

in which such a definition is required. For this purpose *the intended use* is the best basis for such differentiation. In those instances where it becomes important or necessary to reach a decision with respect to the kind of products that should be considered as drugs or foods (such as for control of representations or for tax purposes), the authority for such

purpose could so define the subject to be covered that no controversy should arise.

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Letter to the Editor

PERSONAL EXPERIENCE WITH PAROTID ENLARGEMENT

Dear Sir:

The report of Sandstead, Koehn, and Sesoms regarding enlargement of the parotid gland in malnutrition (AM. J. CLIN. NUTRITION 3: 198, 1955) suggested that some details concerning my personal experience with this disorder may be of interest. Various results of Dr. A. J. Carlson's experiments on my food restriction and fasting were previously described,^{1,2,3} but the development of parotid enlargement was not mentioned. In brief, "asymptomatic" bilateral parotid enlargement began to develop in 1908 when I ate freely after a period of about six months during which I restricted my food intake and, particularly, my protein intake.

The value of protein restriction at that time seemed to be supported by the findings in the studies made by Chittenden.⁴ Although no evidence of parotid enlargement was noted while my total calorie intake was considerably restricted (the food I ate consisted largely of fruit and sugar), I found evidence of excessive water retention.^{2,3} At that time this was assumed to indicate "autointoxication," but between 1917 and 1919 it became evident that the water retention was nutritional edema promoted by semistarvation, and particularly by protein starvation, with the inclusion of some salt in my diet. My food intake was increased in 1908, largely through the addition of potatoes, bread, and cabbage (sauerkraut). This,

incidentally, increased the salt intake considerably and a rapid increase in nutritional edema occurred. My entire body seemed to swell or become bloated and the swelling in the parotid region merely seemed to be the most prominent evidence of this general swelling. I looked as if I had the mumps but the swelling on the right side was somewhat greater than on the left. I also became mentally and physically very sluggish and this suggested in 1909 that I was afflicted with myxedema.

As the increase in my food intake appeared to be responsible for the generalized swelling or assumed increase in autointoxication, I restricted my food intake again and more drastically than before. I believed that fasting would serve best to reduce the swelling, but at that time fasting seemed to be too great an ordeal to be practical. Hence, I tried eating only enough citrus fruit to mitigate hunger. During 17 days in which I ate only oranges and a few lemons, the generalized as well as the local (parotid) swelling disappeared almost completely. Incidentally, I also became more alert mentally.

As a result of a fall I came under the care of a physician who prescribed a liberal diet "with plenty of vegetables like turnips." The generalized and mumps-like swelling consequently became greater than before within



about two weeks. When I complained that the vegetables appeared to be mainly responsible for the disfiguring parotid swelling, this physician said that the swollen glands, which were then assumed to be swollen parotid lymph nodes, could be removed surgically but that vegetables were needed in my diet. After this physician's attention to my injury was no longer needed I stopped eating the seemingly harmful vegetables and made bananas the mainstay of my diet, and my general condition improved. Obviously, the salt added to the vegetables was mainly responsible for their apparent harmful effect.

Details concerning my further experience before 1917 need hardly be presented here except to say that it involved repeated flare-ups in generalized edema and parotid enlargement whenever I did not, or could not, adhere to a fruitarian diet; improvement occurred when I adhered to such a diet or fasted. The most rapid improvement occurred during short fasts and this led me to try a prolonged fast in the hope of obtaining complete relief. A great general improvement was produced by a 26-day fast in 1913 but more fasting seemed to be needed within a year. Photographs of me that were taken in 1917 before and after 15 days of fasting at the University of Chicago, and which were included in Dr. Carlson's report of the study of hunger that he then made on me,¹ clearly show enlargement of the parotid on the right side or a swollen condition of the entire right side of my face before fasting and a more nearly normal appearance at the end of the fast. I do not know when the parotid swelling on the less affected left side disappeared or whether a slight residual enlargement still exists. The difference in improvement here appears to be similar to what I have noticed in regard to the effect of fasting on local fat deposits. The subcutaneous fat seems to disappear fastest or most completely in areas where the least excess exists. Moderately enlarged parotids are evidently often assumed to be normal.

The importance of salt as a factor explaining the flare-up of nutritional edema and enlargement of the parotids after fasting or a period of food restriction was discovered dur-

ing the study made at the University of Chicago in 1917.¹ The value of a liberal protein intake in preventing or limiting the salt-water edema was discovered in our experience in 1918 and 1919. However, the enlargement of my right parotid region or entire right side of my face evidently became chronic or involved some irreversible changes already before 1913 and no significant improvement was produced by fasting even as long as 41 days (in 1925) and using diets high in protein.

As reported by others, one may not experience any discomfort with such parotid enlargement. Only a slight sense of tension in the parotid region was experienced when my parotids were most swollen—in 1909 and 1910. I did not realize how asymmetric my facial appearance remained after 1910 until this was revealed by photographs. A mirror revealed that the mobility of the right side of my face had also become impaired so that I tended to smile only on the left side and the impression I made on others seemed to depend on which side of my face they saw or whether they noted the asymmetry. I did not note any other disturbance until about 1942 when I began to wear glasses. It then became evident that the skin around my right ear was hypersensitive to the slight pressure or pinching occasioned by wearing glasses. It is still necessary to change their position from time to time for relief. About two years ago (1953), I became more concerned about the possible complications, because I began to be troubled with earache on the right side. A further enlargement of the parotid gland did not appear to be responsible for the trouble, although the gland appears to be becoming firmer to palpation. In any case, the earache led me to give my nutritional state closer attention and the earache disappeared with a regimen whereby I got rid of some fat and apparently also some excess fluid ("subclinical" edema).

I was aware long ago that it was much more difficult to get good results in shaving around my right jaw than around my left jaw. This has evidently been due to a poorer skin tone or edematous condition of the skin around the somewhat enlarged submaxillary salivary gland on the right side. This was previously



also thought to be an enlarged lymph node. Sensitiveness to the pull of a somewhat dull razor has served about as well as the palpable size of my right parotid salivary gland as an index of excessive hydration. Some unexplainable variations have been noted to occur from time to time within a few hours but seem most likely to be due to variations in the absorption of products of digestion and fluid from the digestive tract.

In my opinion, only observations of the conditions under which parotid enlargement begins can shed light on this disorder. Fatty infiltration or lipodystrophy⁵ and other changes evidently occur as a result of a more or less chronic edematous state.

Why the parotid salivary glands should become particularly enlarged in some individuals living on diets promoting the development of nutritional edema remains a question. However, the finding of Fawcett and Kirkwood⁶ that salivary glands have a "reverse thyroid" function suggests that they may play a specific role in nutritional edema. I suspect that an iodine deficiency as well as protein deficiency may explain severe nutritional edema. Incidentally, this would link nutritional edema with myxedema.

In any case, parotid enlargement needs closer attention. I have been surprised by the general indifference of physicians to my parotid swelling, even when it was pointed out to them. Sandstead and his associates refer only

to bilateral enlargement but Kenawy⁷ found unilateral enlargement in 18 of 100 cases. Symmetric bilateral enlargement is evidently most common and unilateral enlargement may merely be the result of a failure of acute bilateral enlargement to disappear on one side. The effect of parotid enlargement on neighboring tissues will best be revealed in cases of unilateral enlargement.

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