Britton and Gresty suggest that the sharp low frequency peak may result from an increase in the output of a mechanism that contributes to physiological tremor. In an analysis of the tremor of 127 healthy subjects the averaged spectrum resembled that of a resonant system with broad-band forcing.[2] There was no evidence of a specific input at a low frequency. However, as we noted in our report, a few apparently healthy subjects do have a substantial low-frequency peak in their tremor spectrum. We do not know whether this is a symptom of inchoate disease or a physiological (but in our experience rare) oscillation.

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## Intrapartum fetal monitoring

SIR,—Dr Westgate and colleagues' findings (July 25, p 194) suggest that fetal electrocardiographic waveform (FECG) monitoring reduces the proportion of deliveries for fetal distress. We are doing similar research but so far our results differ from those of Westgate et al: the number of fetal blood samples needed in labour was reduced ten-fold in our study if ST analysis was used to guide management. There was no reduction in the number of operative interventions for fetal distress, and there was an equal number of babies with metabolic acidosis at delivery in each group (pH < 7·2, base deficit > 10).

There may be several reasons for this difference: firstly, although our management guidelines for labour in the trial are broadly similar to those of Westgate et al, monitoring in the FECG group is based wholly on the ST waveform. Conventional cardiotocography (CTG) is not included since it has not been shown to be useful in successive randomised trials[1] and the search for other indices of fetal wellbeing should not be hampered by the use of the CTG. Second, the senior obstetrician in our study withdrew 8% of labours from the FECG group. Third, we found that interference with the ECG signal in the second stage of labour from maternal muscles during pushing led to variation in the signal; this reduced the reliability and interpretability of the ST analysis. On average, only 64% of the second-stage traces printed had ST analysis, and a smaller percentage of the second-stage trace was accompanied by a normal check ECG assuring an acceptable signal for analysis. Prospective assessment is vital in such clinical decisions.

Lastly, technical difficulties with monitors or signals were dealt with by an expert, dedicated team from the labour ward and the medical physics department at Plymouth. Without this immediately available expertise, such difficulties could hamper the use of FECG monitoring by clinical obstetricians. We, too, look forward to improvements in on-line computer analysis techniques, and are continuing our study.

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## Chemoresistance of *Plasmodium falciparum* in central Africa

SIR,—The appearance of drug-resistant strains of *Plasmodium falciparum* in subsaharan Africa has made malaria control more difficult. Our organisation (OCEAC), with the government health services of the six member states, maintains epidemiological surveillance of chemoresistance. In-vivo studies of autochtonous







symptom-free carriers are done with WHO eight-day testing guidelines.[1] Febrile patients in towns were tested with the in-vitro radioisotope micromethod.[2]

The results of in-vivo testing suggest that the level of chloroquine resistance has stabilised or may even be regressing. In patients given 25 mg/kg over three days, the rate of chloroquine resistance in Brazzaville, Congo, was 40% in 1985, 38% in 1986, and 34% in 1990,[3,4] and in Yaoundé, 59% in 1987, 28% in 1988, and 30% in 1989.[5] The results of in-vitro testing were similar. In Yaoundé the Armitage test for trends showed a significant decrease in the rates of chemoresistance from 1987 to 1991 (p = 0·04):

| | *1985* | *1987* | *1988* | *1989* | *1990* | *1991* |
|---|---|---|---|---|---|---|
| *Chloroquine* | 72 | 110 | 45 | 83 | 87 | 28 |
| | *(0%)** | *(60%)* | *(60%)* | *(55%)* | *(42%)* | *(40%)* |

*Number tested and % resistant ($Cl_{50}$ > 100 nmol/l).

Chloroquine resistance also stabilised in Brazzaville: 59% in 1985, 60% in 1987, and 50% in 1990 (Pearson's $\chi^2$, not significant).

Amodiaquine remains effective if the dosage is increased from 25 mg/kg to 35 mg/kg over three days. In 1990, the rate of in-vivo resistance was only 7% in Brazzaville and 4% in Yaoundé.[3–5] Seven years of data in Yaoundé have shown that quinine also remains effective:

| | *1985* | *1987* | *1988* | *1989* | *1990* | *1991* |
|---|---|---|---|---|---|---|
| *Quinine* | 35 | 81 | 44 | 79 | 87 | 36 |
| | *(0%)* | *(1%)* | *(2%)* | *(9%)* | *(3%)* | *(4%)* |

$Cl_{50}$ > 450 nmol/l.

Similar results have been noted for more recently developed antimalarial drugs. In 1991, in-vitro resistance of mefloquine and halofantrine were found, respectively, in only 8% and 4% of strains tested.

The only real problem of chemoresistance is with chloroquine, and the rate of chloroquine resistance has stabilised. These findings are difficult to explain by the classic hypothesis;[6] there has not been an important movement of populations and no change in the vector (well documented in Yaoundé)[7] or in selective drug pressure.[8] Perhaps we are witnessing an encouraging threshold.

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## Impact of BCG on tuberculous meningitis in France in 1990

SIR,—BCG given in childhood has little or no impact on the overall transmission of the tubercule bacillus in a population,[1] as measured by the annual risk of infection (ARI)—ie, the proportion of people infected or reinfected each year by the bacillus. BCG's direct protective effect in children constitutes, however, the main benefit of a vaccination programme. The reduction in incidence of