that should be pursued between teacher, child, and parents.

There is evidence that the pendulum phenomenon is now heralding an era of "narrow punitive education associated with the bullying of children and adolescents,"³ and that this would be welcome in some quarters. Let us hope that sufficient people feel strongly enough to ensure that the next decade does not provide further inspiration for the Pink Floyd.

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¹ McCann E. *Schoolmaster and Career Teacher* December 1977:168-72.

² Koerner JD. *Reform in education*. New York: Delacorte Press, 1968:159-60.

³ Whitehead T. *World Medicine* October 1975:77-81.

Physical indicators of emotional abuse in children

SIR,—A letter (17 November, p 1290) from Dr W R Guirguis requested the recording of physical indicators of emotional abuse in children.

During the last 10 years I have been the medical officer to a primary school for mildly educationally subnormal children where most children have some degree of maladjustment. In the last four years I have also been medical officer to an all-age school for maladjusted children. Both these schools are in Southwark, south-east London, where there is a preponderance of children from social classes IV and V. From this experience may I offer my observations on a sign which I consider is frequently present in maladjusted children? This is a very characteristic indrawing and thinning of the upper lip, which is to me instantly recognisable.

I have never heard anyone else comment on it either verbally or in writing. I have christened it, for local convenience, "Ewart's sign." Not all maladjusted children show this sign but all children who show it are in my opinion maladjusted and, while I have not yet attempted to correlate any sign with emotional abuse, most of the children exhibiting it come from backgrounds of social and emotional deprivation. In addition, it is known that many children who have been overtly abused and neglected suffer emotional damage and because of associated intellectual difficulties and maladjustment require education in special schools¹⁻³. There seems sufficient evidence at least to consider the possibility of emotional abuse in any child who exhibits this characteristic facies.

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¹ Lynch MA. *J Child Psychol Psychiat* 1978;19:175-180.

² Roberts J, Lynch MA, Duff P. *Ther Educ* 1978;6:25-31.

³ Martin HP, Beezley P. *Develop Med Child Neurol* 1977;19:373-87.

The uneasy oesophagus

SIR,—Your leading article "The uneasy oesophagus" (19 January, p 136) began by quoting the words of "the physician out to reassure his worried patient who believes that he has heart disease." It is often the physician himself who first needs that reassurance. The oesophagus has, of course, long been recognised as a source of symptoms masquerading as cardiac pain but the dif-

ferential diagnosis is not always easy. On the one hand, cardiac pain cannot always be excluded by a normal 12-lead exercise electrocardiogram and a normal coronary arteriogram; on the other hand, there has been no entirely satisfactory means of establishing oesophageal spasm as the cause of the intermittent symptom in question.

We have recently reinvestigated 21 patients who had previously had normal coronary arteriograms but continued to experience "angina," leading in many cases to enforced retirement from work and in all except one case to at least one readmission with suspected myocardial infarction.¹ In 16 (76%) of these patients we were able to show that the pain was not cardiac but due to oesophageal spasm. The pain appeared indistinguishable in character from that of cardiac pain, often though not predictably related to exertion, relieved by nitrites, and frequently associated with minor electrocardiographic changes. Full cardiologic investigation confirmed the absence of coronary artery stenosis, ergometrine-induced coronary artery spasm, metabolic evidence of myocardial ischaemia, and other cardiovascular disease. Ergometrine (500 µg intravenously) did, however, reproduce the pain. This offered the means of confirming that the pain was due to oesophageal spasm by oesophageal manometry (recorded through perfused triple-lumen tube), which showed the development of characteristic abnormal motility patterns coincident with the development of the pain.²

The majority of these patients had no other symptoms suggestive of oesophageal disorder and had normal routine barium swallows. Oesophageal spasm appears to be more common than we had appreciated. We suggest that ergometrine provocation offers a means of confirming the diagnosis, though it should be used with caution until coronary artery disease has been excluded.

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¹ Dart AM, Alban Davies H, Dalal JJ, Ruttley MST, Henderson AH. *Eur Heart J* (in press).

² Dart AM, Alban Davies H, Lowndes RH, Dalal JJ, Ruttley MST, Henderson AH. *Eur Heart J* (in press).

SIR,—Our hollow viscera cause much misery and your recent leading article (19 January, p 136) "The uneasy oesophagus" is important to any practising doctor on several counts, but particularly because you stated that oesophageal pain in the chest can be mistakenly considered to be "anginal" by patient or doctor. After 40 years of listening to accounts of chest pains I am sure that oesophageal pain is the commonest to be misdiagnosed, regrettably, as cardiac; and we can do few greater disservices to our patients than diagnose ischaemic heart disease wrongly—but it is commonly done.

Whatever the precise mechanism of the oesophageal pain it is certainly connected with oesophageal contraction, as I have proved at least to my own satisfaction. I have experienced the symptom for at least 15 years, beginning in my 50s, and it can be most unpleasant and, until diagnosed for what it is, most alarming. It mimics anginal chest pain

closely, being retrosternal and radiating to both shoulders; but it is not related to exertion. It is prone, as you say, to occur at night and awakens the victim, and is not typically related to food. I have experienced it when driving the car, without any warning, and it is distracting. Duration is about two to 15 minutes.

My reason for writing to you is that there is a very simple practical method of proving that the pain is arising in the gullet and not the heart: important to me personally because I also have coronary artery symptoms. The trick when you or your patient experiences the pain is to swallow saliva hard, and as the wave of deglutition which you have started descends the pain will subside in seconds; it will usually come back rapidly, but can be repeatedly dispatched by the act of swallowing. Anxiety thus dispelled and equanimity restored, the pain can be tolerated: in the morning small hours, when even the cardiologist's ego is at its lowest, a small tot of neat whisky seems especially efficacious. The whole clinical entity reminds me of proctalgia fugax, an even more catastrophic example of gut contractions gone mad, but obviously without the "angor animi" of the introspective cardiac.

I like your designation of the "uneasy" oesophagus: it matches well the "unhappy colon" of Sir Arthur Hurst. May I suggest "perverted rectum" to take the blame for the atrocious symptom. How fortunate that the pains of the gullet and rectum are fugaceous, though alas not those of the colon.

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Troxerutin in Raynaud's syndrome

SIR,—We read with interest the paper of Dr A J Dods and others (10 November, p 1186) on the treatment of patients with Raynaud's syndrome by weekly plasma exchange. We would like to report our own results in similar patients, also based on an attempt to improve the rheological properties of blood, using intravenous and oral administration of hydroxyethylrutosides (troxerutin) in the form of Paroven capsules. It has previously been shown that this compound in high doses will reduce red cell aggregation and whole blood viscosity at low shear rates and will increase red cell deformability.^{1 2}

We have so far studied a group of seven patients with severe Raynaud's syndrome: three could be classified as having primary Raynaud's syndrome (no demonstrable cause found); one had scleroderma; one had involvement of hands and feet from Buerger's disease; one had vibration-induced disease of the arteries of the hands; one had a vasculitis involving both hands and feet with additional "vibration disease." All patients had severe pains in the digits and some degree of ulceration. They were all investigated by conventional angiograms and also with our recently developed dynamic fluorescein angiography technique.³⁻⁵ These techniques showed organic changes in the arteries of the hands in five patients; the other two patients showed pronounced vasospastic tendencies.

The patients received hydroxyethylrutosides intravenously at a dose of 2-3 g/day (in two injections) for two to four weeks, followed by oral treatment at a dose of 3-6 g/day (in three to four doses) for maintenance therapy. During the parenteral treatment period all patients showed clear clinical improvement, with fewer episodes, healing of ulcers, and a pronounced relief of pain. Five patients showed a definite improvement in peripheral blood flow as shown by fluorescein angio-