Numbers of colony forming units per unit area on right and left sides of tongue and cheek in patient who had undergone left parotidectomy and control

	Patient with left parotidectomy		Colonised subject	
	Right	Left	Right	Left
Cheek (test No):				
1	0	24	3	2
2	3	26	2	2
3	2	10	5	6
4	11	43	2	1
5	7	18	0	0
Tongue (test No):				
1	6	44	3	1
2	1	21	2	1
3	1	11	6	7
4	20	56	2	2
5	11	19	7	6

($p<0{\cdot}05$) and cheek ($p<0{\cdot}01$) in the patient who had had a parotidectomy on all occasions tested (table). In contrast, parallel cultures from a patient with oral candida colonisation and normal salivary flow showed no such differences.

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Events surrounding organ transplantation

SIR,—Dr J D Hill has done a great service to transplantation by describing the events surrounding donation (26 July, p 264). I have a successful kidney recipient in my family, was the father of an organ donor, and am the father in law of a failed kidney recipient awaiting a better match, and I therefore wish to express my personal appreciation of Dr Hill. He highlights three points that merit further consideration.

Firstly, transplantation is inevitably attended by strong emotion not only in the patients and their families but also in the doctors and nurses concerned. Emotion is something we are not all well able to handle, as evidenced by Dr Hill's houseman. The traditional teaching to medical students is, effectively, to switch off emotion. This negative approach is inadequate for transplantation, and we surely need to learn to acknowledge and handle strong feelings constructively rather than to suppress and ignore them.

Secondly, the serious shortage of donors means that publicity for transplantation should be welcomed, not spurned. Indeed, donors' families and recipients may well agree to events being publicised in response to sympathetic inquiry. Journalists should therefore be courted rather than shunned. This, too, is contrary to tradition. My own experience is that cooperation generates cooperation in that a willing interview with a key member of the team, providing the basis for a good story, is more likely to result in confidential matter being omitted than is a reluctantly extracted official statement that leaves the reporter desperately looking for personal detail with which to enliven his copy.

Finally, in practice Dr Hill clearly understood the mechanism of death from brain damage, but his use of the expression "cerebrally dead" seems to be a textual lapse into confusion. Irreversible loss of function of the brain stem is the key event in death. Cerebral death could mean merely a persistent vegetative state unless the word cerebral is stated to include the brain stem.

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Obese deceivers?

SIR,—Dr D A T Southgate's leading article (28 June, p 1692) contains several generalisations which require comment. To state that one should treat with scepticism all claims from obese patients that their dietary intakes are modest is, in effect, to call obese people gluttons and liars. This generalisation is not only scientifically inaccurate but also shows a lack of understanding of the obese patient's problems. Clearly to gain weight one must consume more energy than one expends but this does not imply that energy consumption is always more than that taken by another who remains lean—for example, compare cases 10 and 20 in the study by Dr A M Prentice and others (12 April, p 983)—such is the considerable individual variation in energy expenditure. The obesity prone person could rightly object to the comment that his or her intake is not "modest," and to call such individuals deceivers is unjust.

Dr Southgate has ignored many of the published reports on obesity in stating that one should no longer believe in an inherently lower metabolic requirement, for defects in energy expenditure in response to a meal,[1-5] to overfeeding,[6] and to catecholamine administration[7] have been reported by various groups of investigators in some obese as well as preobese[8,9] and postobese[4,7,10,11] individuals. Not all obese individuals show such abnormalities,[12] as one might expect in a heterogenous population, but Dr Southgate's generalisation prejudges the issue for it has yet to be established whether such abnormalities are relevant or not to the development of obesity.

Recent research has also suggested that obese patients who are mildly hyperphagic have a food intake control mechanism which retains the ability to adapt to energy changes but is "set" at an energy intake above normal.[13] The reason for this is unclear, but to classify such individuals as edacious deceivers could seriously deflect from a proper investigation of the pathophysiology of appetite control. It is therefore not surprising that obese patients are confused about energy needs and have to struggle constantly to reduce intake. Further research on appetite and energy control is required, rather than scepticism and rejection.

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Serial visual evoked potential recordings in Alzheimer's disease

SIR,—Dr A Orwin and colleagues (5 July, p 9) claim that visual evoked potentials can be diagnostic of Alzheimer's disease. In the case cited the patient had abnormal visual evoked potentials when she developed behaviour changes consistent with dementia. Other workers, however, have been unable to show abnormal flash pattern response in patients with mild dementia and have suggested that this abnormality may be seen only in severe dementia.[1] We would be interested to know whether abnormal visual evoked potentials have been noted in other patients before the development of overt dementia.

We are at present investigating visual evoked potentials in dementia and have noted that fixation is very important to obtain responses. We are therefore surprised to read that someone who was too confused to perform simple psychometric tests was able to cooperate enough to have recordings made of her pattern reversal visual evoked potentials.

The authors claim that a delayed flash with a normal pattern reversal response is specific for Alzheimer's disease but this abnormality could theoretically be seen in any disease which affects the visual association areas with relative sparing of the visual cortex.

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AUTHORS' REPLY—Dr Wright and Mr Richardson correctly point out that Coben *et al* failed to find any abnormality of the flash visual evoked potential when comparing non-patients who had been rated, using a clinical dementia rating scale, as mildly demented and non-patients rated as healthy. It may well be that this unique finding from those authors is related to the fact that they were not investigating demented patients. Nearly all other workers have found an abnormality with a delay of the flash visual evoked potential,[1,2] and we first reported this finding of a delayed flash visual evoked potential with a normal latency of a pattern reversal visual evoked potential in 1981.[3]

Although abnormal visual evoked potentials have not been noted in patients before the development of overt dementia, there is certainly a difference in the amount of delay to the flash response between patients with dementia of recent origin and those who have established dementia.[4] We would point out that our finding of a delayed flash visual evoked potential coexisting with the

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