- Craft, I. (1973). *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 80, 46.
- Embrey, M. P., Hillier, K., and Mahendran, P. (1972). *British Medical Journal*, 3, 146.
- Lippert, T. H., and Modly, T. (1973). *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 80, 1025.
- MacKenzie, I. Z., Embrey, M. P., and Hillier, K. (1974). *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 81, 554.

- MacKenzie, I. Z., and Hillier, K. (1974). *Lancet*, 1, 511.
- Midwinter, A., Shepherd, A., and Bowen, M. (1973). *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 80, 371.
- Miller, A. W. F., Calder, A. A., and MacNaughton, M. C. (1972). *Lancet*, 2, 5.
- Read, M. D., Bedford, N. A., and Martin, R. H. (1974). *Lancet*, 1, 214.
- Wiqvist, N., et al. (1972). *Prostaglandins in Fertility Control*, 2, 118.
- Wiqvist, N., et al. (1974). *Prostaglandins*, 6, 193.

Electrocardiographic Abnormalities Associated with Raised Intracranial Pressure

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Summary

Serial electrocardiographic (E.C.G.) recordings were taken in seven patients suffering from intracranial conditions, for which their intracranial pressure was directly and continuously monitored with a Konigsberg extradural transducer. The E.C.G. changes observed in patients with raised intracranial pressure were prominent U waves, ST-T segment changes, notched T waves, and shortening and prolongation of Q-T intervals. Two patients with normal intracranial pressure showed no E.C.G. abnormalities. The results not only confirm the association between central nervous system diseases and E.C.G. abnormalities but also establish a relationship between E.C.G. abnormalities and changing intracranial pressure.

Introduction

Electrocardiographic (E.C.G.) abnormalities are known to occur in a variety of central nervous system lesions like sub-arachnoid haemorrhage, cerebrovascular accidents, head trauma, intracranial space-occupying lesions, meningitis, etc. (Byer et al., 1947; Burch et al., 1954; Schuster, 1960; Finkelstein and Nigaglioni, 1961; Hersch, 1961, 1964; Fentz and Gormsen, 1962; Goldman, 1967; Abildskov et al., 1970; Cruickshank and Dwyer, 1974). Bradycardia, extrasystoles, abnormal ST-T deflection, tall, deeply inverted, or notched T waves, prominent U waves, and prolonged Q-T intervals have been described. The possible mechanism responsible for these changes has been discussed by Abildskov et al. (1970), who concluded that the E.C.G. abnormalities may be mediated by abnormalities of the sympathetic tone to the heart. Raised intracranial pressure as judged by isolated readings of cerebrospinal fluid pressure at lumbar puncture was thought not to be responsible for E.C.G. abnormalities (Hersch, 1964). It is now well established that intracranial pressure is not a static phenomenon and its dynamics can only be assessed by measuring it directly and continuously (Lundberg, 1960; Langfitt, 1969).

We describe here seven patients in whom serial E.C.G.s were recorded while their intracranial pressure was continuously

monitored by placing a pressure-sensitive transducer in the extradural space as part of a different study. To the best of our knowledge similar studies have not been reported in the literature.

Patients and Methods

Seven patients with varying intracranial conditions and with no evidence of cardiovascular disease were assessed. The intracranial pressure was continuously monitored using a Konigsberg extradural transducer. Whenever the intracranial pressure changed 12-lead E.C.G.s were recorded. Serum electrolytes and blood gases were also assessed in all these patients. Intracranial pressure was measured in millimetres of mercury (1 mm Hg = 0.13332 kPa) and 15 mm Hg was considered as the upper limit of normal. The intracranial pressure was above normal limits in five patients. A combination of dexamethasone and glycerol was used to lower the intracranial pressure to within normal limits and E.C.G. abnormalities were recorded several times as the intracranial pressure changed. In the remaining two patients who were admitted with head injuries the intracranial pressure was within normal limits throughout and they did not show any E.C.G. abnormalities.

Case Reports

Case 1.—A 47-year-old man was admitted with a head injury. He had had no previous cardiac disability. His blood pressure remained between 130 and 150 mm Hg systolic and between 80 and 90 mm Hg diastolic throughout the period of illness. The intracranial pressure went up to 45 mm Hg, and with the changing pressure the E.C.G. abnormalities recorded were tachycardia, changes in ST-T segment, U waves, changes in voltage, and shortening of Q-T interval (fig. 1).

Case 2.—A man aged 50 years with benign intracranial hypertension and with no cardiac disability was admitted for investigations and treatment of raised intracranial pressure. His blood pressure throughout was around 140/85 mm Hg. The E.C.G. abnormalities with changing intracranial pressure were tachycardia, S-T depression, U waves, and shortening of Q-T interval as shown in the table.

Case 3.—A 43-year-old man was admitted with right temporal lobe tumour (glioma). His blood pressure throughout was 130/90 mm Hg. With changing intracranial pressure the E.C.G. abnormalities noted in him were a slightly raised S-T segment, U waves, and prolongation of Q-T intervals as shown in the table.

Case 4.—A 45-year-old woman was admitted with recurrent tumour of right cerebral hemisphere (glioma). Her systolic blood pressure varied between 145 and 160 mm Hg and her diastolic pressure was 90 mm Hg. The intracranial pressure at one time had gone up to 65 mm Hg. Bradycardia, progressive T-wave inversion, U waves, and shortening of the Q-T interval were the abnormalities recorded in her E.C.G.s (fig. 2).

Case 5.—A 21-year-old man was admitted with a head injury. He had sustained extensive cerebral contusion and his intracranial

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pressure at one time was 85 mm Hg. He had no past history of cardiac disability and did not drink alcohol. Notched T waves were the only abnormality observed in his E.C.G.s (fig. 3).

FIG. 1—Case 1. E.C.G.s at intracranial pressures of (a) 15, (b) 45, and (c) 5 mm Hg showing tall T waves in (a), flat ones in (b), and normal ones in (c). U waves are seen only in (a) and (b). Voltage decreased with rising pressure.

Results and Discussion

The two patients with normal intracranial pressure showed no E.C.G. abnormalities. The remaining five patients with raised intracranial pressure showed normal sinus rhythm. With changing pressures certain E.C.G. abnormalities were observed.

Rate.—In two patients (cases 1 and 2) the heart rate increased as the intracranial pressure went up to 45 mm Hg and 30 mm Hg respectively. Both these patients were restless. The heart rate in two other patients (cases 3 and 4) became slower as the intracranial pressure went up to 125 mm Hg and 65 mm Hg respectively. These two patients were comatose. It seems that the Cushing response (Cushing, 1902) starts appearing above an intracranial pressure of 45 mm Hg, when the patient is drowsy

FIG. 2—Case 4. E.C.G.s at intracranial pressures of (a) 30 and (b) 65 mm Hg showing progressive T wave changes and presence of U waves.

FIG. 3—Case 5. E.C.G. at intracranial pressure of 85 mm Hg showing notched T waves.

and not restless. According to Burch *et al.* (1954) in patients with cerebrovascular accidents the heart does not exceed 100/min, but in one of our patients (case 1) the heart rate had gone up to 115/min when the intracranial pressure was 45 mm Hg.

T Waves.—In all five patients the T waves were abnormal.

Various Electrocardiographic Abnormalities noted in Five out of Seven Patients with High Intracranial Pressure (I.C.P.). E.C.G. Abnormalities have been Correlated to Changing Intracranial Pressure

	Case 1			Case 2		Case 3			Case 4		Case 5
I.C.P. (mm Hg):	15	45	5	10	30	45	125	10	30	65	85
Rate/min	100	115	100	85	94	71	68	79	70	63	79
T Wave	Tall in V3-6	Flat	Normal	Flat in aVL	Flat in aVL	Flat in I, III, 3aVL	Flat in III, aVL	Flat in aVL	Flat in III, aVL	Inverted in III, aVF	Notched in II, V2-5
S-T Segment	Normal	Slightly depressed in II, III	Normal	Slightly depressed in II, III	More depressed in II, III, aVF	Slightly raised in II, III, aVF	Slightly raised in II, III, aVF	Normal	Normal	Normal	Normal
U Wave	+	+	Absent	-	+	+	+	-	+	+	-
Voltage SV2 + RV5 (mm)	26	19	27	25	24	28	28	16	25	18	38
P-R Period (s)	0.14	0.14	0.14	0.2	0.18	0.18	0.18	0.16	0.18	0.16	0.12
R-R Period (s)	0.60	0.52	0.60	0.7	0.64	0.84	0.88	0.76	0.86	0.96	0.76
QRS	0.08	0.06	0.08	0.08	0.06	0.06	0.06	0.04	0.08	0.08	0.06
Complex (s)											
Q-T Period (s)	0.44	0.39	0.41	0.43	0.40	0.39	0.40	0.39	0.41	0.39	
Corrected											0.42

They were flat in three patients, notched in one patient, and tall in the other. The tall T waves of the type described by Burch *et al.* (1954) and Schuster (1960) in patients with cerebrovascular diseases were seen in only one of our patients (case 1). Burch *et al.* (1954) have said that the amplitude and duration of these T waves are greatest during the period of bradycardia, but tall T waves seen in case 1, when the intracranial pressure was 15 mm Hg, were associated with tachycardia. We suggest that tall T waves are an early E.C.G. manifestation of rising intracranial pressure. In case 1 the tall T waves became flat as the intracranial pressure went up to 45 mm Hg and reverted to normal as the intracranial pressure was brought down to 5 mm Hg. The T waves became progressively inverted in case 4 when the pressure went up to 65 mm Hg. These T wave changes were usually seen in the standard leads II, III, aVL, and aVF. Notched T waves best seen in the chest leads were seen in only one patient (case 5) when the I.C.P. was 85 mm Hg. Though it is reported that notched T waves can also occur in patients with alcoholic heart disease our patient who showed notched T waves was a total abstainer.

S-T Segment.—Only two patients (cases 1 and 2) showed progressive S-T depression with increasing intracranial pressure. These changes, like T wave changes, were best seen in standard leads II, III, aVL, and aVF. In one patient (case 3) the S-T segment was raised with high intracranial pressure, and it became normal when the pressure was brought down to 10 mm Hg. The changes were similar to those seen in myocardial ischaemia but these changes subsided as the raised intracranial pressure was brought down to normal levels. This would also suggest that E.C.G. changes seen in central nervous system lesions are often reversible and therefore unlikely to be due to any structural damage in the central nervous system. Myocardial damage associated with intracranial bleeding has been described (Smith and Tomlinson, 1954; Koskelo *et al.*, 1964; Abildskov *et al.*, 1970); it is seen in only 12.3% of cases and usually affects the subendocardial tissue of the left ventricle (Smith and Tomlinson, 1954). This could account for some of the ST-T segment changes.

U Waves.—Prominent U waves were seen in all the patients with an intracranial pressure of 30 mm Hg and above except in the patient in case 5 who showed notched T waves. Only one patient with an intracranial pressure of 15 mm Hg showed prominent U waves. None of the patients with an intracranial pressure lower than 15 mm Hg showed U waves. Prominent U waves were best seen in the chest leads. In two patients the U waves disappeared as the raised intracranial pressure was brought down to within normal levels.

Voltage.—Changing intracranial pressure has no effect on the voltage of the QRS complex and it remained within the normal range in four patients. In case 1 voltage diminished with the increasing intracranial pressure but it again became normal as the pressure was brought down to 5 mm Hg.

P-R and QRS Intervals.—These intervals remained within the normal range in all the patients with intracranial pressure up to 30 mm Hg. The QRS duration remained between 0.06 and 0.08 seconds when the intracranial pressure rose above 30 mm Hg. In only one patient (case 2) the QRS duration was 0.18 seconds. No other conduction defect was seen.

Q-T Interval.—The Q-T intervals were corrected for their R-R intervals. Initial Q-T intervals ranged from 0.39 to 0.43 seconds. The Q-T intervals became shorter as the intracranial pressure increased except in case 3 where the intracranial pressure went up to 120 mm Hg and the Q-T interval became more prolonged. The relationship between Q-T interval and the intracranial pressure is shown in fig. 4. Contrary to the current concept, the Q-T interval became shorter as the intracranial pressure went up to 65 mm Hg. In case 3 the Q-T interval

became more prolonged as the pressure went up to 120 mm Hg. It is suggested that initially the Q-T interval shortens until the intracranial pressure reaches 65 mm Hg, but thereafter it becomes more prolonged (case 3) when the pressure rises higher than 65 mm Hg. Thus, a prolonged Q-T interval might be an ominous sign.

FIG. 4—Q-T intervals in relation to intracranial pressure (I.C.P.). Q-T interval progressively shortens as intracranial pressure rises except in case 3 in which Q-T interval was prolonged when intracranial pressure was 125 mm Hg.

CONCLUSION

These findings not only confirm the association between E.C.G. abnormalities and central nervous system diseases but also suggest that many of the E.C.G. abnormalities are related to changing intracranial pressure. There has been a certain amount of doubt about the role of intracranial pressure in the production of E.C.G. abnormalities. But never before have E.C.G. changes been correlated with direct and continuous monitoring of intracranial pressure. With the advancement in technology most neurosurgical centres today accept direct and continuous monitoring of intracranial pressure as an important index in the treatment of patients. By using this facility it is hoped to evaluate the pathophysiology of these changes more thoroughly.

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References

- Abildskov, J. A., *et al.* (1970). *Progress in Cardiovascular Diseases*, 13, 210.
- Byer, E., Ashman, R., and Toth, L. A. (1947). *American Heart Journal*, 33, 796.
- Burch, G. E., Meyers, R., and Abildskov, J. A. (1954). *Circulation*, 9, 719.
- Cushing, H. (1902). *American Journal of Medical Science*, 124, 375.
- Cruickshank, J. M., and Dwyer, G. N. (1974). *British Heart Journal*, 36, 395.
- Fentz, V., and Gormsen, J. (1962). *Circulation*, 25, 22.
- Finkelstein, D., and Nigaglioni, A. (1961). *American Heart Journal*, 62, 772.
- Goldman, M. J. (1967). *Principles of Electrocardiology*, 6th edn., p. 282. Los Altos, California, Lange.
- Hersch, C. (1961). *Circulation*, 28, 853.
- Hersch, C. (1964). *British Heart Journal*, 26, 785.
- Koskelo, P., Punsar, S., and Sipila, W. (1964). *British Medical Journal*, 1, 1479.
- Langfitt, T. W. (1969). *Clinical Neurosurgery*, 16, 436.
- Lundberg, N. (1960). *Acta Psychiatrica Neurologica Scandinavica*, 36, Suppl. 149.
- Schuster, S. (1960). *British Heart Journal*, 22, 316.
- Smith, R. P., and Tomlinson, B. E. (1954). *Journal of Pathology and Bacteriology*, 68, 327.