2 to 6% calcium oxide in commercial talcs. When the talc is wetted the boric acid reacts with the calcium hydroxide to produce the highly insoluble calcium borate. Absorption from dusting powder when the surface of the skin is intact is negligible and harmless, but if the surface is broken a dusting powder is probably not the best treatment anyway.

It should be recognized that there is no single blunderbuss method of treating napkin rashes, because they are not all of the same type or due to the same cause. The common ammonia dermatitis, with its diffuse erythema where the wet napkin was in contact with the skin, leaving the creases clear, is due to the liberation of ammonia from the urine by urea-splitting organisms from the stools. This is encouraged by leaving the napkins on in a wet state for prolonged periods. A variety of preparations offer effectual treatment, including quaternary ammonium compounds. A useful lotion is one containing 4% tannic acid in 0.1% proflavine. It is essential to see that the napkins are changed sufficiently frequently—and that the mother has enough of them to make this possible.

Isolated vesicles in the napkin urea are likely to be due to monilia. They may be numerous, and may coalesce. Nystatin ointment usually clears the rash satisfactorily. The so-called psoriasiform napkin dermatitis has been the subject of recent studies.[7,8] A. G. Fergusson and colleagues[7] write that it begins as a napkin dermatitis and later spreads to affect the trunk, limbs, and scalp. They describe 52 cases, and consider that it is a response to monilia in a child with a seborrhoeic type of skin. The growth of monilia may be the result of the administration of antibiotics by mouth. The condition does not respond to nystatin ointment but does to corticosteroid ointment, such as betamethasone five parts and Lassar's paste one part.

All these rashes have to be distinguished from seborrhoeic eczema ; in this the skin of the flexures of the napkin area is attacked, and there are characteristic clear areas round the red patches. It usually responds to triamcinolone-and-chlorhydroxyquinoline ointment with equal parts of Lassar's paste.

## Facial Myokymia

Twitching of the face is common and usually benign. The simple tic or habit spasm is a movement of the facial musculature, often bilateral, which appears involuntary ; but it is probably in the first instance consciously performed to relieve tension, and as the habit becomes established the patient is progressively less aware of the movements. Focal twitching of one corner of the mouth occurs episodically in focal Jacksonian epilepsy, and is usually easy to recognize. Recently it has become clear that certain persistent facial dyskinesias, in which there are pursing, chewing, and other bizarre movements of the lips and tongue, may result from long periods of treatment with phenothiazine drugs. The movements of chorea, whether of the Sydenham's or Huntington's type, frequently involve the facial musculature, and grimacing movements, which are slower but not otherwise dissimilar from those of chorea, are also seen in hepato-lenticular degeneration (Wilson's disease).

More common, and often wrongly diagnosed, is clonic facial spasm (hemi-facial spasm). This benign condition, which is always accentuated by tension or embarrassment, affects only one half of the face ; it often begins with an intermittent and irregular twitching of the muscles around one eye, and later may spread to those around the mouth, so that intermittent sharp and momentary contractions of the entire facial musculature on one side may ensue. The probable cause is compression or irritation of the facial nerve within its bony canal, but regrettably there is no certain method of determining the site of the lesion, so that operations designed to decompress the affected nerve are rarely successful. A similar type of intermittent twitching may be seen following a severe Bell's palsy in association with the facial contracture which can occur as a result of partial regeneration in the facial nerve.

Yet another interesting and unusual form of involuntary movement of the facial musculature has been called "facial myokymia." F. Andermann and his colleagues[1] described four personal cases and found seven in the literature ; of these 11 cases, eight were probably suffering from disseminated sclerosis. More recently, W. B. Matthews[2] has described seven episodes of myokymia occurring in five patients whom he had observed personally over a six-year period. He points out that the onset of the condition is usually abrupt and that it seems to affect the whole musculature on one side. Patients often complain that the face feels screwed up or swollen, and some when looking in a mirror have observed flickering movements. The appearance is highly characteristic in that all of the muscles on the affected side of the face appear to be in a state of slight contraction ; the palpebral fissure is narrowed, the angle of the mouth drawn up, and the lips are pursed. There is a continuous flickering from frontalis to platysma ; this differs from fasciculation, as it recurs with extraordinary rapidity and appears to pass over the face in rapid undulating waves. In Matthews's cases, the duration of the disorder varied ; while showing some waxing and waning in intensity, it usually lasted for anything between three weeks and six months. One patient found all sounds seemed louder in the affected ear, but none had abnormalities of taste sensation. Electromyography showed rhythmical spontaneous discharges occurring in the affected muscles.

As Matthews points out, this syndrome of facial myokymia differs from the form of benign fasciculation in the limbs to which the name "myokymia" has also been given.[3] This benign fasciculation is often widespread and "coarse" and is commonly accompanied by anxiety, muscle cramps, and excessive sweating. Electromyography in such cases usually shows grouped discharges of two or three motor units repeating at intervals, and thus differs from the spontaneous fasciculation activity observed in motor neurone disease, in which the potentials are usually single. Facial myokymia must also be distinguished from the benign myokymia of the lower eyelid which is often seen in normal people.

Facial myokymia has never been described after facial palsy ; its onset is usually abrupt and eventual recovery appears to be invariable, though more than one attack may occur, and both sides of the face may be successively affected. Though there is no histopathological proof, it has been suggested that the lesion is probably intramedullary, lying close to the facial nucleus. H. Oppenheim[4] first described the disorder in patients with multiple sclerosis, and E. H. Lambert and his colleagues[5] reported it in a case of pontine

[1] Andermann, F., Cosgrove, J. B. R., Lloyd-Smith, D. L., Gloor, P., and McNaughton, F. L., *Brain*, 1961, **84**, 31.
[2] Matthews, W. B., *J. Neurol. Neurosurg. Psychiat.*, 1966, **29**, 35.
[3] Denny-Brown, D., and Foley, J. M., *Trans. Ass. Amer. Phycns*, 1948, **61**, 88.
[4] Oppenheim, H., *Neurol. Zbl.*, 1917, **36**, 142.
[5] Lambert, E. H., Love, J. G., and Mulder, D. W., *Newsl. Amer. Ass. Electromyogr., Electrodiagn.*, 1961, **8**, 8.

glioma. In one of the five patients described by Matthews the diagnosis of multiple sclerosis was incontrovertible, and in another it was probable; in the other three the facial myokymia occurred as an isolated event. Matthews's experience suggests that the condition is not uncommon and it is to be hoped that histopathological studies will eventually clarify the pathogenesis of this mysterious but benign affliction.

## Predicting Coronary Disease

All men over 40 have coronary atheroma, and when severe it leads to angina, myocardial infarction, and even sudden death. Research into its cause turned attention at an early stage to disorders of lipid metabolism.

It was soon realized that a high blood level of cholesterol has a close relation to the development of coronary atheroma. The most striking example of this is the death in their early 20s of young men and women with familial hypercholesterolaemia. Then a more detailed series of studies led to the serum lipids being separated by centrifugation and to analysis of the individual lipids. But serious objection to most of this work was the retrospective nature of its inquiry, and the fallacy of *post hoc ergo propter hoc* appeared. Now we have a prospective approach to the prediction of coronary artery disease by means of the Framingham Study in Massachusetts. In this, physicians have followed up 5,127 apparently healthy persons since 1949. The many initial measurements made included determination of the serum level of phospholipids and cholesterol, and a recent report by the directors of the study refers in particular to the predictive value of the cholesterol-phospholipid ratio.[1]

This ratio, they say, "has been especially popular as an indicator of proneness to atherosclerosis and has been interpreted extensively in lipid investigative work as a particularly meaningful value." Various studies had in fact suggested that one of the phospholipids, lecithin, was a stabilizing factor in lipid-protein emulsions which prevented deposition of atheroma. In other words, if a person had a high serum cholesterol but also a high phospholipid level (giving a low cholesterol-phospholipid ratio) he was said to be less likely to develop atheroma than a person with a high cholesterol-phospholipid ratio.

The Framingham group, in analysing a 12-year follow-up of their 5,127 persons initially free from coronary disease, have shown that the relationship of the cholesterol-phospholipid ratio to the risk of developing coronary heart disease is in fact nil, but the individual levels are related to it. The authors have divided the persons in their study according to sex and to their age when the study began. Thus, in those aged 30–49 at the start an initial serum cholesterol of under 220 mg./100 ml. carried half the average risk of coronary disease, while when the level was over 260 mg./100 ml. the risk was doubled in men and increased by half in women. The phospholipid levels were similarly related to risk of disease. The analysis showed that persons with a phospholipid level above the median were in fact slightly more likely than average to develop coronary disease during the 12 years of follow-up. Thus by means of a most careful prospective study it has been conclusively shown that cholesterol-phospholipid ratio is of no value as a predictor of coronary heart disease.

[1] Thomas H. E., jun., Kannel, W. B., Dawber, T. R., and McNamara, P. M., *New Engl. J. Med.* 1966, **274**, 701.

## Uric Acid and Behaviour

Gout has always fascinated doctors. One feature which has contributed to this interest is the high proportion of patients who are out of the ordinary. Many victims have been men of distinction, and several reports have indicated a connexion between uric acid and social or intellectual attainment. Patients with gout attending hospital clinics tend to be of a higher social class than other patients.[1] Executives have a higher mean level of uric acid in the serum than craftsmen,[2] while the intellectual level of soldiers in the United States army has been found to bear a positive relation to that of the serum level of uric acid.[3]

In the most recent study of this type G. W. Brooks and E. Mueller[4] have tried to relate the personal characteristics of 113 professors at the University of Michigan with the level of uric acid in their sera. The professors submitted to a psychosocial analysis derived from questionaries and interviews, and were quantitatively graded with respect to seven variables—namely, drive, achievement, leadership, pushing of self, range of activities, attitude towards pressure, and emphasis on research. From the combined figures was calculated a total behaviour score. The uric acid values in the professors were found to be significantly correlated with their total behaviour scores and with most of the individual behaviour patterns. Higher than average levels of uric acid were most closely associated with the personal characteristics of drive, achievement, and leadership. Full professors had a rather higher mean serum uric acid value than that of associate and assistant professors, but this difference was not significant.

Brooks and Mueller thought their results could be explained best by reference to a theory advanced by E. Orowan,[5] who postulated that uric acid, like some other purines such as caffeine and theobromine, stimulates the cortex. This is a challenging idea, but it will have to be supported by more evidence before it can be accepted. The level of uric acid in the serum is influenced by a number of inborn and environmental factors, some of which were recently reviewed in the *B.M.J.*[6] In their study of the professors Brooks and Mueller noted that there were other characteristics which showed a significant correlation with the level of uric acid, such as obesity, alcohol consumption, and a good appetite, and it seems probable that the interrelationship is a complicated one. Moreover, a raised level of uric acid in the serum is not always associated with intelligence or achievement: there is at least one small group of patients where the opposite is the case, those with the rare and recently described syndrome[7] of choreoathetosis, lip biting, and mental retardation, in which production of uric acid is enormously increased and the serum levels are very high. Nevertheless, it may well be that purine metabolism at some stage does influence or regulate the development and function of the nervous system. No doubt the question will be pursued further by pharmacological experiments.

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[2] Dunn, J. P., *et al.*, *J. Amer. med. Ass.*, 1963, **185**, 431.
[3] Stetten, D., and Hearon, J. Z., *Science*, 1959, **129**, 1737.
[4] Brooks, G. W., and Mueller, E., *J. Amer. med. Ass.*, 1966, **195**, 415.
[5] Orowan, E., *Nature (Lond.)*, 1955, **175**, 683.
[6] *Brit. med. J.*, 1964, **2**, 71.
[7] Lesch, M., and Nylan, W. L., *Amer. J. Med.*, 1964, **36**, 561.

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On 14 July Professor Hedley J. B. Atkins was elected President of the Royal College of Surgeons of England for the ensuing year in succession to Lord Brock.