is a suitable substitute for an appropriate reduction in weight; that tolbutamide does not increase appetite, hence supplements well the benefits of a reducing regimen for the overweight patient; and that the need for drug therapy decreases and may disappear as the weight approaches the ideal. Knowingly to accept the advantages of tolbutamide and to ignore those of a temporarily reduced diet may be likened to the behavior of children who accept privileges but shun responsibilities.

The instruction of patients regarding oral therapy is for the most part an individualized affair but it is pertinent that all know some salient facts about tolbutamide:

a. It is effective in controlling the diabetes in the majority of the older and is rarely effective in young diabetics or in those who acquired diabetes while they were young.

b. It is not a form of insulin nor does it act like insulin.

c. It is not a cure for diabetes.

d. When the diabetes may be controlled by diet alone, the drug is unnecessary.

e. It is ineffective during acute complications, notably acute infections, surgical procedures and ketosis.

f. Its favorable effect is transitory (four to six months) in some patients.

g. It does not reduce the need for attention to *diet, tests for sugar and acetone* in the urine. These measures are of special importance in the first few days of oral therapy.

h. There are no advantages except in rare instances in using insulin and tolbutamide simultaneously.

i. Unlike the great variety of doses of insulin, the dosage of tolbutamide is relatively uniform, preferably at 1.0 or 1.5 gm. and not more than 2 gm. daily given in divided doses for long-term treatment.

j. The dosage of tolbutamide is changed only under the physician's direction. Excessive amounts have been known to increase the level of the blood sugar, the degree of glycosuria and the incidence of unfavorable side effects.

k. The change from insulin to oral therapy is accomplished by different plans according to the severity of the diabetes and the likelihood of a favorable response. For some, indeed for the majority, this can be accomplished on an outpatient basis. For others, hospitalization is highly desirable.

l. Hypoglycemic reactions are most likely to occur during the transition period from insulin to tolbutamide. Such reactions are rare with tolbutamide alone but when they do occur, the symptoms are identical to those of a hypoglycemia due to insulin. The treatment in both cases is the same.

m. Studies, in addition to blood sugar determinations and urinalyses, should be done at frequent intervals until it is certain that the new drug is well tolerated by the patient. These studies will include especially blood counts and the checking of the function of the liver.

n. Research workers and physicians have zealously guarded the welfare of diabetic patients in the development of oral therapy and this keen acceptance of responsibility has been shared by the pharmaceutical firms interested in these new agents.

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OCULAR NERVE PALSY IN DIABETES

Attention has been called recently to the frequency of ocular nerve palsy associated with severe headache occurring in diabetic patients.[1, 2] This syndrome of ocular nerve palsy associated with headache has been recognized for a long time. It is a rather frequent disorder, particularly when the paralysis is that of the oculomotor nerve. It usually comes on suddenly and is accompanied by severe pain on the same side of the head. Most of the patients who show this disease also demonstrate hemorrhages and exudates in the fundus as well as evidences of vascular disease in the retinal vessels.

Dieulafoy[3] in 1905 collected several cases of extraocular muscle paralysis due to diabetes mellitus. The association of such paralyses with diabetes mellitus has been noted in large series of patients by several investigators.[4, 5, 6, 7] Recovery is the rule in these cases. Waind[2] has noted that this is one of the most important features of this syndrome.

Speculation has been made concerning the etiology and exact nature of the lesion. It is suggested that it is of vascular origin, perhaps a minute hemorrhage either into the nerve trunk or about the nucleus of the oculomotor nerve. This theory is supported by the rapidity of onset in a great majority of cases with complete paralysis of the nerves involved. The tendency toward total regression has been mentioned, although recovery of function may require several months. Vitamin deficiencies frequently noted in diabetes mellitus and polyneuritis have also been suggested as causes of the ocular palsies.[6, 8] A recent pathohistologic observation has been made in diabetic oculomotor palsy by

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Adams, Hakin, and Dreyfuss. They noted a gross swelling of the intraorbital portion of the third nerve with extensive destruction of medullated fibers in its central portion.

Controversy exists as to the frequency with which nerves to extraocular muscles are affected; according to Waite and Beetham[4] and Walsh,[8] the sixth cranial nerve is the most commonly involved. Other authors[9] have recorded predominance of third nerve paralysis. The fourth nerve may be affected occasionally.

There are many causes of third nerve involvement from which the syndrome must be differentiated. Lesions of the third nerve fibers within the central nervous system are usually accompanied by other neurological signs and symptoms. Benedict's syndrome is characterized, for example, by homolateral oculomotor paralysis with contralateral intention tremor. When the cerebral peduncle is involved along with the third nerve, Weber's syndrome is diagnosed by the presence of oculomotor paralysis on one side and hemiplegia on the other. When the third nerve is involved in the cavernous sinus and the supraorbital fissure, it is practically always accompanied by involvement of other oculomotor nerves as well. Various inflammatory conditions such as syphilitic and tuberculous meningitis may produce third nerve paralysis. The inflammatory process of these diseases usually catches the third nerve as it emerges from the central nervous system around the chiasm, pons, and temporal lobes. Whenever meningitis of this sort is responsible, the lesion is usually bilateral. Purulent meningitis, on the other hand, usually involves the sixth nerve and not the third. A variety of toxins such as alcohol, lead, arsenic, and carbon monoxide have been said to produce a polyneuritis and produce a similar change as that seen in diabetes mellitus.

A rupture of an aneurysm at the base of the brain may catch the third nerve and produce a characteristic picture. Most of these aneurysms are situated at the branching of the internal carotid. In this site the nerve is especially vulnerable. Usually the individual has severe pain on the side of the face on which the third nerve is paralyzed as well as severe headache.

Migraine has been said to produce recurrent third nerve paralyses. Tumors which involve the third nerve usually impair the function of other oculomotor nerves, along with the third nerve, and often involve the trigeminal nerve.

An awareness of this syndrome and the exclusion of the other possible etiologies in a patient with diabetes mellitus allow one to give a more favorable prognosis. The exact frequency of this complication has not been well established, and further work is required to establish the exact pathogenesis of these palsies.

REFERENCES

1 Jackson, W. P.: Brit. Med. J. *2*:408, 1955.

2 Waind, D. P. B.: Brit. Med. J. 901, 1956.

3 Dieulafoy: Paralysie des nerfs moteurs del 'oeil chez les diabétiques. Press med. *13*:713, 1950.

4 Waite, J. H., and Beetham, W. P.: Visual mechanism in diabetes mellitus. New Eng. J. Med. *212*:367, 1935.

5 Leopold, I. H.: Diabetes mellitus observed in 100 cases for ten years or more; ocular findings. Am. J. Med. Sci. *209*: 60, 1945.

6 Weinstein, E. A., and Dolger, H. P.: External ocular muscle palsies occurring in diabetes mellitus. Arch. Neurol. & Psych. *65*:97, 1948.

7 Eareckson, B. P., and Miller, J. M.: Third nerve palsy with sparing of the pupil in diabetes mellitus. Arch. Ophth. *47*:607, 1952.

8 Walsh, F. D.: Clinical Neuro-Ophthalmology. Baltimore, Williams & Wilkins Co., 1947.

9 Gray, W. A.: Ocular conditions in diabetes mellitus. Brit. J. Ophth. *17*:577, 1933.

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Communication in Medicine

The trials of history taking are only slightly more taxing than the trials of history reading. Forgetting the communicative obstacles that he has had to overcome in getting the patient's story, the student artfully, perhaps vengefully, invents some of his own in setting it down. This is supposed to keep the attending physician on his toes. The patient, reads the professor, gives an Hx (history) of certain Sx (symptoms), consisting mainly of SOB (shortness of breath) and PND (paroxysmal nocturnal dyspnea) which failed to respond to NG (nitroglycerine) under the tongue. The social Hx reveals a lack of TLC (tender loving care) in childhood. After a thorough Px (physical examination), and with the help of a BUN, PSP, EKG, and EEG, the case is Dx'd (diagnosed) and finally Rx'd (treated). I leave you to guess who is Vx'd (irritated).

By Henry T. Ricketts, M.D.,
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