So every little helps, and there is some interesting information in this report. A random sample of 619 women and 440 men in the London borough of Richmond had their heights and weights measured, and they recorded whether they considered themselves to be "underweight," "overweight," or "suitable" for their height. These heights and weights were also analysed and classified (under the same category headings) according to the Metropolitan Life Tables with, perhaps, a somewhat excessive tolerance for mild and moderate overweight. Mild overweight included people whose weights were 11-20% greater than the mean of the medium frame in the tables and moderate overweight included a range of 21-30% above this mean. Since even for young men an apparently slim sedentary man will have about 15% of his body weight as fat, mild overweight would therefore have a range of 11-20% plus 15% or up to 35% of the body weight as fat, and moderate overweight would have a range for men of up to 45% of fat. For women, the equivalent values would be about 10% greater—mild overweight being up to 45% fat, and moderate up to 55% fat. These limits may be realistic for contemporary British society but they are hardly physiological when one considers that the mean proportion of fat in the body of well-fed, physically active men in less advanced societies may be only about 10%.

This questionable classification in the article somewhat distorts the conclusions, since not surprisingly 50% of the Richmond sample were classed as of "suitable" weight. Despite the bias due to the classification 35% of the men and women were overweight, by far the greater majority being over 30 years of age. There were no differences with social class in the men, and only the women from the poorest socioeconomic groups, more than 50% of whom were overweight, were significantly different from the others.

The attitude of the individual to his or her own body weight in general showed reasonably accurate self-assessment. About 60% of those classed as underweight thought they were of suitable weight—and they were almost certainly correct. Almost all the overweight women, of any age and of any social class, knew they were overweight—and here we have objective evidence of a fact of which most men are well aware: that women never, like men, deceive themselves about their crooked nose, squint teeth, big feet, scraggy neck, too big bust, fat thighs, or obesity. There is a remarkable uniformity among the men in their ability to persuade themselves that things aren't really as bad as other more prejudiced observers might think. At all ages and in all social groups, 30-40% of the overweight men thought their weight was suitable.

The impact of the state of overweight on the individual had not always led to attempts to lose weight. Less than half of all the men (although 70% of the men in the higher social groups) had ever tried to reduce their weight, but almost 80% of the women had tried to become slimmer, with social class accounting for comparatively little difference.

There are some salutary conclusions which may be drawn from these findings. With few exceptions, women who are overweight are well aware of the fact, and most of them are sufficiently unhappy about their state to have made attempts to alter it. It seems pointless to waste time or money in an educative attempt to persuade women that obesity is undesirable—they know it already. Whether true mild or moderate obesity (though not the definition given by Ashwell and Etchell) really is undesirable or unhealthy for women is another matter—on which there is extraordinarily little evidence.

Men pose another problem. A large percentage of the men who must clearly have been obese were apparently under the delusion that their body weight was suitable, and more than half of the overweight men had never attempted to lose weight. Even moderate obesity in men is almost certainly a health hazard: so the emphasis on the undesirability of obesity, and on its treatment, might profitably shift from women (who don't need the persuasion and might not even need the treatment) to men, who clearly require both.

[1] Ashwell, M., and Etchell, L., *British Journal of Preventive and Social Medicine*, 1974, 28, 127.

Dopa Decarboxylase Inhibitors

Despite the highly effective results from treating Parkinsonism with levodopa its shortcomings include the large daily dose required, the delay in therapeutic benefit, and the number of side effects which result from its metabolism both inside and outside the brain.[1] The combination of peripheral dopa decarboxylase inhibitors with levodopa is an important advance: it allows a four- or five-fold reduction in dopa dosage, clinical response occurs within one or two weeks rather than several months, and the incidence of vomiting is cut from 80% to 15% of patients. The combination is safe and free from adverse reactions.[2] [3]

The non-specific enzyme L-aromatic acid decarboxylase is necessary for the conversion of dopa to dopamine as well as the decarboxylation of other amino acids such as 5-hydroxytryptophan to serotonin.[4] This enzyme is found in intestine, kidney, and liver, as well as in the brain.[5] The formation of dopamine from dopa can be prevented at these various sites by several inhibitors[6] of decarboxylation which include alpha methyldopa, alpha methyldopa hydrazine (carbidopa, MK 486), and N-DL-seryl-N-(2,3,4-trihydroxybenzyl)-hydrazine (Ro4-4602). In high dosage Ro4-4602 will penetrate and prevent dopamine formation within the brain. In normal dosage both carbidopa and Ro4-4602 penetrate the brain poorly, if at all, despite regional variations, so that their use results in a selective extracerebral rather than intracerebral enzyme inhibition. In contrast to these enzyme inhibitors pyridoxal-5-phosphate, the active form of pyridoxine, is a coenzyme with dopa decarboxylase and will greatly enhance plasma decarboxylase activity.[7]

About 95% of an oral dose of levodopa is metabolized outside the brain and never reaches the cerebral parenchyma, and its combination with pyridoxine will further enhance this metabolism and further decrease the effectiveness of levodopa treatment.[8] [9] In contrast, the combination of levodopa with decarboxylase inhibitors reduces the peripheral metabolism of levodopa, increases peak plasma levodopa concentration, and reduces urinary excretion of dopamine and its metabolites. A daily dose of 200 mg carbidopa will reduce the quantity of levodopa required to produce similar plasma levodopa levels to approximately 20% of that when levodopa is given alone.[10]

Combined therapy with levodopa and dopa decarboxylase inhibitors has now been used in a number of centres for up to five years.[11-14] The results of treatment with carbidopa and Ro4-4602 are similar, apart from minor dose differences. In all trials there has been a very considerable reduction in nausea and vomiting—probably due to the fact that the inhibitor does reach the medullary vomiting centres, which are outside the blood brain barrier.[15] In addition, cardiac arrhythmias caused by levodopa metabolism in the heart are reduced or avoided. The response to combined therapy is unaltered by

the intake of pyridoxine. Patients previously unable to tolerate levodopa because of nausea and vomiting can achieve adequate dosage with the combination, though in other dopa-tolerant patients the optimum response to combined treatment is no greater than to dopa alone. The central side effects of dopa are not altered by combined therapy, and dose-limiting involuntary movements occur at the same plasma dopa levels, the incidence of neuropsychiatric disorders is similar, and possibly there may be problems with the on-off effect earlier in the course of combined treatment. This effect is the abrupt onset of akinesia followed by the equally sudden return of the therapeutic response to levodopa; it may occur in up to 50% of patients after several years of treatment on levodopa alone.

At present only a single fixed ratio of dopa to decarboxylase inhibitor is available, and patients who take a low dopa dosage may not gain any advantages from the combination. Tablets containing 10, 50, and 100 mg of levodopa with a fixed dosage of 25 mg carbidopa should soon be available.[16] Treatment of patients still requires considerable judgement by the physician in balancing the therapeutic and side effects of the different antiparkinsonian drugs, and unfortunately no treatment available at present halts the progression of Parkinson's disease.

[1] Yahr, M. D., in *Advances in Neurology*, ed. M. D. Yahr, p. vi, vol. 2. New York, Raven Press, 1973.
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[15] Lotti, V. I., in *Advances in Neurology*, vol. 2, p. 91, ed. M. D. Yahr. New York, Raven Press, 1973.
[16] Cotzias, G. C., Mena, I., Papavasiliou, P. S., in *Advances in Neurology*, vol. 2, p. 265, ed. M. D. Yahr. New York, Raven Press, 1973.

Surgery for Hemiplegia

The increasing numbers of road traffic accident victims left incapacitated with hemiplegia has stimulated interest in the place of surgery in ameliorating the condition. Many orthopaedic surgeons, faced with a clinical condition in which a severe deformity is obviously due to muscular imbalance, find the temptation to try to restore the balance by tendon transfer difficult to resist. The results of such transfers are almost invariably disappointing. Though deformity may be improved, function seldom is, because of lack of voluntary control of muscle action. The patient has little control over normally situated muscles, so it is not surprising that he cannot control them after transfer. Simple tenotomy, injection of phenol into motor nerves, flexor origin release, and tendon lengthening have all been tried with doubtful benefit.

A recent account by Braun et al.[1] of transfer of the superficialis to profundus tendon in hemiplegic arms has claimed good preliminary results. This is not a new procedure, having been used very effectively in those cases of Volkmann's ischaemic contracture with severe necrosis and contracture of the deep forearm flexors in which some contractile function remained in superficialis. By completely dividing both groups of tendons at different levels and attaching the proximal stumps of the superficialis tendons to the distal stumps of the profundus at the correct tension it is possible at one stroke to relieve the contracture and to restore some active flexion in all the digital joints. The operation is not so much a tendon transfer as a refined tendon lengthening procedure. Though the operation cannot be expected to give such good results in the spastic limb—with its lack of voluntary control—it does combine release of contracture with the possibility of retaining some active flexion of the digits by muscles which, before transfer, had a similar action. In short, one can expect some improvement in deformity with retention of whatever function may have existed before operation.

So far Braun et al. have obtained improvement in deformity in 21 of 24 patients submitted to the operation. It is depressing, however, that only three of the 24 obtained reasonable voluntary control, and these three had demonstrated some control before operation. Possibly in those patients without any pre-operative voluntary control tenotomy and limited excision of the tight tendons might have been just as effective. With such unpromising material it is doubtful whether any but the most simple surgical procedures are justifiable.

At present, then, it seems that only those patients with some degree of voluntary flexor control should be considered suitable for superficialis-to-profundus tendon transfer. Surgeons contemplating this procedure will wish to study the operative technique described in the paper, when any undue optimism should be checked by its first sentence: "Very few patients who have had strokes resulting in hemiplegia can have functional hands restored by surgical procedures."

[1] Braun, R. M., Vise, G. T., and Roper, B., *Journal of Bone and Joint Surgery*, 1974, **56-A**, 466.

Anastomotic Leakage

One of the complications which the abdominal surgeon most fears is leakage of a gastrointestinal anastomosis; it not only reflects on his technical skill and judgement but puts his patient's life at risk from spreading peritonitis, local abscess, or bowel fistula. So since the earliest days of modern surgery the technique of intestinal anastomosis has been a subject of intense interest both in clinical practice and in the experimental laboratory. Certain practical points have been learned from hard experience: anastomoses at either extremity of the alimentary canal, the oesophagus or the rectum, are the most hazardous; small bowel and the stomach usually heal well; while immediate suture in a distended obstructed colon is fraught with danger compared with its comparative safety in resections of obstructed small intestine.

The subject has been reviewed recently by Everett,[1] who stressed three considerations—the blood supply to the ends of the resected bowel, the adherence of surrounding structures, and the technique of anastomosis which is employed.

An adequate blood supply is the first essential for the healing of any wound, and the gut is no exception. Indeed gross leakage is probably due in most cases to inadequate vascularity at the cut ends of the bowel. Careful trimming of the mesentery or mesocolon may make the anastomosis look pretty, but it may result in the production of local ischaemia and consequent dehiscence. It is vital that the surgeon assures a brisk circulation before he attempts to join the bowel ends.