Correspondence

Letters to the Editor should not exceed 500 words.

Choosing Medical Teachers. R. R. M. Porter, F.R.C.S.ED.	312
Cancer and Sex Chromosomes. P. E. Polani, F.R.C.P.	312
Thyroid Asymmetry. Jan D. Wiener, PH.D., and P. Roos, M.Sc.	312
Chemotherapy of Tuberculosis. J. Batten, F.R.C.P.	313
Propranolol in Hypertension. S. N. Tewari, M.B.	313
Antibiotic Cover in Dentistry. R. J. Vecht, M.B., and Celia M. Oakley, M.D.	314

Hypothyroidism and ¹³¹ I Therapy. F. Ellis, M.D.	314
E.E.G. Signs of Death. I. Haider, D.P.M., and others.	314
Ozaena and Iron Deficiency. I. Bernát, M.D.	315
Unusual Cause of Haematemesis. A. P. R. Aluwihare, F.R.C.S.	315
G.P. Obstetric Beds. W. R. Edwards, F.R.C.S.ED.; G. Lloyd, M.R.C.O.G.; J. M. Gate, F.R.C.S.ED.	315
Phenmetrazine Psychosis. Eugenia Negulici, M.D., and D. Christodorescu, M.D.	316
Pancreatic Extracts in Cystic Fibrosis. Lieutenant-Colonel N. G. Kirby, F.R.C.S.	316

Pain in the Face. J. Braham, M.D.	316
Oral Contraceptives and Post-partum Symptoms. P. S. Nemetz, M.D.	316
Barbiturate Poisoning. J. W. Todd, M.D.	317
Immediate Coronary Care. T. W. Preston, M.D.	317
Medical Practice Overseas. J. Fry, M.D.	317
Hospital Career Structure. H. H. Langston, F.R.C.S.	317
An Ingenious Munchausen. D. R. Seaton, F.R.C.P.	317

Choosing Medical Teachers

SIR,—Professor Dorothy Russell's "Personal View" (27 July, p. 248) touches on matters of vital importance to the future members of our profession.

The fact that there is no proper test of ability to teach, and no instruction in teaching methods either demanded from, or offered to, candidates for medical teaching posts, shows an indifference for the welfare of the student which would not be tolerated by any education authority in this country. I was (many years ago) myself pitchforked into a professorship of surgery in India, in the middle of a university term with only a few days' warning, and almost no experience of teaching. This could hardly happen in

Britain, but I have since been a humble member of committees advising on professional and on consultant appointments in this country, and the discussion was mainly on research done, and a few questions on hobbies. In a committee advising on consultant appointments I have been ridiculed when I said that the main consideration was the ability to treat patients skilfully. Research was invariably the deciding factor. I have known only two members of such committees who always took the trouble to read or look up the research articles written by candidates. For the others the "list" seemed to be sufficient.—I am, etc.,

Aberdeen.

R. R. M. PORTER.

Cancer and Sex Chromosomes

SIR,—I would like to draw attention to one other important relationship between sex chromosomes and tumours and/or malignancy arising at the site of the gonad (6 July, p. 5). In females with a Y chromosome in some or all of their cells, there is a possibly considerable risk of neoplasm, malignant or not, forming at the gonadal site and alleged to arise from testicular remnants (which cannot always be confidently identified when a tumour is present) which may exist by virtue of the presence of a Y chromosome, structurally normal or otherwise. In some cases the presence of a Y chromosome may be only presumptive, as for instance when a structurally abnormal chromosome—for example, a ring or a small fragment not quite clearly recognizable as a Y—may, on sex-chromatin and phenotypic grounds, be plausibly assumed to be one.

The frequency of these females is not known, though, among subjects with ovarian dysgenesis and allied conditions, they may be about 2–3%,¹ and I estimate that their prevalence in the general population may be of the order of one in ten thousand females. They may present with ovarian dysgenesis, pure gonadal dysgenesis, Turner's syndrome, mixed gonadal dysgenesis, testicular feminization, male pseudo-hermaphroditism of other types, or as true hermaphrodites; or they may present with a gonadal tumour. The

frequency of neoplastic change is not well known and may vary in the different clinical conditions. For testicular feminization of the classical type it has been estimated at 5% or as high as 22%.²

Most of us with experience of these cases feel that when a Y or presumptive Y is found in a female with one of the above conditions, exploration and gonadectomy, which may mean the removal of streak tissue, is the operation of choice in most cases, with serial or semi-serial histological study of the removed gonad. However, a masculinized male pseudo-hermaphrodite, reared as a male, whose testicular tissue is working well and whose gonad could be replaced in the scrotum, may require special consideration. In the case of testicular feminization, some feel that removal of the testes should take place as soon as the diagnosis is made. Others suggest gonadectomy after natural puberty has taken place, with breast development, etc., but no sex hair nor, of course, menarche. Argument in favour of procrastination is that for the patient spontaneous puberty is psychologically preferable to the artificially induced changes and that the risk of malignancy before puberty is small. Two such cases, however, are known to the writer, though unfortunately an actual risk figure cannot be given.

In all these conditions the question of

what to tell the patient and/or when and what to tell the parents is important. No rules can be laid down and much depends on the nature of the case, the manner of presentation, knowledge and profession of patients and parents. Nurses and doctors, for instance, may require a different handling from the average patient. However, as a generalization, the mention of the word "testis" to these female patients should be avoided where "gonad" would well do. Also the details of the sex-chromosome make-up—namely, the presence of a Y, though not necessarily the fact that there is a chromosome anomaly—need not be divulged, for the obvious reason that full comprehension of the implications of this, and full acceptance, may be very difficult.

May I end by saying that, in contrast, the purely XO female ovarian dysgenesis or Turner's syndrome does not seem to be at an excessive risk, though, if in such a case abdominal exploration is undertaken for other reasons, prophylactic gonadectomy may well be carried out.—I am, etc.,

PAUL E. POLANI.

Paediatric Research Unit,
Guy's Hospital Medical School,
London S.E.1.

REFERENCES

- Polani, P. E., *Guy's Hosp. Rep.*, 1967, **116**, 365.
- Mishell, quoted by Volpe, R., Knowlton, T. G., Foster, A. D., and Conen, P. E., *Canad. med. Ass. J.*, 1968, **98**, 438.
- Goldberg, M. B., and Scully, A. I., *J. clin. Endocr.*, 1967, **27**, 341.

Thyroid Asymmetry

SIR,—Thomson¹ and Orr and Ingham² recently drew attention to the remarkable frequency with which asymmetrical thyroid scans are encountered in subjects without evidence of thyroid disorders. In most of these cases the right lobe was larger than the left lobe. Artagaveytia *et al.*³ reported that single non-toxic cold, warm, and hot thyroid nodules occurred more frequently in the right than in the left lobe (62.8 and 24.4%, respectively, of 408 cases). The same was found to a lesser extent in cases of toxic adenoma.^{4–6} In thyroid hemi-aplasia, the left lobe was absent in 20 out of 26 cases.⁷

These studies prompted us to review the thyroid scans of euthyroid and hyperthyroid patients referred to our institute for thyroid diagnosis since 1966. Scans are made routinely of nearly all patients except some young children. Two categories of patients were excluded: those who previously had undergone thyroid surgery, and those with hyperfunctioning nodules as the only visible thyroid tissue on the scan. The remaining 452 scans, 318 in euthyroid and 134 in hyperthyroid patients, were further divided into four subgroups: (a) normal (no nodules, estimated thyroid weight 30 g. or less); (b) diffusely enlarged; (c) solitary nodule; and (d) multinodular. Normal thyroids were more common in euthyroid than in hyperthyroid patients: 117 out of 318 (37%) and 16 out of 134 (12%), respectively. Diffusely enlarged thyroids and those with multiple nodules were found more frequently in hyperthyroid patients.

TABLE I.—Thyroid Asymmetry in Euthyroid Patients

Subgroup	R > L	R = L	R < L	Total
Normal	56 (48%)	44 (38%)	17 (15%)	117
Diffusely enlarged	34 (60%)	12 (21%)	11 (19%)	57
Solitary nodule	47 (52%)	19 (21%)	25 (28%)	91
Multinodular	22 (44%)	11 (21%)	20 (38%)	53

R, Right lobe; L, left lobe; %, per cent of total number in subgroup.

TABLE II.—Thyroid Asymmetry in Hyperthyroid Patients

Subgroup	R > L	R = L	R < L	Total
Normal	6 (38%)	6 (38%)	4 (25%)	16
Diffusely enlarged	19 (56%)	6 (18%)	9 (27%)	34
Solitary nodule	23 (48%)	11 (23%)	14 (29%)	48
Multinodular	17 (47%)	4 (11%)	15 (42%)	36

R, Right lobe; L, left lobe; %, per cent of total number in subgroup.

The analysis of thyroid asymmetry is shown in Tables I and II. Cases in which both lobes were of approximately equal size occurred more frequently in the normal thyroid subgroups (38%) than in other subgroups (11–23%). In all subgroups except those with multiple nodules, cases in which the right lobe was larger than the left lobe were more common than those in which the reverse was true. This difference appeared to be more pronounced in euthyroid cases (137 v. 53) than in hyperthyroid patients (48 v. 27). Also, solitary nodules were found more frequently in the right lobe than in the left lobe, both in euthyroid and hyperthyroid patients (Table III).

TABLE III.—Position of Solitary Thyroid Nodules

Clinical Diagnosis	Right Lobe	Isthmus	Left Lobe
Euthyroid	39 (43%)	31 (34%)	21 (23%)
Hyperthyroid	20 (48%)	13 (31%)	9 (22%)

%, Per cent of patients with the same clinical diagnosis.

In conclusion, the finding of a high frequency of thyroid asymmetry in normal thyroid glands, with larger right lobes predominating,^{1,2} was confirmed and extended to pathological glands of euthyroid and hyperthyroid patients. In multinodular glands,

however, larger left lobes were about as numerous as larger right lobes. The predominant occurrence of solitary thyroid nodules in the right lobe^{3–6} was also confirmed. The richer vascularization of the right lobe has been proposed as an explanation.⁴—We are, etc.,

JAN D. WIENER.
PETER ROOS.

Department of Medicine and
Clinical Radiochemical Institute,
Academisch Ziekenhuis der Vrije Universiteit,
Amsterdam, Holland.

REFERENCES

- Thomson, J. A., *Lancet*, 1966, 2, 308.
- Orr, F. R., and Ingham, G. K., *Lancet*, 1967, 2, 262.
- Artagaveytia, D., Degrossi, O., Gotta, H., and Pecorini, V., *Rev. clin. esp.*, 1965, 98, 266.
- Conte, N., and Ziliotto, D., *Acta isotop.*, 1964, 4, 197.
- Koenig, M. P., *Minerva med.*, 1965, 56, 1814.
- Ziliotto, D., Conte, N., and Scandellari, C., *Folia endocr.*, 1966, 19, 76.
- Heimann P., and Mortensen, A., *Nord. Med.*, 1967, 78, 1097.

Chemotherapy of Tuberculosis

SIR,—Dr. V. C. Barry, whose work I quoted in the article on "Experimental Chemotherapy of Tuberculosis" (13 July, p. 75), has drawn attention to three errors concerning the phenazines, for which I apologize:

(1) The phenazines were not derived from lichens; they were wholly synthetic.

(2) The correct reference to the description of the rimino compounds is the report by Dr. Barry and his colleagues in *Nature (Lond.)*, 1957, 179, 1013.

(3) Fig. 4 represents anilinoaposafranine. The riminophenazines have an alkyl or cyclo-alkyl group substituted for the H in the imino (=NH) group. Chlorophenyl groups are attached at the R positions in the compound B663.

—I am, etc.,

JOHN BATTEN.

St. George's Hospital,
London S.W.1.

Propranolol in Hypertension

SIR,—I was interested to read the divergent views expressed by Drs. G. S. Humphreys and D. G. Delvin (8 June, p. 601) of the ineffectiveness of propranolol in hypertensive Jamaicans.

I should like to comment on their observations as a result of my experience with propranolol in white hypertensives. I have found that there are two important factors which deserve emphasis, namely, the duration of therapy and the dosage required to lower the blood pressure satisfactorily. Our own work (see Table)¹ shows that a significant response was obtained when using a maximum dose of 400 mg. daily, the effective dose being frequently in excess of that used by Drs. Humphreys and Delvin. Since that time further experience has convinced me that higher dosage may be necessary in order to achieve a therapeutic response in patients who have previously been difficult to manage. Prichard's² work lends additional support to the fact that higher dosage than 360 mg. daily is necessary to achieve a satisfactory control. Furthermore, duration of therapy has also been shown to be important. It

would be interesting to know whether any difference was found between that group who had propranolol continuously for four months and those who had a continuous treatment of two months.

I suggest that a re-analysis of clinical terms should be considered. The current therapy for the treatment of hypertension for the most part involves the sympathetic neurone blocking agents, and while these drugs are capable of lowering blood pressure in the upright position they are not so effective when the person is supine. The same, of course, applies to the control of arterial pressure on exercise. This is a significant point in consideration of any antihypertensive agent. There are several reports suggesting that postural and exercise hypotension hardly ever occur when hypertensive patients are treated with propranolol. Our own work amply confirms these findings.

Prichard³ has postulated that the mode of action of propranolol in hypertension is due to adaptation of baroreceptors, while Frohlich⁴ considers that it is due to heart rate dependent fall in cardiac output. Drs.

Mean Blood Pressures

	Pre-treatment	Week 3	Week 6	Week 12	Standard Error of Difference in Means	Significance
Group 1 (11 Patients)						
Daily Dose of Propranolol (mg.)	—	120	120 240	240 400		
Systolic:						
Lying	200.0	196.7	189.6	178.0	3.3	P < 0.001
Sitting	202.3	196.0	186.6	175.6	3.8	
Standing	199.6	194.3	188.2	173.9	3.3	
Diastolic:						
Lying	110.5	108.3	104.0	96.4	2.5	P < 0.001
Sitting	111.5	107.2	103.6	95.5		
Standing	110.9	107.4	102.4	94.0		
Group 2 (13 Patients)						
Daily Dose of Propranolol (mg.)	—	100	200	300		
Systolic:						
Lying	214.5	208.6	204.3	198.7	2.2	P < 0.001
Sitting	212.2	207.3	203.8	197.3	1.9	
Standing	211.9	206.7	201.7	194.8	2.0	
Diastolic:						
Lying	123.3	119.6	115.2	108.8	1.8	P < 0.001
Sitting	122.8	119.2	114.3	108.9	1.7	
Standing	122.4	118.6	112.3	107.2	1.4	