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Pregnancy and Autonomic Hyperreflexia in Patients with Spinal Cord Lesions

M. B. Wanner, M.D.,[1] C. J. Rageth, M.D.,[1] G. A. Zäch, M.D.[1]
[1] *Swiss Paraplegic Centre, 4055 Basel, Switzerland,* [1] *University Women's Hospital, 4031 Basel, Switzerland*

Summary

Symptoms of autonomic hyperreflexia in patients with complete and incomplete paraplegia above D 7 can be caused by almost any stimulus in the abdominal area or in the lower extremities, specifically during parturition by the uterine contractions. The symptoms vary from pilo-erection and outbreaks of sweating to serious blood pressure crises and cerebrovascular accidents. Epidural anaesthesia and general anaesthesia are effective as therapy and also as prophylaxis. Frequent complications are anemia and urinary tract infections. Changes in bladder function as a result of pregnancy and childbirth were observed. Paraplegic expectant mothers experience premature labour pains more frequently than do others, and this implies the necessity for earlier clinical surveillance up to the time of delivery. The secondary uterine inertia frequently requires an operative termination of the birth. The perception of labour pains is clearly possible also with lesions above D 10.
Key words: *Pregnancy; Spinal cord lesion; Autonomic hyperreflexia; Prevention.*

Introduction

Definition. Under autonomic hyperreflexia, the main effects of the unrestrained activities of the sympathetic system, due to spinal cord lesions above the splanchnic outflow can be summarised as follows.

The symptomatology of autonomic hyperreflexia, caused by dilatation of the bladder, was described for the first time in 1917 by Head and Riddoch. Autonomic hyperreflexia occurs in patients with complete and incomplete lesions (Bors and Comarr, 1971), the damage to the spinal cord must lie in the upper thoracic or cervical region. Various sources indicate a niveau of D 7 (Nath *et al.*, 1979; Schumacher, 1974; Stirt *et al.*, 1979). With lesions above D 5 one can expect, according to this textual evidence, that in 85% of the cases, under the corresponding stimulation, there will be symptoms of autonomic hyperreflexia (Desmond, 1970; Guttmann and Whitteridge, 1947).

Precipitating mechanisms

Practically any stimulus below the lesion can lead to autonomic hyperreflexia: including increased pressure in the abdominal cavity, dilatation of viscera, especially of the pelvic organs, percussion of the bladder, stimulation of the rectum, renal or biliary colic, vesico-ureteral reflux, manipulations of indwelling catheters, contraction of the uterus, introduction of a vaginal speculum (Bedbrook, 1981; Corbett *et al.*, 1975, Guttmann and Whitteridge, 1947; Hardy and Warrell, 1965; Rossier *et al.*, 1969; Roussan *et al.*, 1966). Summing up, the most frequent causes are filling of the bladder, meteorism and uterine contractions.

Symptoms and findings of autonomic hyperreflexia

As an expression of the sympathetic activity brought about by the causes mentioned above, there is a very varied picture, both quantitatively and qualitatively. Subjective symptoms can appear, such as nausea, anxiety, malaise, a prickling sensation in the skull region, a ringing in the head and characteristic throbbing headache. The objective signs vary also from light sweating and blushing, pilo-erection, tremor, nasal obstruction, increasing spasticity in all extremities, twitching, rise in blood pressure, temporary loss of consciousness, to a state of unconsciousness, bleeding into the retina as well as subarachnoid haemorrhage, and cerebro-vascular stroke that can lead to death (Abouleish, 1980; Frankel *et al.*, 1965; Guttman, 1963; Göller and Paeslack, 1970; Hardy and Warrell; 1965, Kurnick: 1956, McGregor, 1985). A characteristic feature is reactive sinus bradycardia (Frankel *et al.*, 1965; Nath *et al.*, 1979; Stirt *et al.*, 1979). Other rhythm disturbances are found in the ECG: prolongation of the PV-interval, AV-blocks of the second degree, multitope ventricular extrasystoles, disappearance of the P-waves and transition to an AV-rhythm (Frankel *et al.*, 1965). A bigeminal rhythm after bladder dilatation was described (Guttmann and Whitteridge, 1947). The composition of the blood is changed: the plasma volume is reduced by 10—15%, the hematocrit raised by 9.5% and the plasma protein concentration increased (Naftchi *et al.*, 1978).

Pathological physiology

The causal mechanisms mentioned above are conducted via the afferent, somato- and viscerosensory pathways over the posterior roots into the lateral horns of the spinal cord (Kurnick, 1956). The afferents for the pelvic organs are carried through the pelvic and pudendal nerves and enter the spinal cord in the sacral segments 2—4 (vagina and cervix). A different pathway to the hypogastric plexus to the ganglion of the sympathetic trunk at the level of the lumbar segments. Rising in the sympathetic trunk to approximately D 11 and 12, the stimuli finally enter the spinal cord (Aminoff, 1978; Netter, 1983; Rossier *et al.*, 1969). From there the impulses find their way over ascending tracts into the spino-thalamic tract and the posterior white columns (Nath, 1979; Rossier *et al.*, 1969) the brain stem and higher centres. Because of spinal cord lesions (Guttmann and Whitteridge, 1947; Head and Riddoch, 1917; Ravindran *et al.*, 1981) there

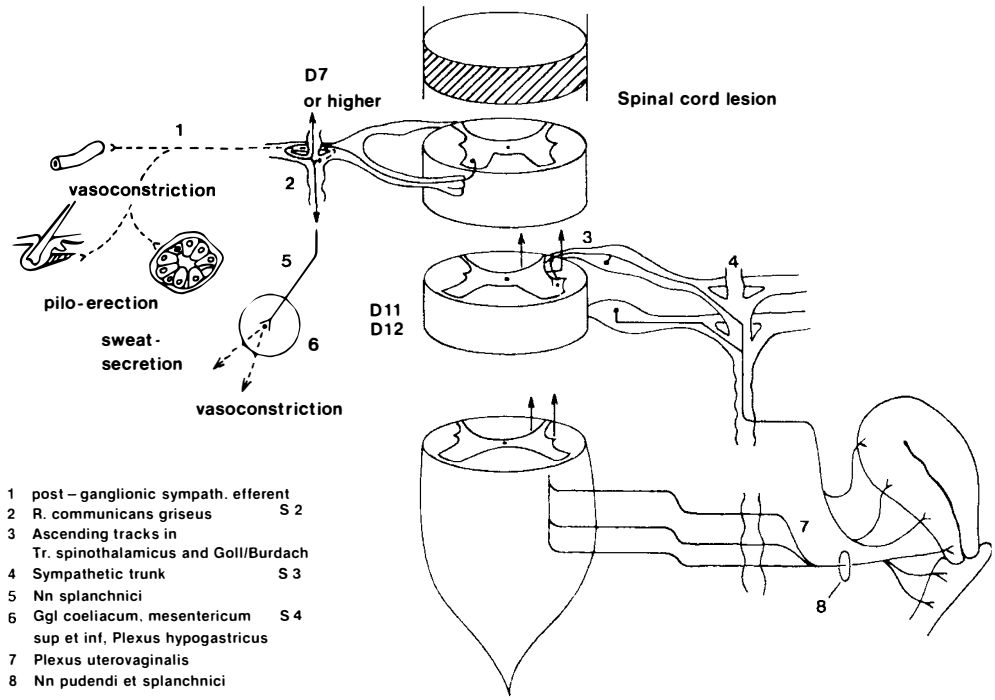

Figure 1 Afferent and efferent tracts in cases of autonomic hyperreflexia (Cole *et al.*, 1967; Frankel *et al.*, 1965; Kahle, 1984; Netter, 1983: Ramzin and Hinselmann, 1979; Ravindran *et al.*, 1981; Rossier *et al.*, 1969; Watson and Downey, 1980)

is no influence exerted on higher centres to centrally counterbalance the increased sympathetic activity below the lesion. As a result of stimulation of the vaso-motor neurones, vasoconstriction takes place, especially in the splanchnic region, which leads to a rise in blood pressure (Nath *et al.*, 1979; Ravindran *et al.*, 1981).

Sweating attacks also occur by sympathetic stimulation. The headache accompanying the autonomic hyperreflexia is explained by stretching of the pial arteries (pia mater) when the intravascular pressure is raised (Desmond, 1970; Nath *et al.*, 1979; Rossier *et al.* 1969), but also through the increased spill of prostaglandins which, it is assumed, leads to direct cerebral vasodilation (Naftchi *et al.*, 1978).

An additional pathogenetic factor for the rise of blood pressure could be with tetraplegics increased 'vascular hypersensitiveness' (Corbett *et al.*, 1971).

An increased sensitivity of the vessels to noradrenalin was noted (Debarge *et al.*, 1974). The concentration of the catecholamines in the blood is raised by an attack of autonomic hyperreflexia (Debarge *et al.*, 1974; Naftchi *et al.*, 1978). However, the noradrenalin increase is significantly higher than that of adrenalin, so that one can assume that the release of adrenalin is a relatively unimportant factor, contributing little to the increase in blood pressure (Guttmann and Whitteridge, 1947; Naftchi *et al.*, 1978).

As a counter reaction to the raised blood pressure, bradycardia is achieved via

the baro-receptors (Frankel *et al.*, 1965; Rossier *et al.*, 1969; Watson and Downey, 1980) and vagal stimulation (Ravindran *et al.*, 1981; Rossier *et al.*, 1969). The bradycardia is the sole mechanism the body still has (Cole *et al.*, 1967) for adaptation to the altered circumstances of the circulation. The cardiac output is not increased (Naftchi *et al.*, 1978; Ravindran *et al.*, 1981).

Differential diagnosis

In the medical literature, pre-eclampsia and pheochromocytoma are identified as diagnostically differential manifestations (Naftchi *et al.*, 1978). Nevertheless it would seem to be the case that toxemia is not more frequent in pregnant paraplegics than it is in other pregnant women (Bradley *et al.*, 1957; Hardy and Warrell, 1965; Robertson, 1972; Robertson and Guttmann, 1963; Rossier *et al.*, 1969). An important differential diagnostic feature, in addition to observing the other symptoms of severe EPH-Gestosis such as increasing oedema and proteinurea, is the exact documentation of the beginning of labour (Watson and Downey 1980). It has been observed that the symptoms mentioned above usually appear simultaneously with labour pains in patients with autonomic hyperreflexia, whereas in eclampsia labour pains are usually absent (Frankel *et al.*, 1965). If a lesion exists in the cervical region of the spinal cord or below it, then an intensification of the proprioceptive reflexes of the normally innervated muscle implies pre-eclampsia rather than autonomic hyperreflexia (Watson and Downey, 1980).

Prophylaxis and therapy of autonomic hyperreflexia during birth

As an urgent measure to lower the blood pressure the Ca-antagonists (Verapamil, Nifedipin) and Dihydralazin, that has no negative effect on the uterus perfusion, are considered (Rageth *et al.*, 1986). Secondly, ganglion blockers (Trimetaphancamsilat) can be used.

Nitroprusside alone proved inadequate in one patient, and only when it was used in combination with epidural anaesthesia could the hypertensive crisis be brought under control (Ravindran *et al.*, 1981). At the present time, the best known and most effective method for the prevention and treatment of autonomic hyperreflexia is epidural anaesthesia with Lidocain or its derivatives (Baraka, 1985; Nath *et al.*, 1979; Rageth *et al.*, 1986; Ravindran *et al.*, 1981; Tabsh *et al.*, 1982; Watson and Downey, 1980).

It is inadvisable to make use of beta blockers, on account of the vasoconstrictive effects on the uterine vessels, and the cardio-depressive effects. The epidural use of morphia, by blocking the afferents, can also be considered as a therapeutic procedure.

Other complications during pregnancy and birth

As a result of the increasing size of the uterus, bladder voiding can be less complete. Thus it is necessary to insert an indwelling catheter when there is an increase in the residual urine (Robertson and Guttmann, 1963). It sometimes happens that the postpartum function of the bladder undergoes a change. A case

was recorded of a patient who lost her reflex type of bladder (Göller and Paeslack, 1970). Usually, however, the bladder functions just as normally after, as before the birth. A frequent complication is infection of the urinary tract (Comarr, 1966; Robertson, 1972; Robertson and Guttmann, 1963; Rossier *et al.*, 1969; Stöhrer *et al.*, 1984). In cases of chronic infection, exacerbations during pregnancy are possible (Robertson and Guttmann, 1963). Precipitating factors are the increasing quantity of residual urine during the gestation period (consequently a stricter control than usual of the amount of residual urine) and the frequent occurrence of anemia.

Besides the sensibility loss and trophic disturbance brought about by the paralysis, anemia also, is a factor for the increased risk of pressure sores in the paralysed patient during pregnancy and childbirth (Aminoff, 1978). An anemia under 80% Hb (= 11.2 g%) significantly increases the pressure sore frequency and makes blood transfusions and/or substitutes necessary (Aminoff, 1978; Robertson and Guttmann, 1963). Hypoproteinemia should also be regarded as a pathogenetic factor and should be rectified.

The increased susceptibility to venous thrombosis in the spinal paralysed is increased even further by pregnancy. (Oppenhimer, 1971).

Premature onset of labour happens more frequently with paraplegic and quadriplegic patients than with others (Frankel *et al.*, 1965; Rageth, 1986; Ramzin and Hinselmann, 1979; Robertson, 1972). Furthermore, labour pains in case of lesions above D 10 are not identifiable as such with absolute certainty (Aminoff, 1978; Robertson, 1972; Robertson and Guttmann, 1963). It is nevertheless possible to be certain about labour pains by the palpation method (Göller and Paeslack, 1970; and our own observations). An examination of the labour pain activity is necessary when autonomic hyperreflexia is present, where uterine contractions and the sympathetic manifestations appear at the same time. It can happen that labour pains may not actually be felt, so there have been repeated cases of unperceived parturition. It is consequently recommended that pregnant paraplegic and quadriplegic patients be hospitalised if cervical dilation is diagnosed, at the latest, however, 2—3 weeks antepartum (Robertson, 1972).

Clinical features

The data for the clinical features table were compiled from medical histories from the delivery hospitals and from the Paraplegic Centre, as well as from answers given by the patients themselves to questions by telephone. All 17 deliveries of the 13 patients took place between 1974 and 1984. Of the 13 patients five showed symptoms of sympathetic nervous stimulation (autonomic hyperreflexia) through contractions of the uterus. In our patient data the symptomatology ranged from subjective pressure sensations in the head and headaches to objective features such as pilo-erection, outbreaks of sweating, epiphora simultaneous with labour pains and a massive rise in blood pressure over 200 mm Hg systolic.

Our findings corroborated the evidence in the medical literature that autonomic hyperreflexia can occur both in cases of complete and incomplete lesions. In one case a general anaesthetic had to be given during the labour pains because

of uncontrollable, high blood pressure values. In two other cases, in which no serious autonomic hyperreflexia occurred, it was not necessary to employ general anaesthesia or epidural anaesthesia. One patient was given a general anaesthetic for other reasons, while another patient was treated prophylactically with an epidural anaesthetic. Our patient data reveals only one instance of serious autonomic hyperreflexia. Of the 13 patients, seven had lesions above D 7, and of these 5 showed symptoms of autonomic hyperreflexia, while 2 had no signs whatsoever of symptoms concurrent with labour pains. These two received antepartum general anaesthesia. Our patient data has thus shown that all patients with lesions above D 7 had symptoms of autonomic hyperreflexia during labour.

The data also shows that there was no instance of symptoms concurrent with labour pains where the lesion was below D 9. For most of the patients, labour pains were subjectively and clearly recognised, even if, clinically, the examiner could not easily identify them. Of 13 patients, three were unable to recognise labour pains. One of these three patients had either complete or incomplete lesions of D 8, D 6 and D 4. One patient found it impossible to distinguish the labour pains from abdominal spasms.

The medical literature states that in patients with lesions above D 10, the reliable perception of labour pain is not possible (Ohry *et al.*, 1978). We found that our patients quite clearly perceived labour pains also in cases of complete and incomplete lesions of D 4, D 5, C 8 and C 6. In all patients there was no sacral sparing. Our patients recognised the labour pains by sympathetic induced symptoms concurrent with them. The labour pains were described by the women as (mostly) strong abdominal spasms. In addition, spasms of the legs, difficulty of breathing, pains in the back or convulsive abdominal pains occurred. While it was difficult to describe the difference between labour pains and normal abdominal spasms, the difference was, however, quite clear, although none of the patients had experienced deliveries before.

The postpartum bladder function is, in most cases, unchanged. Only two of the patients have experienced a continuous impairment of the bladder function after delivery: in one case, the residual urine amount was increased, in the other, delivery caused 'loss of the reflex bladder'. This patient has required an indwelling catheter since then. A new development since childbirth in one case is pilo-erection when the bladder fills. Infections of the urinary tract are frequent peripartum complications. In 6 of 17 births urinary tract infections occurred.

The occurrence of anemia as is described in the medical literature was also confirmed by us: in 3 of 17 births, where data was available, anemia was found. Premature labour pains between the 31st and 36th week of pregnancy were experienced in 2 of the 13 patients.

The birth mode was spontaneous in 8 of the 17 cases, in 5 a sectio was carried out, and in four patients an operative termination of parturition had to be undertaken because of secondary uterine inertia. A satisfying outcome was that during pregnancy, delivery and puerperium no new pressure sores developed.

Among the 17 births there were two instances of malformation. In one case there was anal atresia and small-for-date baby, the child died 5 months postpartum. Subsequently the same patient gave birth to two healthy children. The accident that caused the paralysis happened some years prior to the first pregnancy. In another patient where the accident occurred before the beginning

Table Clinical features

Patient No	Birth No	Age	Niveau	Lesion	AHR symptoms	Labour pains clearly recognizable	Urinary tract	Anemia	Premature labour pains, WP	Hosp. date, WP	Delivery time, WP	Mode of birth	Pressure sores	Birth weight	Anaesthesia	Specific features
1	1	24	D4	compl.	yes 9	yes	1,2	+	31	31	36	spontan.	∅	2 930	none	3 transfusions
2	2	26	D9	compl.	∅	yes 8	3,4	?	∅	42	42	spontan.	∅	3 070	none	
	3	29	D9	compl.	∅	yes 8	3,4	?	∅	41	41	spontan.	∅	3 660	none	
	4	32	D9	compl.	∅	yes 8	3,4,6	?	∅	42	42	spontan.	∅	norm.	none	
3	5	27	L1	incompl.	∅	yes	3,4	?	∅	39	40	spontan.	∅	3 050	none	UTI puerperium
	6	32	L1	incompl.	∅	yes	3,4	?	∅	39	39	spontan.	∅	2 930	none	
4	7	24	D5	compl.	yes 10	yes	1,4,5	+	36	37	39	forceps	∅	2 650	EDA prophylact.	Decubitus foot (occurred previously)
5	8	20	D12	incompl.	∅	yes	3,4,6	+	36	34	37	forceps	∅	2 300	gen. anaesthesia	Accident in 12th WP
6	9	23	D8	compl.	∅	no	3,4,5	∅	∅	29	36	spontan.	∅	2 340	none	Chordotomy years ago
7	10	22	D12	incompl.	∅	yes	3,4,5	∅	∅	38	42	forceps	∅	2 620	EDA prophylact.	Anal atresia, small-for-date-baby
8	11	24	C6	incompl.	∅	yes	1,4	∅	∅	39	40	1° Sectio	∅	2 720	gen. anaesthesia	V. septum defect, thrombosis
	12	30	C6	incompl.	∅	yes	1,4	∅	∅	38	40	1° Sectio	∅	2 720	gen. anaesthesia	prophylactically
9	13	28	D4	incompl.	∅	no	1,4	?	∅	36	41	2° Sectio	∅	3 610	gen. anaesthesia	prophylactically
10	14	23	D12	compl.	∅	yes	1	?	36	36	37	spontan.	∅	2 910	none	
11	15	25	D6	compl.	yes 9,11,13	no	3,4,6	?	∅	37	40	Sectio	∅	2 700	gen. anaesthesia	Sectio because of AHR
12	16	24	C8	incompl.	yes 9,12	yes	3,4	?	34	34	40	forceps	∅	3 260	none	
13	17	27	D6	incompl.	yes 9,12 *	yes	3,7	∅	∅	33	34	Sectio	∅	1 750	gen. anaesthesia +	Sectio because of neurol. deterioration

1, Indwelling catheter during birth; 2, Bladder function worse postpartum (permanent); 3, No indwelling catheter during birth; 4, Bladder function postpartum idem; 5, Relapsing Urinary tract infection (UTI); 6, Chronic Urinary tract infection (UTI); 7, Large amount residual urine; 8, At times, hard to differentiate abdominal spasms; 9, Headaches, pressure felt in head; 10, Pilo-erection, shivering fits before delivery; 11, Epiphora; 12, Sweating attacks * before delivery; 13, Massive rise in blood pressure; UTI, Urinary tract infection; WP, Week of pregnancy; AHR, Autonomic hyperreflexia; EDA, Epidural anaesthesia.

of pregnancy, a child was born with a septum defect. The birth defect was operatively cured, and the child is now 12 years old and is healthy.

Discussion

Experience shows that symptoms of autonomic hyperreflexia or autonomic dysreflexia are extremely frequent in paraplegic patients (Frankel *et al.*, 1979). We were able to confirm the great variability of symptomatology as has been described in the medical literature. It is possible that a distinction can be drawn between the relatively harmless intensified sympathetic activity, and the more threatening autonomic hyperreflexia, and thus would be of positive value. Under the first concept we would include the less pronounced symptoms of sympathetic activity; under the second, the state created by actual autonomic hyperreflexia, requiring special medical therapy. In our patient data, in one case serious autonomic hyperreflexia could be controlled by general anaesthesia, and by the preventive employment of general anaesthesia or epidural anaesthesia it was possible in four cases to stop the development of serious symptoms.

The perception of labour pains is not merely confined to transmission via the afferents in the spinal cord. Further afferents are postulated, for example, via the vagus, as in one case of complete lesion of C 6 the labour pains were also clearly felt.

The danger of pressure sores in paralysed patients was plainly recognized, and by taking appropriate measure it was possible to prevent the formation of new pressure sores.

Although infections of the urinary tract can be a frequent complication, we ascertained that there was no case of urosepsis.

As far as the deformity rate is concerned, it is not possible, with the small number we were able to examine, to make an unequivocal statement.

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