	No	Serum Magnesium Concentration					
		mmol/l.			mmol/100 g protein		
		Mean	S.D.	Significance of difference from normal mean	Mean	S.D.	Significance of difference from normal mean
Group A	151	0·77	0·05	P<0·001	1·04	0·08	P<0·001
Group B	16	0·79	0·05	N.S.	1·02	0·10	P<0·02
Group C	59	0·78	0·05	P<0·001	1·04	0·10	P<0·01
All epileptics	226	0·78	0·05	P<0·001	1·04	0·09	P<0·001
Control subjects	95	0·81	0·04		1·08	0·07	

Group A: Patients treated with phenytoin.
Group B: Patients treated with phenobarbitone or primidone.
Group C: Patients treated with phenytoin and phenobarbitone or phenytoin and primidone.
N.S. = Not significant (P>0·05).

tion, like calcium absorption, tends to increase after vitamin D administration;[2] and vitamin D deficiency is accompanied by impairment of both calcium and magnesium absorption.[3] It is known that children with florid rickets often exhibit hypomagnesaemia in addition to hypocalcaemia.[4] After completing our recently published article on anticonvulsant osteomalacia[1] we have investigated the possibility that epileptic patients on anticonvulsant therapy show hypomagnesaemia.

Serum magnesium was determined by atomic absorption spectrophotometry (Perkin-Elmer, model 403) and serum protein refractometrically in the 226 epileptics and 95 control subjects, details of whom have been given.[1] Mean duration of anticonvulsant treatment was seven years. The coefficient of variation of duplicate measurements of serum magnesium was 0·9% and of serum protein 0·3%. The results are given in the table. The epileptics had serum magnesium concentrations significantly lower than the control subjects, whether the concentrations were expressed as mmol/l. or as mmol/100 g serum protein. Fourteen and 12% of the patients had a serum magnesium concentration below the normal range (mean±2 S.D.) in terms of mmol/l. and mmol/100 g serum protein respectively. Accordingly, the lower serum magnesium concentration in the epileptics was not due to differences in serum protein concentrations and protein-bound magnesium. Serum magnesium was not correlated to serum calcium nor to the duration of anticonvulsant therapy.

Lowering of the magnesium concentration in extracellular fluid would tend to increase neuromuscular irritability and is therefore probably an undesirable side effect of anticonvulsant therapy. The clinical significance of such minor degrees of hypomagnesaemia in epileptic patients remains to be established.—We are, etc.,

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Adverse Reactions to Alclofenac

SIR,—The intent of Dr. D. Mansel Jones's letter (26 January, p. 160) is fully supported by this company, which has co-operated with his colleagues in the exchange of information on adverse reactions.

Our data sheet on alclofenac is currently under revision, but present circumstances will produce a delay in the distribution of this to prescribers. May we, therefore, use your columns to point out that this company considers the role of alclofenac is in the treatment of the chronic arthritides, where it is being shown to be of particular value and safety in long-term treatment. To our knowledge, all adverse reactions have occurred in the first three weeks of treatment, while treatment over months and years has not been accompanied by any sign of toxicity. We would appreciate receiving any information that appears to contradict this statement.—I am, etc.,

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Fluoride and Osteoporosis

SIR,—We feel compelled to reply to the comments by Drs. J. Dequeker and A. Burssens (3 March 1973, p. 551). They make three main criticisms of our report on the treatment of osteoporosis with fluorides[1]: they question the diagnosis in our cases, our choice of fluoride dosage, and the validity of our measurements. All of our 11 patients had the syndrome of postmenopausal osteoporosis as defined by Albright, and they did not have osteomalacia. We have previously reported[2] that osteoid borders are somewhat wider in osteoporotic persons than in control persons of the same age, but the width is still significantly less than that found in clinical osteomalacia.

One of the purposes of our study was to determine the optimum dose of sodium fluoride for effective treatment. The dose was chosen as 50 mg of sodium fluoride daily because this was approximately halfway between the ineffective dose, less than 45 mg, and the maximum, 60 mg, which may lead to an undesirable effect on mineralization of new bone. Monitoring serum fluoride values might be of value, particularly because the intestinal absorption of fluoride is variable, but this is both tedious and impracticable in a large group. As Drs. Dequeker and Burssens state, renal sclerosis decreases fluoride elimination. Our patients were noted to have normal renal function. Indeed, the use of sodium fluoride and, more particularly, the administration of large doses of vitamin D and calcium would be contraindicated if there were any evidence of impaired renal function.

Finally, Drs. Dequeker and Burssens have made an error in questioning the validity of our quantitative values. Estimates of error are expressed as percentages of the absolute values. In this case the absolute values of our measurements are also percentages (percentage of total surface). Thus the increase in bone formation that we reported, 4·4% in absolute terms, is approximately 100% in terms of the percentage differences. In addition, measurement errors are generally random and the P value of less than 0·001 indicates a highly significant, non-random difference between the before-treatment and after-treatment values for bone formation.—We are, etc.,

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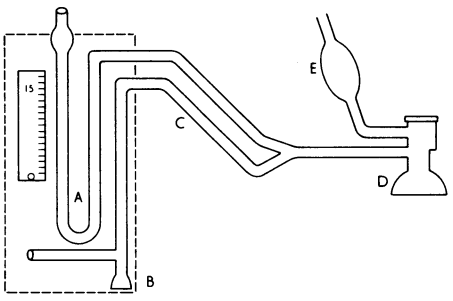
New Constant Positive Pressure Respiration Apparatus

SIR,—Gregory *et al.*[1] described a method whereby constant positive pressure respiration is made possible for the newborn infant, making use of the simple apparatus described by them. There are, however, two major disadvantages in Gregory's method: (1) tracheal intubation in the neonate for an extended period of time is hazardous; and (2) the aneroid manometer is adversely affected by high humidity, and personal observation has shown that very often the pressures indicated are incorrect. The former pitfall may be avoided by the use of the Bennet face mask, and the latter by using a water manometer.

A simple and highly effective apparatus (see fig.) has been designed and has been in use in our department for more than a year. It consists of: a U-tube (*a*), with scale for reading off the positive pressures attained in the system; an escape valve (*b*) for regulating these pressures; connecting tubes (Bird) (*c*) from the above to the Bennet face mask; (*d*) and an anaesthetic bag and connecting gas line (*e*) from an air/O_2 blender.



The U-tube and escape valve are assembled on a plastic board fitted with a Gabler snaplock to fit a Gabler wall rail. The Bennet face mask is applied snugly to the patient's face. The constant positive pressure is adjusted according to the needs of the patient.

The apparatus has been used with success in our neonatal units and is a very effective, simple, and cheap method of treating the idiopathic respiratory distress syndrome of the neonate weighing more than 1,500 g. We