mentation, for energy supply. Hence, during the few days following the chemical treatment, these seeds, in comparison with the control, exhibit a low oxygen uptake and a high carbon dioxide evolution, and, consequently, a high CO_2:O_2 ratio. This effect is very similar to that caused by germinating seeds under low oxygen tension, which has been fully described by Taylor (*3*). As a result of O_2 deficiency in the medium, aerobic respiration is reduced, and this reduction is to some extent compensated for by the fermentative activity of the germinating seeds, as evidenced by the high CO_2:O_2 ratios under such a condition. If they cannot furnish the energy necessary for germination fermentatively, most seeds will fail to germinate at all. If, as in rice, the seed is especially gifted with a highly functional fermentative mechanism, it can proceed to germinate, although with some delay, even after 2,4-D treatment or under anaerobic condition. However, this condition cannot go on indefinitely. Even rice cannot continue to grow in the complete absence of oxygen.

During recent years evidence has accumulated which shows that auxin is involved in the 4-carbon acid respiratory system (*4*). The increased respiration due to auxin treatment was found to parallel the increase in elongation of, and the acceleration of protoplasmic streaming in, the *Avena* coleoptile. This statement is probably true only at certain low concentrations of auxin.

Our experiments have shown that 2,4-D, at low concentrations (0.01 per cent), promotes germination; but, at higher concentrations (0.1 per cent), it begins to inhibit aerobic respiration and checks germination.

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Prevention of Respiratory Embarrassment in Therapeutic Curarized Convulsions

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Transitory asphyxia and cyanosis occur so frequently in spontaneous and therapeutically induced convulsions that cerebral anoxia was cons dered a possible explanation for the beneficial effects of convulsions in mental disorders. However, it has been demonstrated that the production of cerebral anoxia by the inhalation of nitrogen has no such therapeutic value (*4*). On the contrary, asphyxia contributes clinically to cardiac strain and plays a role in the fatalities and near-fatalities that occasionally occur in convulsions. The treatment of asphyxia by the use of chemical stimulants has proved futile (*3*). Administration of oxygen is ineffectual unless the air passages are open. The presence of trismus makes the installation of apparatus to clear the respiratory passages *after* a convulsion difficult (*1*) and often impossible. We have found that although administration of the convulsant electric current during inspiration facilitates postconvulsive breathing, since the first respiratory movement is then an expiration which clears the passages, this so increases intrapulmonary and venous pressure as to make the procedure not without risk. This procedure has, therefore, been discarded.

Certain few patients are "hard breathers" in that they persistently have asphyxial episodes during and after convulsive seizures. They seem to fall into two main categories: (1) those whose illness is characterized by agitation, depression, rejection of food and sleep, and other psychological "oral" qualities; and (2) patients with evidence of generalized arteriosclerosis or other neurologic complications, in whom there appears to be some inefficiency in the respiratory apparatus. The physician is sometimes compelled to treat a patient with coronary disease, whose agitation is so marked and dangerous to the heart as to necessitate the administration of shock treatment in an attempt to terminate the mental disorder. It is precisely those patients falling into both categories who are apt to show respiratory difficulties and in whom asphyxia is most dangerous. In these patients it is impossible to soften the convulsion adequately by means of curare because it increases respiratory difficulty and asphyxia. By premedication with sodium pentothal or sodium amytal (*2*) one can increase slightly the dosage of curare. The sedation seems to diminish the preconvulsive anxiety and restlessness and to diminish pharyngeal spasm. Unfortunately, these barbiturates increase the postconvulsive apnea.

We have found a simple means of avoiding asphyxia and consequently diminishing cardiac strain. With this technique a Guedel rubber airway is installed *during* the convulsion.

The patient is prepared as usual for the treatment. He is placed supine on a flat bed without hyperextension, since the latter increases respiratory difficulty. The barbiturate and curare, or curare alone, are injected. In electroshock convulsions, a cloth gag is placed between the teeth to protect the tongue and lips. The current is then applied. During the initial tonic flexion phase, the gag is forcibly bitten. This is followed by a moment of relaxation in which the mouth is opened widely. At this point a Guedel rubber airway is introduced to the hilt. Where metrazol is used, the cloth gag is not necessary. The initial movement of the mouth is an opening one, and the airway is similarly introduced. With the airway in place there may be respiratory exchange in the m dst of the convulsion. Before the end of the convulsion, the patient is turned on the side with the mouth down. Mucus and saliva will flow from the airway. Respiration usually begins shortly after the convulsion is over. The airway is removed a few minutes after the convulsion, when respirations are normal and the mouth and jaw relaxed.

With this technique asphyxia and cyanosis are now rare. There are occasional periods of apnea, which seem to be of central origin. In these apneic periods it is a simple matter, with the air passages open, to institute respiration, if one so desires, by abdominal pressure. The airway acts as an efficient mouth gag and bitten tongues and lips are infrequent. A theoretical objection to the method is the possibility of dislocation of the jaw and trauma to the pharynx, but in practice neither occurs. Postconvulsive headache, nausea, and confusion have been diminished considerably. Larger amounts of curare can be administered safely to hard breathers. In one patient with known severe coronary disease, the postconvulsive electrocardiographic changes were less marked and of much

shorter duration with this method. Patients who are not sedated are less apt to show postconvulsive restlessness. The postconvulsive hypertension is reduced, but not entirely eliminated. Observations with different methods of therapy indicate that there are at least four factors in postconvulsive hypertension: (1) anxiety and restlessness, which can be alleviated by means of intravenous sodium pentothal or sodium amytal; (2) muscular exertion, which can be alleviated by means of curare; (3) asphyxia, which can be prevented by the method described; and (4) a cardiac convulsant action. The heart seems to take part in the generalized convulsion, cardiac irregularities having been reported during seizures. These are less marked when asphyxia is prevented. Hypertension does not always accompany partial convulsions, but is an invariable component of generalized seizures, although it is less marked and of shorter duration with the technique described. In the postconvulsive period, especially with metrazol, premature cardiac beats coincide with mild, generalized twitches. These coincide too well temporally to be explained except on the basis of a common stimulus. The fact that this hypertension can be temporarily arrested by carotid sinus pressure suggests that there is a nervous pathway through the autonomic nervous system. Clinically, however, with the prevention of asphyxia, anxiety, restlessness, and severe muscular exertion by means of the technique outlined (barbiturate, curare, intraconvulsive airway) most cardiac patients seem able to stand the convulsion with minimal evidence of cardiac strain.

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Photoinactivation of Milk Fat Lipase and the Origin of Bitter Flavor in Milk

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H. D. Kay recently reported in a letter to the editor of *Nature* (*2*) that an enzyme which hydrolyzes tributyrin in milk (tributyrinase) is sensitive to light and that the atmospheric oxygen plays an important part in the photodestruction of the enzyme. This letter prompts the present writer to release information pertaining to the mechanism of the photochemical inactivation of milk fat lipase and to the origin of bitter flavor in milk, obtained in connection with studies of the interrelationship of ascorbic acid oxidation in milk and the reaction which produces the tallowy flavor (*4*). It has been found that the activity of milk fat lipase, as produced by cooling, warming, and the subsequent holding of milk at 0–5° C. from 24 to 48 hours (*5*), varied with the ascorbic acid content of the milk reduced to various levels by exposure to light (Fig. 1B). The phenomenon suggested the possibility that H_2O_2, formed in the course of the photooxidation of ascorbic acid (*1*), might be responsible for the inactivation of milk fat lipase.

This postulation made it necessary to determine the effect of H_2O_2, added to milk in different quantities but not in excess of that required to oxidize the ascorbic acid completely, upon the activity of milk fat lipase. The results were that both processes, namely, the oxidation of ascorbic acid and the destruction of lipase, were promoted simultaneously in the milk (Fig. 1A).

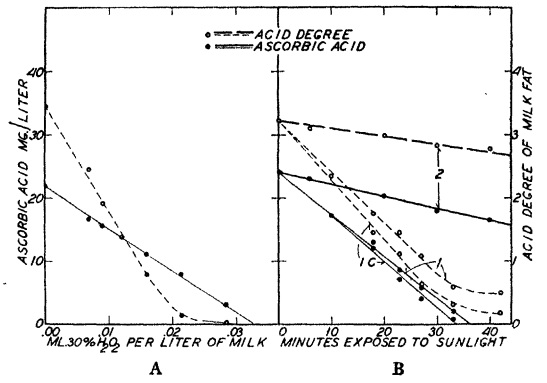

Fig. 1. Simultaneous promotion of ascorbic acid oxidation and of inactivation of milk fat enzyme lipase in the milk either in the presence of added H_2O_2 or photochemically by the exposure of milk in Erlenmeyer flasks to direct sunlight (B, 1) or northern daylight (B, 2). Sample 1 C was exposed in the presence of added catalase. Lipolysis was activated by cooling-warming-recooling of milk, and activity measured by increase in the acid degrees of fat at the end of a 48-hour holding period at 0–5°C. Ascorbic acid content of the milk was followed by direct titration with 2,6-dichlorophenol-indophenol in acid solution.

Consequently, to prove that the photooxidation of milk fat lipase resulted from H_2O_2 formed in the course of ascorbic acid oxidation, a portion of lipolytically active milk was fortified with catalase prior to exposure to sunlight. The amount of catalase (*6*) added to milk was sufficient to prevent ascorbic acid oxidation in the presence of 0.028–0.03 ml. of 30 per cent H_2O_2 solution/l. of milk, added subsequently. The activity of catalase, determined again at the end of 40 minutes of exposure to sunlight by the addition of ascorbic acid to milk depleted of its content and of H_2O_2, was found to be the same as at the starting point. The photoinactivation of milk fat lipase was not prevented, however, by the addition of catalase to milk (Fig. 1B). In fact, the enzyme was inactivated at a slightly faster rate in the presence of catalase as compared with that in the control milk. It was apparent, therefore, that the photoinactivation of lipase is an independent reaction, and that H_2O_2 formed in the course of ascorbic acid oxidation might play an auxiliary part. This evidence was supported further by data concerning the effect of the depletion of the ascorbic acid content of milk, prior to exposure to light, by cucumber juice oxidase. Since the ascorbic acid–cucumber juice oxidase system uses one atom of oxygen per molecule of vitamin C (*1*), it was safe to assume that no H_2O_2 was present in the milk at the time of exposure. Again, the sensitivity to light of the milk-fat-splitting enzyme was found to be approximately the same as in the control portion of milk containing ascorbic acid. It was also found that sensitivity of milk lipase to light varied appreciably between the samples of milk from different cows and that, of the 20 samples studied, only two samples showed from 80 to 90 per cent loss in the lipolytic activity due to 30 minutes' exposure to light. The losses of lipase in the remaining samples of milk varied from 50 to 80 per cent. The inactivation of milk