apparatus of breathing, and "mismatch" no more than that at least one ventilatory variable is outside the normal range of values. In other words, the subject or patient is unhealthy. To say that in all cases of dyspnoea there is "length-tension inappropriateness" or "mismatch" is to say little more than that dyspnoea is a symptom of ill-health. Conversely, identification of "length-tension inappropriateness" or of "mismatch" is not diagnostic of ill-health. An inspiratory effort against a closed glottis is an example of extreme "length-tension inappropriateness," and voluntary hyperventilation an example of extreme "mismatch" between ventilation and lung perfusion, but neither condition need cause breathlessness or dyspnoea.

But the main criticism of these expressions is not a semantic one. It is that they are in danger of hampering our understanding and investigation of the physiology of breathlessness. We need to measure the factors associated with breathlessness and dyspnoea; yet there are no units of inappropriateness or mismatch (how about the "Odd"?), and therefore no way of assessing their contribution to respiratory sensation. The reason that we do not understand the cause of dyspnoea is that we cannot yet measure all the variables, or determine their interrelationships, which underlie respiratory sensation. The length-tension relationships of respiratory muscles could be measured, and also many of the ventilatory variables, which may be matched or not; but other factors, such as afferent nervous activity and psychological influences, defy quantitative analysis. In the meantime the use of "length-tension inappropriateness" and "mismatch" may

delude us into thinking that we understand a process because we can label it. Both expressions are more appropriate as the jargon of marriage guidance counsellors than as that of medical scientists.—I am, etc.,

New College,
Oxford.

J. G. WIDDICOMBE.

Massive Overdose of Adrenaline

SIR,—I would like to question the use of trimetaphan camsylate (Arfonad) in the treatment of adrenaline overdosage as reported by Dr. M. A. Lewis (7 October 1967, p. 38) and supported by Dr. B. J. Freedman (21 October 1967, p. 171). Trimetaphan camsylate is a rapidly acting antihypertensive agent which acts predominantly by ganglionic blockade and may actually increase pressor responses to catecholamines. The treatment of choice is surely the use of specific alpha and beta blocking agents such as phentolamine (Rogitine) and propranolol (Inderal). In individuals with normal hearts the provision of alpha blockade is particularly important. In the case described by Dr. Lewis it is likely that the use of phentolamine was the important therapeutic procedure. The use of combined alpha and beta blocking agents has recently been reported to be effective in the treatment of pheochromocytoma,¹ which is a naturally occurring model for therapeutic overdosage with catecholamines.—I am, etc.,

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Montreal, Quebec, Canada.

LORNE KATZ.

REFERENCE

- ¹ Crago, R. M., Eckholdt, J. W., and Wiswell, J. G., *J. Amer. med. Ass.*, 1967, 202, 870.

Nail-gun and Masonry Nail Accidents

SIR,—The current resurgence of reports of serious injury from industrial nail-guns (30 December, p. 784, and 20 January, p. 181) reminds me that it is exactly six years since the danger of these tools was first stressed in these columns.¹⁻³ Colleagues have since told me of their own experiences of such injuries, which are clearly far from rare. The real point is that lethal industrial nail-guns still require no licence, can still be used by any untrained, careless, or irresponsible workman, may still lack foolproof safety attachments, and have seemingly aroused no interest among our legislators. Having regard to the proposition that crash-helmet and car safety-belt regulations (for example) would not exist but for medical advocacy, I urge all doctors concerned by the problem to combine their data and forces to lobby Members of Parliament so that an appropriate Bill is formulated before further tragedies accrue from this preventable hazard of modern life.—I am, etc.,

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Guy's Maudsley Neurosurgical
Unit,
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REFERENCES

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- ² Oldfield, M. C., *ibid.*, 1962, 1, 262.
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SIR,—May I add to your list of nail-gun accidents (30 December, p. 784, and 20 January, p. 181) four cases of injury using the

so-called masonry nail but not in a gun? The nails may be driven into masonry with a hammer, and, unless struck absolutely vertically, they are easily fractured and the broken piece rebounds with tremendous force.

Case 1.—A plumber was driving a masonry nail into a concrete block when the nail fractured and he was struck in the right eye by the broken piece. This entered the eye at the limbus and actually punched a quarter segment out of the lens, the remains of which were left in situ. Portions of iris and vitreous lay on the cheek, and the end of the piece of nail could be clearly seen. The eye was removed and a one-inch (2.5 cm.) length of a one-eighth-inch (0.3 cm.) nail was recovered from it.

Case 2.—A masonry nail broke while a do-it-yourself worker was driving it into a wall. The broken fragment rebounded and shattered the left lens of his spectacles, which were fortunately a strong plus correction. Apart from a few fragments of glass on the cornea, the eye was not injured.

Case 3.—A builder was using masonry nails in the course of his work when a fractured piece struck him in the left eye, causing a complete hyphema. When this had absorbed a tear in the iris could be seen. The eye did not recover normal visual acuity.

Case 4.—On Christmas eve a workman was fixing a shelf in his house when a masonry nail rebounded and struck him in the left eye. The cornea was ruptured from limbus to limbus across the lower one-third. The eye was bleeding profusely from the anterior chamber, but there was no evidence of an intraocular foreign body. Owing to the jagged edges of the laceration

it was not possible to effect a satisfactory repair, so a thick conjunctival flap was fashioned from below and secured over the injury with four mattress sutures. There was no resorption, and no perception of light. The anterior chamber was still full of blood, and, as it was likely that the torn iris was incarcerated in the wound, the eye was removed.

This collection of four cases in 12 months in a small non-industrial society (population 46,000) emphasizes the grave dangers of the masonry nail both in the hands of the professional and in those of the do-it-yourself worker.—I am, etc.,

FRANK R. NEUBERT.

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Guernsey.

Infantile Enteritis

SIR,—Your leading article and the preliminary communication by Dr. E. S. Anderson (3 February, pp. 263 and 293) on the recent outbreak of infantile enteritis on Teesside prompt me to describe a small investigation which we carried out last year in a Dublin children's hospital. The investigation was made because, in 1966, we had isolated multiresistant R⁺ *E. coli* from a large number of infants in this hospital.¹ We did not know if these R⁺ strains had been acquired in the hospital. Therefore we selected 22 infants who had faeces in their napkins on arrival at the hospital and no recent history of diarrhoea. Faecal swabs were taken in the admission unit to ensure that the bacteria we isolated from the swabs were not acquired in the hospital. Faecal swabs were obtained at one- or two-day intervals until the infants were discharged.

We found no *E. coli* of known enteropathogenic serotypes; all resistant strains carried transmissible R factors. The results of culture of the admission swabs are given below.

No. of admission swabs	No. without R ⁺ <i>E. coli</i>	No. with R ⁺ <i>E. coli</i>
22	7 (32%)	15 (68%)
Resistance patterns	No. with each pattern	
A S T C N	8	A = ampicillin resistance
A S T C	3	S = streptomycin "
A S T N	1	T = tetracycline "
A S T	1	C = chloramphenicol "
A T C	1	N = neomycin "
S T	1	

Fifteen of the 22 infants (68%) excreted resistant R⁺ *E. coli*. Nine of the 22 were admitted from other hospitals and these included 7 excreting resistant strains. Of 13 admitted direct from their own homes, 8 were excreting R⁺ *E. coli*; exclusion of children who had recently been in hospital or who had had antibiotic therapy left only 9 children whose intestinal flora might be expected to be normal. Of these, 6 were excreting R⁺ *E. coli*. During their stay in hospital all 22 infants at some time excreted multiple resistant R⁺ strains, some of them, presumably, acquired in the hospital. A majority (86%) of faecal specimens from children in three of the wards which housed some of our 22 children yielded R⁺ *E. coli* strains. The fact that multiresistant R⁺ *E. coli* were excreted by 6 out of 9 infants with no previous history of hospitalization or antibacterial therapy indicates that R factors are

common in the enterobacteria of the infant population of Dublin.

Dr. Anderson suggests that the R factors in the Tees-side strains had probably been transferred from non-pathogenic R⁺ *E. coli*. Our findings support his view. One possible method of preventing the spread of R factors within a hospital is to isolate all infants excreting R⁺ enterobacteria on admission. As it is a routine practice in most children's hospitals to culture the faeces of infants on admission for pathogenic enterobacteria, I suggest that a direct sensitivity test should also be done on the faeces. All infants carrying R⁺ strains should be isolated within the hospital. It would not be possible or necessary to screen for R factors, because it is most probable that if the organism is multi-resistant it is R⁺.—I am, etc.,

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Microbiology,
Royal College of Surgeons,
Dublin 2.

REFERENCE

- ¹ Moorhouse, E. C., *Irish J. med. Sci.*, 1966, 375.

Misuse of Antibiotics

SIR,—In the last paragraph of your leading article on "Transferable Antibiotic Resistance" (3 February, p. 263) you raise the question of whether or not antibiotics should be prescribed for mild respiratory infection in view of their possible effect on intestinal flora. The same question may be asked with regard to mild sporadic gastrointestinal upsets in children.

For a period of about a year between 1965 and 1966 I was in charge of a military families' clinic in Malaya. Diarrhoea was a common cause of attendance and stool specimens were obtained from all but a small minority of patients. In one child a pathogenic *E. coli* was isolated, and in one woman a clinical diagnosis of dysentery was confirmed. In all the other cases recovery followed dietary restriction with the prescription of kaolin or codeine phosphate, and no bacterial pathogens were isolated.

One wonders how often antibiotics are prescribed "for safety." It has certainly been my impression that the admission of a child to hospital sometimes appears to be precipitated by vomiting following the administration of (usually) sulphonamides. These same children get well as a rule without further antibiotics, and bacterial pathogens are not isolated.

It would be of interest and value to know how frequently antibiotics are prescribed for diarrhoea and vomiting in England, and whether recovery is in fact speeded by their use. Perhaps this would be a suitable subject for the Royal College of General Practitioners to investigate.—I am, etc.,

Cambridge Military Hospital,
Aldershot, Hants.

J. CARSON.

Fatal Embolus During "Pill" Administration

SIR,—I wish to record a case of sudden death in a young woman of 23 who had been taking an oral contraceptive containing mestranol and ethynodiol diacetate for three months. I might add that this was not prescribed by me.

Post-mortem examination revealed a large pulmonary embolus, and loosely adherent antemortem thrombus was found in both iliac veins and their tributaries. There was no other lesion present which might have predisposed to thrombosis.

The facts have been reported to the Dunlop Committee.—I am, etc.,

Cleethorpes, Lincs.

R. G. ALLEN.

Degos's Syndrome

SIR,—Mr. R. E. May's well-documented medical memorandum (20 January, p. 161) describes a case under the title "Degos's Syndrome" (malignant atrophic papulosis). The photographs illustrating the case, and the description of the skin lesions, do not substantiate a diagnosis of this rare syndrome. Degos,¹ in a private communication, maintains that the cutaneous lesions are so characteristic that the diagnosis is easily made from a photograph. Case reports such as those of Nomland and Layton,² Naylor *et al.*,³

and Winkelmann *et al.*⁴ are illustrated with photographs which show typical lesions of malignant atrophic papulosis. These are well shown in the accompanying photograph of the first reported British case.⁵

The early lesion is a pale rose papule of 2-5 mm. diameter which soon becomes umbilicated and porcelain-white in colour, though some lesions may show a yellowish scaly centre; a striking feature is the sharply defined and elevated enclosing pink halo. Ulceration of the skin is rare. The eruption occurs in crops at intervals of days or weeks and the development of a characteristic lesion with a porcelain-white centre may occur over a period of 7-10 days. The black centre and tenderness of the lesions in the case described would make most dermatologists query the diagnosis; also, in Degos's syndrome the patient is almost invariably asymptomatic in the early cutaneous phase of the disease, which may precede the onset of abdominal symptoms by months and sometimes years.

A diagnosis of Degos's syndrome cannot be upheld on the basis of a histopathology

which shows a non-specific vasculitis; in our present state of knowledge probably "allergic vasculitis" might serve as a convenient label for this most interesting case.—I am, etc.,

Hove, Sussex.

PATRICK HALL-SMITH.

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- ¹ Degos, R., personal communication, 1964.
- ² Nomland, R., and Layton, J. M., *Arch. Derm.*, 1960, 81, 181.
- ³ Naylor, D., Mullins, J. F., and Gilmore, J. F., *Arch. Derm.*, 1960, 81, 189.
- ⁴ Winkelmann, R. K., Howard, F. M., jun., Perry, H. O., and Miller, R. H., *Arch. Derm.*, 1963, 87, 54.
- ⁵ Hall-Smith, S. P., *Proc. roy. Soc. Med.*, 1964, 57, 519.

Budd-Chiari Syndrome after Oral Contraceptives

SIR,—At the time of the report by Drs. K. Sterup and J. Mosbech (16 December, p. 660) we were treating a very similar patient who had been taking the same oral contraceptive, lynoestrenol 5 mg. and mestranol 0.15 mg. (Lyndiol) for six months, and for the previous six months another oral contraceptive, Gynovlar 21 (norethisterone acetate ethinyloestradiol).

A 37-year-old housewife with two children had been perfectly well until a few weeks before admission on 3 December 1967, when she had developed a slight sensation of distension in the upper abdomen. About 24 hours before admission she had vague epigastric pain, which became severe within a few hours, radiating to the left scapular region and right hypochondrium and associated with vomiting of bile and a little altered blood. The pain continued and was accompanied by the passage of several greenish loose stools.

On admission the patient was shocked and dehydrated, with a pulse rate of 134/min. and a systolic blood pressure of 70 mm. Hg, the diastolic pressure being unobtainable. There was possibly slight icterus, and the urine contained a trace of bile and albumin. There was marked tenderness in the epigastrium and right hypochondrium, and a smooth mass could be palpated in the epigastrium. There was on admission no evidence of ascites, and bowel sounds were normal. The possibilities of a posterior perforation of peptic ulcer into the lesser sac or acute pancreatitis were suspected. The dehydration and shock were treated with intravenous fluids, and repeated gastric aspiration was performed. The serum amylase was normal (160 units/100 ml.), and the plasma electrolytes were normal except for a raised potassium of 5.2 mEq/l. and an alkali reserve of 12 mEq/l. The blood urea was 47 mg./100 ml., the haemoglobin 16.5 g./100 ml., P.C.V. 57.5%, and white count 23,000/cu. mm. with a polymorphonuclear leucocytosis. After intravenous fluids and ampicillin the patient's general condition improved over the next 36 hours, though her daily output of urine was never more than 100 ml. Two days after admission she deteriorated again, with hypotension and drowsiness, but no fetor hepaticus or liver flap. The blood urea had risen to 120 mg./100 ml. By this time she had developed a large ascites and was slightly icteric. The liver-function tests were reported subsequently as bilirubin 2.5 mg./100 ml., S.G.O.T. 2,000 R.F. units/ml., and alkaline phosphatase 18 K.A. units/100 ml. With the rapid development of ascites a diagnosis of Budd-Chiari syndrome was made, but, as the possibility of generalized peritonitis could not be excluded, a laparotomy was performed which revealed a large amount of clear ascitic fluid, a grossly enlarged, engorged purple liver, and a moderately enlarged spleen. No other organs were obviously diseased. A wedge liver biopsy was taken, which showed necrosis of most