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# ANAESTHESIA OF THE SPINAL CORD-INJURED PATIENT: CARDIOVASCULAR PROBLEMS AND THEIR MANAGEMENT[1]

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SPINAL CORD-INJURED PATIENTS deserve particular attention when undergoing anaesthesia. Altered cardiovascular function in those patients includes sympathetic hypofunction in the acute stage and autonomic hyperreflexia in the chronic stage (Desmond, 1970). During the life of the spinal cord-injured patient, haemodynamic responses to volume changes, the administration of drugs, and the stress of an operative procedure, can cause clinical management problems. These abnormal responses are often complicated by initial overtransfusion in the patient with an acute spinal cord injury or low blood volume in chronic patients. Frequently, some degree of respiratory dysfunction complicates the clinical status in both acute and chronic patients. Any anaesthetic superimposes major stresses on the cardiovascular system, already compromised by the spinal cord injury in the absence of anaesthesia. Attempts to provide better anaesthesia for these patients must rely upon improved methods of monitoring cardiovascular function.

The purpose of this paper is to demonstrate how information obtained from recently available methods of monitoring may be applied during the management of cardiovascular problems occurring in the spinal cord-injured patient, intra- and post-operatively.

## PATIENTS AND METHODS

Since cardiovascular problems occur most frequently in patients with a cervical or a high thoracic (T1-T6) lesion, at Yale we employ the following monitoring system during the anaesthesia of these patients (fig. 1).

As in the anaesthesia of the non-spinal cord-injured patient, an airway is established, the electrocardiogram is recorded continuously and routine measurement of rectal temperature and urine output is done. In the spinal cord-injured patient an indwelling percutaneous arterial catheter is used to provide continuous recording of systemic systolic, diastolic and mean perfusion pressures. It is also used to obtain blood gases, thereby, avoiding repeated trauma to peripheral arteries.

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# MONITORING OF THE SPINAL CORD-INJURED PATIENT DURING ANESTHESIA

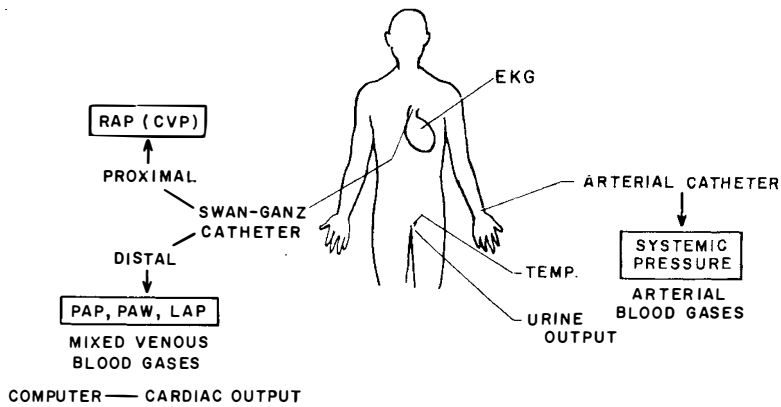


FIG. 1

Parameters monitored during the anesthesia of the spinal cord-injured patient.

The flow-directed balloon-tipped catheter described by Swan, Ganz, *et al.* (1970) is placed in the pulmonary artery via percutaneous insertion into the brachial vein. The thermistor, located in the distal end of the catheter, permits computerised cardiac output measurement by the thermodilution method (Loughman, 1973). The Swan-Ganz catheter[1] is guided into pulmonary wedge position with the balloon inflated, using constant pressure monitoring via a transducer. In this position the pulmonary artery wedge pressure (P.A.W.) can be obtained repeatedly simply by inflation of the balloon. With the balloon deflated, pulmonary artery pressures (P.A.P.) (systolic, diastolic and mean) are measured. The central venous pressure (C.V.P.) can be obtained from the catheter by recording from the proximal lumen which is located at the junction of the superior vena cava and the right atrium. Correct positioning of the catheter may be confirmed by a portable chest X-ray.

Once the catheter is positioned in the pulmonary artery, a sample of blood may be obtained for the determination of mixed venous oxygen tension ($p\bar{v}O_2$). By comparing $p\bar{v}O_2$ to the arterial oxygen tension ($paO_2$), both the amount of oxygen extracted at the tissue level and the amount of intrapulmonary shunting can be calculated accurately.

## ILLUSTRATIVE CASES AND DISCUSSION

Circulatory instability and susceptibility to pulmonary oedema represent major hazards to the spinal cord-injured patient during anaesthesia. An unstable cardiovascular system may be expected if the level of the lesion is at T6 or above thereby causing sympathetic denervation, the detailed physiology of which has

[1] Edwards Laboratories, Inc., Santa Ana, California.

TABLE I

Comparison of cardiovascular responses in patients with and without spinal cord injury: volume alterations

| | Hypovolaemia | | Normovolaemia | | Hypervolaemia | |
|---|---|---|---|---|---|---|
| | Non-spinal cord-injured patient | Spinal cord-injured patient | Non-spinal cord-injured patient | Spinal cord-injured patient | Non-spinal cord-injured patient | Spinal cord-injured patient |
| C.V.P. | ↓, → or ↑* | ↓ | → | → or ↓ | ↑ | → or ↑ |
| P.A.W. | ↓ | ↓ | → | → | ↑ | ↑ |
| P.A.P. | ↓ | ↓ | → | → | ↑ | ↑ |
| C.O. | → or ↓ | → or ↓ | → | → or ↓ | → or ↓ | → |

* ↑ if there is increased pulmonary vascular resistance.

been reviewed by Greene (1962). Haemodynamic alterations in acute quadriplegic patients consist of bradycardia, arterial hypotension and an increased venous capacitance (Meyer *et al.*, 1971). It can be hazardous to subject such patients to an anaesthetic with myocardial depressant properties. A decreased venous return, caused by controlled ventilation during anaesthesia, also reduces cardiac output (C.O.) in spinal cord-injured patients because they lack compensatory cardiovascular reflexes (Quimby *et al.*, 1973).

Intravascular volume replacement is commonly used to treat hypotension in patients with spinal cord injury. Blood pressure (B.P.) and C.V.P. have been the conventional guides used to determine the amount of transfusion necessary (McSweeney, 1968); however, the value of C.V.P. measurement, particularly in the spinal cord-injured patient, has been questioned recently (Meyer *et al.*, 1971). The vascular space is expanded in the acute quadriplegic patient allowing infusion of large fluid volumes without the expected rise of C.V.P. (Meyer *et al.*, 1971) (Table I).

Pulmonary oedema occurred in 44 per cent of the acute quadriplegic patients reported by Meyer *et al.* (1971). The C.V.P. showed an apparent increase in patients with pulmonary oedema relative to those without pulmonary oedema, but the difference was not statistically significant.

A high incidence of pulmonary oedema in spinal cord-injured patients (Cheshire, 1964; Wolman, 1965), despite careful clinical observation and C.V.P. measurement, emphasises the need for better monitoring techniques. By measuring P.A.P. directly, a high pulmonary diastolic pressure may be detected early and treated before pulmonary oedema develops. Elevated P.A.W. may be an early manifestation of left ventricular failure and immediate therapeutic intervention should prevent the occurrence of pulmonary oedema. Usually large changes in left ventricular filling pressure and/or P.A.P. may occur before these alterations are reflected in C.V.P. (Civetta and Gabel, 1972). This delayed response may be further prolonged in spinal cord-injured patients because of sympathetic hypofunction and increased venous capacitance (Table II).

TABLE II

Comparison of cardiovascular responses in patients with and without spinal cord injury: alterations of myocardial function

| | Left heart failure* | | Right heart failure | | Left and right heart failure | |
|---|---|---|---|---|---|---|
| | Non-spinal cord-injured patient | Spinal cord-injured patient | Non-spinal cord-injured patient | Spinal cord-injured patient | Non-spinal cord-injured patient | Spinal cord-injured patient |
| C.V.P. | → | → or ↓ | ↑ | ↓ , → or ↑ | ↑ | ↓, → or ↑ |
| P.A.W. | ↑ | ↑ | → | → | ↑ | ↑ |
| P.A.P. | ↑ | ↑ | → | → | ↑ | ↑ |
| C.O. | ↓ | ↓ | ↓ | ↑ | ↓ | ↓ |

* Early; before involvement of right heart.

If pulmonary oedema is associated with normal P.A.P. and P.A.W., then abnormal pulmonary capillary permeability might well be responsible for the oedema. In such non-cardiogenic pulmonary oedema, P.A.P. and P.A.W. monitoring may be helpful in the differential diagnosis. Guttman (1973) states that, following transection of the cervical spinal cord, impaired renal function resulting in oliguria is seen. Associated changes in hydrostatic and colloid osmotic pressures lead to increased transudation of fluid from the intravascular space to the interstitial space and, therefore, are responsible for the oedema formation.

As diuretics and/or positive end expiratory pressure (P.E.E.P.) ventilation are commonly used in the treatment of patients with pulmonary oedema, the Swan-Ganz catheter is particularly useful, since C.O. and $p\bar{v}O_2$ can be determined.

Available data specific to spinal cord-injured patients are limited and further investigation is needed. The following cases illustrate how the simultaneous monitoring of haemodynamic parameters facilitates the management of cardiovascular problems.

## CASE REPORTS

**Case I** (fig. 2). A 16-year-old male sustained an acute anterior subluxation of the cervical spine at the C4-5 level. He presented for cervical decompressive laminectomy and posterior cervical fusion.

The preoperative course was complicated by atelectasis of the entire upper lobe of the right lung. Resulting respiratory failure required controlled mechanical ventilation with P.E.E.P. to treat the intrapulmonary shunt which amounted to 13 per cent of C.O.

Intraoperative vital signs remained stable during the first four hours of halothane (0·5 to 1·0 per cent) anaesthesia. An increasing intrapulmonary shunt, with a concomitant decrease in the $paO_2$, required elevation of P.E.E.P. from 10 to 15 cm. $H_2O$. Within minutes B.P. fell from 120/70 to 80/60 mm. Hg, despite an apparently adequate blood volume as evidenced by P.A.P. of 26/15 mm. Hg and P.A.W. of 10 mm. Hg. A fall in C.O. from 7 l./min. to 4 l./min. was associated with a rise in P.A.P. to 35/28 mm. Hg

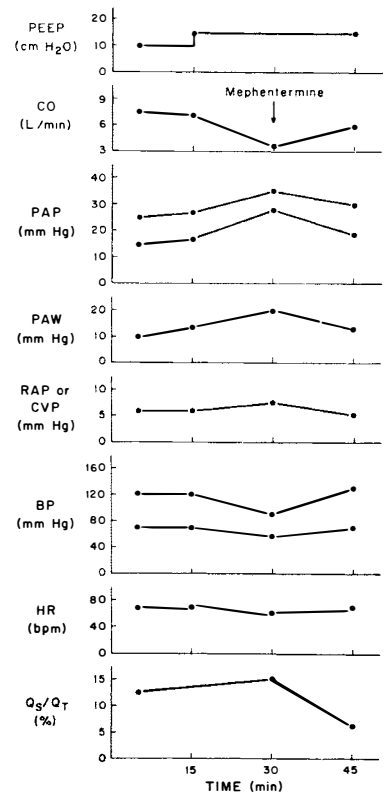


FIG. 2

Case I: Changes in cardio-respiratory function and the response to mephentermine.

and a corresponding rise in P.A.W. to 20 mm. Hg. The C.V.P. only rose from 5 to 7 mm. Hg.

Administration of mephentermine[1] improved C.O. and systemic B.P. The increased C.O. was associated with a return of P.A.P. and P.A.W. to normal levels. The operation was completed uneventfully.

*Comment.* Patients with sympathetic denervation secondary to acute spinal cord trauma may have a limited cardiac reserve (fig. 3). First, an increased cardiac output from a rise in heart rate (H.R.) may fail to occur. Secondly, myocardial contractility is reduced because of loss of the normal sympathetic tone (Guyton, Jones & Coleman, 1973). This limits the ability to increase stroke volume. Impaired myocardial contractility in patients with sympathectomy can be restored to some extent with a positive inotropic agent. A larger stroke volume also can be achieved by increasing the venous return. Quadriplegic patients may be deprived of this mechanism as the venous blood tends to pool peripherally.

The cardiovascular measurements obtained in Case I illustrate some of above-

[1] Wyamine, Wyeth Laboratories, Philadelphia, Pennsylvania.

mentioned alterations of function. When P.E.E.P. is raised, C.O. usually falls but is partially restored by an increase in H.R. This compensatory tachycardia failed to occur in this patient.

The increase in P.A.P. and P.A.W. seen in Case I can also occur in the non-spinal cord-injured patient during the use of P.E.E.P. (Qvist *et al.*, 1975). An increased P.A.P., reflecting increased pulmonary vascular resistance, and an increase in P.A.W., reflecting left ventricular filling pressure, must be interpreted with caution when a change in airway pressure occurs. It has been shown that during mechanical ventilation with P.E.E.P., ventricular filling pressure rises relative to atmospheric pressure (Qvist *et al.*, 1975). In the same study by referring the measured pressures to intrapleural pressure instead, a fall in the ventricular filling pressure was noted (Qvist *et al.*, 1975). Thus, the apparent increased filling pressure, as usually interpreted from a rise in P.A.W. in the face of increased P.E.E.P., may actually be a falling filling pressure relative to intrapleural pressure. This will result in a fall of C.O. (fig. 4).

In the quadriplegic patient, a decreased ventricular filling pressure is quite detrimental because myocardial contractility often is impaired. If P.E.E.P. has to be maintained to treat hypoxia from intrapulmonary shunting, an effective way to improve C.O. is to increase myocardial contractility using a positive inotropic agent. This treatment proved helpful in Case I.

The differential diagnosis of acute left ventricular failure must be considered also. A rise in P.A.W. with an associated fall in C.O. may represent decreased myocardial contractility on the downslope of the ventricular function curve (fig. 3). Treatment is similar in that a positive inotropic agent is required to restore C.O.

Low C.O., from a failing ventricle with high filling pressure or from decreased filling pressure secondary to an elevated airway pressure, can be differentiated only with reference to intrapleural pressure. Thus far, this measurement is not clinically available except for esophageal pressure monitors.

The Swan-Ganz catheter serves as a sensitive monitoring device to differentiate hypovolaemia from decreased myocardial contractility, both of which result in a low C.O., a particularly serious condition in the spinal cord-injured patient.

**Case II** (fig. 5). This 14-year-old male patient was admitted with acute traumatic paraplegia secondary to a compression fracture at the T4-5 level. A thoracic decompressive laminectomy, using halothane anesthesia, was tolerated well.

The postoperative course was complicated by the development of massive left lower lobe atelectasis. The ensuing respiratory failure required controlled ventilation, P.E.E.P. and 100 per cent oxygen ($F_IO_2$ 1·0) to maintain the $paO_2$ above 70 mm. Hg. On the sixth post-operative day, he became clammy, cold and cyanotic with B.P. of 80/60, H.R. 140, C.V.P. 3 mm. Hg and an $paO_2$ of 66 mm. Hg with $F_IO_2$ 1·0. Hypovolaemia was suspected and a Swan-Ganz catheter was re-inserted during resuscitation. The findings of a low P.A.P., low P.A.W. and low C.O. confirmed the diagnosis and an infusion of albumin was started. P.A.P. and P.A.W. were used as guidelines and the administration of colloid was continued until reaching the upper normal limits of P.A.P. and P.A.W., 20/14 and 13 mm. Hg respectively. B.P., H.R. and C.O. returned to normal with a marked improvement of $paO_2$. Throughout the time of colloid infusion, the C.V.P. showed a considerable lag when compared to P.A.P. and P.A.W.

*Comment.* Such a wide discrepancy between C.V.P. and P.A.W. usually is not seen in non-spinal cord-injured patients unless they are in congestive heart failure.

Since the respiratory status was already compromised in the patient in Case II, it was imperative to avoid overtransfusion. Overtransfusion may have occurred if the C.V.P. was used to determine the amount of fluid replacement, because there is a delayed rise

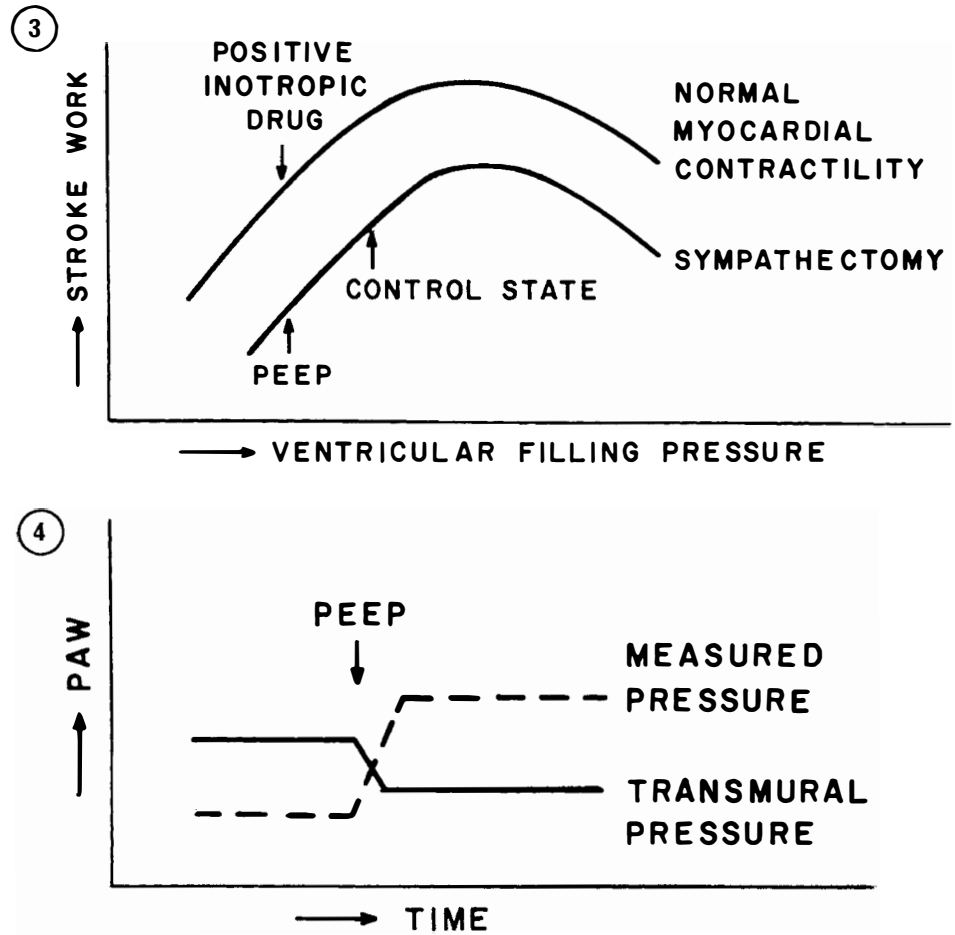


FIG. 3

In the sympathectomised patient the myocardial contractility is decreased relative to that of the non-sympathectomised patient (arrow-control state). The addition of P.E.E.P. will result in a lower stroke work due to decreased ventricular filling pressure (arrow-P.E.E.P.); however, at this point the administration of a positive inotropic drug to the sympathectomised patient on P.E.E.P. will restore myocardial contractility to a level approximating normal for the corresponding ventricular filling pressure (arrow-positive inotropic drug).

FIG. 4

P.A.W. measured during the use of P.E.E.P. rises relative to atmospheric pressure but falls relative to transmural pressure. (Transmural pressure is the measured pressure relative to atmospheric minus pleural pressure relative to atmospheric.)

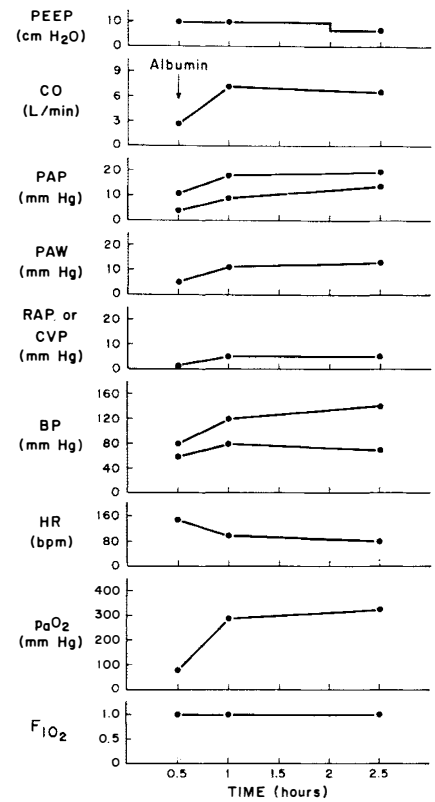


FIG. 5

Case II: Effect of albumin on cardio-respiratory function.

of the C.V.P. to expected levels when a spinal cord-injured patient is returned to a normovolaemic state (Table I).

## SUMMARY

An increased venous capacity and a decreased myocardial contractility can be expected in patients with an acute spinal cord lesion at or above T6. Both factors may contribute to a high incidence of arterial hypotension and pulmonary oedema in these patients especially during anaesthesia.

We feel that the Swan-Ganz catheter provides valuable information concerning prevention, diagnosis and treatment of arterial hypotension and pulmonary oedema. Although there may be occasional difficulty in interpretation of measurements from the Swan-Ganz catheter if high airway pressures are used, it is a more sensitive monitor than C.V.P. measurement and is particularly useful in patients with a sympathectomy secondary to spinal cord trauma.

## RÉSUME

On peut s'attendre à trouver une capacité veineuse accrue et une diminution de la contractilité du myocarde chez les malades atteints de lésion aigue de la moelle épinière au niveau T6 ou au-dessus. Ces deux facteurs peuvent contribuer a une incidence élevée de l'hypotension artérielle et de l'oedème pulmonaire chez ces malades, en particulier pendant l'anesthésie.

Nous pensons que la sonde Swan-Ganz fournit des renseignements précieux pour ce qui est de la prévention, du diagnostic et de traitement de l'hypotension artérielle et de l'oedème pulmonaire. Bien qu'il puisse y avoir des difficultés occasionnelles dans l'interprétation des mesures à partir de la sonde Swan-Ganz si l'on utilise des pressions élevées de la voie d'aérage, c'est un moyen de contrôle plus sensible que la mesure au CVP et il est particulièrement utile pour les malades ayant subi une sympathectomie suite à une lésion de la moelle épinière.

## ZUSAMMENFASSUNG

Eine vermehrte venöse Kapazität und eine verminderte myokardiale Kontraktibilität kann in Patienten mit akuten Rückenmarksverletzungen in Höhe oder oberhalb von T6 erwarted werden. Beide Faktoren können häufig zu Hypotension und Lungenoedem beitragen.

Der Swan-Ganz Katheter erlaubt gute Information in der Verhütung, Diagnose und Behandlung von arterieller Hypotension und Lungenoedem. Er ist ein weit sensitiver Monitor als CVP Messungen und ist besonders wertvoll in Patienten, die eine Sympathektomie sekundär zum spinalen Trauma haben.

## ABBREVIATIONS

| | |
|---|---|
| B.P. | Systemic blood pressure. |
| C.O. | Cardiac output. |
| C.V.P. | Central venous pressure. |
| $F_IO_2$ | Fraction of inspired oxygen. |
| H.R. | Heart rate. |
| L.A.P. | Left atrial pressure. |
| P.A.P. | Pulmonary artery pressure. |
| P.A.W. | Pulmonary artery wedge pressure. |
| P.E.E.P. | Positive end expiratory pressure. |
| $paO_2$ | Arterial oxygen tension. |
| $p\bar{v}O_2$ | Mixed venous oxygen tension. |
| $Q_s/Q_t$ | Intrapulmonary shunt. |
| R.A.P. | Right atrial pressure. |

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