adenoids were much less: snoring 18%, nasopharyngeal obstruction 15%, ear infection and deafness 18%, frequent colds 20%, and speech defects 8%. At operation 54% had insufficient adenoid tissue to warrant removal and only 19% had adenoids causing gross obstruction.

I think the opponents of adenotonsillectomy are influenced by the knowledge that the symptoms due to adenoids will clear as the child develops. I have much sympathy with this view, and I am very reluctant now to operate unless there is a history of recurrent tonsillitis, certainly not until conservative treatment has been tried for several months. Recurrent tonsillitis is a common disease of childhood; 741 children out of the 1,043 in this survey suffered from this type of infection before operation. An immunity does not usually develop with growth. Possibly because of the conservative attitude of the Cornish doctors who refer our cases, and of the surgeons, we are now admitting for tonsillectomy an increasing number of adults. The present ratio is one adult to three children, and they nearly all date the onset of their attacks in their schooldays.

Surely it is folly, if operative treatment becomes necessary, to remove adenoids alone, which we know will regress as the child develops, and leave behind the tonsils, which so frequently remain infected into adult life?—I am, etc.,

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T. M. BANHAM.

SIR,—Every discussion of this operation raises arguments on just how many or how few deaths are caused by it each year. Even if there were no mortality this would be no argument in favour of the operation which, like the once popular uvulectomy, should be discontinued.

Unfortunately one of the indications for tonsillectomy often cited is adenoidectomy. Tonsillectomy is likely to continue as long as adenoidectomy is performed. I know of no evidence that adenoidectomy decreases the incidence or favourably alters the course of acute or chronic otitis media. I have never understood why the adenoids, which are centrally placed, should be blamed for blocking the Eustachian tubes, which enter the pharynx on either side. I was not therefore surprised, as Mr. Stuart R. Mawson says he was (8 February, p. 379), that the King's College Hospital group found that adenoidectomy led to no significant reduction in the incidence of otitis media.

Your leader writer (25 January, p. 203) correctly states that "tonsillo-adenoidectomy does not prevent virus infections of the respiratory tract," but nor does it influence the incidence or prevent the complication of haemolytic streptococcal infection, and I do not understand what is meant by "it is important to distinguish this group from true recurrent tonsillitis." There are contra-indications. In one place it is written "No doctor doubts the value of the operation when performed for right reasons," implying that there are right reasons, yet later "... there are no finite pathological or clinical criteria by which to judge." Also I cannot agree with the conclusion, which is that "The final medical opinion must be the

surgeon's." Surely it is not medical opinion but medical treatment that is required.—I am, etc.,

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"Hyperphenylalaninaemia"

SIR,—The majority of recent articles and correspondence¹⁻³ continue to use the term phenylketonuria when the emphasis has now turned entirely to the testing of blood for raised levels of phenylalanine. Only Hutchison⁴ has moved away from the horde and heads his article "Raised Phenylalaninaemia in the Newborn."

May I suggest that the time has come to change the name of the disease and offer the title "hyperphenylalaninaemia" with the abbreviation, which we will need, of "hupa"?—I am, etc.,

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Eating and Corticosteroid Levels

SIR,—We were most interested in Miss Katharine Montagu's observations that plasma 11-hydroxycorticosteroid (11-OHCS) concentrations rose dramatically after a normal lunch in healthy volunteers (28 December, p. 833), but we are rather concerned by her suggestion that breakfast might interfere with stimulatory tests of hypothalamo-pituitary-adrenal axis function.

It has not been our practice to fast patients prior to any of the standard tests of hypothalamo-pituitary-adrenal axis function other than the insulin hypoglycaemia test. In previous publications from this centre,¹⁻⁴ where these tests have been employed utilizing the standard fluorimetric technique of Mattingly⁵ for the estimation of plasma 11-OHCS, we have noted that basal plasma 11-OHCS levels have tended to be slightly higher than in other published series⁶ using the same technique. We attributed this to the fact that tests in this centre are normally commenced at 8.30 a.m., which is probably an hour or so earlier than is usually the case elsewhere, and is at a time when the plasma 11-OHCS level falls rapidly in the course of its diurnal rhythm. However, it is also one hour after the patients have breakfast.

We have therefore estimated the plasma 11-OHCS in 13 patients with a variety of chronic rheumatic diseases immediately prior to breakfast and again 60 minutes later at 8.30 a.m. None of the patients had evidence of renal, hepatic, nor endocrine disease and none had had recent corticosteroid therapy. Each patient remained at rest in bed, and smoking was not permitted. The results are listed in the Table and show a small drop in plasma 11-OHCS following breakfast. The results of a further similar study on nine patients whose plasma 11-

OHCS were estimated 30 minutes before and 60 minutes after a standard hospital lunch are also listed and show a minimal and insignificant rise.

Plasma 11-OHCS Levels ($\mu\text{g./100 ml.}$) in Two Groups of Patients Before and One Hour After a Meal

Before Breakfast	After Breakfast	Before Lunch	After Lunch
27	21	14	13
17	15	13	15
26	18	12	23
8	10	21	15
27	17	17	15
22	18	28	13
14	10	13	16
23	19	3	8
16	14	18	26
19	18		
6	5		
22	16		
17	15		
Mean 18.8 $\pm$ Standard error 1.9	15.1 1.3	15.4 2.3	16.0 1.9

We are unable to explain adequately the difference in our findings from those of Miss Montagu. She does stress the fact that when the plasma-11-OHCS level is over 16 $\mu\text{g./100 ml.}$ food seems to be ineffective in causing a rise, and many of our patients had levels above 16 $\mu\text{g./100 ml.}$ However, the Mattingly method would be expected to give higher values than the method of Spencer-Peet,⁷ as no correction is made for non-specific fluorescing substances. However, if the discrepancy was due to the lesser specificity of the Mattingly technique one would have to postulate a fall in the non-steroidal fluorescing substances of the plasma after food, which seems rather improbable. It is of course possible that hospital patients are simply not as sensitive as healthy young adults to changes in plasma 11-OHCS after a meal, or is it possible that Miss Montagu's volunteers sneaked a quick cigarette or pint of beer with their lunch? At all events, perhaps the effect of food on plasma "cortisol" levels should be further investigated in various situations using a really specific method.⁸

In the meantime we feel that stimulatory tests of hypothalamo-pituitary-adrenal axis function can be safely conducted after breakfast. Indeed, fasting may act as a stress and lead to higher basal levels.—We are, etc.,

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