babies should thereafter be given either 25 μg daily or 1 mg weekly orally from 1 week until 3 months of age. All suspected cases of haemorrhagic disease of the newborn were collected by a surveillance system similar to that in Germany and Britain and based on active monthly reporting by all paediatric departments. From 1 October 1992 until 31 August 1994 no cases of serious bleeding or intracranial haemorrhage were reported. Only one case could be validated as idiopathic late haemorrhagic disease of the newborn.[3] This was in a healthy 8 week old Turkish boy who was admitted with persistent nasal bleeding. He had been exclusively breast fed and had never received extra vitamin K.

Thus over 23 months only one case of idiopathic late haemorrhagic disease of the newborn was reported, in a boy who had not received prophylaxis. In the Netherlands an incidence of 7/100 000 was found before prophylaxis was introduced[4]; this is comparable to the incidence in Britain[5] and Germany.[6] As about 200 000 babies are born in the Netherlands each year and about 70% are breast fed it is striking that we did not encounter a single case in which prophylaxis failed; the surveillance system obtained a mean response rate of 90% (range 87-93%). In contrast, many cases of failure of oral prophylaxis were found in Germany, where a response rate of 77-88% was achieved.[2] We assume that this is because only two or three doses are given in Germany. In accordance with our data, the British Paediatric Surveillance Unit stated in 1993 that a considerable proportion of babies in Britain probably received repeated oral doses and that it had not been notified of any failure (unpublished report).

MARLIES CORNELISSEN
Trainee paediatrician

Department of Paediatrics,
University Hospital Nijmegen,
PO Box 9101,
6500 HB Nijmegen,
Netherlands

REMY HIRASING
Paediatrician

Dutch Paediatric Surveillance Unit,
PO Box 124,
2300 AC Leiden,
Netherlands

1 Newburn M, Dodds R. Vitamin K for neonates. *BMJ* 1994;**309**: 668-9. (10 September.)
2 Von Kries R, Göbel U. Oral vitamin K prophylaxis and late haemorrhagic disease of the newborn. *Lancet* 1994;**343**:352.
3 Von Kries R, Hanawa Y. Neonatal vitamin K prophylaxis. Report of the scientific and standardization subcommittee on perinatal haemostatis. *Thromb Haemost* 1993;**69**:293-5.
4 Widdershoven JAM. Vitamin K deficiency in infancy. Nijmegen: University of Nijmegen, 1991. (Medical thesis.)
5 McNinch A, Tripp JH. Haemorrhagic disease of the newborn in the British Isles: a two year prospective study. *BMJ* 1991;**303**: 1105-9.
6 Von Kries R, Göbel U. Vitamin K prophylaxis and vitamin K deficiency bleeding (VKDB) in early infancy. *Acta Paediatr Scand* 1992;**81**:655-7.

Should obesity be treated?

Patients with sleep apnoea should be treated

EDITOR,—We believe that there are two further aspects to the problem of whether obesity should be treated.[1] Firstly, conditions in which obesity is life threatening and not just a marginal statistical risk must be considered. We frequently see patients with the obesity hypoventilation syndrome and respiratory failure. In these dangerously obese patients weight loss is critical if life expectancy is to be greater than just a few months. We take an aggressive approach to dieting in this condition and generally achieve a good response.

Secondly, obese patients who are a potential hazard to others should be treated. An appreciable number have the sleep apnoea syndrome, in which an excess of road traffic accidents occur because patients fall asleep at the wheel. The driving authorities in Britain ban people with this condition from driving public service or heavy goods vehicles until they have received 12 months of effective treatment. We have seen more than 20 professional drivers (of buses, taxis, and heavy goods vehicles) who are unwilling to accept investigation or effective treatment for sleep apnoea because of the potential effect on their ability to drive and hence their standard of living. We have a major moral dilemma here. Should we inform the driving authorities about these patients or should patient confidentiality come first? It is difficult to resist the temptation to blackmail patients into losing weight with threats of driving bans.

We are faced daily with the problems of managing obesity in patients with disordered breathing during sleep. We feel duty bound to take an active approach to weight loss in view of the adverse effects of obesity on the patients and the community. We strongly support J S Garrow's proposal for community based slimming clinics, although, while women find these useful, our predominantly male patients with sleep apnoea decline to attend them.

ASHLEY WOODCOCK
Consultant respiratory physician
PATRICIA STONE
Staff grade physician

Sleep Laboratory,
North West Lung Centre,
Wythenshawe Hospital,
Manchester M23 9LT

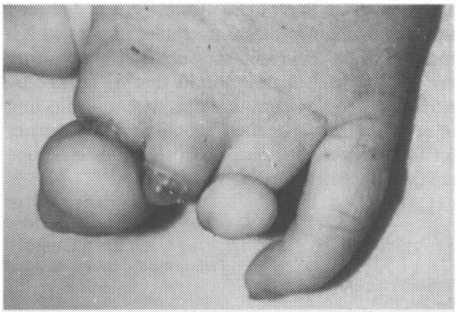
1 Garrow JS, Wooley SC, Garner DM. Should obesity be treated? *BMJ* 1994;**309**:654-6. (10 September.)

Amniotic band sequence in child of thalidomide victim

EDITOR,—Andrew P Read[1] and Richard W Smithells[2] dismiss W G McBride's hypothesis that thalidomide might be a mutagen.[3] After this correspondence was published we saw a child with limb reduction defects born to a thalidomide victim.

The baby had the following isolated anomalies of the left hand: almost complete absence of the third and fourth fingers, with necrotic tissue attached to the tip, while the second finger had a deep constriction ring at the proximal phalanx with a hypertrophic distal segment and hypoplastic nail. Annular constriction was also present between the first and second phalanxes of the thumb and was associated with a fibrous remnant; the fifth finger was normal (figure).

Pregnancy had been uneventful; ultrasound examinations did not show any limb defect, but a probable amniotic band was noted at the 22nd week of gestation. The mother, who was 34, had first been seen in 1986 when she received genetic counselling; her history showed maternal intake of thalidomide during the first trimester of pregnancy in 1959. Physical examination showed defects typical of thalidomide victims: bilateral symmetrical reduction defects of the arms (complete absence of the left forearm and hand with severe hypoplasia of the distal segment of the left humerus; absence of the right forearm with fusion of the hypoplastic humerus with a bony stump, possibly the radius, and the presence of only one finger) and shortening of the left femur, the rest of the legs being normal.



Limb reduction defect in baby born to thalidomide victim

The birth prevalence of limb reduction defects in our region (north east Italy malformation registry, a EUROCAT local registry—EUROCAT is a European network for epidemiological surveillance of congenital anomalies) was 5·3/10 000 newborn babies during 1981-93, when 570 872 live births and stillbirths occurred. Three of the 304 cases were familial: two babies had a split hand or split foot malformation and one had a defect not due to thalidomide. In the same period only 10 cases due to amniotic bands, all sporadic, were registered. These data are not significantly different from those reported by other registries.[4,5]

This is the third report of limb reduction defects in children of thalidomide victims; to our knowledge no anomalies other than limb reduction defects have been noted. These three reports raise the possibility that thalidomide could be involved in the cases. Read pointed out that thalidomide is unlikely to be a mutagen because a mutagen is not specific and thus malformations other than limb reduction defects should also be expected. Limb reduction defects could, however, be more easily reported than other defects because they are evident to the doctor, especially when a parent has a limb reduction defect. Furthermore, the hypothesised split hand or split foot deformity and Holt-Oram syndrome in the two cases reported by McBride could be the result of a new mutation.

Finally, although these three cases might have arisen by chance, a causal relation between limb reduction defects in the offspring of thalidomide victims and parental exposure to thalidomide cannot be excluded because the three cases are statistically unexpected on the basis of available epidemiological data and because the offspring of thalidomide victims are few (owing to the limited number of the victims and their presumed reduced fitness).

Thus retrospective and prospective studies should be carried out, especially because thalidomide is still used to treat leprosy.

R TENCONI
Professor of medical genetics
M CLEMENTI
Academic research assistant
L NOTARI
Resident in paediatrics
V R LO VASCO
Resident in medical genetics

Servizio di Genetica Medica,
Dipartimento di Pediatria,
University of Padua,
35128 Padua,
Italy

1 Read AP. Thalidomide may be a mutagen. *BMJ* 1994;**308**:1636. (18 June.)
2 Smithells RW. Thalidomide may be a mutagen. *BMJ* 1994;**309**: 477. (13 August.)
3 McBride WG. Thalidomide may be a mutagen. *BMJ* 1994;**308**: 1635-6. (18 June.)
4 EUROCAT Working Group. *Surveillance of congenital anomalies 1980-90.* Brussels: EUROCAT Central Registry, 1993. (EUROCAT report No 5.)
5 Froster UG, Baird PA. Amniotic band sequence and limb defects: data from a population-based study. *Am J Med Genet* 1993;**46**:497-500.

Resuscitation and patients' views

EDITOR,—N J Dudley suggests that the Medical Defence Union and the Medical Protection Society should re-evaluate their advice on cardiopulmonary resuscitation and expresses the view that our advice is "both ill considered and . . . incorrect."[1] If our advice had been quoted correctly we would accept his objection, but unfortunately it was not.