the deciduous dentition in the infant, if these diseases occur during the time when the teeth are forming. It is also understood that these same diseases in infants and young children, occurring during the period when the permanent teeth are forming, will similarly affect these teeth.

A partial study of the available dental literature on this subject has revealed an interesting discussion which includes the presentation of a case similar to those found by Evans (*Med. J. Aust.*, 1944, **2**, 225). This was reported in 1893 by J. J. R. Patrick as follows (*Dent. Rev.*, 1893, **7**, 439):

"I have entered thus far into a description of the origin and development of the teeth, in order to illustrate the detrimental influence of eruptive fever on the teeth during the periods of their formation; and while eruptive fevers are not the only factors to be recognized in producing abnormal conditions in tooth development, I consider such fevers a prolific source of faulty nutrition in these organs, as the following cases observed in my practice will fully exemplify.

"Mrs. S., during the fifth month of gestation was afflicted with scarlet fever—recovering, in the course of time gave birth to a child. This child's teeth appeared unusually early, the enamel discolored, pitted and granular and absent at the incisive margins, and the teeth barely extended beyond the gum. He is now fifteen years old with all his permanent teeth except the third molars. These second teeth, with the exception of a little decay, are in good condition, the enamel smooth and white and all the teeth large and strong.

"Three years later another child was born, and soon afterward the first child contracted the measles and the second child also contracted the disease. In the course of time the deciduous teeth of the second child appeared, all in excellent condition, but were lost unusually early, and the second set appeared in the following order: The sixth year molars at four years. The central incisor at five years. The premolars between the sixth and seventh years; and the cuspids between the eighth and ninth years. All these teeth are discolored, pitted and granular, and the enamel is worthless as a protection to the dentine."

Later in the same year Dr. Patrick attended the World's Columbian Dental Congress at Chicago, where a paper on the pathology of congenital defects of the deciduous dentition was presented by Otto Zsigmondy. During the discussion of this paper Dr. Patrick made the following remark (*Trans. World's Columbian dent. Congr.*, 1894, **1**, 65):

"All eruptive diseases during gestation must necessarily affect the enamel of the deciduous teeth if these eruptive diseases take place during the formative process of the enamel. How could it be otherwise?"

The quotations given above not only show that Dr. Patrick had developed a clear concept of the role of acute disease of the mother in the causation of congenital defects of the infant in the field of dental development, but also point out two factors which are of importance in the current efforts to clarify the medical aspects of this problem. The teeth are relatively permanent structures, and their embryology has been worked out in considerable detail. It is therefore apparent that the presence or absence of defects in the deciduous teeth will be of great importance in the evaluation of the effects of various disturbances of pregnancy on the fetus during the period of tooth formation. A study of the simultaneous occurrence of dental defects and malformations in other organs will also be of considerable interest.

Dr. Patrick's second case points out that the problem of developmental anomalies is not limited to the period of gestation, but also extends for some time beyond the birth of the child. This postnatal aspect of the problem deserves more study from a medical point of view. The importance of the presence or absence of dental defects, especially in conjunction with defects in other parts of the body, is again apparent. (R. J. M. HORTON, *Harvard School of Public Health*, and J. M. DUNNING, *Harvard School of Dental Medicine.*)

To express in a mathematical formula a complicated physiological response such as that involved in body-temperature reactions of cattle to climate, the fundamental requirement is that it be based on as complete an accumulation of facts as possible. To modify a formula that satisfies this condition by introducing a factor based on an assumption unsupported by experimental evidence is to confuse rather than to advance the knowledge of the subject.

In a recent paper R. F. Gaalaas (*J. dairy Sci.*, 1947, **30**, 79–85) has modified the formula

$$HT = 100 \div [10(BT - 101.0)]$$

originated by Rhoad (*J. animal Sci.*, 1942, **1**, 85) to

$$HT = 100 - [14(BT - 101.0)].$$

In the Rhoad formula the factor 10 is used merely to remove the decimal from the result within the parentheses. In this manner a whole-number coefficient in a scale of 100 is obtained, with 100 equal to perfect efficiency in maintaining a normal body temperature of 101.0°F., under prescribed rules of a test procedure (A. O. Rhoad. *Trop. Agric.*, 1944, **21**, 162–164).

The factor 10 was changed by Gaalaas (p. 80) "based on the assumption that a body temperature of 108°+ at 90°F. air temperature and under otherwise normal conditions would indicate a complete loss of control in the regulation of body temperature, or zero per cent efficiency in eliminating surplus body heat. Conversely, a body temperature of 101°F. is considered normal and a cow that would maintain that body temperature at 90°F. air temperature would be considered 100 per cent efficient. . . ."

The reader is led to believe that the factor 14 was derived by dividing the 7° range between 101 and 108 into 100 equal parts, thereby placing a value of 0.14285716 . . . ∞ on each tenth of a degree on the clinical thermometer.

In correspondence, Gaalaas states that the factor 14 is a correction factor necessary because his cows had "access to shade instead of requiring them to be held in the sun. . . . The factor 14 is enough larger to correct for the observed average difference in body temperatures." A check on the results published earlier by Gaalaas (*J. dairy Sci.*, 1945, **28**, 555–563) gives credence to this interpretation of the origin of the factor 14 and is acceptable as a correction factor, as it is properly supported by satisfactory data.

One wonders, therefore, why an unsupported assumption is given as the base for changing the factor 10 to 14 in the original formula! (ALBERT O. RHOAD, *Inter-American Institute of Agricultural Sciences, Turrialba, Costa Rica.*)