Origin and course of an afferent component of the facial nerve within the central nervous system[1]

P. M. Fuller

Department of Anatomy, University of Louisville, School of Medicine and Dentistry, Health Sciences Center, Louisville (Kentucky 40201, USA), 18 February 1978

Summary. The mesencephalic nucleus of trigeminal gives rise to an afferent component of the facial nerve. This nucleus contains large unipolar afferent cell bodies which give rise to an axon which courses caudally through the brainstem and exits via the facial nerve to terminate distal to the stylomastoid foramen.

The precise origin and course of the proprioceptive component of the facial nerve within the central nervous system remains one of the numerous unanswered questions in the field of neurology. It is known that the mesencephalic nucleus of trigeminal is concerned with proprioceptive impulses from teeth and jaw muscles[2]. It has also been reported that this nucleus gives rise to the proprioceptive component of the facial nerve, but that the fibres exit via the trigeminal nerve, and then join with the facial nerve at the periphery[3]. Another report suggests that the proprioceptive fibres of the facial nerve originate from the main sensory nucleus of trigeminal and pass via the trigeminal branches to their point of termination[4]. Studies in the cat have suggested that the proprioceptive component of the facial nerve may arise from the spinal trigeminal nucleus[5]. It is generally accepted that the facial nerve does possess a proprioceptive component; however, the question remains as to its origin and course through the central nervous system.

Part of the difficulty in establishing the precise origin and pathway of the proprioceptive pathway has been technical in nature. Many of the anatomical studies have relied on the retrograde changes occurring within a nucleus resulting from lesioning the facial nerve. Using cobalt iontophoresis, one of the newer neuroanatomical methods, we are able to trace the origin of a particular pathway and its course through the central nervous system.

Materials and methods. Using the cobalt procedure as reported previously[6-8], the animals were anesthetized, then perfused with saline. The brains were removed and pinned to the bottom of a petri dish lined with Sylgard (Dow-Corning). A plastic tubing suction electrode is then attached to the cut end of the facial nerve and filled with a 30% solution of fresh cobalt chloride. Using a voltage divider, a DC current of 10–12 μA is used to drive the cobalt into the cut end of the nerve. The experiment is run for 24 h at room temperature, the suction electrode is removed and the brain is treated with a 4% solution of ammonium sulphide for 30 min. The brains are then embedded in egg yolk and serial sections cut at 33 μm. Alternate sections are stained with either the Tyrer and Bell[9] procedure or with a Cresyl Violet stain.

Results and discussion. Previous studies have shown that the mesencephalic nucleus of trigeminal in the frog is located in the rostral $^1/_3$ of the optic tectum[10]. The cells are located in layers 2 and 4 and are composed of large unipolar neurons[11]. Work in our laboratory has confirmed that this nucleus gives rise to the proprioceptive component of the trigeminal nerve[6].

Filled cells within the frog mesencephalic nucleus (figure 1) were identified after filling the 7th nerve, thus indicating that they give rise to fibres which enter the facial nerve. These cells are located in the lateral portion of the ipsilateral mesencephalic nucleus. The fibres pass laterally from the nucleus toward the edge of the tectum and then turn abruptly ventral and caudal. Followed caudally the fibres pass lateral to nucleus isthmi and the cerebellar nucleus. The fibres then course further caudally and pass lateral to the motor nucleus of the trigeminal nerve, and ultimately exit with the motor fibres of the 7th nerve.

A very careful dissection was made of the 7th nerve prior to filling of the nerve with cobalt so as to insure that the results would be from filling only the 7th nerve, and not aberrant filling of a portion of the trigeminal nerve. Also,

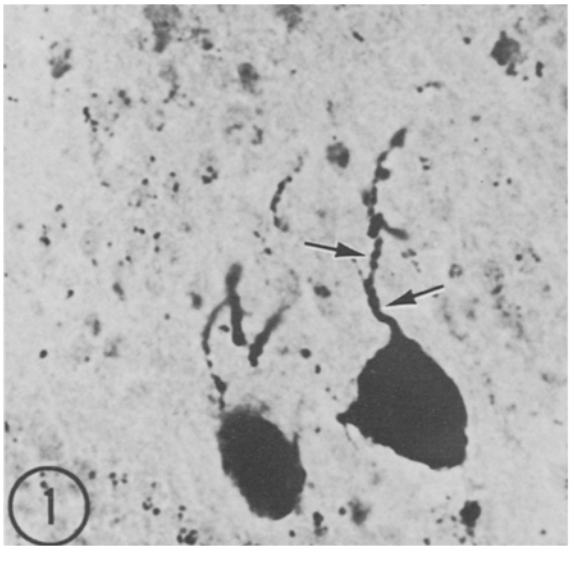

Fig. 1. Photomicrograph of 2 cells within the frog mesencephalic nucleus after filling of the 7th nerve with cobalt. Note that the cells are completely filled with the dark precipitate. Arrows indicate the axon arising from the cell body. × 200.

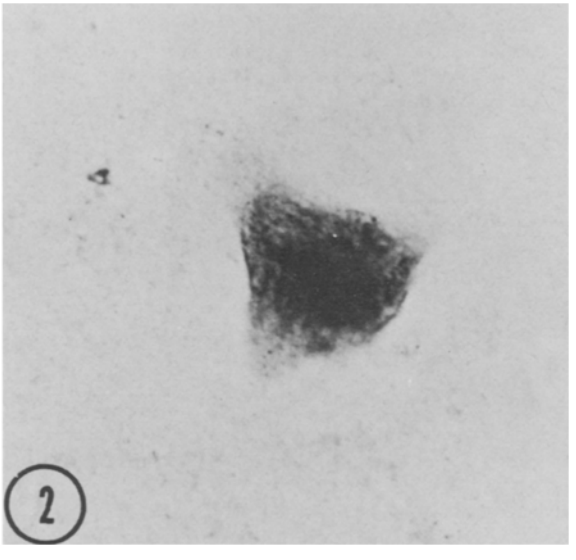

Fig. 2. Photomicrograph of a cell within the mouse mesencephalic nucleus after filling of the 7th nerve distal to the stylomastoid foramen. The cell body is labelled with the cobalt, indicating that it is giving rise to a fibre which enters the facial nerve. × 200.

close examination of the sections showed that only the motor nucleus of the 7th nerve was filled, and no portion of the motor nucleus of trigeminal was filled - thus indicating that the 7th nerve was the only nerve filled with cobalt.

In the mouse, the mesencephalic nucleus of trigeminal is located in the mesencephalon at the level of the oculomotor nucleus and lateral to the 3rd ventricle. As a result of filling of the facial nerve in the mouse, scattered cells within the ipsilateral mesencephalic nucleus were identified as giving rise to afferent fibres which enter the facial nerve (figure 2). The fibres course ventrally from the nucleus and pass medial to the lateral lemniscus on the ipsilateral side of the brain. Passing caudally through the brainstem, the fibres pass lateral to nucleus locus coeruleus and lateral to the motor nucleus of trigeminal. Further caudally they exit the brainstem with the motor fibres of the facial nerve.

To insure that only the afferent fibres from the facial muscles of the mouse were filled, the 7th nerve was isolated at its exit from the skull at the stylomastoid foramen, and filled with cobalt at this point. Therefore, the labelling of the cells within the mesencephalic nucleus of trigeminal are from afferent fibres which probably arise from the facial musculature.

We have been able to demonstrate that the facial nerve, in at least 2 species, contains an afferent component which arises from the ipsilateral mesencephalic nucleus of the trigeminal complex, and that the fibres pass caudally through the brainstem to exit with the motor branch of the facial nerve. It is quite possible that this afferent component may indeed be the proprioceptive fibres of the facial nerve.

1 This work was supported by National Aeronautics and Space Administration Grant No. NAS 9-15213.
2 R.D. Smith and H.Q. Marcarian, J. dent. Res. *47*, 616 (1968).
3 J.J. Baumel, Archs. Otolar. *99*, 34 (1974).
4 A. Tokunaga, M. Oka, T. Murao, H. Yokoi, T. Okumura, T. Hirata, Y. Miyshita and S. Yoshitatsu, Med. J. Osaka Univ. *9*, 397 (1958).
5 N. Iwata, S.T. Kitai and S. Olson, Brain Res. *43*, 662 (1972).
6 D.J. Prior and P.M. Fuller, Brain Res. *64*, 472 (1973).
7 P.M. Fuller and D.J. Prior, Brain Res. *88*, 211 (1975).
8 M.R. Martin and C.A. Mason, Brain Res. *121*, 21 (1974).
9 N.M. Tyrer and E.M. Bell, Brain Res. *73*, 151 (1974).
10 E. Weinberg, J. comp. Neurol. *46*, 249 (1928).
11 K. Rubinson, Brain Res. *19*, 3 (1970).

Coffee and cola beverage consumption as heart disease risk factors in men[1,2]

Marge F. Stanton, R.A. Ahrens and L.W. Douglass

Food, Nutrition and Institution Administration Department, College of Human Ecology and Dairy Science Department, College of Agriculture, University of Maryland, College Park (Maryland 20742, USA), 26 January 1978

Summary. The relationship of coffee drinking and cola beverage consumption to recognized heart disease risk factors was studied in 365 adult men. Cola beverage consumption was not positively related to any risk factor. Heavy coffee drinking seemed to reflect a life style characterized by infrequent eating, which was significantly correlated to all the heart disease risk factors studied.

The possible involvement of coffee drinking as a risk factor in heart disease has been controversial. In 1973, the 'Boston collaborative drug surveillance program' reported that patients hospitalized following a heart attack had drunk more coffee prior to hospitalization than patients hospitalized for other reasons[3]. Their analysis showed that this effect of coffee was independent of cigarette smoking. Analysis of the Framingham[4] and Kaiser-Permanente[5] epidemiological studies failed to find any relationship between coffee drinking and heart disease death rate, except for that connected with associated cigarette smoking. Maugh has suggested that all 3 studies are invalid because they failed to take into account the consumption of cola beverages, which supply more caffein to many people than does coffee[6]. The present study specifically measured cola beverage consumption in men, 17-70 years of age, and compared its association with known heart disease risk factors to that of coffee drinking.

Methods and materials. Records were obtained from men who attended a 'heart screening' project of the Heart Association of Southern Maryland. Of these, only complete records from men who were taking no prescription medication were analyzed. That left 365 subjects, aged 17-70 years, who completed a medical history and a dietary questionnaire. Screening personnel recorded sex, age, height, weight and measured blood pressure by arm cuff and sphygmomanometer. A 12-h fasting blood sample was analyzed for serum cholesterol[7] and triglyceride[8] levels. The dietary questionnaire was a 7-day recall that attempted to get an estimate of average weekly consumption by the subject. The short method of dietary analysis[9] was used to judge serving sizes and total food intakes, which were then adjusted to a daily basis. Body fatness was estimated by calculating the ponderal index (height divided by cube root of weight) of each subject. Subjects with the smallest ponderal index tended to be most obese. Frequency of eating was estimated by adding the meals and snacks reported by each subject and dividing by 7 to get a daily average. Smoking refers to cigarette smoking. Several biomedical data description and step-up linear regression computer programs were used to select variables that were closely related to one another.

Results and discussion. The correlation coefficients of selected risk factors (cigarette smoking, body fatness, blood pressure, serum cholesterol and triglyceride levels) with selected dietary components are presented in the table. Cola beverage consumption was not positively correlated with any of the heart disease risk factors measured. There were significant negative correlations with blood pressure and body fatness ($p < 0.05$). However, it seems probable that this is a reflection of the strong negative correlation ($r = -0.29^{**}$) between age and cola beverage consumption. Coffee drinking, on the other hand, was positively correlated with age ($r = 0.14^{**}$), cigarette smoking ($r = 0.32^{**}$) serum cholesterol ($r = 0.18^{**}$) and triglyceride ($r = 0.15^{**}$) levels. This would tend to indicate that any correlation between coffee drinking and recognized heart disease risk factors is unrelated to the caffein content of the coffee. Rather, the relationship of coffee drinking to heart disease risk factors seems to be indirect and a reflection of the life style associated with coffee drinking. To that extent, our findings confirm the earlier reports from the Framingham[4] and Kaiser-Permanente[5] epidemiological studies that any effect of coffee drinking on heart disease risk