# CARDIAC IRREGULARITIES DURING LABOUR IN PARAPLEGIC WOMEN

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IN previous publications, attention has been drawn to transient irregularities of the cardiac rhythm accompanying hypertension as a result of visceral activity, especially of the bladder, in patients with complete lesions of the spinal cord at and above the splanchnic outflow (Guttmann & Whitteridge, 1947; Guttmann, 1953, 1954). These irregularities were demonstrated electrocardiographically and consisted of extra-systoles of sino-auricular and auriculo-ventricular origin and increase in the size of T- and in particular U-waves at the height of bladder distension. It was assumed that an increase in the load of the heart was the cause of these disturbances of cardiac rhythm, resulting from a vasoconstriction in the paralysed parts of the body elicited by the distension of the bladder. Actually, in some of these patients with high cord lesions, the heart shadow showed an increase in diameter of several centimetres at the height of the visceral activity due to distension. However, the disturbances of cardiac rhythm as well as hypertension disappeared promptly with cessation of bladder distension.

In a recent paper on pregnancy and labour in 20 paraplegic women (Guttmann, 1963), attention was drawn to various reflex responses of the autonomic system, in particular, cardiovascular changes in relation to uterine contractions, which were observed in a young woman with tetraplegia below C6 following fracture-dislocation of the 5th cervical vertebra. Although, during delivery, the blood pressure rose to 190/100 and the pulse rate fell to 48/50, no irregularities of cardiac rhythm were noted on auscultation (electrocardiographic examination was not carried out in this case). The hypertension disappeared promptly after delivery and the bradycardia was followed by a temporary tachycardia of 100 per minute.

Since that publication, several more paraplegic women with complete and incomplete cord lesions were studied in detail during labour, amongst them two with complete lesions below T5 and T6. They confirmed the previous findings that uterine contractions during labour elicited profound reflex responses of autonomic mechanisms, the most important being intermittent hypertension combined with bradycardia coinciding with the uterine contractions. However, one young woman with a complete lesion below the 5th dorsal segment, following fracture-dislocation of the spine, developed, in addition to the usual changes of the vascular system described above, profound cardiac irregularities during the last stages of labour. These were studied in detail electrocardiographically and are presented in this paper.

**Case Report.** Miss J. W., then aged 18, who had previously enjoyed good health, was a pillion passenger on a motor-cycle involved in a road accident on 11.5.58. She suffered from:

1. Fractured skull, remained unconscious for six weeks.

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2. Fracture-dislocation T3/4 (two weeks after injury had a laminectomy and spinal fusion) leaving a transverse spinal cord syndrome complete below T5.
3. Fracture shaft right femur.

On 8.8.58 admitted to National Spinal Injuries Centre, Stoke Mandeville Hospital, when her mental condition was good, she has however remained rather talkative and excitable, she had a right abducens paresis which recovered and complete anosmia which has persisted. She had a complete spastic paraplegia below T5 segment.

She was treated and rehabilitated here and was discharged home on 13.12.58. Although the paraplegia remained complete below T5 she was independent, able to dress herself, get into and out of her wheelchair. Her urine was sterile and an intravenous pyelogram was normal. She had about one minute's warning of automatic bladder emptying; this warning consisted of tingling and sweating on her forehead, bowel action produced similar sensations.

She lived with her parents and took up full-time secretarial work until her marriage in 1962. She attended Stoke Mandeville for check-ups in 1960, 1961 and 1962, on each occasion her neurological lesion was unchanged. Her urine was sterile and intravenous pyelograms normal. Her menstrual periods restarted two months after the accident and remained regular 5/28. (During intercourse she had no sensation in lower part of body.)

*Pregnancy*

Last menstrual period 27.11.62.

Expected date delivery 4.9.63.

The pregnancy was uneventful, B.P. remained at 120/80 or less, there was a trace of albumin on 23.7.63, a sub-clinical urinary infection was treated with Furadantin.

As premature labour is anticipated in paraplegics she was admitted to Stoke Mandeville Hospital on 8.7.63. A vaginal examination on 15.7.63 showed the cervix to be effaced, a further vaginal examination on 25.7.63 showed no change. A further examination on 1.8.63 showed the cervix to be 2 fingers dilated.

*Labour*

At 8 p.m. on 3.8.63 regular uterine contractions every 5 to 7 minutes started, no abdominal sensations, but with each contraction noticed tingling and flushing of face. During contraction B.P. rose from 120/75 to 130/80 and pulse rate dropped from 64 to 56. The cervix was still 2 fingers dilated. As it seemed that patient was in early labour she was transferred to Royal Bucks Hospital. During the next seven days irregular, mild contractions continued but there was no further dilatation of cervix.

On 11.8.63 at 8 p.m. uterine contractions became stronger and regular 1 : 8 with each contraction B.P. rose to 150/100, pulse dropped to 60, at the end of each contraction the patient felt tingling, sweating, flushing of face and a burning feeling in the feet. Her cervix was 3 fingers dilated. Contractions continued through the night and by 11 a.m. on 12.8.63 were regular and strong 1 : 3 with similar subjective sensations. A vaginal examination was performed and the membranes ruptured, the cervix was nearly dilated, there being only a rim $\frac{1}{2}$ cm. all round. During this vaginal examination patient developed a frontal headache which persisted and became more severe until the baby was delivered. From this time on there were regular strong contractions 1 : 3 with each contraction the headache became more severe, there was sweating and flushing of face and neck, she vomited several times. During the last half-hour of labour, each uterine contraction was accompanied by strong, rapid clonic spasms in legs.

During the rest of labour, there was a progressive rise in B.P. and fall in pulse rate.

At 1.47 p.m., B.P. had risen to 210/105 and the headache was severe. As the cervix was fully dilated, the patient was catheterised (6 oz.), an episiotomy was performed, forceps applied (Dr. A. Moolgaoker), and a healthy premature male infant 5 lb. 8 oz. delivered at 1.56 p.m. The placenta was delivered at 2 p.m. and the episiotomy repaired.

The headache was most intense during delivery, then disappeared, returned during delivery of the placenta, disappeared again and was replaced by a moderate pain in the back of the neck with slight neck stiffness which lasted 20 minutes.

No drugs were given, apart from an i.m. injection of 1 ml. of syntometrine given with delivery of the anterior shoulder. No local anaesthetic was given for repair of episiotomy; the insertion of each suture produced a strong contraction of the anal sphincter but no subjective sensation and no rise in B.P.

Figure 1 demonstrates the changes in blood pressure and pulse rate before, during and after delivery.

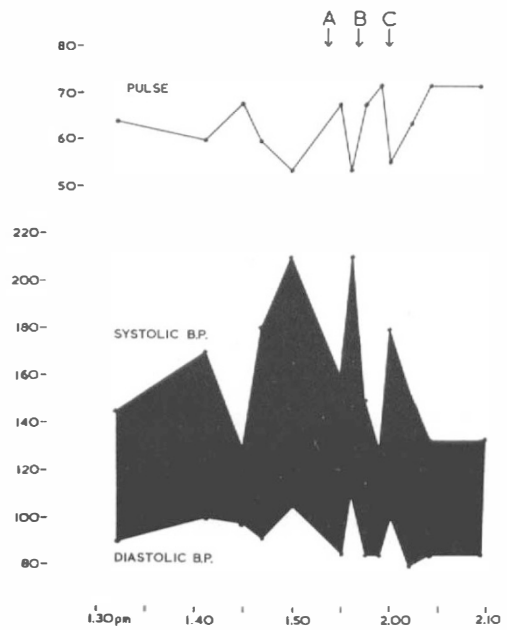


FIG. 1

At *A* the forceps were applied, at the *B* delivery was completed, at *C* the placenta was delivered. As will be seen, the bradycardia coincides with peak of the raised blood pressure.

Of special interest in this case were cardiac irregularities developing during the various stages of labour, which could be recorded electrographically.

At 1 p.m., during a uterine contraction, an irregularity of the pulse was first noted; clinically, this seemed to be due to extra-systoles. E.C.G. recordings started at 1.25 p.m. During the rest of the labour, a number of arrhythmias were recorded, which are demonstrated in detail in Figures 2 to 11. Various arrhythmias were demonstrated, starting with prolongation of the P-R interval with regular ventricular extra-systoles (figs. 2 and 4) followed by 2nd degree A-V block with A-V escape beats (fig. 3). On occasions when there was only a moderate rise of blood pressure, a bradycardia with normal sinus rhythm was found (fig. 5). As the hypertension increased (1.52 p.m., fig. 6), 2nd degree A-V block returned and there were ventricular extra-systoles differing in shape and direction from those previously seen (fig. 7). During the actual delivery the P waves disappeared and regular ventricular extra-systoles occurred firstly at every third beat (fig. 8) and later at every alternate beat (fig. 9).

As soon as the baby had been delivered, the electrocardiogram became normal except that during the delivery of the placenta there were two more ventricular extra-systoles (fig. 10). The three standard leads after delivery are shown in Figure 11.

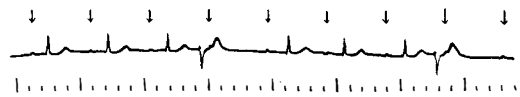

FIG. 2

1·28 p.m. Towards end of 1st stage of labour. One and a half hours after rupture of membranes.

Lead I. P-R interval 0·28 seconds. Every third conducted beat is followed by a ventricular extra-systole which is followed by a compensatory pause.

The arrows marking the P-waves show the fourth and eighth P-waves distorting the S-T segments of the extra-systoles.

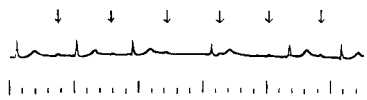

FIG. 3

1.30 p.m. Lead I. P-R-interval 0·32 seconds. P-waves marked by arrows. The third P-wave is not followed by a ventricular complex and the ensuing interval is terminated by an A-V escape beat, the S-T segment of which is deformed by the next P-wave.

FIG. 4

1.31 p.m. Lead I. Every third conducted beat is followed by a ventricular extra-systole. In contrast with Figure 2 the P-R interval is normal.

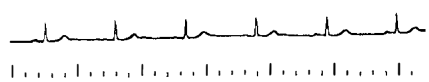

FIG. 5

1.38 p.m. Lead I. Bradycardia, rate 54 per minute with normal sinus rhythm during uterine contractions with moderate rise of blood pressure.

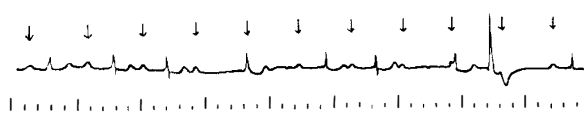

FIG. 6

1.52 p.m. Height of hypertension. Lead I. P-waves marked by arrows. The first P-R interval is 0·34 seconds, the next two are 0·4 seconds, the fourth P-wave is not conducted and the ensuing interval is terminated by an A-V escape beat, the fifth P-wave is hidden in the ventricular complex, thus the first part of the tracing shows 2nd degree A-V block 4 : 3. The sixth and seventh P-waves are conducted (P-R interval 0·4 seconds), but the eighth is not; the interval is again terminated by an A-V escape beat, the R-wave of which is deformed by the next P-wave (2nd degree A-V block 3 : 2). Following this, there is a ventricular extra-systole the S-T segment of which is deformed by the next P-wave. This extra-systole differs in shape and direction from those seen in Figures 2 and 4.

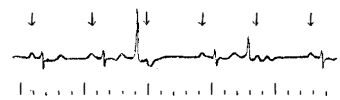

FIG. 7

1.52 p.m. Height of hypertension. Lead I. Two ventricular extra-systoles of different shape and size; the third and fifth arrows show P-waves distorting the S-T segment of the extra-systoles.

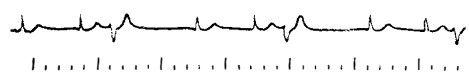

FIG. 8

1.55 p.m. During delivery. Lead I. No P-waves seen. Every 2nd supra-ventricular beat is followed by a ventricular extra-systole. The extra-systoles are again similar to those seen in Figures 2 and 4.

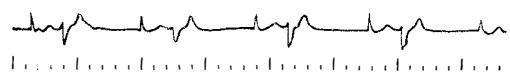

FIG. 9

1.55 p.m. During delivery. Lead I. No P-waves seen. Each supra-ventricular beat is followed by a ventricular extra-systole ('coupling' or 'bigeminus').

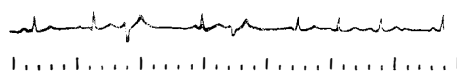

FIG. 10

1.59 p.m. During delivery of placenta. Lead I. Sinus rhythm, two ventricular extra-systoles.

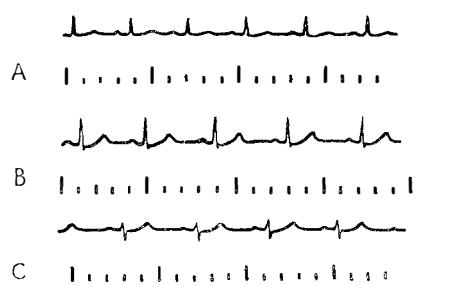


FIG. 11

2.32 p.m. 35 minutes after delivery of placenta. Leads I, II, III. Return to normal.

## DISCUSSION

Although irregularities of cardiac rhythm associated with hypertension in response to bladder distension have been described in patients with high transection of the spinal cord (Guttmann & Whitteridge, 1947; Kendrick *et al.*, 1953; Garnier *et al.*, 1963), this seems to be the first account of the occurrence of cardiac irregularities accompanying intermittent hypertension related to uterine contractions

during labour. We have demonstrated bradycardia, abnormalities of A-V conduction, varying from simple prolongation of the P-R interval to second degree block with A-V nodal escape beats, disappearance of the P-waves and, during the actual delivery of the child, ventricular extra-systoles which often occurred regularly after every 3rd or 2nd conducted beat, and coupling. The ventricular extra-systoles occurred in different directions and were of different shape. There has been a close relation between uterine contractions, hypertension and the arrhythmias. In the intervals between the uterine contractions and after delivery of the child, the electrocardiogram was normal.

It is of interest to compare our findings with cardiovascular changes occurring in normal women during labour. The electrocardiogram during labour in non-paraplegic women, unless they are suffering from heart disease, show no abnormalities apart from occasional extra-systoles in sympatheticotonic subjects. Hendricks and Quilligan (1956), using pulse pressure methods, found an increase in cardiac output of 30·9 per cent. during normal uterine contractions. Adams and Alexander (1958) found that, during normal uterine contractions, cardiac output increased by 19·7 per cent. (dye dilution technique), and there was a rise in mean blood pressure from 94 to 120 mm. Hg., accompanied by a rise in pulse rate of 14 per cent. This is quite different from the findings in patients with high spinal cord lesions described previously (Jung & Schmidt; 1962, Guttmann, 1963) and also from those in the present case, where the rise of blood pressure with uterine contractions was infinitely larger and associated with a fall in pulse rate.

In none of the paraplegic patients with high lesions studied so far during labour were cardiac irregularities of the type described in the present case found. Although no electrocardiograph readings were made in the case of tetraplegia described by Guttmann in 1963, such profound disturbances of cardiac rhythm as found in the present case would certainly not have escaped notice by auscultation.

The case of complete lesion below T5 described by Jung and Schmidt (1962) at first developed intermittent hypertension rising to 230/110 mm. Hg., coinciding with every uterine contraction. These hypertensive attacks were associated with 'dramatic convulsions and profound headaches'. Very soon, the intermittent blood pressure crises were replaced by a slow continuing rise to 230/145 associated with vomiting. Although immediate Caesarian section was performed and a healthy boy was delivered, the mother had developed a cerebral haemorrhage which left her with a paralysis of the left arm, paralysis of ocular movements and a left facial paralysis. The authors explain the intermittent hypertension, which started following rupture of the membranes when the child's head was just above the interspinous plane, as a viscero-spinal reflex response, in accordance with Guttmann's and Whitteridge's investigations on the effect of bladder distension on autonomic mechanisms, while the continuing increase of the blood pressure is explained as a result of acute cranial pressure due to cerebral haemorrhage from a post-traumatic aneurysm resulting from the woman's accident and elicited by the hypertension during labour.

In analysing the mechanism of the irregularities of cardiac rhythm observed in the present case, no evidence was found that they are peculiar to pregnancy and labour in women with high spinal cord lesions as such, but are just another manifestation of the exaggerated viscero-spinal reflex response described following distension of the bladder. The difference in the intensity and extent of the cardiac irregularities is explained by the differences in duration, frequency and intensity of the visceral

stimulation. The diagnostic distension of the bladder can be stopped very soon after the appearance of cardiac irregularity, while labour and delivery in the present case had to take its natural course. On the other hand, it must be remembered that, in the woman with tetraplegia below C6 described previously (Guttmann, 1963), where visceral stimulation during labour and delivery must have been no less severe than in the present case, no such irregularity of cardiac rhythm was noticed, although the bradycardia (48/54 per minute) and hypertension (190/100 mm. Hg.) were profound. Although, in both cases, the increase in blood pressure is most likely to be responsible for the observed bradycardia as part of a depressor reflex aroused by the intact aortic and carotid sinus nerves and the prevalence of vagal stimulation, one cannot exclude the response of the autonomous innervation of the heart itself to the visceral stimulation as an important if not decisive factor in eliciting these arrhythmias. Whether and to what extent a humoral factor—*i.e.* increased level of pressure amines locally and/or circulating may be responsible as initiator of the marked disturbances of cardiac rhythm needs further investigation.

In this connection, it is of interest to compare our findings with those described in paroxysmal hypertension due to phaeochromocytoma, where during acute attacks the heart rate is usually increased (Hegglin & Holzmann, 1937; Raab, 1950), but occasionally bradycardia is also found during hypertensive paroxysms (Porter & Porter, 1930; Hegglin & Holzmann, 1937; Mortell & Whittle, 1945). Irregularities of cardiac rhythm may occur during and immediately after the acute hypertensive paroxysms, and these include extra-systoles and auricular fibrillation (Hegglin & Holzmann, 1937; Espersen & Jorgensen, 1947), atrio-ventricular block, dissociation with interference, and wandering pacemaker (Burgess *et al.*, 1936; Hegglin & Holzmann, 1937; Mortell & Whittle, 1945; Espersen & Jorgensen, 1947). Raab (1953) concluded that some of these arrhythmias could be interpreted as a result of continued primary sympathetic and simultaneous secondary vagal stimulation.

## Summary

Paraplegic women with complete lesions of the spinal cord at and above the splanchnic outflow (T5) develop during the final stages of labour, especially just before and during delivery, the classical symptoms of reflex-hyperactivity of autonomic mechanisms (hypertension, bradycardia, outburst of vaso-dilatation of the face, headaches, and sweating). These symptoms are closely related to the uterine contractions and disappear or are greatly diminished during the free intervals.

A case of a woman with a complete transverse lesion below T5 is described, where, during the uterine contractions in the later stages of labour and during delivery, profound disturbances of the cardiac rhythm were observed and studied in detail electrocardiographically. These arrhythmias accompanying the intermittent hypertension were closely related to the uterine contractions.

The mechanism of the cardiac arrhythmias is discussed.

## Résumé

Les femmes paraplégiques avec une lésion médullaire complète, au niveau, et au-dessus, de l'émergence du sympathique (D5) présentent au cours des dernières phases du travail, juste avant et pendant la naissance, les symptômes classiques d'hyperréflectivité autonome (hypertension, bradycardie, bouffées de vaso-dilatation faciales, céphalées et transpiration).

L'apparition de ces symptômes est en relation directe avec les contractions utérines, disparaissent ou s'atténuent considérablement pendant les périodes de repos.

Le cas d'une femme, avec lésion transverse complète au dessous de D5, est présenté, qui, pendant les contractions à la phase terminale du travail, et au cours de la délivrance, a présenté des troubles marqués du rythme cardiaque qui ont été étudiés à l'électro-cardiogramme. Ces arythmies accompagnées d'hypertension intermittente seraient en relation avec les contractions utérines, et leur mécanisme discuté.

## ZUSAMMENFASSUNG

Paraplegische Frauen mit kompletter Lähmung im oder über dem Niveau der segmentalen Splanchnikus Innervation (T5) zeigen die klassischen Symptome der Übererregbarkeit des autonomen Nervensystems (hoher Blutdruck, Bradycardie, Vasodilatation des Gesichtes, Kopfschmerzen und Schwitzen) während des Spätstadiums der Entbindung, besonders unmittelbar bevor und während der Geburt.

Diese Symptome sind eng verbunden mit den Wehen und werden zwischen ihnen viel schwächer oder verschwinden.

Der Fall einer Frau mit einer kompletten T5 Läsion wird beschrieben, in dem während der Wehen im Endstadium der Entbindung schwere Störungen des Herzrhythmus beobachtet und in allen Einzelheiten elektrocardiographisch untersucht wurden. Diese Arrythmien begleiteten den intermittierenden Hochdruck und die Bradykardie und gingen mit den Wehen parallel.

Die Pathogenese der kardialen Arrhythmie wird erörtert.

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