is represented by the effort to find a nonvolatile agent for complete narcosis or, failing that, a nonvolatile agent to serve for so-called basal anesthesia, supplemented by the use of a volatile anesthetic. Lendle's paper seems to show that neither Avertin nor the barbital derivative "Pernocton" is a satisfactory solution of the problem. Safe and efficacious new methods of anesthesia may result from the modern work in this field; but all the evidence indicates that the products thus far proposed for so-called basal anesthesia are hypnotics or sedatives and should be used as such. They cannot be safely used for complete anesthesia and can be safely used in combination with other agents for the production of complete anesthesia only by those thoroughly experienced in the administration of anesthetics and closely familiar with the studies of the use of nonvolatile agents for anesthesia. Thus far their intravenous use must be considered unsafe.

PULMONARY ASBESTOSIS

In 1928 The Journal,[1] commenting on an article by W. E. Cooke,[2] suggested that pulmonary asbestosis, because of its dangers and its unique pathologic features, deserved more attention than had been accorded it. Two and a half years later Mills[3] reported the first case from the United States. His patient had worked in an asbestos mine in South America prior to 1898, had returned to the United States, and again had been employed in South America, this time in an unstated occupation, between 1911 and 1913. He died in Minnesota in 1929. The history of the case indicates that the asbestos which was found in the subject's lungs at necropsy was inhaled prior to 1898. If so, the profound pulmonary changes which evidently were induced by the asbestos, and which seemingly contributed to the patient's death, were present thirty-two years after the agent that caused them was introduced. Even if the exposure to the asbestos occurred at the time of the patient's later visit to South America, the pulmonary injury endured at least seventeen years. Accordingly, Mills has concluded that pulmonary asbestosis is an incurable disease, from which the patient may or may not die. The cause of death, if tuberculosis is avoided, seems to be pulmonary fibrosis with its indirect effect on the heart. At necropsy of Cook's patient, pulmonary fibrosis and evidence of extensive tuberculosis were found; it was not certain whether the fibrosis was due more to the tuberculosis or to the asbestosis. In Mills's case, evidence of tuberculosis was not found but the fibrosis was severe. The conjecture seems warranted, therefore, that asbestos is capable of producing two types of injury, somewhat as iron dust is capable of producing two types of pulmonary siderosis. According to Bohrod,[4] who cited the work of Hart, the red type of iron lung is found in persons who work in iron mines and in factories using certain iron pigments; pneumonoconiotic induration predominates and there is little tuberculosis. Metal grinders and polishers display the black type of iron lung; induration is less evident and tuberculosis is unrestrained. That siderosis and asbestosis are allied seems likely in view of the evidence that the iron-containing[1] portions of asbestos constitute the danger to health in the asbestos industry. Concerning the relation of physicians of the United States to this industry, Mills pointed out that asbestos is mined and manufactured in many parts of this country and that pulmonary asbestosis surely will be encountered. In England the workmen's compensation act has recently been extended to include this condition.[5]

INSENSIBLE PERSPIRATION AND BASAL METABOLISM

The estimation of basal metabolism has proved to be unexpectedly valuable as a diagnostic aid. The measurements of the respiratory exchange, which are now depended on to furnish the requisite information, have been greatly simplified in recent years, so that the procedure can be made applicable even in the office of the individual practitioner. There are, however, refinements of technic that ought to be applied more widely than they usually are. This refers particularly to the proper preparation of the patient and to the maintenance of strictly "basal" conditions. In the case of infants and children, obvious difficulties present themselves in the measurement of respiratory exchange as well as in direct estimations of heat output. Fortunately an alternative procedure has been proposed in the relatively simple method of ascertaining the insensible losses represented by perspiration. The determination of loss by insensible perspiration as an index of metabolism was discussed in detail in 1926 by Benedict and Root[6] in its application to adults. Its use in relation to children has been championed by S. Z. Levine and his co-workers[7] in the Department of Pediatrics of the Cornell University Medical College, New York. Basal standards of insensible perspiration were proposed by these workers in reference to the normal infant. The latest researches of Levine and Marples[8] have been concerned with the validity of the proposed methods and their basis of reference to predictions. A statistical study was made of the published data responsible for the standards at present available for the basal metabolism and basal insensible perspiration of normal infants. The investigation included consideration of the correlation between the two variables in simultaneous calorimetric measurements as well as in independent measurements with the respiration chamber and the balance. The evidence secured points to a high positive correlation between the physiologic mechanisms of heat production and insensible perspiration in the human subject.

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