information gleaned by having the protein independently labelled was not much more than had been known from studies of crude transcobalamins with labelled vitamin alone. A poor return, one might think, for having to produce, for labelling, transcobalamins purified several million-fold from several hundred litres of plasma.

More original, although also predicted, was the data obtained on the fate of the TCII following its uptake into tissues. After a 30-min time lag radioactive iodine appeared in the urine in rapidly increasing amounts and with a molecular weight of less than 1,000, indicating linkage to, at most, a small peptide. That can only mean that TCII had been rapidly degraded in the tissues. In contrast the vitamin was either retained in the tissues or returned to the blood stream, with little appearing in the urine.

Whereas it is easy to understand why the vitamin should be retained it is, as Schneider *et al.* point out, harder to explain the wasteful way in which TCII is destroyed in the process (compare transferrin). Various microorganisms appear to be able to release vitamin B_{12} from TCII and it is difficult to see why cell surface enzymes could not have evolved to do the same.

Tissue uptake of transcobalamin–vitamin B_{12} complexes probably involves pinocytosis, a process that threatens to come back into vogue in relation to receptor modulation (Raff, *Nature*, **259**, 265; 1976). Regardless of that there is little doubt that the transcobalamins are destined to receive more attention in the near future. In part that is thanks to the possibilities opened up by Allen's elegant techniques and in part it is motivated by despair. This year is the 50th anniversary of the discovery by Minot and Murphy (for which they were to share a Nobel prize with Whipple) that a diet of raw liver could control the previously fatal pernicious anaemia. But we are still at a loss to understand the biochemical basis for the prevention of that disease by vitamin B_{12}, the active component of liver. □

Central anomalies: why so strong?

from Peter J. Smith

WHEN marine magnetic anomalies were first analysed in detail in the early 1960s, it immediately became clear that their amplitudes decrease with distance from the corresponding oceanic ridge. Possible explanations for this phenomenon were not long in coming. For example, the magnetic constituents of the oceanic lithosphere newly formed along ridge axes may change chemically with time, giving rise to a gradual decrease of magnetisation. Another suggestion was that older igneous crust would be overlain by a thicker deposit of sediment; so the anomalies as measured at the ocean surface would be attenuated with respect to those observed above younger crust with less accumulated sediment.

But although these and some other processes, acting either individually or together, could possibly explain a gradual decrease in anomaly amplitude, they were apparently insufficient (at least in the form originally envisaged) to account for the precise form of decrease actually observed. For there is usually a particularly large reduction in amplitude immediately beyond the central anomaly followed by a much more gradual decrease outwards towards the continental margins. In the case of slow-spreading ridges (<30 mm yr^{-1} half-rate), for example, the central anomaly amplitude can be at least twice as high as those of near neighbours, although for faster-spreading ridges the difference is less marked.

More recently, near-bottom magnetic measurements have shown that not only does the central anomaly have a relatively high amplitude, there is often also a large-amplitude, short-wavelength (<15 km) anomaly within it. With slow-spreading ridges the latter is not generally observed separately at the water surface because of loss of resolution with distance from the ocean floor; the wider and narrower anomalies combine in such cases to give a very large central anomaly. Over fast-spreading ridges, on the other hand, the two anomalies can usually be clearly distinguished from each other.

One possible explanation of the relative strength of the wider central anomaly, first proposed by Matthews and Bath (*Geophys. J.*, **13**, 349; 1967), is that all magnetised oceanic crustal blocks except the central one are contaminated with material of opposing polarity, thereby reducing the strength of their respective anomalies. Harrison (*J. geophys. Res.*, **73**, 2137; 1968), on the other hand, concluded that this phenomenon would be insufficient, suggesting that there must also be a 'demagnetising effect' outside the central region. Klitgord (*Earth planet. Sci. Lett.*, **29**, 201; 1976) has now taken up the question of the demagnetising effect in the particular context of the narrower central anomaly. From a new analysis of both surface and near-bottom magnetic data from many different spreading ridges, she concludes that the narrower anomaly is due to a zone of high magnetisation coinciding with the region of most recent extrusion. The absolute strength of this magnetisation is regarded as that to be expected from newly-formed basalt recently quenched in seawater. Its large magnitude relative to that of the adjacent older basalt, however, is attributed to the fact that the latter rock has undergone low-temperature oxidation.

This is not the first time that oxidation has been seen as the cause of decreasing anomaly amplitudes. Klitgord's point, however, is that in the light of recent work oxidation can be invoked to explain not only the gradual reduction of anomaly amplitude away from a ridge but also the more sudden drop at the edge of the central anomaly. The recent work concerned is partly that of Butler (*J. geophys. Res.*, **78**, 6868; 1973) who concluded that small stable single-domain titanomagnetite grains become superparamagnetic when oxidised to titanomaghemite and thus lead to a greater reduction in magnetisation than the oxidation of pseudo-single-domain or multi-domain grains, and partly that of Watkins *et al.* (*Earth planet. Sci. Lett.*, **8**, 322; 1972) who found that fresh pillow basalt has a zone of fine grains immediately below the glassy outer rim.

The argument is that many of the fine grains in this zone will be single domains with high magnetisation, which means that they will contain a high percentage of a pillow's total magnetisation. A given amount of alteration in the zone will therefore have a much greater effect on the overall magnetisation than the same amount of alteration in the larger-grained pseudo-single-domain interior of the pillow. Moreover, being both fine-grained and close to the exterior, the zone will be much more susceptible than the interior to alteration in the first place. Rapid oxidation of the fine-grained zone is thus envisaged as the cause of the sudden amplitude drop outside the narrower central anomaly, whereas slower oxidation of the interior is seen as the cause of the more gradual amplitude decrease away from the ridge.

The widths of the narrow anomalies in the ridge systems studied by Klitgord range from 2 km to 10 km, which compare with several tens of kilometres for the main central anomalies. The magnetic relationship between the two anomalies never clearly emerges, however. At one stage Klitgord suggests that "the narrow magnetisation high probably does not account completely for the difference in amplitude between the central anomaly and the flanking anomalies", implying that it accounts for some, and perhaps even a high (but unspecified) proportion, of the difference. On the other hand, it is acknowledged that in some cases the narrow anomaly does not exist at all.