Papers and Originals

Post-concussional Sequelae*

ALEX. R. TAYLOR,† M.A., F.R.C.S.

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After a year in which several papers have appeared describing a quantitative relationship between the late symptoms of head injury and measurable deviations from physiological and psychological norms (Houdart *et al.*, 1966; Lishman, 1966; Taylor and Bell, 1966), it would seem to be an appropriate time to re-examine a one-hundred-year-old question about the aetiology of post-concussional sequelae.

The same argument has ranged back and forth almost unchanged over this hundred years. In 1866 Erichsen presented 63 cases of severe injury sustained mostly on American railroads. He was concerned with the severity of the symptoms in the absence of much evidence of external injury. He thought the symptoms were due to molecular derangement of the spinal cord. In 1879 Regler, who had noted a great increase in the severity of symptoms after the introduction of compensation awards in 1871, expressed the view that they were actuated by fright, long inactivity, and a desire for financial gain. He thought there was no evidence for molecular derangement. This was the so-called "railway spine" controversy.

In 1962 Symonds reviewed the aetiology of concussion, and stressed, *inter alia*, the finding of cellular and diffuse white-matter damage. In 1966 Miller, analysing his experience of many head-injured litigants, wrote: "I cannot believe that the clinical features of this disorder justify its speculative attribution to cellular damage." This was a judgement about the writer's beliefs and not about concussion, but there is a good historical precedent for this too. In 1863, rejecting the then current vascular theories, Hilton wrote: "I cannot conceive it possible that a long continuance of the symptoms of concussion can depend upon any temporary error of this kind." Or again, Strümpell (1888) wrote: "I cannot free myself from a feeling of uncertainty in these cases of head injury."

It is worth while noting that in the early days of the argument the symptoms of "spinal concussion" preceded the inauguration of compensation payments, and that they were the same before as after, though their severity apparently increased. Indeed, the difference of opinion about aetiology has been bedevilled for 100 years by the inability of physicians to disentangle the symptoms occurring after concussion from the symptoms that follow the initiation of a claim for compensation. I have yet to read an article on the subject of post-concussional sequelae in the British literature which does not contain the words "litigation" or "psychogenic" in the first dozen lines. Even a critical leading article about post-traumatic headache, written in April 1966, which concluded that treatment was not easy but was well worth while, contained the statement that in at least two-thirds of the patients there is a psychogenic element. This is certainly an advance on a similar leader of 1961, which said: "Above all, the general physician . . . should find it advisable to refer a patient to the psychi-

atrist as soon as possible . . ." and concluded: "In the ultimate analysis it [accident neurosis] is a social disease and a function of industrial morale."

Symonds (1962) pointed out that it was valueless to inquire whether the symptoms were organic or functional, because they must always be both. While agreeing with this, I feel that a periodic critical analysis of degrees and probabilities is necessary with each advance in physiological or biochemical knowledge about head injury. The treatment of a disorder predominantly organic must be different from one predominantly psychiatric.

Prima facie, it seems strange that 66% of the head-injured, necessarily a random sample of the population, should be afflicted with neuroticism or excessive cupidity while most of the victims of limb, abdominal, or thoracic injury escape these stigmata. The evidence for neuroticism should be carefully examined.

Evidence for Neuroticism

There are no Abnormal Signs on Physical Examination

This is a notoriously difficult obstacle to precise diagnosis. That a patient should complain of organically determined symptoms of headache, dizziness, lack of energy, poor memory, and difficulty in concentrating and have no physical signs is to some doctors incredible. But the central nervous system is unique in its clinical silence.

A meningioma may attain the size of a cricket ball without causing more than an occasional epileptic fit or transient headache and give rise to no physical signs. A frontal or temporal lobe may be excised without definable abnormality, and thousands of patients have had all the white fibres of both pre-frontal regions divided in the operation of leucotomy without detectable physical changes. Patients whose character, mood, and behaviour change after meningitis may have "negative" physical examinations. As Strauss and Savitsky (1934) concluded after a lengthy review of this question: "Failure to understand a clinical phenomenon is no proof of its psychogenicity."

There is an Inverse Relation Between Severity of Injury and Persistence of Post-concussional Sequelae

According to Miller (1961) the more trifling the injury the longer and more vehemently the litigant complains. Of his patients with a post-traumatic amnesia of less than 15 minutes 37% had gross unequivocal psychoneurotic complaints. Only 6% of patients with amnesia of over 72 hours had such complaints. Brock (1960), Russell and Smith (1961), and Symonds (1962) came to opposite conclusions about the significance of post-traumatic amnesia. They believed that though imperfect

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† Department of Neurological Surgery, Royal Victoria Hospital, Belfast 12.

it was the most reliable and sensitive index of the severity of brain injury.

The other comparison made by Miller to show that symptom severity varied inversely with physical damage was between those who had skull fractures and those who had not. Of nine cases with compound fractures none was complicated by neurosis, and "neurotic" symptoms were three times as common in those without as in those with fractures.

These data prove exactly the opposite of what was intended. Percivall Pott (1808) realized that a fracture might protect the patient against the general effects of brain deceleration. He listed vomiting, giddiness, loss of speech and sense, loss of voluntary motion, etc., as general symptoms, and then wrote: "On the other hand the bones of the skull are sometimes cracked, broken, nay even depressed, and the patient suffers none of these symptoms."

Denny-Brown and Russell (1941) found that an animal is rendered unconscious by a much lesser blow if the head is freely movable. The head not free to move is more easily fractured and suffers less of the effects of "cerebral commotion." A great deal of energy is absorbed in fracturing the skull, which is then diverted from the production of shearing stresses in the brain. For this reason biomechanics experts are exploring the possibilities of "tuning" a crash helmet so that it fractures under a certain stress and so protects the contained brain. One expects the patient with a fractured skull to suffer less from concussional effects than his unfractured counterpart who absorbed the same amount of energy on impact.

Unskilled Workers and Less-intelligent Patients Suffer More Prolonged Disability

This is a matter of interest and motivation, and is not confined to the field of trauma. During the last war Commando and Special Service personnel who were off duty for more than 21 days had to transfer to another unit. One could be certain that such selected men after operations for hernia, cartilage derangement, or disc protrusion would be out of bed as soon as permitted and back to their units probably suffering in silence within the statutory three weeks. Some prisoners of war or the more lowly performers of menial tasks in the Services could with difficulty be prised out of hospital in twice the time and be discharged, complaining bitterly, on sick leave.

Symonds and Russell (1943) found that head-injured R.A.F. air crews returned to duty twice as rapidly as ground personnel.

Similarly in civilian practice, the man with the more interesting job and the bigger personal stake in his recovery complains less and returns to his desk sooner.

Personal hazard is also a factor. A civil servant may be able to do his job efficiently in spite of some headache and a sense of abnormal movement, but a scaffold erector or steeplejack will not. Professional men will return to work sooner than manual workers, but it is incorrect to assume that they do not therefore have symptoms. Though 80% of Russell's (1933-4) patients (not entitled to compensation) returned to full work within six months of injury, 40% still had symptoms at the end of 18 months.

It is sometimes said that doctors do not suffer from post-concussional sequelae, but published accounts do not support this belief. Durand Wever (1929) described typical symptoms from an injury for which she was not entitled to claim compensation. Mayer (1929) found that his symptoms continued after his claim was settled, and advised other doctors to believe that their patients' complaints were both real and disabling. Strauss and Savitsky (1934) described a case in which a doctor had symptoms for five months after head injury without loss of consciousness. He continued to work but suffered from headaches, a sense of abnormal movement, lack of concentration, and poor effort tolerance. Compensation was not claimed.

Injuries Sustained during Sporting Activities do Not Have Sequelae

This generalization is partly true. It does not apply to severer ski-ing or rock-climbing accidents nor to "missile" injuries suffered on the hockey field. Hunting and rugby football are usually mentioned as "no-sequelae" sports, though American football, because of the dangerous "protective" devices worn, has abundant records of college boys who never played again after a head injury.

In physical terms this is a matter of speed at impact. The rugby player seldom achieves 20 ft. (6 m.) per second, the huntsman seldom comes off his horse at more than 30 ft. (9 m.) per second. The 30 mile (48 km.) an hour motorist is travelling at 44 ft. (13.4 m.) per second and twice that at 60 miles (96 km.) per hour. Graduation of severity according to speed of impact is well recognized in the Mid-western cities of America, where in hot weather small children stray to the stairs of tenement houses and fall through one or more flights. A fall of one flight, or 15 ft. (4.6 m.) (21 ft. (6.4 m.) per second), seldom results in injury requiring long detention in hospital, two flights (34 ft. (10.4 m.) per second) means a severe head injury, and three or more (44 ft. (13.4 m.) per second) may be fatal.

The question of motivation is again important; the sportsman does not want to give up a pleasurable form of activity, and, if necessary, will suffer the inconvenience of a headache.

Summary of Evidence for Neuroticism

The evidence for the predominance of neurotic symptoms after head injury is not very convincing, particularly if one disregards the litigants who obviously have a powerful motive, conscious and unconscious, for continuing to suffer.

If neurosis is such an important factor one would expect the literature to contain many controlled randomized studies of head-injured patients treated or not treated by psychiatric methods. No such studies exist. Instead are the statements that psychiatric treatment is of no value until claims for compensation are settled—patients not claiming compensation are never mentioned in this connexion. Surprisingly there is an absence of correlation between post-traumatic sequelae and history of previous psychiatric illness, which is mentioned in most reviews.

Though it is often said in our courts of law that recovery will begin at the conclusion of the case and be complete in two years, this is not borne out by 10-year surveys of the German head-injured of the first world war (Goldstein, 1942), or of the American Korean war head-injured (Caveness, 1966).

Of Goldstein's patients 20% became normal workers, 32% worked at one-half to one-third capacity, 30% had a below normal working capacity, and 18% did no work. These were largely missile injuries and not comparable to a civilian group, but the Korean series is equally divided between penetrating and blunt injuries. Of these patients 84% had returned to work after 10 years, but 40% still had symptoms and 27% had not made a satisfactory social and economic adjustment.

Known Organic Factors

That the initial signs of concussion and the symptoms immediately following recovery of consciousness are organic there is no doubt. Loss of consciousness, mass contraction of the limbs, cessation of respiration, rise in blood pressure, loss of corneal reflexes, and dilated pupils are followed by headache, perhaps vomiting, dizziness, confusion, and amnesia. These are all symptoms and signs which could arise from damage to the cranial nerve nuclei between the medulla (X) and the upper brain stem (III), the arousal centre in the reticular formation, the pyramidal tracts, and the vasomotor centre. If damage is

sought in the brain of the concussed animal this is the area in which it is mainly found. The nerve cell count in the lateral vestibular nucleus is invariably reduced even after a mild concussional blow. That this is not transitory was shown by Groat and Simmons (1950), who made cell counts in the brain-stem nuclei of guinea-pigs 13 months after a single concussive blow to the movable head.

The mechanism of the medullary injury is uncertain. It may be, as Friede (1961) suggested, a blow to the cervico-medullary junction dealt by the odontoid process as the anterior surface is hyperflexed over it. The brain stem and cord are more probably stretched than struck, as the neck is hyperflexed through about 110 degrees and rapidly decelerated. Breig (1960) demonstrated that the cord is stretched and elongated on neck flexion. As it is fixed by the filum terminale, dentate ligaments, and cranial nerves, especially V, VII, and VIII, this pull will be sharply felt all through the brain stem and forward through the long tracts to where the forebrain is fixed by the optic nerves, olfactory bulbs (sometimes pulled out), and carotid arteries. As a result of this stretching white fibres from the brain stem to the centrum ovale are actually fractured or snapped. Biggart (1936) demonstrated these "fractures" and the resulting end bulbs on the axons by Bielschowsky staining in the brains of patients dying after head injury. But these lesions are present not only in fatal injuries. Nevin (1964) found them all through the white matter of rats subjected to even minor concussive blows. They took about three days to develop (Fig. 1) and were present as constantly as the chromatolysis in the brain stem nuclei described by Windle *et al.* (1944) (Fig. 2). In the same areas he found irregular thickening and fragmentation of the myelin sheaths (Fig. 3).

If concussion causes widespread lesions throughout the white matter it is not surprising that Becker *et al.* (1946) found a deterioration in the maze-running performance of guinea-pigs a month after concussion. Animals already trained lost their skills, while untrained animals were much more difficult to train and retained their new learning badly compared with control animals. It seems more reasonable, therefore, to suppose that injured human subjects concentrate and learn badly, find difficulty in retaining new information, are irritable, anxious,

and lacking in insight because they have multiple lesions throughout the white matter, than to label them neurotic because they do not have any of the easily elicited physical signs.

Variations from the Physiological Norm

Though patients after uncomplicated concussion do not have abnormal physical signs, they have been found by special examination to show deviations from normality in the permeability of the blood/C.S.F. barrier, cerebral blood flow, and vestibular responses to threshold stimuli (electronystagmography). Zetterholm (1947) examined 149 head-injured patients six months to two years after head injury for permeability of this blood/C.S.F. barrier to bromine. Of 104 who had normal permeability quotients 63 had no sequelae and only four had severe symptoms. Only eight of 45 with abnormal permeability quotients had no sequelae, and 15 had severe symptoms.

After two years 73 patients were re-examined: 57 were normal, of whom 47 had no sequelae and none was severely

FIG. 1.—Retraction balls on the end of nerve fibres from a rat 10 days after concussional head injury. (Bielschowsky. $\times 350$.)

FIG. 2.—Nerve cells in the medulla oblongata showing chromatolysis three days after injury. (Gallocyanin. $\times 265$.)

FIG. 3.—Myelin sheaths showing fusiform swelling and vacuole formation seven days after concussion. (Weigert Pal. $\times 320$.)

disabled. Of the 16 with abnormal permeability 12 had sequelae, seven of a severe degree. These figures are statistically significant. The findings have been confirmed by Haxhe (1963), using radioactive bromine. They indicate that one of the effects of brain injury is altered blood/C.S.F. permeability and that its degree correlates with the severity of postoperative sequelae.

Vestibular Function

Though much has been written about labyrinthine damage in head injury Friedman *et al.* (1945) showed that tests of vestibular function did not place the lesion causing post-concussional dizziness in the labyrinth. Since then testing has been greatly refined and includes the electrical recording of nystagmus (electronystagmography). In this way Houdart *et al.* (1966) studied 50 patients with "a subjective syndrome following injuries to the head." In more than half they found a vertical nystagmus appearing in all positions of the head. There were additional minor abnormalities of rhythm and amplitude which they thought were "specific to the traumatic syndrome and apparently authenticating its organic basis." Wilmot (1966), using threshold stimulation and electronystagmography, found the abnormal post-traumatic responses quite different from those arising from a damaged labyrinth; and similar to those found in vertebrobasilar insufficiency. This accords with the experimental finding of chromatolysis in the vestibular nuclei after concussion in animals.

Vascular Factors

Vasomotor instability has been postulated as an aetiological factor in the sequelae of head injury since the earliest days of inquiry. By this was originally meant instability of blood pressure on change of posture with resulting dizziness, faintness, and tachycardia. Taylor and Bell (1966) showed that patients with symptoms after head injury with loss of consciousness had increased cerebral circulation times. Recordings were made of the first passage of an intravenously injected radioactive stream through the head. The mean circulation times of 70 patients with head injury was 20% greater than the mean of 70 controls matched for age and sex. Longitudinal studies of individual patients showed that the circulation times approached normal as the symptoms became less. Patients with compensation claims were excluded from the series. The cause of this slowing of cerebral circulation is still a matter for speculation, but it could be either increase in vasomotor tone at the arterial or capillary level or increased capillary permeability.

Discussion

There is now a great deal of pathological, physiological, and biochemical data to show that patients who absorbed a blow on the head sufficient to cause alteration in consciousness have lesions of varying severity in the medullary pontine nuclei and the white matter, with consequent disorders of vestibular function, capillary permeability, and cerebral blood flow. Minor intellectual disorders are found if sought for by objective psychological testing (Zangwill, 1966). There is also evidence of acute disorders in glucose and pyruvate metabolism (Kurze *et al.*, 1966), water and electrolyte distribution (Van Harreveld, 1966), adenosine triphosphate metabolism (Ishii, 1966), and a rapidly accumulating number of other biochemical reactions.

It is no longer good enough to stand firm on clinical convictions that head-injured patients, if properly managed, socially adjusted, and carefully rehabilitated, will not have "neurotic" symptoms. The evidence from the few series so managed all points in the other direction. We cannot therefore say that the patients have their symptoms because they have been

neglected, spoiled, or legally implicated. It is increasingly clear that closed head injury may present any degree of chronic complication from the slightest abnormality to dementia and quadrispasticity (Strich, 1961). Patients who have minor difficulties in concentration and performance which irritate them and make them act unusually are not "neurotic," they are "cerebrally disorganized," and for good organic reasons. How else can we explain the child who comes in from play and asks to be allowed to lie down because he has a headache, the active young man who discovers to his surprise that he is sensitive to the effects of alcohol, or the older man whose family says "he has never been the same since his fall"?

These symptoms are important, firstly, because for all we know they may be preventable, and, secondly, because the number of sufferers increases steadily every year.

They may be preventable—the investigation of the concussed patient stops after neurological examination at skull x-ray examination, and treatment is rest in bed until he is well enough to get up. The number of sufferers increases—head injury is now by far the commonest neurological disorder. In Great Britain traffic accidents, industrial injuries, and miscellaneous falls cause over half a million head injuries each year. Of these, 80% are classified as non-dangerous, but about 40% have "concussion" and 15% have continuing symptoms. In round figures there are about 75,000 new patients with post-traumatic symptoms added to the existing number each year. The profession will soon be forced by the magnitude of the problem to take some note of it.

It does not appear as if propaganda or prophylactic measures will reduce the number of traffic accidents. They continue to increase annually in every country along with the climbing traffic density. We shall have to treat the resulting patients, and as a necessary prerequisite we shall have to discover much more than we now know about the physiological and biochemical disorders of acute and chronic post-concussional states. The present surgical endeavour in the treatment of head trauma is almost wholly absorbed in the diagnosis of intracranial bleeding and its treatment. We do everything for the severely damaged 10% and neglect the non-serious 90%. The only head injury sequelae we remotely begin to understand is epilepsy, because it can be observed, assessed, and treated. The less tangible symptoms—headaches, vertigo, irritability, poor concentration, faulty memory, sensitivity to alcohol, loss of libido—and all the other pieces of this so-called syndrome should be analysed, dissected, and investigated until it is known what they are, what they express, and how they can be corrected.

Summary

The history of the relationships between litigation and head trauma and its neurotic sequelae is presented. It is noted that there is a higher rate of so-called neuroticism after head trauma than after trauma to other parts.

The evidence that concussional sequelae are in fact neurotic is examined in detail. It is concluded that, except in litigants, the evidence is poor.

The known organic factors in patients who have symptoms after concussional head injury are reviewed. The influence of lesions seen in the midbrain and white matter of experimentally concussed animals is considered.

A relationship is suggested between these diffuse minor lesions and the emotional and intellectual abnormalities found after head trauma.

Physiological and biochemical changes are described affecting the blood/C.S.F. barrier, cerebral circulation times, and tests of vestibular function.

It is concluded that management of the head-injured patient is based on too many unwarrantable assumptions about the incidence of neuroticism and the absence of important physical

and biochemical abnormalities. It is suggested that research into the large and important group of "non-serious" head injuries has been neglected, to the detriment of their management.

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REFERENCES

- Becker, R. F., Groat, R. A., and Windle, W. F. (1946). *Fed. Proc.*, 5, 7.
 Biggart, J. H. (1936). *Pathology of the Nervous System*. Edinburgh.
 Breig, A. (1960). *Biomechanics of the Central Nervous System*. Stockholm.
 Brock, S. (1960). *Injuries of the Brain and Spinal Cord and Their Coverings*, 4th ed. New York.
 Caveness, W. F. (1966). In *Head Injury Conference Proceedings*, edited by W. F. Caveness and A. E. Walker, p. 209. Philadelphia.
 Denny-Brown, D., and Russell, W. R. (1941). *Brain*, 64, 93.
 Durand Wever, A. M. (1929). *Munch. med. Wschr.*, 76, 1879.
 Erichsen, J. E. (1866). *On Concussion of the Spine, Nervous Shock, and Other Obscure Injuries of the Nervous System*. New York.
 Friede, R. L. (1961). *Arch. Neurol. (Chic.)*, 4, 449.
 Friedman, A. P., Brenner, C., and Denny-Brown, D. (1945). *J. Neurosurg. (Chic.)*, 2, 36.
 Goldstein, K. (1942). *After Effects of Brain Injuries in War*, p. 220. London.
 Groat, R. A., and Simmons, J. Q. (1950). *J. Neuropath. exp. Neurol.*, 9, 150.
 Haxhe, J. J. (1963). Blood C.S.F. barrier breakdown. Personal communication.
 Hilton, J. (1863). *Lectures on Rest and Pain*. London.
 Houdart, R., Cathala, F., Pialoux, P., Raby, D., Fontelle, P., and Vollmer, D. (1966). *Presse méd.*, 74, 449.
 Ishii, S. (1966). In *Head Injury Conference Proceedings*, edited by W. F. Caveness and A. E. Walker, p. 276. Philadelphia.
 Kurze, T., Tranquada, R. E., and Benedict, K. (1966). *Ibid.*, p. 254. Philadelphia.
 Lishman, W. A. (1966). *Proc. roy. Soc. Med.*, 59, 261.
 Mayer, E. (1929). *Munch. med. Wschr.*, 76, 2135.
 Miller, H. (1961). *Brit. med. J.*, 1, 919.
 — (1966). *Proc. roy. Soc. Med.*, 59, 257.
 Nevin, N. C. (1964). "Pathology in Cerebral Concussion." Thesis, The Queen's University of Belfast.
 Pott, P. (1808). *The Surgical Works*. London.
 Regler, J. (1879). *Über die Folgen der Verletzung auf Eisenbahnen*. Berlin.
 Russell, W. R. (1933-4). *Trans. med.-chir. Soc. (Edinb.)*, 129.
 — and Smith, A. (1961). *Arch. Neurol. (Chic.)*, 5, 4.
 Strauss, I., and Savitsky, N. (1934). *Arch. Neurol. Psychiat. (Chic.)*, 31, 893.
 Strich, S. J. (1961). *Lancet*, 2, 443.
 Strümpell, A. (1888). *Über die traumatischen Neurosen*. Berlin.
 Symonds, C. (1962). *Lancet*, 1, 1.
 — and Russell, W. R. (1943). *Ibid.*, 1, 7.
 Taylor, A. R., and Bell, T. K. (1966). *Ibid.*, 2, 178.
 Van Harreveld, A. (1966). *Brain Tissue Electrolytes*. Washington, D.C.
 Wilmot, T. J. (1966). *J. Laryng.*, 80, 1156.
 Windle, W. F., Groat, R. A., and Fox, C. A. (1944). *Surg. Gynec. Obstet.*, 79, 561.
 Zangwill, O. L. (1966). *Proc. roy. Soc. Med.*, 59, 266.
 Zetterholm, S. (1947). *Acta psychiat. scand.*, Suppl. No. 45.

Prolapse of the Posterior Leaflet of the Mitral Valve: A Clinical, Familial, and Cineangiographic Study

MARY STANNARD,* M.B., B.S.; J. G. SLOMAN,† M.B., B.S.C., M.R.C.P., M.R.C.P.ED., M.R.A.C.P.
 W. S. C. HARE,‡ M.D., M.R.A.C.P., F.F.R., F.C.R.A.; A. J. GOBLE,§ M.D., M.R.C.P., F.R.A.C.P.

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Mid and late systolic clicks and late systolic murmurs have in the past been regarded as "innocent," and were thought to be due to pericardial adhesions (Wells, 1957; McKusick, 1958; Ongley *et al.*, 1960; Segal and Likoff, 1964). Recently there has been evidence to show that these auscultatory findings are due to an abnormality of the mitral valve and sometimes are associated with a history of anterior chest pain and an abnormal electrocardiogram (Barlow *et al.*, 1963; Barlow, 1965; Tavel *et al.*, 1965; Barlow and Bosman, 1966; Criley *et al.*, 1966; Leon *et al.*, 1966; Linhart and Taylor, 1966). The purpose of this paper is to present our findings in 13 patients with mid or late systolic clicks and late systolic murmurs, with particular reference to the familial nature of this entity and the findings on left ventricular cineangiography and selective coronary arteriography.

Methods and Materials

Of the 13 patients studied six were referred with anterior chest pain, four because a murmur had been detected on routine examination, and three were siblings of patients already known to us. All were assessed clinically, and an electrocardiogram and a phonocardiogram were made. In six cases haemodynamic studies and left ventricular cineangiography were carried out.

* Research Assistant, Cardiac Department, Royal Melbourne Hospital. Grant in Aid No. G.428, National Heart Foundation of Australia.

† Director, Cardiac Laboratory, Royal Melbourne Hospital.

‡ Professor of Radiology, University of Melbourne.

§ Cardiologist, Royal Melbourne Hospital.

Selective coronary arteriography was performed in two patients who presented with severe anterior chest pain.

Findings

The clinical features are set out in Table I. There was a definite female preponderance in the series, 11 patients being female and two male. Their ages ranged from 28 to 67, with a mean of 46 years.

Chest pain was a significant symptom in six patients. The pain occurred anteriorly in the chest, was stabbing in quality, and was not related to exertion. In two cases it was severe enough to require analgesics by injection, but most patients had been regarded as suffering from "cardiac neurosis." In no case was there a past history of rheumatic fever or a history of an illness which in retrospect could have been pericarditis. One patient had been involved in a motor-car accident shortly before the murmur was detected, but she could recall no chest injury. Two patients had thyrotoxicosis; one had symptoms of this at the time her murmur was detected and the other developed it subsequently. One patient had been thought to have had a myocardial infarct some years previously, but there was no evidence of this on her electrocardiogram.

The family history was of significance in only one patient whose father and four brothers were said to have coronary artery disease. However, because a familial incidence of this entity had been suggested, we examined the siblings of our