Horner's syndrome, the components of which appear in this order: (1) narrowing of the palpebral fissure, (2) contraction of the pupils, (3) enophthalmos, and (4) absence of sweating on the side of the injection. To this must be added a noticeable increase of warmth on the injected side. Also, in half to three-quarters of the cases a conjunctivitis appears on the affected side.

Technique

In my series of cases an anterior approach to the stellate ganglion was used. This approach differs from that used by most other authors. It is done with a No. 1 serum needle on a 10-ml. syringe. The needle is passed medial to the sternal head of the sternomastoid muscle a finger-breadth above and lateral to the sternal notch. With the middle and ring fingers of the left hand the carotid artery is palpated and then moved laterally. The serum needle is inserted at a right angle and is pushed straight through just lateral to the trachea. In most cases there is no difficulty in hitting the transverse process of the seventh cervical vertebra. The stellate ganglion is situated between the transverse process of the seventh cervical vertebra and the neck of the first rib. Then 10 ml. of 1% procaine hydrochloride is injected. In my later cases, when there has been cerebral thrombosis I have injected bilaterally. With this method, as opposed to the more usual anterior approach through the two heads of the sternomastoid, most of the complications are avoided. These are: puncture of the subclavian artery, puncture of the meninges, and surgical emphysema or pneumothorax (Leriche and Fontaine, 1934).

The Table shows the results of 124 cases of cerebral thrombosis treated in this way. It is apparent that improvement was present in 17 out of the 23 cases seen within

the course of the last year: two had complete relief, one had slight improvement, and in four there was no effect.

Summary

The stellate ganglion has been injected 342 times in 139 patients. These patients were seen in a general practice in South London and the injections took place in the patient's own home, the consulting-room, nursing-homes, an L.C.C. welfare home, and the out-patient department of a general-practitioner hospital. A technique of the anterior approach to the stellate ganglion is described, and it is suggested that this is a safe procedure for use in general practice. The general practitioner obviously sees more cases of cerebral thrombosis first, and it is comparatively easy for him to inject the stellate ganglion within the first 24 hours. With this short time lag the prospect of a complete recovery or great improvement is very great. In this series of cases the procedure itself has not given me a moment's anxiety.

I should like to thank my colleagues for allowing me to anaesthetize the stellate ganglion in many of their patients.

REFERENCES

- Butt, T. C., and Mathers, F. (1948). *J. Fla med. Ass.*, 35, 285.
 Gilbert, N. C., and de Takats, G. (1948). *J. Amer. med. Ass.*, 136, 659.
 Leriche, R. (1952). *British Medical Journal*, 1, 231.
 — and Fontaine, R. (1934). *Presse méd.*, 42, 849.
 — (1936). *Rev. Chir., Paris*, 55, 755.
 Mackey, W. A., and Scott, L. D. W. (1938). *British Medical Journal*, 2, 1.
 Villaret, M., and Cachera, R. (1939). *Les embolies cérébrales*. Masson, Paris.
 Volpito, P. P., and Risteen, W. A. (1943). *Anesthesiology*, 4, 403.

Results

Time Lapse	No.	Complete Recovery	Good	Fair	No Improvement
Within 24 hours ..	23	12	5	0	6
" 2-7 days ..	27	8	6	9	4
" 1-4 weeks ..	18	1	5	5	7
" 1-6 months ..	34	0	0	8	26
" 6-12 " ..	22	0	0	4	18
Total ..	124	21	16	26	61

24 hours. The interesting feature at the other end of the scale is that of the long-standing hemiplegias. Within 6 to 12 months there was some improvement in 4 out of 22 cases. Leriche (1952) gives one injection of 10 ml. a day for 15 days for the "strokes" of recent origin, and in the longer-standing cases he gives 12 to 36 injections. It has not been possible for me to give as many as this, but if some degree of recovery has not taken place within three injections I count it as a failure.

Raynaud's Disease.—Three cases were treated: one had relief for one week, and one for one month, in the third there was no effect. This is a very small number of cases, but it was interesting to see that the effects on the second arm were identical with those on the first.

Cardiac Pain in Angina Pectoris.—In general practice one is confronted always with a few cases of long-standing angina pectoris. Some of these patients may have had a coronary thrombosis 10 to 15 years ago, and in others the electrocardiogram is normal or almost so. I have been very careful to select cases of long standing for injection. Of the five cases treated, two had complete relief, two had some relief, and in one there was no effect. In all these cases bilateral ganglion blocks were done.

Stiff Shoulder.—In all cases these were post-traumatic. There is a time in treating this type of shoulder when active exercises and heat are producing very little result and the interval is too short since the time of injury for manipulation under an anaesthetic. This small series of seven cases represents those patients seen in general practice during

GYNAECOMASTIA IN TWO BROTHERS

BY

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In 1950 I reported on a youth of 17 suffering from gynaecomastia. Subsequently the same condition developed in the younger brother. The latter has been investigated, and further studies have been carried out on the elder brother.

Case 1

The elder brother was first seen in 1947, aged 17. He complained of breast enlargement, which started at the age of 14. He had never shaved and had had neither erections nor emissions. Libido seemed to be absent. His hobbies were those of a boy. He had suffered no injuries to or disease of the testicles.

Pronounced gynaecomastia was present (see Fig. 1). The penis was small and the urethra opened at the base of the glans. Scrotum and testicles appeared normal. The pubic hair was of feminine distribution and axillary hair was scanty. The face was covered with a fine down. His height was 63½ in. (161 cm.) and span 66 in. (168 cm.); head to pubis measured 32 in. (81 cm.) and pubis to ground 31½ in. (80 cm.). His voice was soft and his manner retiring. There was no plethora or glycosuria, and the blood pressure was normal.

His bone age corresponded to chronological age. 17-ketosteroid excretion was considered to be within normal limits. Although the original estimate was rather high, this was not confirmed by subsequent tests, which yielded figures of 12, 18, and 14.5 mg. within 24 hours. The original estimate of F.S.H. excretion was regarded as being within normal limits (Dr. A. M. Hain). Another, some months after a period of testosterone therapy, showed a 24-hour output of more than 40 and less than 160 units

(Dr. G. I. M. Swyer). A further estimate six months later revealed less than 10 units per 24 hours (Dr. G. I. M. Swyer).

A breast biopsy by Mr. Page showed hyperplasia of duct elements and fibrous tissue (Dr. J. F. Heggie). Laparotomy revealed neither uterus nor Fallopian tubes, and the adrenals appeared normal on palpation. Testicular biopsy and mastectomy were refused.

Case 2

The younger brother was first seen in 1950 at the age of 18. He complained of breast enlargement, starting at the age of 17. He had never shaved nor had emissions, but he had experienced erections for one year. His hobbies were those of a boy. He had suffered no injury to or disease of the testicles.

Physically he closely resembled his brother (see Fig. 2). Breasts, penis, testicles, hair distribution, voice, manner, and general appearance were almost identical. His height was 60 in. (152 cm.) and span 63½ in. (161 cm.); head to pubis measured 29 in. (74 cm.) and pubis to ground 31 in. (79 cm.). Two 24-hour estimates of 17-ketosteroid excretions were 8.9 and 17.5 mg. (Dr. A. B. Anderson). Two estimates of F.S.H. excretions were performed: one revealed less than 5 units in 24 hours, and the other between 5 and 10 units in 24 hours (Dr. G. I. M. Swyer).

Further investigations, including testicular biopsy and mastectomy, were refused.

FIG. 3.—Family tree.

Family History

The father and mother are related, as shown in Fig. 3. The father had one brother and four sisters, and the mother four brothers and six sisters. No abnormality resembling the present condition was known of in the family, nor was

there any history of primary amenorrhoea in the females; all but one of the nine married aunts and uncles have had children.

Case 1 at the age of 22 and Case 2 at the age of 20 were both 161 cm. in height. So far as was known, the female members of the family on both sides were of the same size or taller, and the men with one exception distinctly taller. The two brothers therefore seem to be unusually small.

Discussion

The condition is clearly hereditary in nature. The consanguinity of the parents and the apparent absence of similar abnormality in other members of the family suggest a recessive mode of inheritance.

In the absence of a biopsy, the precise gonadal lesion and the diagnosis must remain in doubt. Ovotestes may have been present, when the diagnoses of hermaphroditism would apply; the failure to find a uterus or Fallopian tubes at laparotomy in the eldest brother is somewhat against this explanation. Testicular hypoplasia is a possibility. The low figures for F.S.H. excretion may indicate a pituitary origin for such hypoplasia, and the tendency to dwarfism—consistent with a deficiency of growth hormones—may therefore be significant. The one high figure for F.S.H. excretion in the elder brother followed a period of testosterone therapy, and may well be a rebound phenomenon. Alternatively, the testicular cells responsible for secretion of the substance inhibiting gonadotrophin production may have been spared in a primarily gonadal hypoplasia.

The mechanism for the development of gynaecomastia in testicular hypoplasia is obscure. Oestrogen excretion has been investigated in a number of cases and found within normal limits (Klinefelter *et al.*, 1942; Heller and Nelson, 1945). Excess of circulating gonadotrophin is clearly not the explanation in these two patients.

Summary

Gynaecomastia has been found in two brothers. Testicular biopsies have not been obtained, and aetiology is uncertain. Ovotestes with hermaphroditism and testicular hypoplasia are possibilities. The condition appears to have been inherited in a recessive manner.

I would like to thank Dr. G. I. M. Swyer for the gonadotrophin assays in these patients, and Mr. C. W. Hall for the photographs.

REFERENCES

- Ferriman, D. G. (1950). *British Medical Journal*, 1, 162.
Heller, C. G., and Nelson, W. O. (1945). *J. clin. Endocr.*, 5, 1.
Klinefelter, H. F., Riefenstein, E. C., and Albright, F. (1942). *Ibid.*, 2, 615.

Medical Memoranda

A Case of Inversion of the Uterus

Inversion of the uterus is such a rare condition that the following case of inversion of the non-puerperal uterus caused by a fibroid is worth reporting.

The incidence of puerperal inversion is about 1 in 30,000 labours (Das, 1940) and the non-puerperal condition is even more uncommon. Das, in a study of 391 cases of inversion, found that 16.35% were non-puerperal, and McCullagh (1925), in an analysis of 233 cases recorded in the literature between 1911 and 1924, found 15%.

CASE HISTORY

A 0-gravida, aged 35, was admitted to hospital on December 15, 1951, complaining of severe vaginal bleeding for the past seven days. Since the age of 15 her periods had been regular, lasting five days and recurring in a 28-day cycle. The loss was not heavy except on rare occasions, and there was no dysmenorrhoea. Seven days before admission a period had started on the expected date. The loss was very heavy from the beginning and the patient was confined to bed. There were no other symptoms except slight lower abdominal discomfort. She had no pain or nausea, no difficulty on micturition, and no bowel symptoms. On the seventh day her face and hands became numb. Her doctor arranged for her immediate transference to hospital.

On admission she was found to be extremely exsanguinated. Her temperature was 97.8° F. (36.6° C.), pulse rate 116, and blood pressure 115/65. Haemoglobin