Clinical and Laboratory Circulatory Effects of Trifluoroethylvinyl Ether.

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TRIFLUOROETHYLVINYL ether (Fluoromar®) has been under investigation as an anesthetic agent at our institution for some time both in the laboratory and in the operating room. It is our purpose to present some observations of the effects of this agent on the circulatory system.

In the laboratory, dogs showed a consistent, repeatable fall in systolic and diastolic blood pressure and a decrease in pulse pressure with deepening anesthesia whether or not assisted respiration was used. This was easily reversible by lightening the plane of anesthesia. Cardiac arrhythmias failed to occur in twelve dogs anesthetized with trifluoroethylvinyl ether vaporized by oxygen, through a to-and-fro carbon dioxide absorption system meticulously guarding against hypoxia and hypercarbia. One other dog showed nodal rhythm and a 2 to 1 heart block with deep anesthesia, but this reverted to a sinus rhythm when fresh soda lime was substituted for the exhausted absorber.

In the clinical investigation, 134 patients were anesthetized with trifluoroethylvinyl ether by various techniques and combinations with other agents for nearly all types of operations. Thirteen patients were monitored with continuous electrocardiographic recordings. An occasional A-V nodal rhythm occurred. Ten of the 134 anesthetizations were judged unsatisfactory from the circulatory standpoint; seven because of hypotension. A wide variation in blood pressure response was noted but attempts to provide muscle relaxation by deep anesthesia frequently resulted in moderate to severe falls in blood pressure. One cardiac arrest occurred but was attributed to causes other than the agent.

In summary, trifluoroethylvinyl ether seemed to have certain definite cardiovascular effects. Attempts to produce deep anesthesia and good muscular relaxation were met with moderate to severe hypotension. The cardiac rhythm, however, remained remarkably stable even in deep anesthesia. Judgment of depth of anesthesia required close observation since its speed of action led to rapid shifts in degree of respiratory and circulatory depression. Used as an adjuvant agent with nitrous oxide, trifluoroethylvinyl ether seemed superior to most agents presently employed.

Validity of Features of Decompensatory Phase of Shock as Signs of Irreversibility.

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IRREVERSIBLE experimental shock is commonly characterized by a decompensatory phase. Visceral pooling of blood, especially in the liver and bowel, is an important cause of decompensation and is generally considered a critical component of irreversibility. The ferritin system of the liver and the breakdown of bacterial defense barriers of the bowel have been extensively related to irreversible shock. Previous studies in our laboratory and by many others, have demonstrated various protective modalities against an ordinarily lethal stress. Experimentally protected animals do not develop the characteristic hemodynamic and visceral features of the decompensatory phase. In the protection experiments in hemorrhagic shock on rats, whose tissues are normally sterile, positive bacterial cultures of the liver and blood were observed. Repetition of these experiments using careful aseptic technique instead of conventionally clean technique eliminates the positive bacterial findings. In these sterile experiments animals show a higher survival rate and absence of decompensatory visceral congestion and hemorrhage. When subjected to more severe stress a significant number of these sterile rats become irreversible but still show no decompensatory take-up of blood. Fatalities cannot be differentiated from survivals by gross autopsy or histological examination of tissues.

In attempting to determine whether the sterile shock preparation could be protected by the same modalities as the conventional preparation some sterile groups were pretreated with chlorpromazine and others with various antibiotics—two reliable protective

modalities. Results indicate that chlorpromazine is without significant protective effect but that significant added protection results when any one of most of the antibiotics used is given. Again the typical decompensatory phase does not develop in either group of animals regardless of whether they die or survive.

The observations indicate that shock may be irreversible in the absence of the classic decompensatory phase. This suggests that decompensation is not a basic component of the shock syndrome. When a decompensatory phase is allowed to develop, however, it is clearly an unfavorable influence. Its development may be less the consequence of the stress per se than the result of species characteristics (dog vs human, rat) or experimental methods. In this regard other studies indicate that other components of experimental techniques namely dose ranges of heparin and anesthetics, predispose to the onset of the typical decompensatory phase. The purer form of shock, without decompensation, should be a less confusing entity to study since the stress responses are not clouded by uncontrolled or unintentional ancillary factors.

Correlations of the Electroencephalogram with Trifluoroethylvinyl Ether (Fluoromar®) Anesthesia. J. R. HOUSEHOLDER, M.D., AND L. E. MORRIS, M.D., Department of Anesthesiology, University of Washington School of Medicine, Seattle, Washington.

TRIFLUOROETHYLVINYL ether (Fluoromar®) has the property of producing rapid anesthetic induction and emergence and is capable of causing respiratory arrest in the presence of high concentrations of oxygen. Because of these properties a study of this agent was pursued to see if it would produce EEG patterns significantly different from those resulting from other anesthetic agents, and to see if the EEG patterns could be correlated with venous blood level of trifluoroethylvinyl ether and clinical estimates of depth of anesthesia.

Sixteen patients, ranging in age from 89 to 33 years, the majority of whom were to have operations involving the lower extremities, were selected on a random basis. Premedication consisted of atropine sulfate 0.2 to 0.4 mg., one hour prior to induction of anesthesia. In the operating room bipolar fronto-occipital needle electrodes were placed midline in the scalp and the leads were connected to a multi-channel Gilson recorder. Lead I of the electrocardiogram was recorded simultaneously. Pre-induction waking state electroencephalogram patterns were obtained. Induction of anesthesia was with nitrous oxide-oxygen, semiclosed circle absorption, adding trifluoroethylvinyl ether as rapidly as tolerated. The system was closed as soon as possible and anesthesia maintained with trifluoroethylvinyl ether and oxygen through an endotracheal tube. Venous blood samples were obtained through an indwelling plastic catheter which had been threaded through the cephalic vein up to the junction with the external jugular vein. Samples were drawn when there was a definite change in the electroencephalogram pattern or when there was judged to be a clinical change in depth of anesthesia. Forty-nine blood samples were chemically analyzed, utilizing a bromination of the trifluoroethylvinyl ether in an excess of methanolic bromine and back titration of the excess bromine with normal thiosulfate.

Since nitrous oxide was used as an induction agent, initial electroencephalogram patterns were probably influenced by this agent. Patterns observed after the system was closed were probably more representative of trifluoroethylvinyl ether effect. Interpretations of the electroencephalogram patterns were limited to the planes of Stage III, since often excitement stages created too much interference and artefacts.

Examination of the records of the sixteen patients revealed patterns that recurred frequently. Blood samples were obtained with the appearance of these patterns and an attempt was made to correlate the blood level with the electroencephalogram pattern. The electroencephalogram patterns at progressively increased venous blood levels were:

9.00 mg.%—14–16 cps, moderate voltage
15.75 mg.%—3–4 cps, high voltage
18.20 mg.%—2.5–3 cps with beginning flattening 1¾ sec. duration
29.60 mg.%—1.5–1.0 cps with suppression periods of about 1 sec.