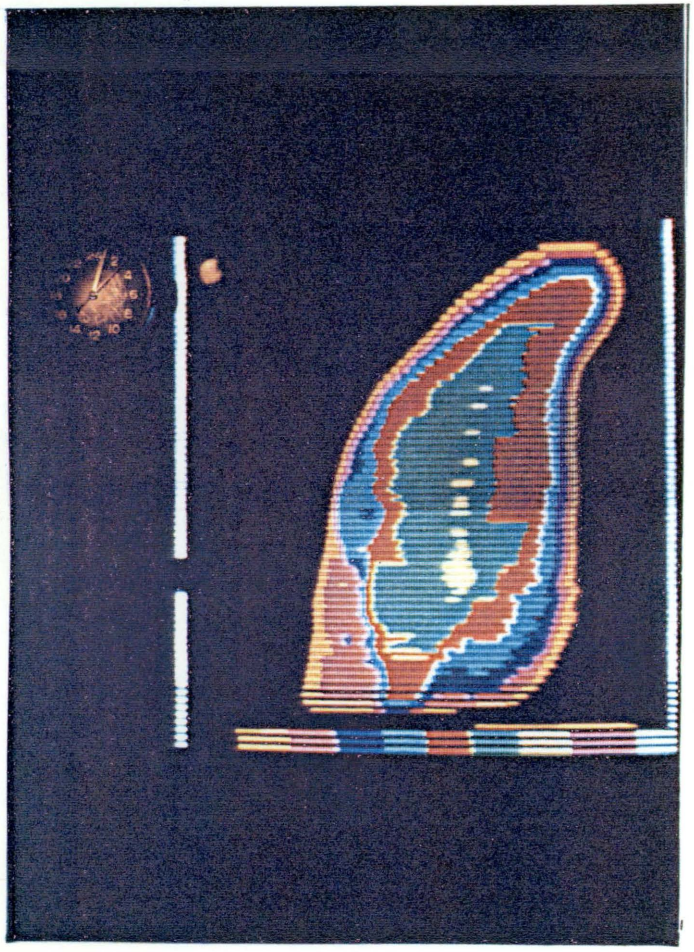


Time: $t = t_o$

Experiment 2

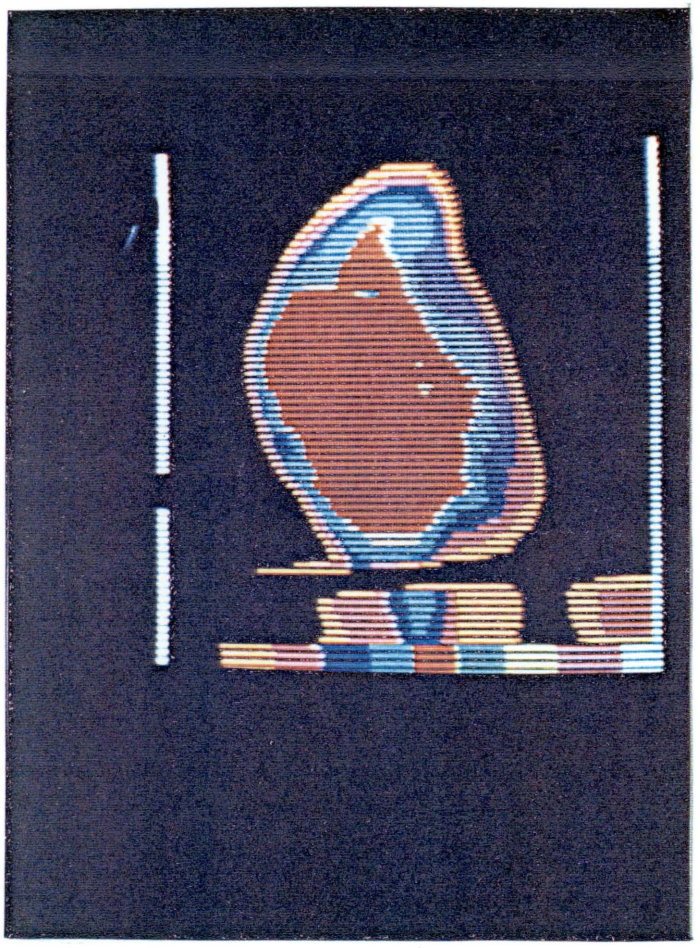


Time: $t = t_o + 34$ min.

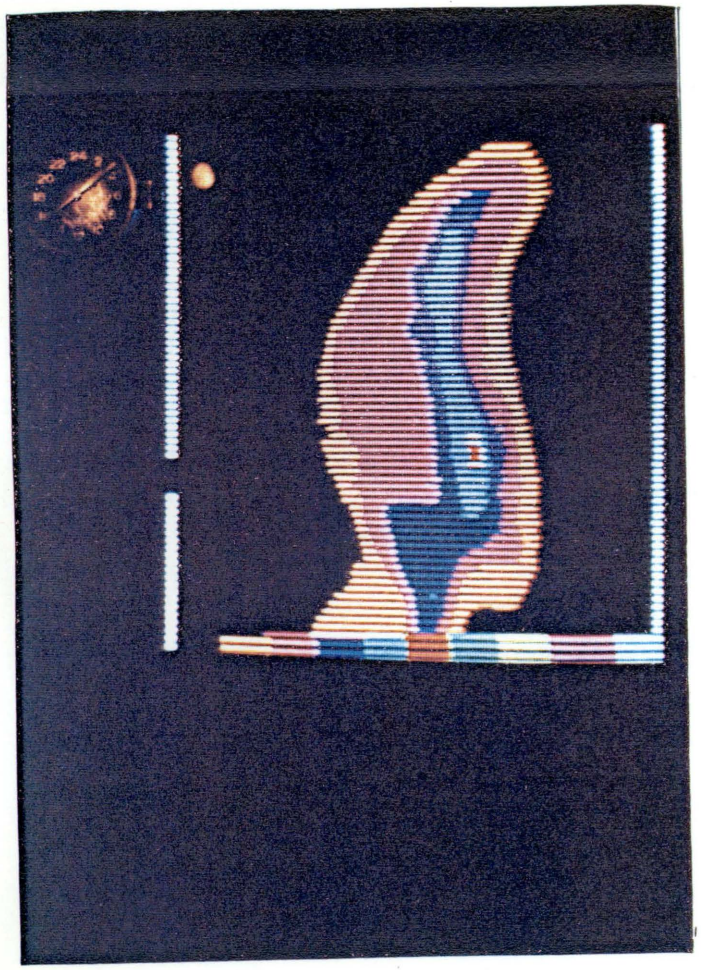


Time: $t = t_o$

Experiment 2

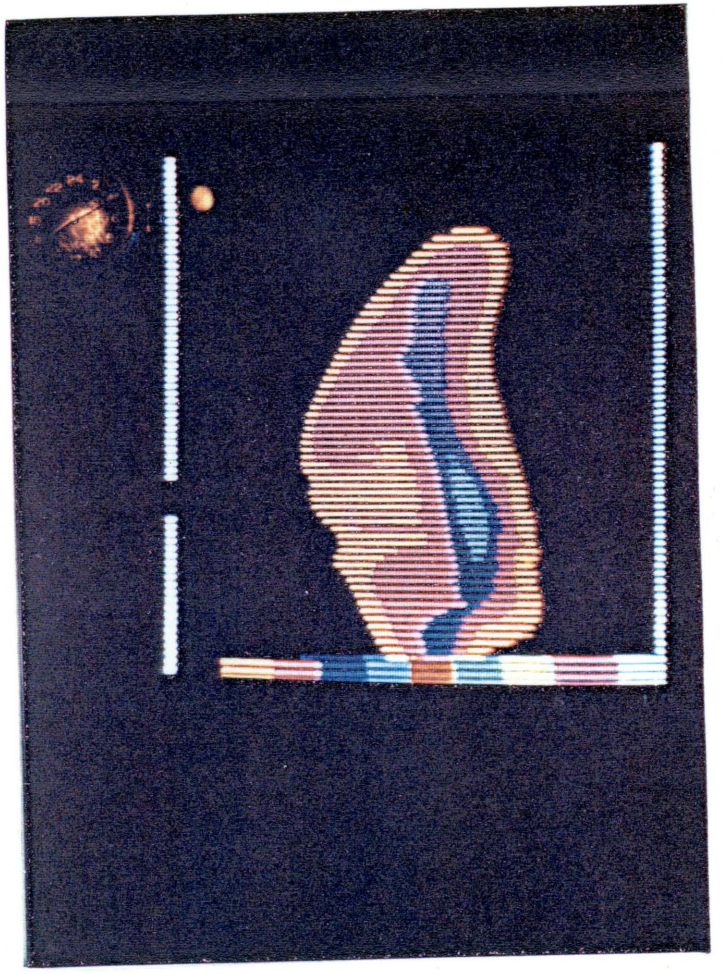


Time: $t = t_o + 36$ min.

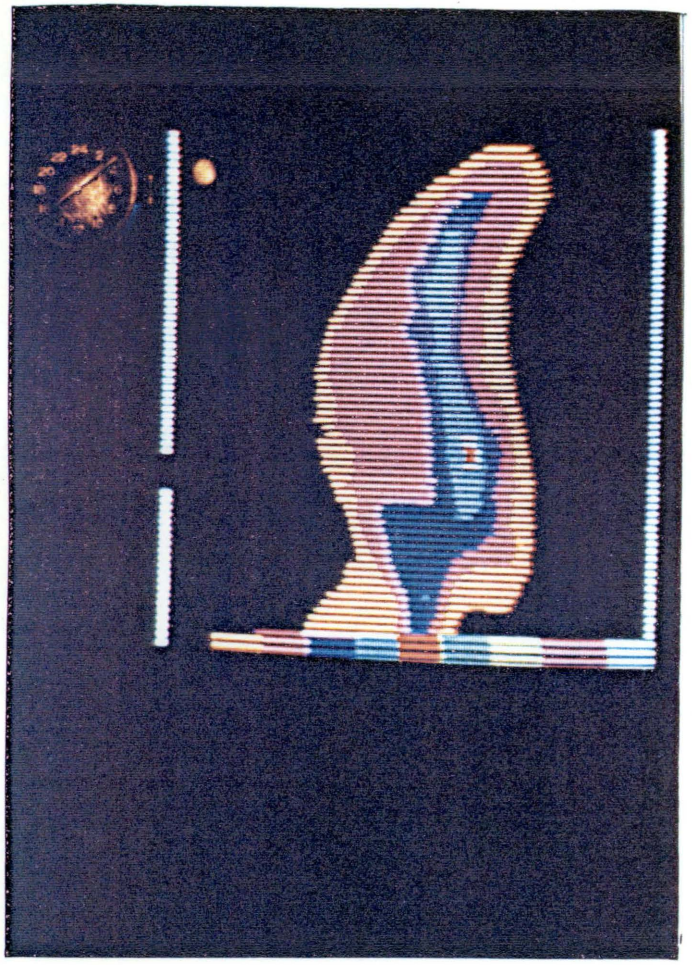


Time: $t = t_o$

Experiment 3

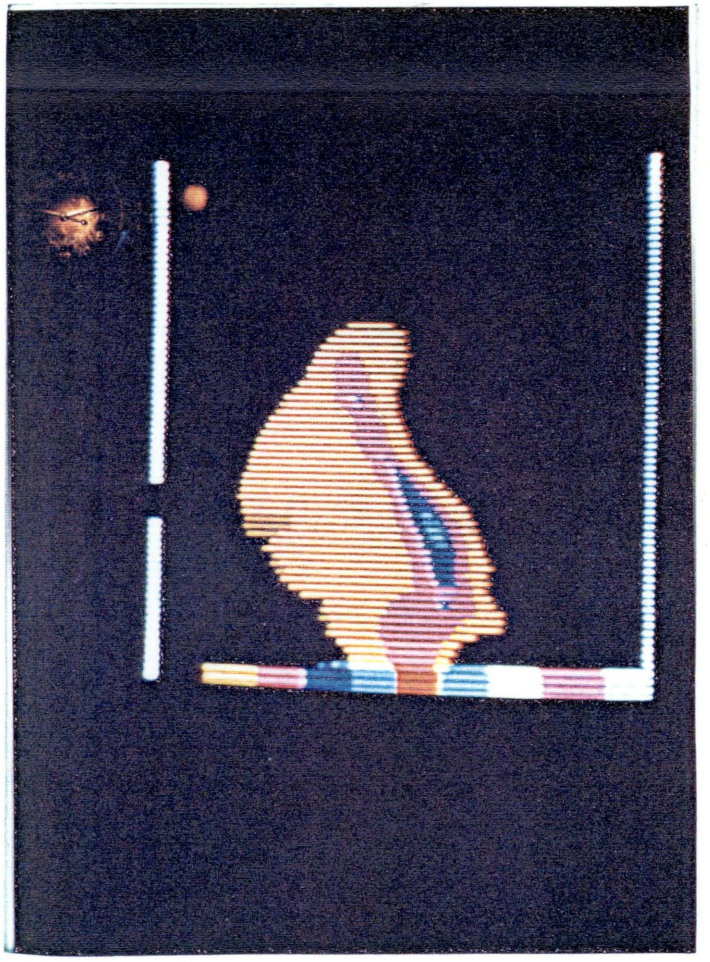


Time: $t = t_o + 2$ min.

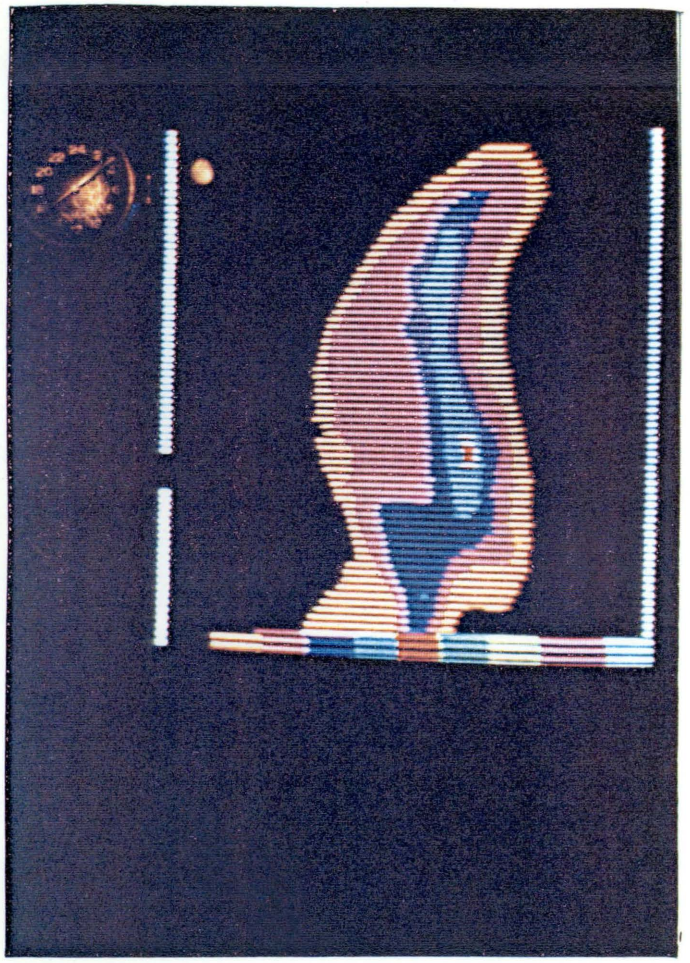


Time: $t = t_o$

Experiment 3

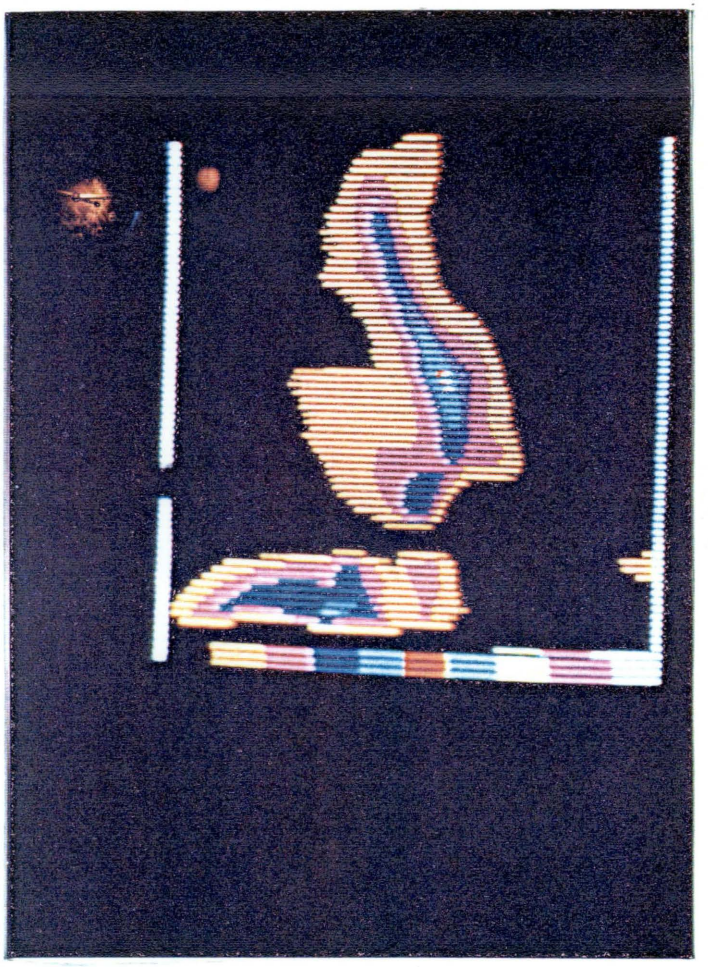


Time: $t = t_o + 4$ min.

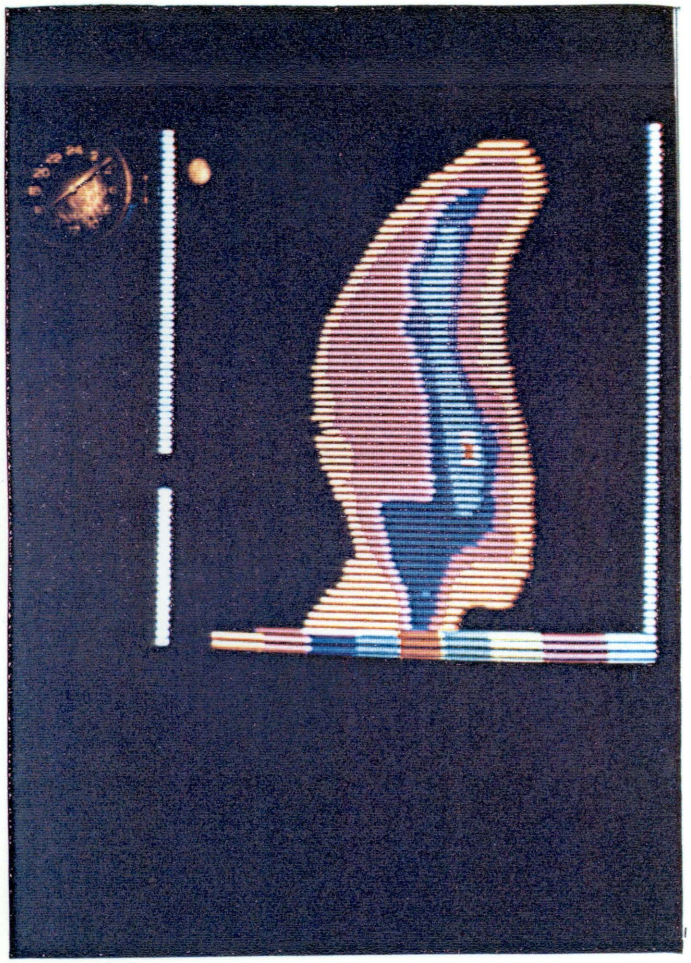


Time: $t = t_o$

Experiment 3

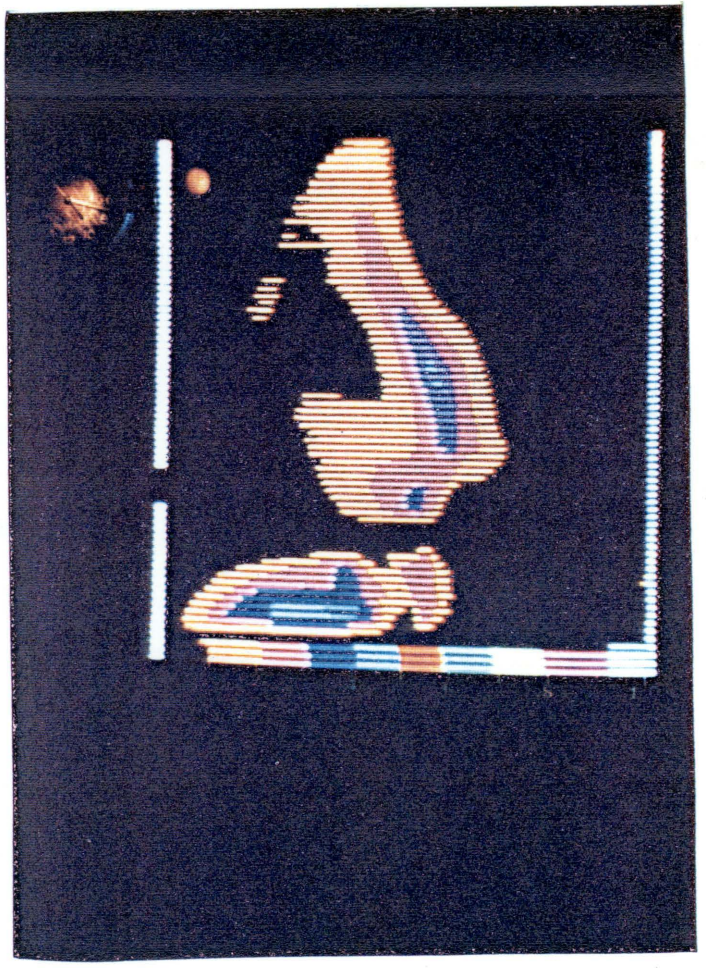


Time: $t = t_o + 6$ min.

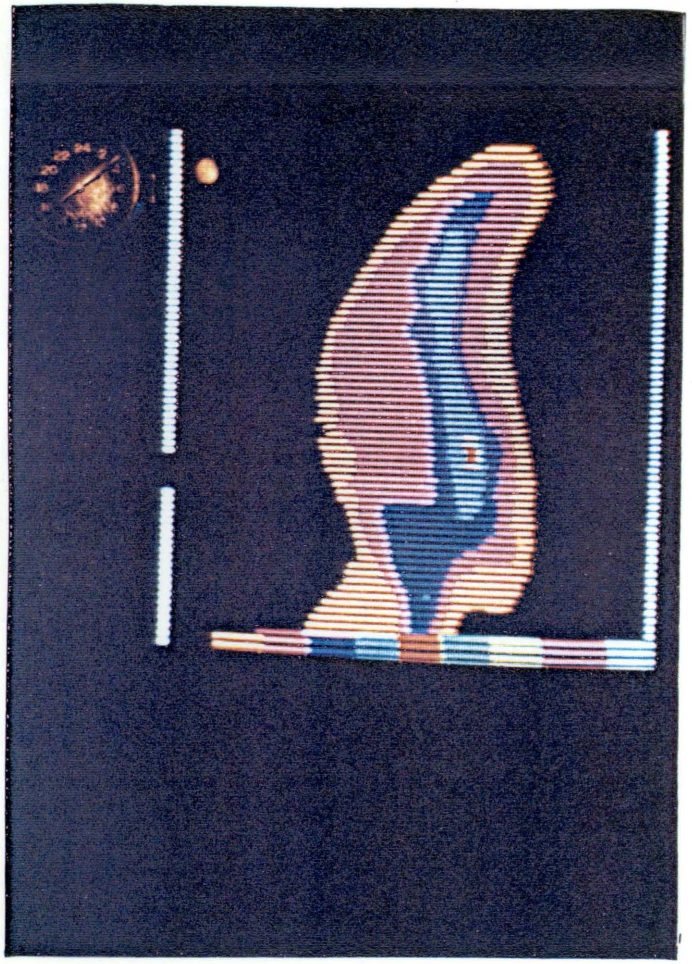


Time: $t = t_o$

Experiment 3

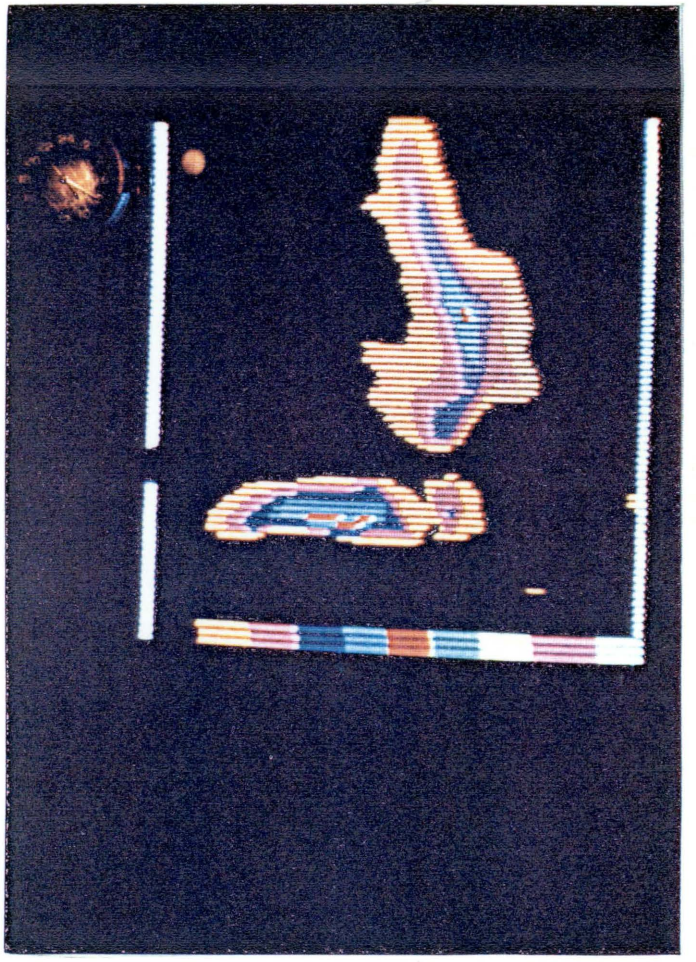


Time: $t = t_o + 8$ min.

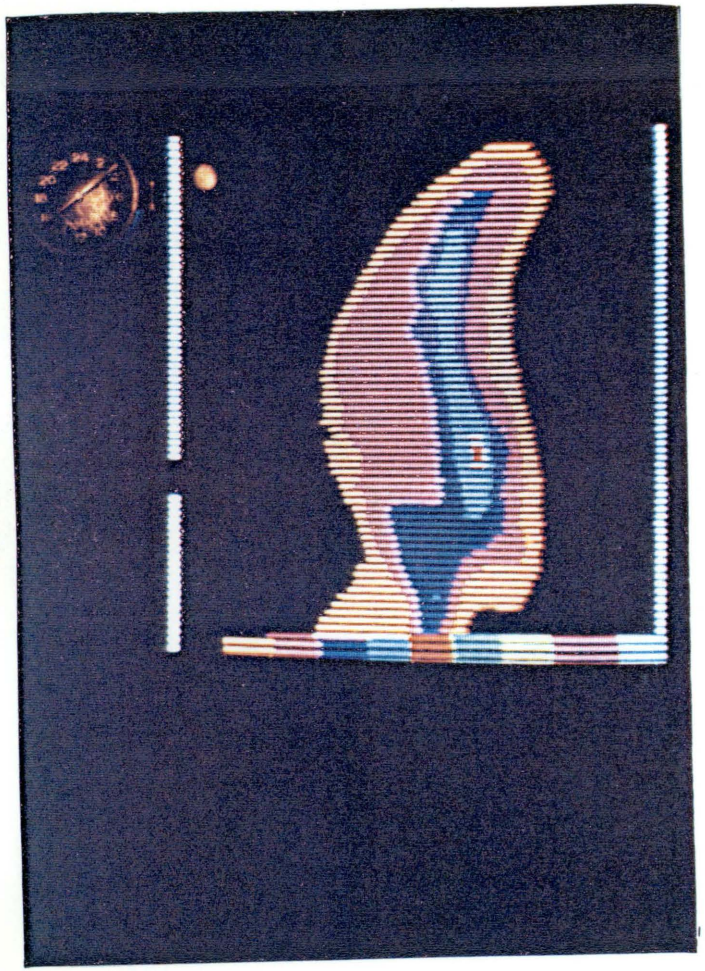


Time: $t = t_o$

Experiment 3

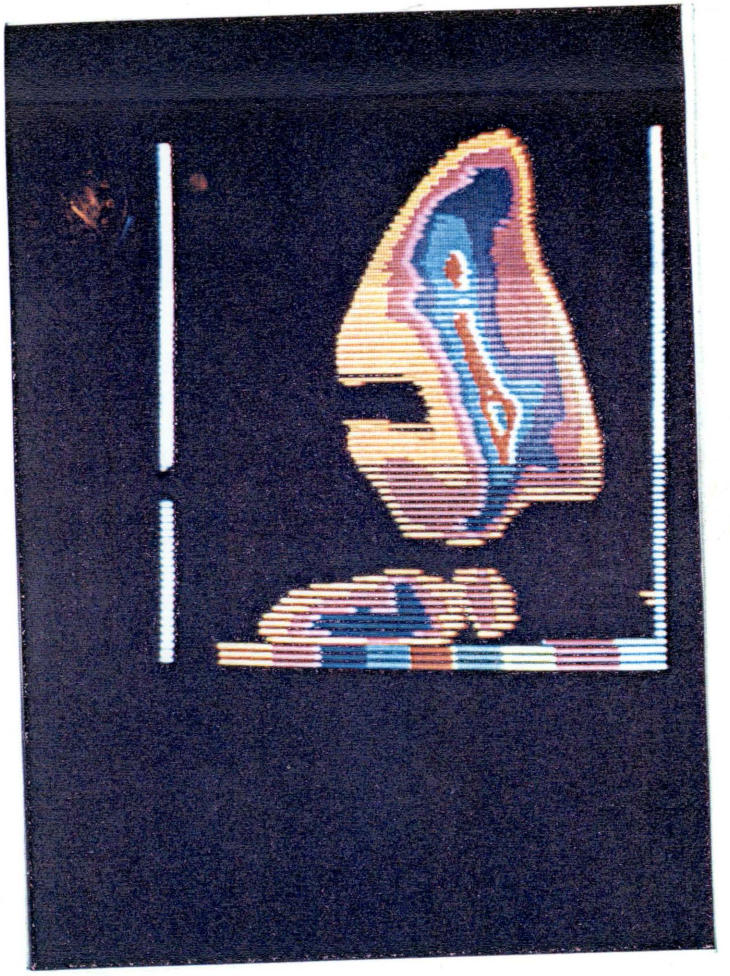


Time: $t = t_o + 11$ min.

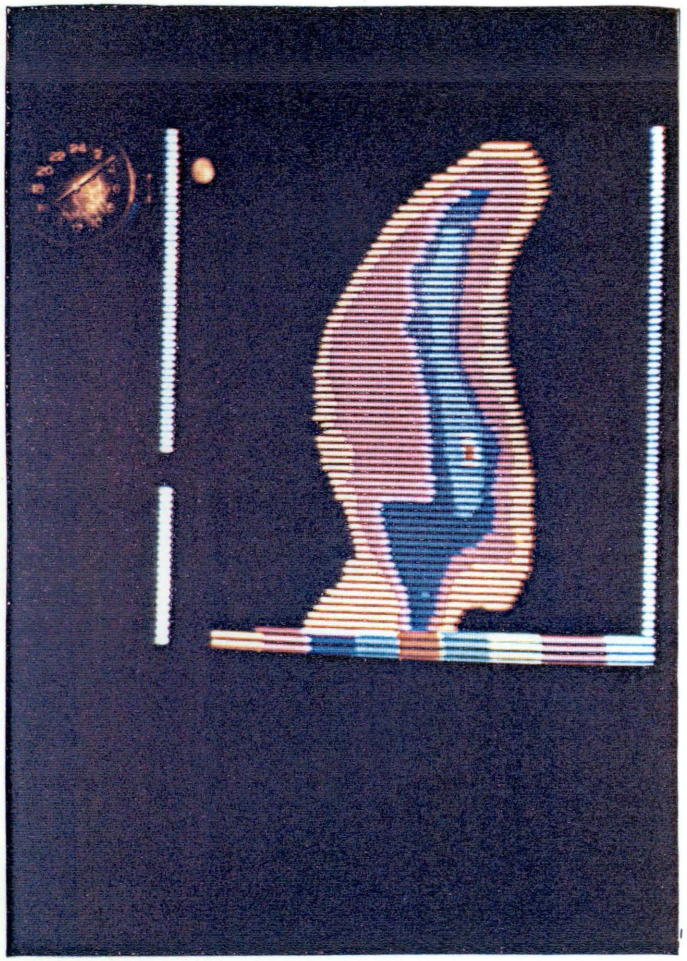


Time: $t = t_o$

Experiment 3

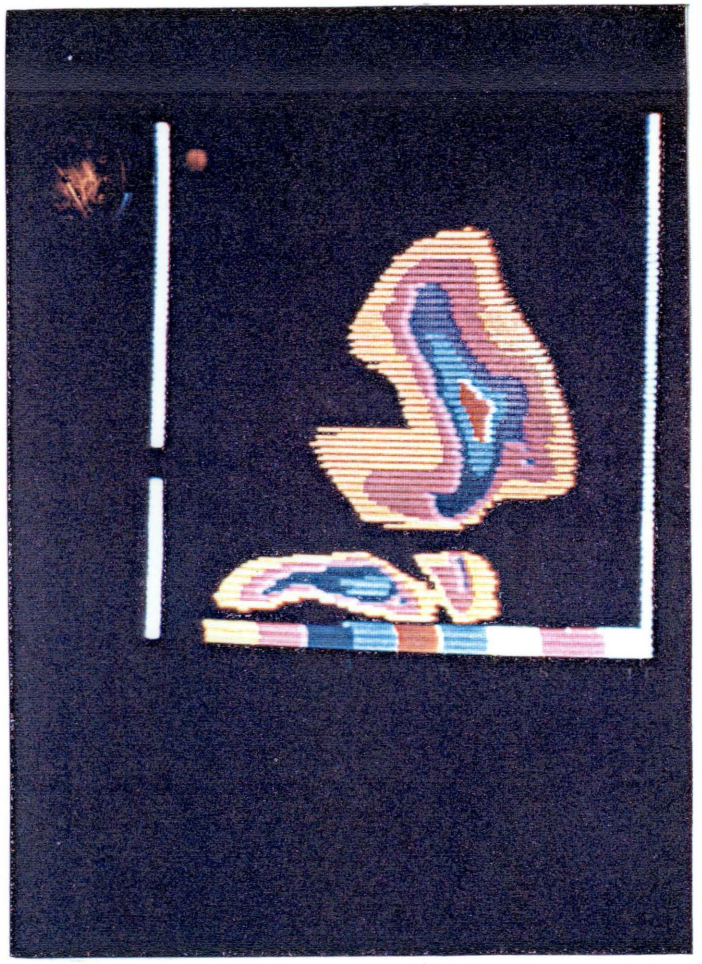


Time: $t = t_o + 13$ min.

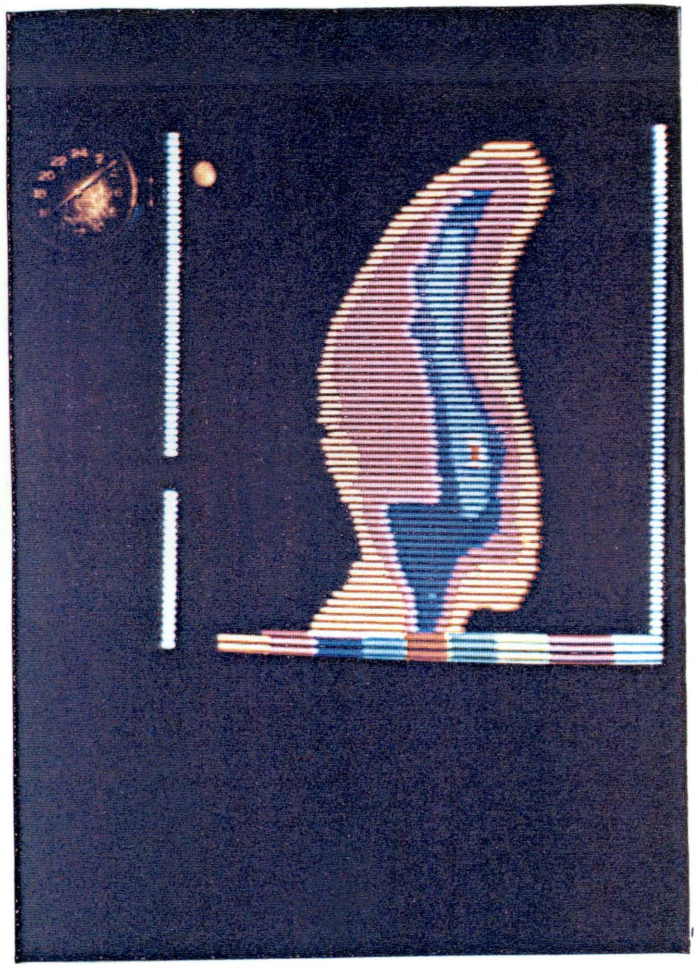


Time: $t = t_o$

Experiment 3

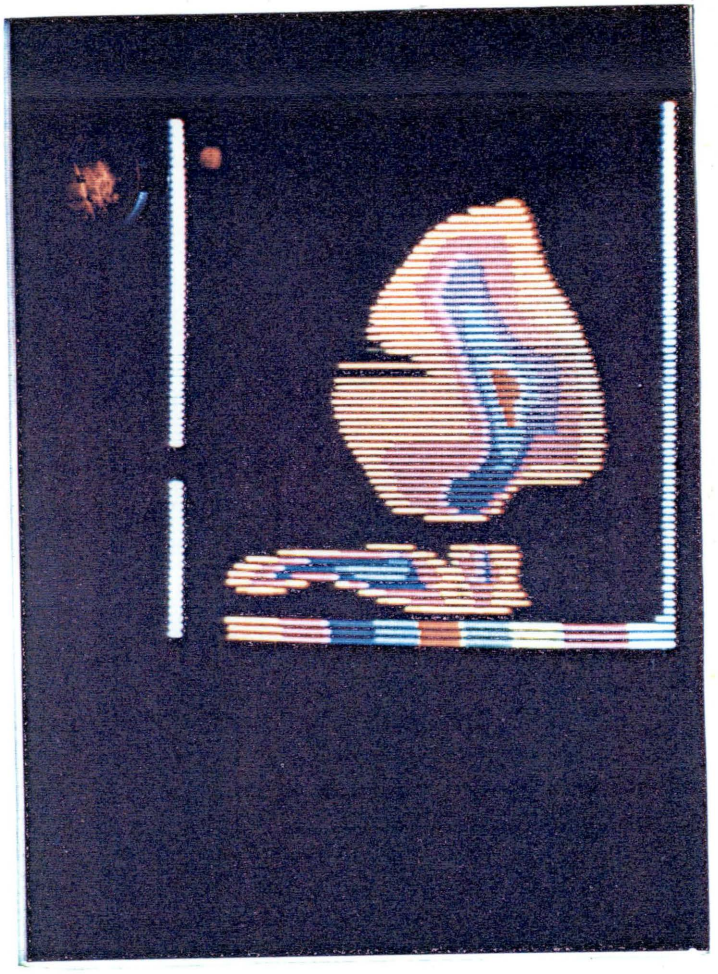


Time: $t = t_o + 15$ min.

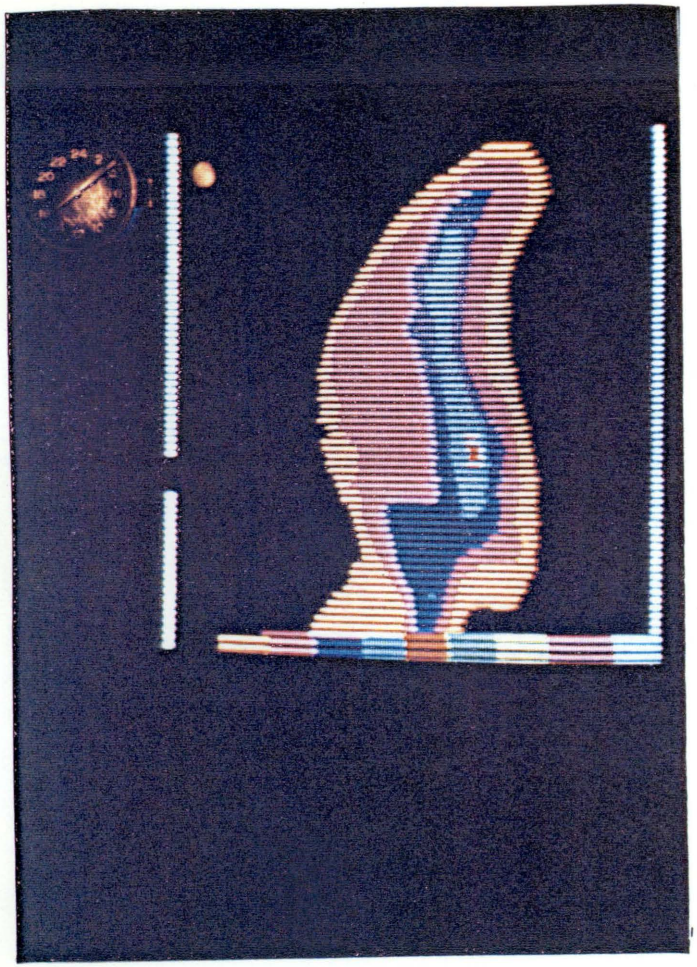


Time: $t = t_o$

Experiment 3

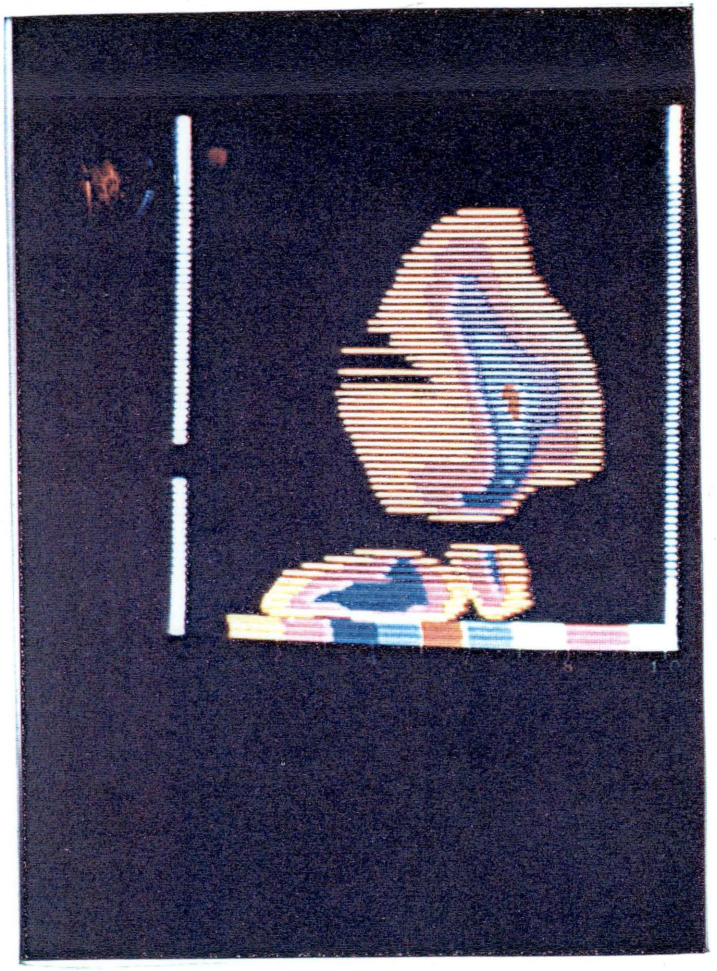


Time: $t = t_o + 17$ min.

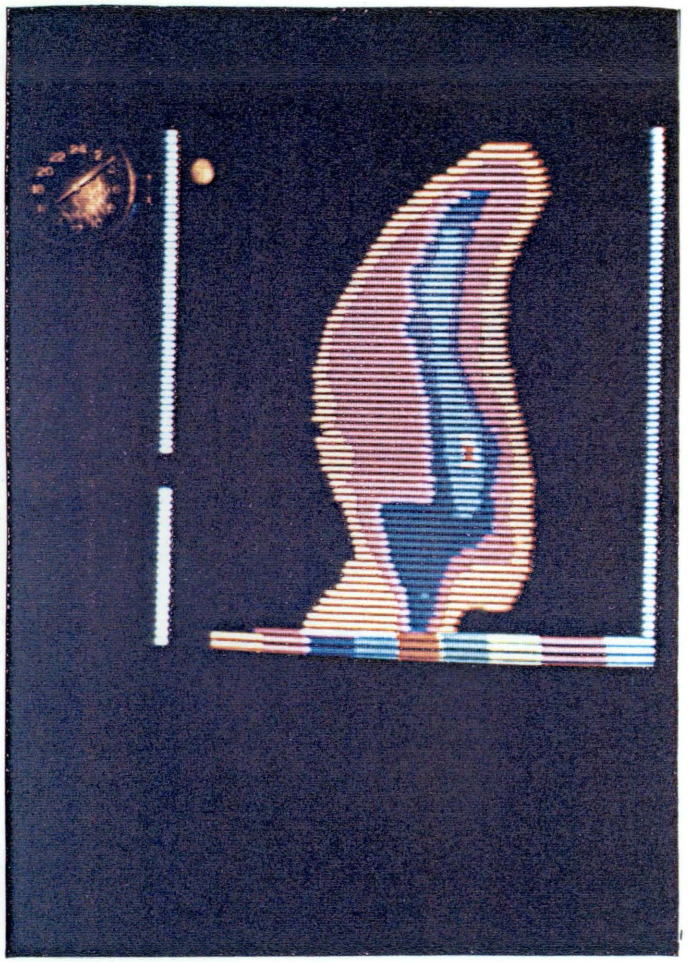


Time: $t = t_o$

Experiment 3

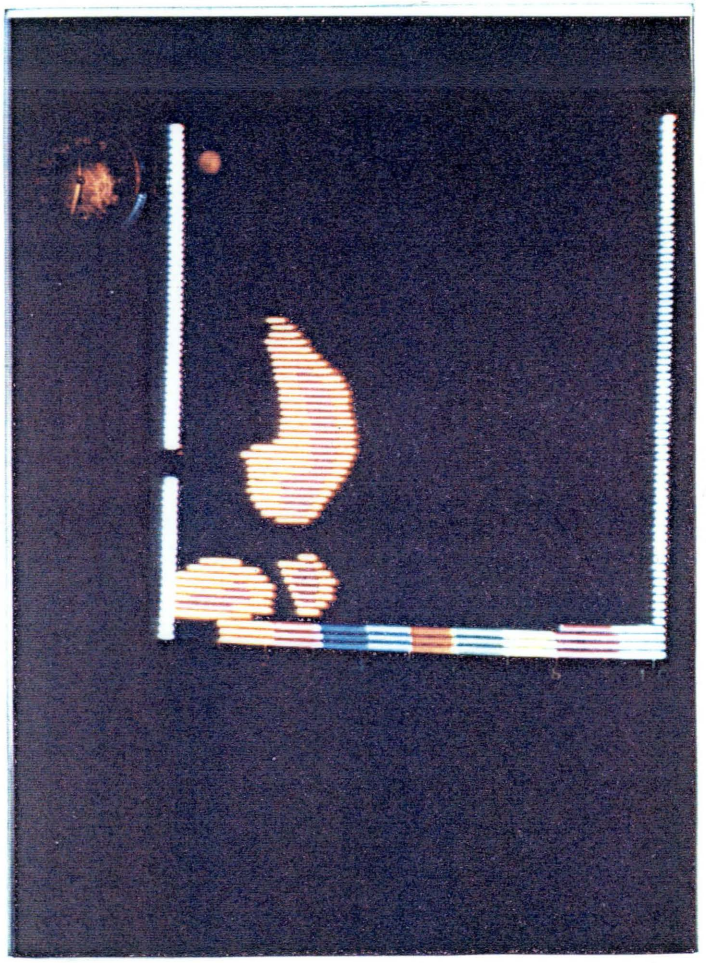


Time: $t = t_o + 19$ min.

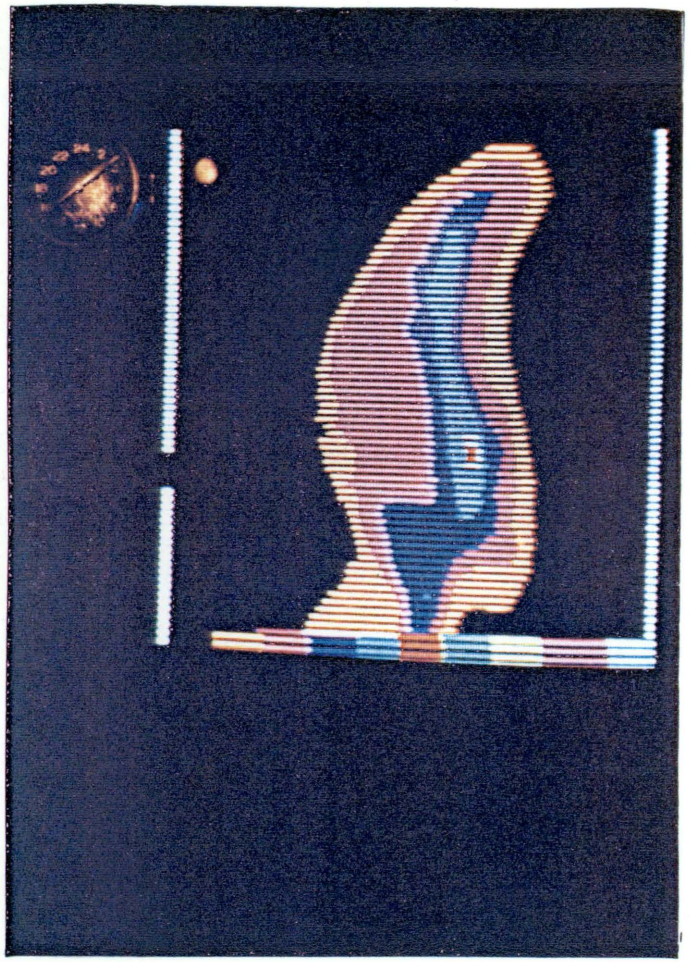


Time: $t = t_o$

Experiment 3

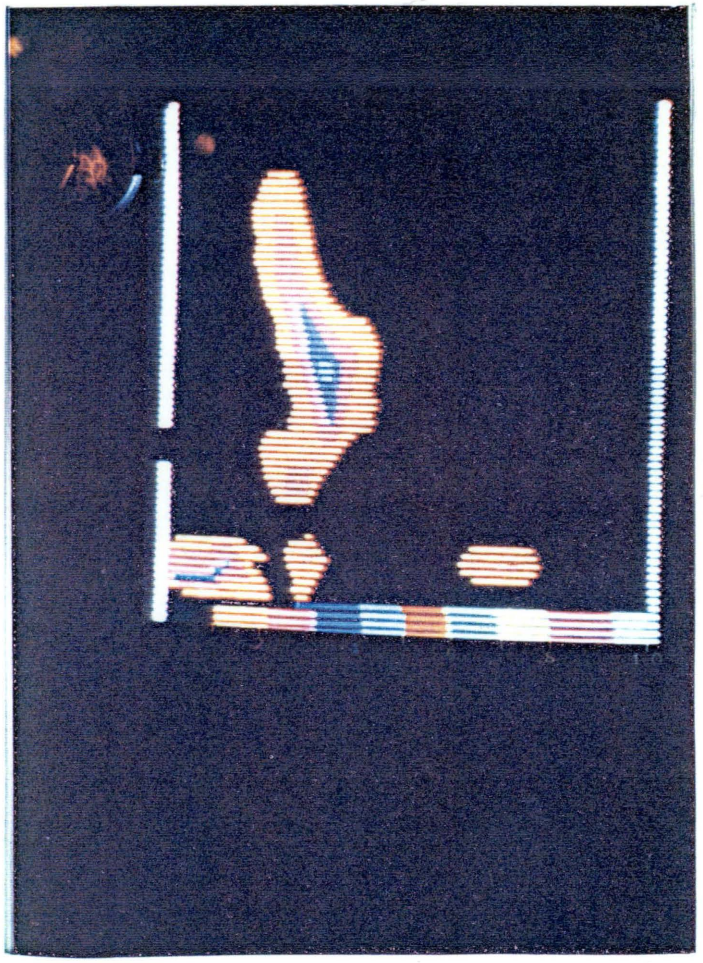


Time: $t = t_o + 22$ min.

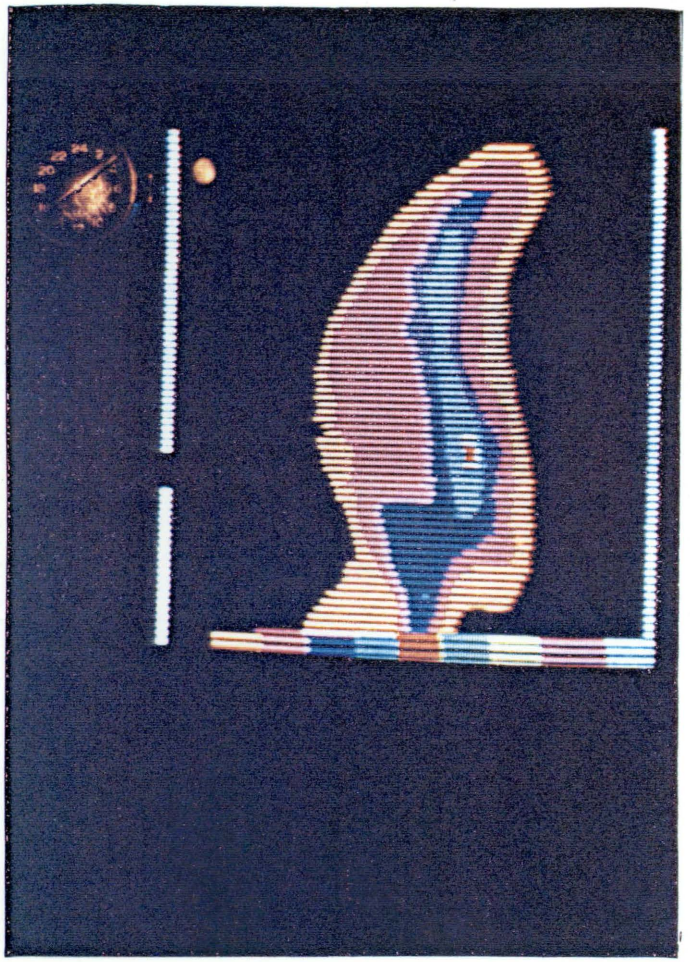


Time: $t = t_o$

Experiment 3

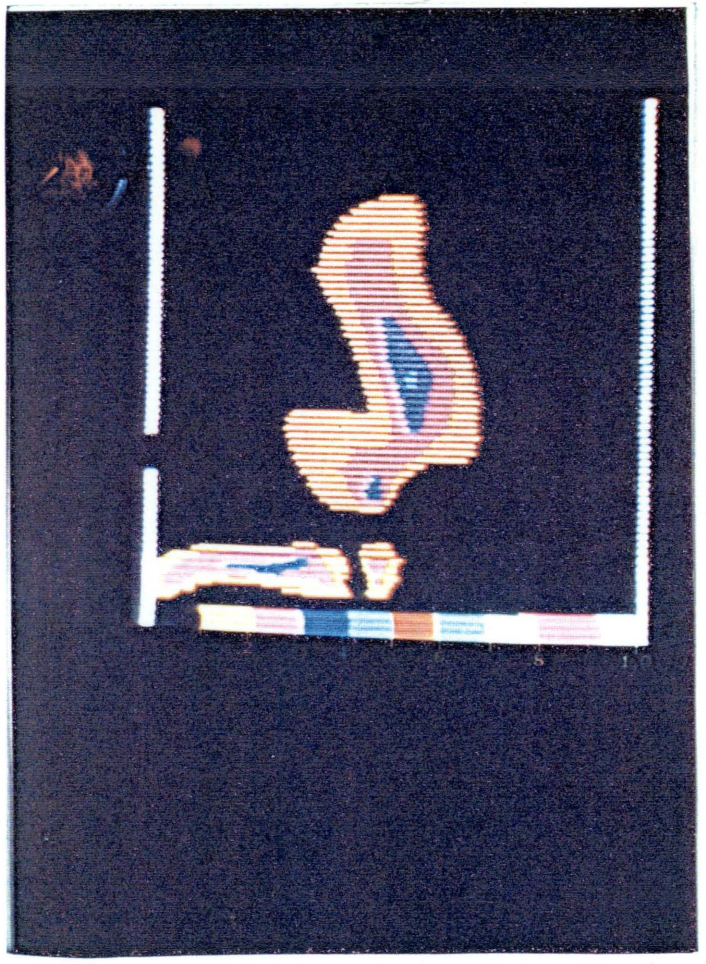


Time: $t = t_o + 24$ min.

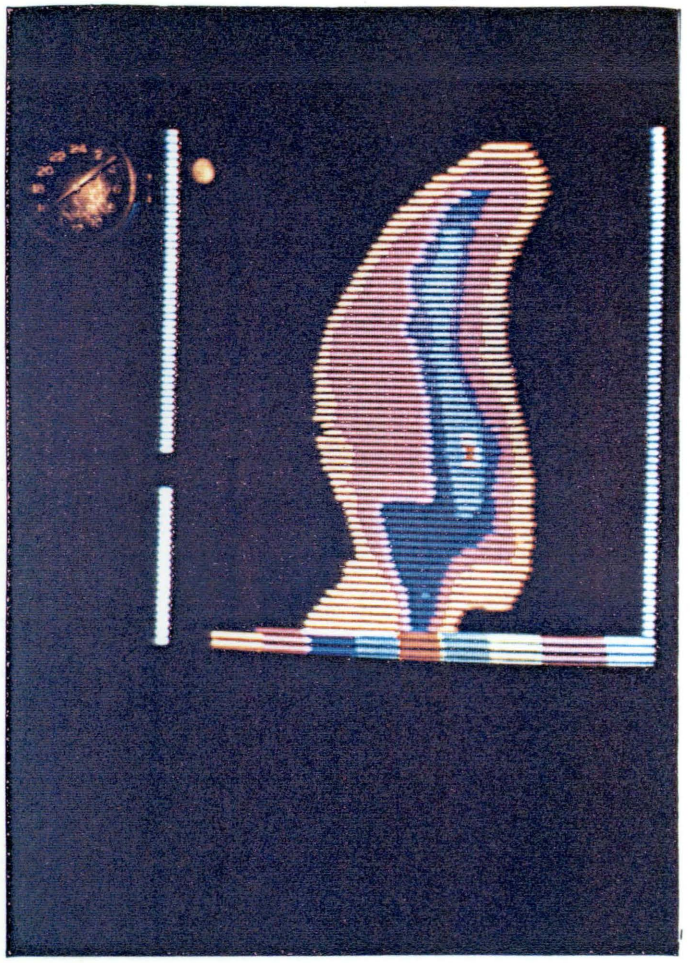


Time: $t = t_o$

Experiment 3

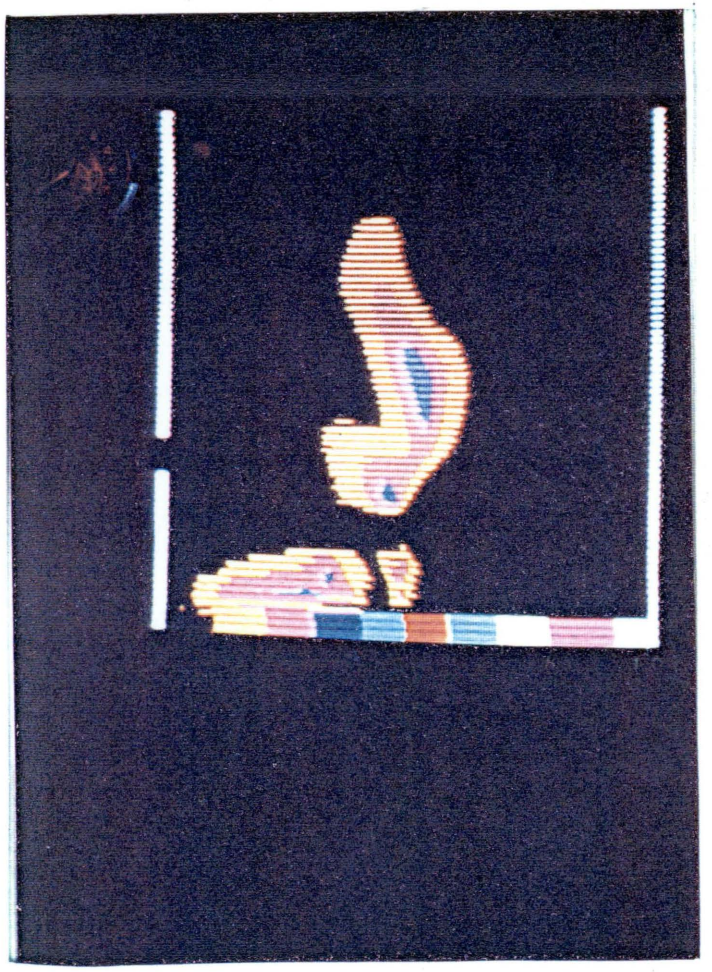


Time: $t = t_o + 26$ min.



Time: $t = t_o$

Experiment 3



Time: $t = t_o + 29$ min.

Table 3

SUMMARY OF TEST 1

Time	Comment	Photograph Page No.	Temperature Decrease from Baseline (°C)	Ozone Concentration (ppm)
1:12 p.m.	Pre-ozonation	52	-	0.08
1:14	Pre-ozonation	52	0	0.09
1:16	Pre-ozonation	53	0	0.06
1:20	Ozone introduction	54	1	0.47
1:22	Ozone introduction	55	1 - 2	0.70
1:24	Ozone introduction	56	2	0.77
1:26	Ozone introduction	57	1 - 2	0.64
1:28	Ozone introduction	58	1	0.61
1:30	Ozone introduction	59	1	0.76
1:33	Post-ozonation	60	1 - 2	0.25
1:35	Post-ozonation	61	1	0.27
1:37	Post-ozonation	62	1	0.22
1:29	Post-ozonation	63	1	0.16
1:41	Post-ozonation	64	1	0.18

Table 3 (continued)

SUMMARY OF TEST 2

Time	Comment	Photograph Page No.	Temperature Decrease from Baseline (°C)	Ozone Concentration (ppm)
5:42 p.m.	Pre-ozonation	65	-	0.15
5:44	Pre-ozonation	65	0	0.16
5:46	Pre-ozonation	66	0	0.14
5:48	Pre-ozonation	67	0	0.15
5:50	Ozone introduction	68	0	0.21
5:52	Ozone introduction	69	1	0.23
5:54	Ozone introduction	70	0	0.35
5:56	Ozone introduction	71	0	0.80
5:58	Ozone introduction	72	0 - 1	0.75
6:00	Ozone introduction	73	0 - 1	0.83
6:02	Ozone introduction	74	0 - 1	0.80
6:04	Ozone introduction	75	1	0.91
6:06	Ozone introduction	76	1	0.99
6:08	Post-ozonation	77	1	0.65
6:10	Post-ozonation	78	1	0.35
6:12	Post-ozonation	79	1	0.27
6:14	Post-ozonation	80	1	0.23
6:16	Post-ozonation	81	1	0.21
6:18	Post-ozonation	82	1	0.19

Table 3 (continued)

SUMMARY OF TEST 3

Time	Comment	Photograph Page No.	Temperature Decrease from Baseline (°C)	Ozone Concentration (ppm)
7:55 a.m.	Pre-ozonation	83	-	0.11
7:57	Pre-ozonation	83	0	0.11
7:59	Pre-ozonation	84	1	0.21
8:01	Ozone introduction	85	1	0.51
8:03	Ozone introduction	86	1	0.75
8:06	Ozone introduction	87	1	0.72
8:08	Ozone introduction	88	1	0.77
8:10	Ozone introduction	89	1	0.77
8:12	Ozone introduction	90	1	0.72
8:14	Ozone introduction	91	1	0.83
8:17	Ozone introduction	92	2	1.06
8:19	Post-ozonation	93	2	0.98
8:21	Post-ozonation	94	2	0.49
8:24	Post-ozonation	95	2	0.35

V. DISCUSSION OF RESULTS

Prior to the introduction of ozone in the first test, the predominant temperature of the rabbit's ear corresponded to the color violet (as seen in photographs 1 - 3). Two minutes after ozone exposure began photograph number 4 was taken. The ozone concentration at this point was near 0.4 ppm. This picture revealed a predominance of light yellow. This indicates a temperature reduction of 1° C, since light yellow is one unit to the left of violet on the color scale. Thermograph number 5 was virtually identical to 4 with slightly more light green. Thermograph 6 revealed even more light green coloration, indicative of another decrease. The pattern of light yellow characterized in photograph 5 was prevalent again in pictures 7 through 9. Picture 10 showed more light green encroachment and numbers 11 through 14, taken after the ozone generator was switched off, revealed the basic light yellow pattern again.

Approximately 3 hours and 40 minutes after the termination of test 1, the second experimental exposure began. Pre-exposure thermographs revealed a base color of light green in the middle portion of the ear. The next three pictures, taken before ozone was introduced, showed little variation. In number 6, regions of red occupied areas previously denoted by light green coloration. From this, one could infer a 1° C temperature decrease, but photographs 7 and 8

did not reveal significant changes. Photographs 9, 10 and 11, however, once again show the trend of red replacing light green. Twelve through 15 strongly suggest a 1° C temperature decline and in 16 through 19, red has virtually replaced light green in the middlemost portions. Even though pictures 14 through 19 were taken after the ozone generator was switched off, no evidence of recovery was noted.

An ear temperature corresponding to the color magenta existed in large proportions in the pre-ozonation state of test 3, which began approximately 12 hours and 40 minutes after experiment two's completion. (When comparing one test with another, it should be noted that pre-exposure base temperatures, i.e. colors, were not the same in all cases. Such differences probably reflect such variable parameters as ambient temperature and time of day and should thus behoove caution when making interexperimental comparisons.) Upon the introduction of ozone, zones of yellow began to encroach upon territory previously denoted by a magenta coloration. This action signaled a temperature decrease of 1° C. This general pattern showed little variation through thermograph 10. At this point, an increase in oxygen feed resulted in a jump in ozone concentration from 0.73 ppm for picture 10 to 0.93 for thermograph 11. The color pattern in picture 11 was substantially different from that of 10. Large portions of black occupied areas corresponding to the outer regions of the rabbit's ear. This indicated that the

temperature in those zones was below the range being scanned, thus, at least 2° cooler than the original magenta. At this point the ozone generator was turned off and the oxygen feed terminated. Three succeeding photographs spanning 5 minutes failed to indicate any significant evidence of recovery.

Conclusions and Recommendations

The purpose of this study was to investigate the effects of ozone on the skin temperature patterns of a rabbit's ear. The results of the three tests performed indicated a correlation between ozone introduction and periods of decreased ear temperature.

A possible explanation of this correlation is linked to edema, which in this case is an abnormal accumulation of fluid in the pulmonary tissues and air spaces due either to changes in hydrostatic forces in the capillaries or to increased capillary permeability (120). The fluid's presence in the lungs tends to decrease the oxygen uptake efficiency, thus hypoxia results. In order to counter the mechanism responsible for reducing the oxygen supply, peripheral vasoconstriction may occur. This results in a reduction of blood flow to non-essential parts of the body, i.e. ears, in order to increase the oxygen feeding more essential body members. Thus, the cooling phenomena is noticed.

Through personal communications with Drs. F. M. A. McNabb

and A. G. Heath of the Virginia Polytechnic Institute and State University Biology Department (121), it was learned that a reduction in heart rate is associated with peripheral vasoconstriction. This metabolic state occurs to prevent a damaging rise in blood pressure which would result from the decreased blood vessel caliber if flow were not reduced.

It is, therefore, recommended that additional work be done incorporating electrocardiography, since verification of the peripheral vasoconstriction theory could be accomplished by noting a decreased heart rate.

A close examination of test two's results reveals a slower response to ozone when compared to the first or third tests (using the skin temperature parameter). Influences of a tolerance mechanism could be the cause, since test two began only 3 hours and 40 minutes after the first test was completed. Test three, by comparison, began over 12 hours after the second test ended.

Further tests, initiated at progressively varying time intervals, would aid in evaluating the validity of this hypothesis.

With respect to stated objectives, the use of thermography as a non-destructive temperature measurement technique proved reliable. No difficulties were encountered regarding its use. The unique role of Reynolds Film as an infrared window was a valuable feature of the test chamber design,

for it allowed skin temperature observations to be made during exposure periods. The only detriment of this material is a lack of physical integrity which prevented higher flow rates through the chamber. In the future, larger air inlets may not only serve to lessen this problem but also allow greater mixing to occur.

In conclusion, the objectives established in the early stages have been met. A call for further work is, therefore, issued by the author with the hope of further understanding the close ozone exposure-skin temperature relationship.

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A METHOD FOR REAL TIME EVALUATION OF PHYSIOLOGICAL STRESS DUE TO OZONE EXPOSURE

by

Randy Dwight Atkinson

(ABSTRACT)

A study was undertaken to determine what effects, if any, exposure to ozone would have on a rabbit, as indicated by changes in its ear temperature patterns.

By using a thermographic camera, thermal contours were obtained prior to, during and after exposure to low levels of ozone (near 1.0 ppm).

Results from three ozonation experiments indicated a decrease in ear temperature of up to 2° C upon ozone's introduction. No evidence of recovery was noted within 12 minutes following exposure.