- George, J. T. A., McDonald, J. C., Payne, D. J. H., and Slade, D. A. (1958). *Brit. med. J.*, **2**, 1381.
- Goldsmith, H. J., Cowan, M. A., and Gooder, E. (1958). *Lancet*, **2**, 674.
- Goslings, W. R. O., Valkenburg, H. A., Bots, A. W., and Lorrier, J. C. (1963). *New Engl. J. Med.*, **268**, 687.
- Hardin, R. A., Quinn, R. W., and Avery, R. C. (1956). *J. infect. Dis.*, **99**, 84.
- Higgins, P. M. (1965). *J. Coll. gen. Practit.*, **9**, 136.
- Holmes, M. C., and Williams, R. E. O. (1958). *J. Hyg. (Camb.)*, **56**, 43.
- Kleinman, H. (1954). *Minn. Med.*, **37**, 479.
- Manser, R. W. E., and Wilson, M. M. (1952). *Med. J. Aust.*, **2**, 339.
- Mitchell, E. S. (1962). *J. clin. Path.*, **15**, 231.
- Rammelkamp, C. H. (1957). *Harvey Lectures*, 1955-6, Series 51, N.Y.
- and Weaver, R. S. (1953). *J. clin. Invest.*, **32**, 345.
- and Dingle, J. H. (1952). *Trans. Ass. Amer. Phycns.*, **65**, 168.
- Rantz, L. A., Spink, W. W., and Boisvert, P. J. (1945). *Arch. intern. Med.*, **76**, 278.
- Ravenswaay, A. C. van (1944). *J. Amer. med. Ass.*, **126**, 486.
- Reed, R. W. (1953). *Canad. med. Ass. J.*, **68**, 448.
- Seegal, D., Seegal, B. C., and Lyttle, J. D. (1935). *J. Amer. med. Ass.*, **105**, 17.
- Siegel, A. C., Johnson, E. E., and Stollerman, G. H. (1961). *New Engl. J. Med.*, **265**, 559.
- Rammelkamp, C. H., and Griffeath, H. I. (1955). *Pediatrics*, **15**, 33.
- Stetson, C. A., Rammelkamp, C. H., Krause, R. M., Kohen, R. J., and Perry, W. D. (1955). *Medicine (Baltimore)*, **34**, 431.
- Valkenburg, H. A., Goslings, W. R. O., Bots, A. W., de Moor, C. E., and Lorrier, J. C. (1963). *New Engl. J. Med.*, **268**, 694.
- Wertheim, A. R., Lyttle, J. D., Loeb, E. N., Earle, D. P., Seegal, B. C., and Seegal, D. (1953). *J. clin. Invest.*, **32**, 359.
- Williams, R. E. O. (1958). *Bull. Wld Hlth Org.*, **19**, 153.
- Wilmsers, M. J., Cunliffe, A. C., and Williams, R. E. O. (1954). *Lancet*, **2**, 17.

Fluoresceinretinography: Exudates and Microaneurysms

F. SKOVBORG,* M.D.; E. LAURITZEN,* M.D.

Brit. med. J., 1965, **2**, 1160-1162

Novotny and Alvis's (1961) technique for investigation of the retina by intravenously injected fluorescein has opened up new possibilities for examining the vessels in different pathological conditions.

Fluoresceinretinography gives an impression of the permeability of the vessels, and in proliferative retinopathy many more new vessels are revealed than by ordinary ophthalmoscopy. A bright fluorescence is seen in the cotton-wool exudates, but not in the waxy exudates (Dollery *et al.*, 1962; Hodge and Dollery, 1964; Scott *et al.*, 1964; Skovborg and Lauritzen, 1964).

Dollery *et al.* (1962), Hodge and Dollery (1964), and Scott *et al.* (1964) state that fluoresceinretinography reveals many more microaneurysms in the retina among diabetics and hypertensive patients than can be seen by ordinary ophthalmoscopy.

From our results and the study of other papers we conclude that most of the small fluorescent dots are not microaneurysms, but are probably small exudations of the same type as the cotton-wool exudates. This paper is primarily concerned with the diagnostic problems of differentiating by fluoresceinretinography between microaneurysms and small cotton-wool exudates.

Method

The method used was that described by Novotny and Alvis (1961) with Dollery *et al.*'s (1962) modifications. Instead of Ilford 717 we used Fluorodak (Kodak) film (Jensen, personal communication) because this film is especially sensitive to fluorescent light.

Fluoresceinretinography has been carried out on 110 diabetics. Our main results are in agreement with other publications (Dollery *et al.*, 1962; Hodge and Dollery, 1964; Scott *et al.*, 1964).

Many of the cotton-wool exudates are loaded with fluorescein. Fig. 1 demonstrates such an example. There is a vigorous fluorescing circular area, which appeared in the arteriolar phase. At the same place on the colour print a cotton-wool exudate was seen.

Fig 2 demonstrates a single fluorescent dot. At exactly the same spot on the colour print there was a small exudate.

In Fig. 3a the fluorescein picture shows many more fluorescent dots without corresponding blood dots on the colour photograph (reproduced as Fig. 3b). Fig. 3c shows the same area of retina two months later. Some of the fluorescent dots that were seen earlier cannot now be identified.

Discussion

As Fig. 3 demonstrates, fluoresceinretinography reveals many more fluorescent dots than correspond to the blood dots on the colour pictures. This has been previously shown by other authors (Novotny and Alvis, 1961; Dollery *et al.*, 1962; Scott *et al.*, 1964). These authors state that these fluorescent dots are microaneurysms filled with fluorescein. Such fluorescein-filled microaneurysms have also been observed in hypertensive patients without diabetes, according to Hodge and Dollery (1964).

It is possible that the fluorescent dots which can be identified with blood dots are microaneurysms, but on the other hand it is also possible that the fluorescein may have leaked out of the vessel at the point where the haemorrhage occurred, and the fluorescent dot thus represents a small haemorrhage and not a microaneurysm.

Most of the fluorescent dots cannot be identified with blood dots on the colour pictures, and there is no proof that these dots are microaneurysms. However, there are some characteristics which indicate that at least some of the fluorescent dots are microaneurysms—they all have the same dimension as the blood dots. Some of them seem to connect with the vessels. Furthermore some of them appear and disappear simultaneously with the filling and clearing of fluorescein in the vessels.

The last group may indicate microaneurysms with open connexion with the vessels. Fig. 1, however, demonstrates an example of a cotton-wool exudate where the fluorescein-filling took place very quickly—namely, in the arteriolar phase.

Fig. 2 shows a fluorescein dot of the same size as a blood dot, but this fluorescein dot could be identified with a nearly invisible small exudate. Moreover, it appeared and disappeared simultaneously with the filling and clearing of fluorescein in the vessels. Scott *et al.* (1964) have made a similar observation, and, according to Friedenwald (1950),

* Hvidøre Hospital, Klampenborg, Denmark.

they interpret these single small dots as thrombosed microaneurysms. However, it seems unlikely that such a closed microaneurysm could fill with fluorescein. Since such dots are yellow and are quickly filled with fluorescein, just as are cotton-wool exudates, it is more reasonable to assume that they are, in fact, small cotton-wool exudates.

Many of the small fluorescent dots are delayed in their appearance relative to the appearance of the fluorescein in the vessels; furthermore, they disappear more slowly (Scott *et al.*, 1964). It is difficult to imagine that a microaneurysm should have the above-mentioned properties, but the cotton-wool exudates often show these features of delayed filling and emptying by fluoresceinretinography.

As Hodge and Dollery (1964) showed, the fluorescent dots may not be reproduced two months later, as can be seen in Fig. 3. The phenomenon is well known so far as the cotton-wool exudates are concerned (Esmann *et al.*, 1963) but a similar fast regression of blood dots is a rare phenomenon. They may disappear, but as a rule they persist for many months.

The fluorescent dots which seem to connect with the vessels are mainly found in hypertensive patients (Hodge and Dollery, 1964). It is, however, exceptional to demonstrate as much as

FIG. 1.—There is a vigorous, fluorescing circular area, which appeared in the arteriolar phase. At the same place on the colour print a cotton-wool exudate was seen.

FIG. 2.—The arrow points to a little fluorescent dot which corresponds to a nearly invisible exudate on the colour print.

FIG. 3a

FIG. 3b

FIG. 3c

FIG. 3a.—The fluorescein picture shows many more fluorescent dots without corresponding blood dots on the colour photograph (Fig. 3b). FIG. 3c.—The same area of retina two months later. Some of the fluorescent dots that were seen earlier cannot now be identified.

a single blood dot by ordinary ophthalmoscopy of these patients. By microscopy of the retina it is also rather unusual to find microaneurysms. These facts combined with the observation that fluorescent dots disappear quickly during treatment of the hypertension (Hodge and Dollery, 1964) indicate that the fluorescent dots are due to exudates which are not ophthalmoscopically visible.

Summary

Some of the results from fluoresceinretinography of diabetic patients are demonstrated, and the interpretation of some of the observations is discussed. It is concluded that most of

the small fluorescent dots which appear in diabetics and hypertensive patients can be interpreted as small exudates rather than microaneurysms.

REFERENCES

- Dollery, C. T., Hodge, J. V., and Engel, M. (1962). *Brit. med. J.*, **2**, 1210.
 Esmann, V., Lundbæk, K., and Madsen, P. H. (1963). *Acta med. scand.*, **174**, 375.
 Friedenwald, J. S. (1950). *Amer. J. Ophthalm.*, **33**, 1187.
 Hodge, J. V., and Dollery, C. T. (1964). *Quart. J. Med.*, **33**, 117.
 Novotny, H. R., and Alvis, D. L. (1961). *Circulation*, **24**, 82.
 Scott, D. J., Dollery, C. T., Hill, D. W., Hodge, J. V., and Fraser, R. (1964). *Brit. med. J.*, **1**, 811.
 Personal communication, Jensen, H. J., Århus Kommunehospital.

Galvanic Stimulation of the Tongue as a Prognostic Index in Bell's Palsy

O. A. PEIRIS,* M.D., M.R.C.P., M.R.C.P.E.D.; D. W. MILES,* M.B., CH.B., B.SC., M.R.C.P.

Brit. med. J., 1965, **2**, 1162-1163

Bell's palsy is one of the commonest lesions seen in neurological practice. Between 35% and 45% of patients affected show evidence of denervation, which may result in troublesome sequelae such as continuing muscle-weakness, "associated movements," and "crocodile tears" (Taverner, 1955, 1959). The correct management of such patients depends on the early detection of denervation. Electrical conduction studies enable a firm prognosis to be made seven days from the onset of the lesion (Langworth and Taverner, 1963). The present report concerns the value of anodal galvanic stimulation of the anterior two-thirds of the tongue in making an even earlier prognosis in Bell's palsy.

Material and Methods

Bell's palsy was diagnosed according to the following criteria (Taverner, 1955): (1) the sudden onset of complete or partial paralysis of the muscles of expression on one side of the face; (2) the absence of any symptoms or signs of any other disease of the central nervous system; (3) the absence of any symptoms or signs of any disease of the ear or posterior fossa; and (4) the absence of herpetic vesicles.

Studies were made of 69 cases, the method of electrogustometry (Krarup, 1958a) being used. A 120-volt dry battery is connected through a circuit which is shown in the Diagram. The cathode was held firmly by the patient and the anode was applied to the tongue. This stimulating electrode was a circular disk 7 mm. in diameter, made of platinum. The stimulus was applied to the lateral aspect of the tongue over the area that lies against the canine tooth when the tongue is placed on the floor of the mouth. The electrode was applied to the normal side first and the stimulus strength was increased gradually until the patient was able to appreciate a distinct acidic taste. In some cases it was necessary to do this a few times so that the patient understood what was required. The same procedure was then repeated on the side showing the paralysis and any alteration in threshold was noted. In 90% of cases the patients could appreciate stimuli of 30 μA or less on the normal side. In the remaining 10% the threshold on the normal side was raised above 30 μA , and this was associated with heavy smoking, coated tongues, or low intelligence. Since a comparison of the

two sides was the essential observation no regard was paid to the threshold level on the normal side provided that this could be recorded. The disparity in threshold on the two sides was recorded, and the values thus obtained were classified into three groups in the following manner: (1) those patients with an elevation of threshold to more than 100 μA above the normal side; (2) those in whom the threshold was at least 10 μA higher than on the normal side; and (3) those with a normal response; here there was no measurable difference in the thresholds on the two sides.

Clinical assessment of function in the face was estimated in terms of a percentage of normal function in the orbicularis oris, the orbicularis oculi, and the frontalis muscles. Electromyography and facial-nerve-conduction studies were done in every case. All patients in the series were seen at the end of three or more months in order to assess the outcome of the lesion. In this follow-up special care was taken to note the presence of associated movements in the face and fibrillation and "blink-burst" activity in the electromyogram. The pre-

* Department of Medicine, University of Leeds.