patients were shocked, and three had muscular twitchings and carpopedal spasms.

Thirty-four patients had electrocardiographic evidence of myocarditis—that is, inversion of the T waves in several leads, significant deviation of the RST segment, prolongation of Q-T_c and conduction defects with complete restoration to normal in three to six days.

Toxic myocarditis has not been previously described after scorpion stings. Death is usually ascribed to a toxic effect on the medullary centres. Myocarditis is the commonest complication following stings by *Tityus trinitatis*, and it would be reasonable to postulate that it is a frequent cause of death in this condition.

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NUCLEAR SEX OF EMBRYONIC TUMOURS

BY

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Many workers have reported chromatin-positive teratomata from males, mainly those of the testis (review by Lennox, 1961). As the sex-chromatin pattern of an individual may be used to infer his sex chromosomes, this suggests the presence of two X chromosomes in the teratomatous cells of normal (XY) males.

In a recent series I have found sex-chromatin discrepancies in 8 out of 50 teratomata (three from males and five from females) and in 4 out of 49 other embryonic tumours studied (one from a female and three from males).

Material, Methods, and Results

Fifty teratomata (24 from boys, 26 from girls) and 49 other embryonic tumours (21 from boys, 28 from girls) were examined. This material forms part of the collection of the Department of Morbid Anatomy, Hospital for Sick Children, Great Ormond Street.

Most of the material was fixed routinely in formol-saline, paraffin-embedded, sectioned, and stained in haematoxylin-eosin and haematoxylin-Van Gieson. Some of the material was fixed in Bouin's fixative. When available, spare sections were stained with the Feulgen method, counterstaining with light green or with orcein, and additional sections were cut when possible and necessary.

The incidence of sex-chromatin-positive cells for each tumour was determined by counting several random groups of 100 nuclei each. All pyknotic or folded nuclei were rejected. Only masses attached to the nuclear membrane were scored as positive. Neuroglial cell nuclei were not included in the counts, since in both males and females these frequently contain single or double sex-chromatin-like masses. In large areas of some malignant tumours identification of sex chromatin was impossible. When the main tumour mass was unreadable small metastases were sometimes suitable for examination. The results are listed in the Table.

Analysis of Sex-chromatin Discrepancies in 12 Out of 99 Embryonic Neoplasms Studied

Case No.	Sex	Average Sex-chromatin Incidence Percent of Tumour		Type of Tumour
		One Mass	Two Masses	
1	F	4	—	Malignant cranial teratoma
2	F	2	—	" ovarian
3	F	48	33	Histologically benign ovarian teratoma
4	F	47	27	" " retroperitoneal
5	F	60	14	Malignant retroperitoneal teratoma
6	M	31	—	Histologically benign cervical teratoma
7	M	57	—	Partly well-differentiated cranial teratoma with metastases
8	M	27	—	Histologically benign high abdominal teratoma
9	F	7	—	Embryonic sarcoma
10	M	51	Few	Nephroblastoma
11	M	30	"	"
12	M	28	"	"

Discussion

Hunter and Lennox (1954) suggested that teratomata might be derived from the fusion of two gametes, "or, at least, of haploid cells," giving only tumours with an XX sex-chromosome complement in females, but with XX, XY, or YY complements in males. Tavares (1955) suggests that division of haploid cells followed by chromosome reduplication gives rise to either XX or YY tumours in males. This assumes that YY cells are viable in teratomata.

The main objection to these suggestions is that they do not easily account either for sex-chromatin discrepancies in extragerminal teratomata or for the mosaic teratomata which were described by Maltzeff and Sacco (1958) and Myers (1959) after the formulation of these hypotheses. Furthermore, we must reckon with the possibility that sex-chromatin discrepancies in teratomata and other tumours may be the expression of other abnormal sex-chromosome complements such as XO, XXY, XY/XXY, XX/XXX, XO/XXX, etc.

Recent studies have shown that these complements are viable and not altogether very uncommon in man, and the discrepancies observed in the tumours under consideration might be produced by one or more errors of mitosis such as non-disjunction or anaphase lagging of the sex chromosomes at the inception of the tumour or soon afterwards.

In some cases the discrepancy between nuclear sex and inferred sex-chromosome complement of the tumour and of the patient may be more apparent than real. Both Carpentier *et al.* (1956) and Hienz (1961) have described dysgerminomata in chromatin-negative women with ovarian dysgenesis. Hunter and Lennox (1954) described the case of a boy with a chromatin-positive pineal teratoma who was later shown to have chromatin-positive Klinefelter's syndrome (Lennox, 1960), and W. M. Davidson (personal communication 1962) has recently seen a further patient with Klinefelter's syndrome and a chromatin-positive teratoma.

Summary

Sex-chromatin discrepancies were found in 8 out of 50 teratomata and 4 out of 49 other embryonic tumours

studied. It is suggested that sex-chromatin discrepancies in these tumours may be the expression of abnormal sex-chromosome complements such as XO, XXY, XY/XXY, produced by one or more errors of mitosis such as non-disjunction or anaphase lagging at or soon after the inception of the tumour.

The work referred to in this communication is part of a Ph.D. thesis approved by the University of London. The study was undertaken in the Department of Morbid Anatomy, Hospital for Sick Children, Great Ormond Street, London. The provision of material and facilities and the encouragement and advice of Dr. Martin Bodian are gratefully acknowledged. I also thank Mr. J. L. Hamerton and Professor P. E. Polani for their advice and constructive criticism.

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BERYLLIOSIS: REPORT OF TWO CASES, WITH SPECIAL REFERENCE TO THE PATCH TEST

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[WITH SPECIAL PLATE]

In Germany, in 1933, Weber and Engelhardt published the first description of a disease due to beryllium, and subsequently various papers appeared in European literature, including a report of 46 cases in Russia by Berkovits and Izrael (1940). Van Ordstrand *et al.* (1945) described a chemical pneumonia in workers in Ohio who were extracting beryllium from beryllium ore: since then many cases have been reported in the United States, and the historical, industrial, and clinicopathological features have been fully reviewed by Hardy and Stoeckle (1959) and in a report of the symposium on beryllium disease held at the Massachusetts Institute of Technology in 1958 (Symposium, 1959). In 1952 a beryllium case registry was established at the Massachusetts General Hospital, and by 1958 this contained the records of 601 patients with berylliosis.

The respiratory system is primarily involved in all cases of beryllium disease, and in some the clinical picture is confined to the lungs, when the term "beryllium lung" is used. There may also be evidence of systemic involvement, when, in addition to pathological changes in the lungs, there are lesions in the liver, spleen, lymph nodes, and skin, with alteration in the serum proteins and a raised sedimentation rate. When such systemic disease is present the term "berylliosis" may be used.

Although many papers have been published in the United States describing cases of berylliosis and surveying the industrial environment, relatively few have appeared in the British literature. Agate (1948) published the first account of beryllium disease occurring in this country, and since then other cases have been described by Lederer and Savage (1954), Sneddon (1955,

1958), Rogers (1957), Jordan and Darke (1958), Wood *et al.* (1958), Sita-Lumsden (1959), and McCallum *et al.* (1961). We now describe the cases of two men with beryllium disease who presented with respiratory symptoms in the out-patient department of a general hospital. One of them demonstrates the picture of berylliosis with systemic manifestations as well as pulmonary changes. The other man has chronic beryllium lung disease with the process apparently confined to the lungs.

These patients presented in an interesting manner. The first man (Case 1) was referred to the medical clinic with a history of dyspnoea gradually increasing over the previous 12 months. A radiograph of the chest showed generalized mottling suggestive of tuberculosis or sarcoidosis, and he was admitted for further investigation. As the sedimentation rate was normal and tests for tuberculosis were negative, a provisional diagnosis of sarcoidosis was made; the patient was observed as an out-patient. Shortly afterwards the second man (Case 2) presented with dyspnoea of rapid onset, and a history of recent exposure to beryllium was readily obtained. Radiographs of the lungs showed appearances similar to that seen in the previous patient, and further questioning of the first man revealed that both patients worked in the same factory. The detailed industrial histories were obtained from both men, and subsequently the diagnosis of berylliosis was established. We stress this rather unusual presentation because the first patient might well have remained undiagnosed had not the second man presented at the same time.

Case 1

A man aged 41 presented in July, 1959, with a one-year history of dyspnoea on exertion and a history for many