the psychiatric disturbance in these cases was presumably relatively mild (only four were schizophrenics).

Although these figures do not prove that termination would have prevented the later ill-health of a number of the women, they do show that pregnancy was for many of them more than a temporary embarrassment. According to Höök the unwanted pregnancy and the emotional difficulties that followed were probably decisive in causing actual illness in some cases.

Since Dr. Sim is concerned about the legal risks of advising abortion, it may be pointed out that, whatever "permanent mental damage" means, it does not necessarily mean psychosis. In *Rex v. Bourne* (1938) it is clear from reports of the trial (*Lancet*¹; *B.M.J.*²) that the defendant and the psychiatric witness for the defence both stressed the risk of later neurotic illness, "anxiety states of a serious kind," which would give rise to mental instability, difficulties in achieving successful marriage, and fear of sex. The risk of later schizophrenia was alluded to briefly, the risk of puerperal psychosis or suicide not at all. The point is that, although not the only factor, the psychiatric evidence was without doubt most important in securing acquittal. If Dr. Sim believes that termination was not justified in this case, can he say in what way the data he presents in his paper support his belief?

Psychotic illness in the puerperium and emotional disorders connected with pregnancy are two largely unrelated conditions which should not be confused. In the latter the psychiatrist's problem is to assess the patient's capacity for eventual adjustment if the pregnancy continues. The fact that this is difficult and that indications for termination are not yet generally agreed does not mean that it is "bad medical practice" to advise it in certain cases.—I am, etc.,

D. W. K. KAY.

Department of Psychological Medicine,
Royal Victoria Infirmary,
Newcastle upon Tyne.

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Psychiatry and the Medical Curriculum

SIR,—The problem seems to be what to leave out. I suggest that if time is limited we might with advantage leave out the teaching of any psychological theory purporting to explain or predict human behaviour on a scientific—that is, deterministic—basis as they are non-proven. I feel that beyond the syndromes we are dealing not with matters of fact but with matters of opinion, and that this is not always made clear to the uninitiated. The only concepts which remotely resemble "underlying explanations" are mental mechanisms such as projection, dissociation, rationalization,

etc. These processes are real in the sense that they stem from the prohibitions of our traditionally authoritarian culture; what more can be taught in the way of explanations without descent into speculation and personal world view?

As Professor Denis Hill says (September 21, p. 743), if I understand him correctly, our culture is based on teleology and free will (responsibility) and not on determinism. Society clearly depends on our regarding others as responsible for their actions, and it is difficult to see how it could be otherwise. Would it not therefore be correct to say that any psychology which is not orientated around the cultural realities of authority, responsibility, and dependence and which is not teleological is blinking reality and hence no basis for a true medical psychology? And does it necessarily follow that the success of determinism in the material world proves its superiority to teleology in the human world? It seems to me that in teaching students we would do well to leave aside problems of a psychological nature which cannot in any case be separated from background problems in philosophy, history, and theology and confine ourselves to more immediate issues. It is not intended by implication to decry the value of non-specific psychotherapy, but simply to suggest a negative solution to the problem of "what psychology."—I am, etc.,

R. T. D. FRANKLIN.

St. Ann's Hospital,
Poole, Dorset.

Protopsychology and the Medical Curriculum

SIR,—Pertinax (August 10, p. 382) drew our attention to Best's work on maze learning by primitive worms with a nervous system so rudimentary that their nerve cells can hardly be distinguished from the surrounding tissue. He did not, however, refer to the "rebellious" negativism which Best found affecting the worms when the maze may have appeared to them punitively restrictive, a condition from which they only recovered when provided with an open door. This kind of emotional response is also prevalent, Best was informed, in rats, cats, and monkeys undergoing psychological study, but is a poorly understood phenomenon even in such higher animals.

The parallel with the dramatic improvement in behaviour and manageability of patients in our psychiatric hospitals in response to "open-door" policies¹ suggests that there is good reason for including such studies as Best's in the first year medical curriculum. From the worm undergraduates could then proceed to similar learning experiments on cockroaches,² and thence to sense organ values in the behaviour of dogfish.^{3,4} The amphibian stage of practical zoological dissection could be co-ordinated with a visit to laboratories undertaking the Hogen test perhaps, thus initiating the student into the role played by endocrines

in motivation, and the complexities of mammalian function, by a demonstration of the opposite endogenous mood change⁵ produced in the dog and cat by large doses of serotonin.⁷—I am, etc.,

J. P. CRAWFORD.

Ide Hill, Kent.

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Cure of Hodgkin's Disease

SIR,—Dr. E. C. Easson and Miss Marion H. Russell (June 29, p. 1704) have presented the results obtained in a series of patients with Hodgkin's disease treated at Manchester in the years 1934–56. From their data they concluded that there was good evidence that localized Hodgkin's disease is 40% curable, and that even in generalized disease "the outlook is certainly not hopeless." Drs. E. W. Emery and T. A. J. Pranker (July 13, p. 114) have criticized these conclusions and suggested that the figures do not support the hypothesis that Hodgkin's disease is curable because the slope of the survival curve at 15 years is based on "a single point." In view of this criticism it is of interest to subject Easson and Russell's data to a more sophisticated statistical analysis in order to see how strong is the evidence for the existence of a cured group.

A number of mathematical models^{1–3} have been proposed to fit the survival curves of patients treated for cancer, and have been found not only to be a reasonable fit in many series followed over long periods but also to be able to make reliable early prediction of late results. Dr. Easson and Miss Russell have very kindly made available to me their detailed data on 309 cases of localized Hodgkin's disease treated in 1934–57 (one more year than was available for full five-year follow-up when they prepared their paper) and on 158 cases of generalized Hodgkin's disease treated in 1934–47. The data were in a form which enabled me to make estimates of the fraction cured, "c," using the exponential model proposed by Berkson and Gage,² and also the extrapolated actuarial model developed by myself.³ The results of the estimates of "c" with their standard errors are shown in Table I. It is seen that using either model, estimates of "c" are obtained which are much larger than twice their standard errors and are therefore significantly different from zero. The cured group in the localized series is about a third of the total, while in the generalized series it is about one-eighth.

The goodness of fit of the models to the data has been tested, as shown in Tables II and III. In compiling these Tables, one patient in the localized group and three in the generalized group, who were lost sight of in the first year after

treatment, have been omitted, while the total deaths in each interval have been predicted using the estimated parameters of each model and the mortality rates for a normal population of the same age distribution. The withdrawals listed at five, ten, and fifteen years in Table II arise from the fact that the localized data were available in three periods, 1934-47, 1948-52, and 1953-7. Also in Table II the first three years have been grouped together because the models are not expected to be a close representation of the early part of the survival curve in a situation where the progress of disease in uncured patients is relatively slow. It may be seen that the estimated deaths are a good fit to those actually observed.

TABLE I.—Estimates of Proportion Cured

Model	Localized Hodgkin's Disease	Generalized Hodgkin's Disease
Exponential	0.36±0.05	0.13±0.03
Extrapolated actuarial	0.32±0.06	0.13±0.03

TABLE II.—Localized Hodgkin's Disease

Interval (Years)	Observed		Predicted	
	Deaths	Withdrawals	Exponential	Extrapolated Actuarial
0-3	98		101.5	99.7
3-4	17		21.3	21.0
4-5	22		17.2	16.4
At 5		83	80.0	81.5
5-7	12		12.8	12.7
7-10	9		11.1	12.4
At 10		36	33.9	34.0
10-15	5		5.2	5.9
At 15		26	25.0	24.4
Total	163	145	308.0	308.0
			$\chi^2 = 3.066$	4.064
			$P = 0.80$	0.67

TABLE III.—Generalized Hodgkin's Disease

Interval (Years)	Observed Deaths	Predicted	
		Exponential	Extrapolated Actuarial
0-1	64	57.6	66.6
1-2	31	33.1	26.4
2-3	18	19.3	16.1
3-4	5	11.1	9.3
4-5	8	6.5	5.9
5-7	5	10.4	6.5
7-15	5		7.1
>15	19		17.1
Total	155	155.0	155.0
		$\chi^2 = 4.879$	5.042
		$P = 0.30$	0.41

These results, therefore, provide strong supporting evidence for Easson and Russell's contention that some patients with Hodgkin's disease are curable. It may be argued that the estimate of "c" by a mathematical model can be unreliable if the model does not in fact represent the true state of affairs—e.g., the analysis may mistake for cured a group with a death-rate that, although low, is still greater than that in the normal population. In view of the experience of the use of these models on series of patients followed for longer periods than fifteen years, it seems unlikely that an estimate of "c" as large as 32% in local-

ized Hodgkin's disease could be obtained, if there were in fact no patients cured at all. The lower estimate of "c" in generalized Hodgkin's disease may be more suspect, but in contrast to this result I have found that similar analysis on a group of patients with inoperable carcinoma of the bronchus and on a group with stage IV carcinoma of the breast led quite unambiguously to values of "c" in the neighbourhood of zero.—I am, etc.,

J. L. HAYBITTLE.

Radiotherapeutic Centre,
Addenbrooke's Hospital,
Cambridge.

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Mortality in Heart Disease

SIR.—I was very interested to read Dr. Maurice Campbell's two articles on death rate from diseases of the heart (August 31, p. 528, and September 21, p. 712), but feel that his rather novel calculations may have led him to draw an erroneous conclusion. The crude death rate for diseases of the heart is affected by the changing population structure; a simple method of overcoming this is to compare the death rate at a particular age group for a chosen sex. The following death rates can be calculated from the information given in each of the Registrar-General's Statistical Review of England and Wales for the appropriate years.

The death rate per 100,000 living males aged 45-54 and 55-64 years for all diseases of the heart in the quinquennia 1920-4 was 149 and 400 respectively: this has risen to 249 and 754 for the quinquennia 1957-61. Put another way, the chance of a man aged 45-54 dying from heart disease rose by 67% from 1920-4 to 1957-61, and for a man aged 55-64 it rose by 89% over the same period. These changes are statistically significant ($P < 0.01$) and would indicate that at least part of the increase in coronary artery disease is real and not just a combination of an ageing population and altered diagnostic practice. The accuracy of the coding of the underlying cause of death from the death certificate affects mortality statistics, and we are at present engaged in a two-year investigation in this city which will provide information on this problem.—I am, etc.,

M. R. ALDERSON.

Bristol Investigation of Mortality,
Bristol 1.

Oestrogens and the Myometrium

SIR.—The *B.M.J.* of November 3, 1962, containing the interesting article of Dr. A. I. Klopfer and Mr. K. J. Dennis (p. 1157) unfortunately reached us with some delay. According to the findings of these authors neither oral oestriol nor stilboestrol affect the induction-delivery

interval or the length of labour. Kelly¹ showed that the intravenous administration of oestrogen did not affect spontaneous motility of the non-pregnant uterus. In other words, up to the present date, although we suspected that oestrogens had an effect on uterine activity, nobody had shown it *in situ*.

We tried oestradiol-17 β by intravenous drip in patients with retention of dead foetuses or with delayed labour. We used 100 mg. of a colloidal suspension in 500 ml. of dextrose serum (Baxter) at a rate of 10-40 drops (100 to 400 mg.) a minute and we obtained rhythmic uterine activity 30 minutes after starting the infusion (see graph). The tracing shows it as it was registered by a Sanborn pressure-transducer with a Sanborn recording Poly Viso. The intensity of

contraction varied between 15 and 20 mm. Hg and the frequency was from one every seven to eight minutes to five per minute. Generally the contractions were of the Braxton Hicks painless kind, sometimes there was a slight menstrual-like distress. In the case of dead and retained foetuses the activity was more evident and there was a higher frequency of contractions. With living foetuses and term-pregnancies maturation of the cervix was stimulated and the uterus sensitivity to oxytocin was increased. In patients with dead foetuses, although the uterine activity was increased, the maturation of the cervix was not so evident; we ascribed this to the lack of action of progesterone of placental origin.

We are sending you this note in the belief that it is the first time an increase in the uterine activity has been attained using oestradiol-17 β in intravenous drip; this may explain the important part played by oestrogens in the onset of labour, prior to the use of oxytocin. Our detailed paper will be published in the near future.—We are, etc.,

ROBERTO MARTIN PINTO.

ROBERTO A. VOTTA.

ESTEBAN MONTUORI.

HECTOR BALEIRON.

Maternity Hospital Ramos Mejia,
University School of Medicine,
Buenos Aires, Argentina.

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