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[Intervention Review]

Cerebrospinal fluid drainage for thoracic and thoracoabdominal aortic aneurysm surgery

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ABSTRACT

Background

During aortic aneurysm surgery, cross-clamping can lead to inadequate blood supply to the spinal cord resulting in neurological deficit. Cerebrospinal fluid drainage (CSFD) may increase the perfusion pressure to the spinal cord and hence reduce the risk of ischaemic spinal cord injury.

Objectives

To determine the effect of CSFD during thoracic and thoracoabdominal aortic aneurysm (TAAA) surgery on the risk of developing spinal cord injury.

Search methods

For this update the Cochrane Peripheral Vascular Diseases Group Trials Search Co-ordinator searched the Specialised Register (last searched May 31 2012) and CENTRAL (2012, Issue 5) for publications describing randomised controlled trials of cerebrospinal fluid drainage for thoracic and thoracoabdominal aortic aneurysm surgery. Reference lists of relevant articles were checked.

Selection criteria

Randomised trials involving CSFD during thoracic and TAAA surgery.

Data collection and analysis

Both authors assessed the quality of trials independently. SNK extracted data and GS verified the data.

Main results

Three trials with a total of 287 participants operated on for Type I or II TAAA were included.

In the first trial of 98 participants, neurological deficits in the lower extremities occurred in 14 (30%) of CSFD group and 17 (33%) controls. The deficit was observed within 24 hours of the operation in 21 (68%), and from three to 22 days in 10 (32%) participants. CSFD did not have a significant benefit in preventing ischaemic injury to the spinal cord.

The second trial of 33 participants used a combination of CSFD and intrathecal papaverine. It showed a statistically significant reduction in the rate of postoperative neurological deficit ($P = 0.039$), compared to controls. Analysis was undertaken after only one third of the estimated sample size had entered the trial.

In the third trial TAAA repair was performed on 145 participants. CSFD was initiated during the operation and continued for 48 hours after surgery. Paraplegia or paraparesis occurred in 9 of 74 participants (12.2%) in the control group versus 2 of 82 participants (2.7%) receiving CSFD ($P = 0.03$). Overall, CSFD resulted in an 80% reduction in the relative risk of postoperative deficits. Meta-analysis showed an odds ratio (OR) of 0.48 (95 % confidence interval (CI) 0.25 to 0.92). For CSFD-only trials, OR was 0.57 (95% CI 0.28 to 1.17) and for intention-to-treat analysis in CSFD-only studies, the OR remained unchanged.

Authors' conclusions

There are limited data supporting the role of CSFD in thoracic and thoracoabdominal aneurysm surgery for prevention of neurological injury. Further clinical and experimental studies are indicated.

PLAIN LANGUAGE SUMMARY

Cerebrospinal fluid drainage for thoracic and thoracic abdominal aortic aneurysm surgery

An aneurysm is a local bulging of a blood vessel that carries a risk of rupture. Surgery for an aortic aneurysm requires clamping the aorta, the biggest artery in the body. This reduces the supply of blood and oxygen to the spinal cord (ischaemia) and tissue damage can lead to the partial or incomplete paralysis of the lower limbs (paresis) and paraplegia (paralysis of the legs and lower part of the body). These deficits are frequently irreversible. The cerebrospinal fluid (CSF) pressure increases during clamping further decreasing the perfusion pressure of the spinal cord. As more of the blood supply to the spinal cord is interrupted, the likelihood of paraplegia is increased. Various treatments are used to reduce the ischaemic insult to the spinal cord including temporary blood shunts (such as distal atriolfemoral bypass and re-connection of intercostal and lumbar vessels), pharmaceutical interventions (to protect the heart and cerebral blood vessels), epidural cooling and CSF drainage. Draining CSF from the lumbar region may lessen the CSF pressure, improve blood flow to the spinal cord and reduce the risk of ischaemic spinal cord injury.

The available evidence does not fully establish CSF drainage as a method of protection. The review authors made a thorough search of the medical literature and identified three randomised trials involving a total of 287 participants operated on for high-risk thoracoabdominal aortic aneurysms. All of the studies used CSF drainage in addition to other measures of spinal cord protection. In the first trial of 98 patients, neurological deficits in the lower extremities occurred in about one third of patients with or without drainage. The deficit was observed within 24 hours of the operation in 21 (68%), and from three to 22 days in 10 (32%). The second trial of 33 patients reported that a combination of CSF drainage and papaverine in the region of the spinal cord (intrathecally) reduced the rate of postoperative neurological deficit compared to controls. In the third trial involving 145 patients, drainage was begun during the operation and continued for 48 hours after surgery. Paraplegia or paraparesis occurred less with CSF drainage (2.7% of patients with drainage versus 12.2% in the control group).

BACKGROUND

The spinal cord like the brain is exquisitely sensitive to ischaemia (inadequate blood flow leading to reduced oxygen in the tissues). In fact ischaemia of less than ten minutes can lead to infarction (tissue death due to a local lack of oxygen) of the spinal cord. In aortic aneurysmal surgery, aortic cross-clamping leads to decreased blood supply to the spinal cord resulting in dysfunction, a condition that is frequently irreversible.

The blood supply of the distal spinal cord is mainly via the anterior and posterior radicular branches of the intercostal and the lumbar arteries. These connect to form single anterior and double posterior spinal arteries which run longitudinally along the cord (Bolton 1939; Gillian 1958). These also form connections with the spinal branches of vertebral, deep cervical and lateral sacral arteries arising from the internal iliac arteries. Frequently, one of these is a dominant vessel; the artery of Adamkiewicz, or *arteria radicularis magna* (Gray 1973). This artery tends to arise between the ninth thoracic and the second lumbar vertebrae in the majority (85%) of cases (Grace 1997). This is considered to be the major blood supply that gives the critical branches to the distal two thirds of the cord (Gray 1973), interruption of which can lead to paraplegia.

The incidence of paraplegia following infrarenal aortic aneurysm is in the order of 1% (Joseph 1989). However, the incidence tends to increase with ruptured aneurysms, in acute dissection of the aorta and in thoracic or thoracoabdominal aneurysm surgery (Rosen 1988; Zull 1988).

Spinal cord ischaemia following aortic ligation was first noted experimentally by Stenonis in 1667. However, spinal ischaemia in relation to aortic aneurysmal surgery was not reported until 1962 by Mehrez (Mehrez 1962). As more of the blood supply to the cord is interrupted during thoracic aneurysm surgery, the incidence of cord ischaemia rises. Crawford (Crawford 1986) was the first to report a large case series of thoracic aortic aneurysm (TAA) repair with an overall paraplegia rate of 9%. A later, larger series showed a paraplegia rate of 5% to 40%. It was during this period that Cox (Cox 1992) reported the incidence of paraplegia as 38% with Types I and II, and 12% with Types III and IV aortic aneurysms (Crawford classification) (Crawford 1986). See additional tables for classification of aortic aneurysm (Table 1).

Postoperative paraplegia remains the most devastating complication of surgery of the descending and thoracoabdominal aorta. Control of proximal hypertension that follows cross-clamping of the thoracic aorta to repair aneurysms of the descending and thoracoabdominal aorta is necessary to prevent left ventricular failure, myocardial infarction, and haemorrhagic cerebral events. Both pharmacological and mechanical modalities used to control central hypertension during aortic occlusion affect cerebrospinal fluid (CSF) dynamics and spinal cord perfusion pressure (SCPP).

In order to reduce the incidence of such a dreadful complication, different groups have looked into various treatments to reduce the ischaemic insult to the spinal cord. In this regard temporary shunts, pharmaceutical interventions either parenterally or intrathecally (Agee 1991; Laschinger 1984; Qayumi 1994; Suzuki 1994; Svensson 1990), epidural cooling (Marsala 1993; Vanicky 1993), cerebrospinal fluid drainage (CSFD) and a variety of intraoperative manoeuvres

(de Mol 1990; North 1991; Okamoto 1992; Schepens 1995; Shiiya 1995), have been employed.

Spinal cord perfusion pressure (SCPP) is the difference between blood pressure and CSF pressure. Thus theoretically, decreasing the cerebrospinal pressure or increasing the blood pressure will improve the SCPP (Blaisdell 1962; Kazama 1994). Miyamoto 1960 and McCullough 1988 reported a decreased incidence of paraplegia in canine models on drainage of CSF following cross-clamping of aorta. In one study (Grubbs 1988), CSFD significantly increased SCPP from 9.4 to 21.8 mmHg. Similar observations by other researchers lead to the introduction of CSFD in routine practice of thoracic aortic aneurysm surgery aiming to keep the CSF pressure at less than 15 mmHg intraoperatively, and for several days thereafter (McCullough 1988). It has been observed that simple drainage of CSF is not enough but that the lowering of CSF pressure is necessary to improve the SCPP (Crawford 1991). Beneficial effects have been reported by other authors (Murray 1993; Safi 1994).

OBJECTIVES

To assess the effects of cerebrospinal fluid drainage (CSFD) on lowering the incidence of spinal cord dysfunction following aneurysm surgery.

METHODS

Criteria for considering studies for this review

Types of studies

We considered randomised trials in which cerebrospinal fluid drainage (CSFD) in patients undergoing thoracic aneurysm surgery was randomly put to the test. Only open surgery trials were included as no randomised trial for endovascular stenting of TAA and CSFD effect on paraplegia has been identified. Non-randomised observational cohort studies and non-randomised with historical controls were also identified but were excluded from analysis for reasons described under excluded studies.

Types of participants

Males and females of any age with a diagnosis of Type I or II TAA made by an expert clinician on clinical and investigative assessment (computed tomography (CT) scan, magnetic resonance (MRI) scan, angiogram). The clinical presentation and the size of the aneurysm had to indicate to the surgeon that reconstructive surgery was justified.

Types of interventions

Drainage of cerebrospinal fluid (CSF) via the lumbar route to decrease the CSF pressure in patients undergoing Type I or II TAA repair. Other proposed spinal cord protection manoeuvres such as distal atriocaval bypass and re-anastomosis (re-connection) of intercostal and lumbar vessels were undertaken in both the controls and the cases.

Types of outcome measures

The outcome measure was postoperative neurological deficit in terms of paresis (slight or incomplete paralysis), and paraplegia (paralysis of the legs and lower part of the body) affecting the lower limbs.

Search methods for identification of studies

For this update the Cochrane Peripheral Vascular Diseases Group Trials Search Co-ordinator (TSC) searched the Specialised Register (last searched May 31 2012) and the Cochrane Central Register of Controlled Trials (CENTRAL) 2012, Issue 5, part of *The Cochrane Library*, www.thecochranelibrary.com. See [Appendix 1](#) for details of the search strategy used to search CENTRAL. The Specialised Register is maintained by the TSC and is constructed from weekly electronic searches of MEDLINE, EMBASE, CINAHL, AMED, and through handsearching relevant journals. The full list of the databases, journals and conference proceedings which have been searched, as well as the search strategies used are described in the [Specialised Register](#) section of the Cochrane Peripheral Vascular Diseases Group module in *The Cochrane Library* (www.thecochranelibrary.com).

Data collection and analysis

Selection of trials

Both authors (SNK and GS), carried out the selection of trials for inclusion in the review. The criteria for selection of trials was as specified in the above section 'Criteria for considering studies for this review'.

Quality of trials

SNK and GS independently assessed the methodological quality of the trials using the checklist recommended by the Peripheral Vascular Diseases Review Group with particular consideration given to concealment of randomisation. Each reviewer gave the trial an allocation score of A (clearly concealed), B (unclear if concealed), C (clearly not concealed) and also a summary score of A (low risk of bias), B (moderate risk of bias) and C (high risk of bias). Any discrepancies between authors in the above scores were discussed and consensus reached. Trials scoring A for concealment or bias were included, C excluded, and B discussed in more detail.

Data extraction

The data collected on each trial included information on the participants (age and sex distribution, available only in Massachusetts study ([Svensson 1998](#)), classification of aneurysm); the interventions (type of TAA repair, CSF pressure monitoring, control intervention, lower limb neurological deficit, usual care in both groups); and the outcomes as specified in criteria for considering trials for a review. One reviewer (SNK) collected the data which were verified by the second reviewer (GS).

Statistical analysis

The trials were assessed and measures used for statistical pooling as per the guidelines published by the PVD Group. The data synthesis comprised a comparison of group results, where feasible.

RESULTS

Description of studies

See [Characteristics of included studies](#) and [Characteristics of excluded studies](#). Ten trials were identified, seven of which were excluded ([Acher 1990](#); [Acher 1994](#); [Acher 1998](#); [Hollier 1992](#); [Murray 1993](#); [Safi 1998](#); [Svensson 1988](#)).

Three randomised controlled trials, one conducted in Massachusetts ([Svensson 1998](#)), and the other two in Houston Texas, USA ([Coselli 2002](#); [Crawford 1991](#)) were included. None of the studies used CSFD alone as the protection against spinal cord damage; other adjunct measures of spinal cord protection were employed as well.

In the first Houston trial ([Crawford 1991](#)), 100 participants gave written fully-informed consent for operation with CSFD. Thirty-three of these participants had aortic dissection, and 67 had non-dissection disease. The follow-up period was reported for up to six months. Later, two people withdrew consent for CSFD, a third person had fever and a urinary tract infection, and an intrathecal catheter could not be inserted in a fourth person. Operations were performed in these four people without CSFD, and thus they were included in the control group. Surgery was aborted in one person because of acute pulmonary oedema during induction of anaesthesia. Forty-seven participants received CSFD, and 52 acted as controls. One person who received CSFD died of myocardial infarction at the end of operation, and thus 46 participants who received CSFD and 52 control participants survived long enough to evaluate neurological complications. Nineteen (41%) in the CSFD group and 20 (38%) of the controls underwent aortofemoral bypass while 38 (83%) who received CSFD and 40 (77%) controls underwent intercostal and/or lumbar arteries re-attachment.

In the Massachusetts trial ([Svensson 1998](#)), 33 participants met the inclusion criteria. There were 25 men and eight women with a median age of 66 years (range, 34 to 79 years). The number was limited as the interim results showed a significant difference in outcomes between groups and the institutional review board decided to stop the study early. All participants underwent aortofemoral bypass with active cooling before cross-clamping of the aorta. Seventeen of the participants received CSFD along with 3 ml of warmed (37°C) preservative-free papaverine solution (30 mg). Intercostal and lumbar arteries below T6 were preserved, where possible. Cerebrospinal fluid was allowed to drain freely during aortic cross-clamping and stopped after unclamping. Intrathecal papaverine was instilled before cross-clamping. Postoperatively, CSF drained freely if the pressure was more than 10 cm H₂O. Other possible spinal cord protective measures were used for example, distal aortic perfusion (DAP) using aortofemoral bypass, aortic segments sequentially repaired to maintain proximal and distal perfusion, and segmental artery re-attachment; these were equally distributed between the two groups. However, in the group of participants with good outcome, the use of active cooling using bypass was higher (16 of 24) compared with the group with postoperative neurological injury (2 of 7; $P = 0.02$). Aortic cross-clamp times were also longer (32.3 min $\pm$ 15.1 min versus 50.3 min $\pm$ 19.3 min; $P = 0.008$) in the group with neurological deficits. Neurological outcome evaluation was graded by a blinded neurologist. Overall, neurological deficit rates were 2 of 17 (12%) in the CSFD and papaverine group compared with 7 of 16 (44%) in the control group ($P = 0.039$). The authors concluded that the combination of CSFD and intrathecal papaverine significantly reduced the incidence and severity of neurological injury, and this effect was additive if combined with aortofemoral bypass with hypothermia.

In the second Texas study ([Coselli 2002](#)), 156 participants entered the trial. All participants underwent graft repair with standard techniques and a consistent strategy for spinal cord protection

of left heart bypass, moderate heparinisation, permissive mild hypothermia, and aggressive re-attachment of available critical intercostal and lumbar arteries (T7 to L2). The experimental group underwent CSFD in addition to these adjuncts. Cerebrospinal fluid was allowed to drain freely by gravity whenever CSF pressure exceeded 10 mmHg. In participants without a spinal cord deficit, the drain was removed on the second postoperative day. In the presence of neurological injury however, the catheter was kept in place beyond two days.

In all the studies the operating team was not blinded to the procedure. In the first Texas study of CSFD alone (Crawford 1991), this was initially attempted, but it was impossible to implement successfully. Postoperative observers were unaware of the randomisation in the first two studies. However, the first Texas study (Crawford 1991), only referred those patients on whom some degree of neurological deficit was already noted. In the second Texas study (Coselli 2002), neurological assessment was not done independently.

Risk of bias in included studies

Both authors (SNK and GS) independently assessed the methodological quality of the trials using the checklist recommended by the PVD group. Particular attention was paid to concealment of randomisation.

In the first Houston trial (Crawford 1991), initial randomisation was clearly concealed in 100 participants however four were excluded from the CSFD group and included in the controls due to reasons mentioned earlier. The participants were block randomised prospectively to either the control group or the CSFD group by computer-generated sequence in opaque, sealed envelopes immediately before surgery (score A). The observers documenting the outcome were blinded only in cases initially documented by the operating team to have a neurological deficit.

In the Massachusetts trial (Svensson 1998), randomisation was done by computer-generated assignment and the use of folded cards within serially numbered opaque envelopes (score A). In this trial only 33 participants were recruited, at which point the institutional review board decided to discontinue the study as a result of interim analysis that revealed a significant difference. The observers documenting neurological deficit were blinded in this study.

In the second trial from Houston (Coselli 2002), randomisation was computer-generated and assignments were placed in sequentially numbered opaque sealed envelopes preoperatively without preoperative exclusion criteria (score A). The surgical team was not blinded to group assignment. All randomised participants were followed in accordance with intention-to-treat principles. The observers documenting the outcome were not blinded.

Effects of interventions

In the first Houston trial (Crawford 1991), neurological deficits in the lower extremities of varying intensity and duration occurred in 31 (32%) of the 98 participants; 14 (30%) of those who received CSFD and 17 (33%) controls ($P = 0.80$). The deficit was observed immediately (within 24 hours) of the operation in 21 (68%) and from 3 to 22 days (delayed) in 10 (32%) of those participants affected. The deficit was severe (paraplegia) at onset in 21 participants and

milder (paraparesis) in 10 participants. By the time of death or hospital discharge, nine participants had fully recovered. However, 22 participants (22%) continued to have deficits; 15 had paraplegia, and seven had paraparesis. Of the 90 participants (93%) surviving 90 days, six (7%) were paraplegic and six (7%) had paraparesis, four of whom were able to walk with assistance (cane, walker, brace). No significant difference was found in the incidence or degree of neurological deficits between the CSFD and the control groups, and regardless of CSF pressure levels maintained during operation. The incidence of neurological complications was in the range of historical cases.

The volumes of CSFD, including an estimate of loss during insertion of catheter, varied from 24 to 120 ml (median volume, 52.5 ml). No post operative CSFD was carried out. In only 20 of 46 participants was CSF pressure reduced to less than 10 mmHg.

In the Massachusetts trial (Svensson 1998), neurological injury developed in two of the 17 participants receiving CSFD plus intrathecal papaverine (IP) (11.8%) and seven of the 16 controls (43.8%, $P = 0.0392$). The lowest mean motor score was 3.88 in the CSFD plus IP group and 3.25 in the control group ($P = 0.0340$, Student's t test; $P = 0.17$, Kruskal-Wallis test). Only one person from the 33 members of the control group (3%) had complete paraplegia (score = 0); all the other participants had various grades of paraparesis. Of the two CSFD plus IP participants who had paraparesis, one was transient and the other permanent. Neurological injury developed in all three participants (one CSFD plus IP and two controls) who had more than seven intercostal arteries oversewn because of acute dissection.

The total volume of CSF drained averaged 447.2 ml (range: 12 to 1157 ml) and included 18.2 ml lost at the time of needle and catheter insertion, 101.8 ml during aortic cross-clamping, and 304.3 ml during subsequent drainage in the intensive care unit. The median postoperative length of CSFD was 40 hours (range: 2 to 60 hours). CSF was allowed to drain freely if the CSF pressure exceeded 7 to 10 cm H₂O. Neither of the two participants who died had a neurological injury. On complete follow up after discharge, all except two participants (94%, 29/31) had recovered and were walking without support.

In the second Houston trial (Coselli 2002), neurological deficit occurred in 2 of 82 participants (2.7%) who underwent CSFD versus 9 of 74 participants (12.2%) in the control group ($P = 0.03$). The overall operative mortality rate was 7.1% (11 of 156), which included six deaths within 30 days (range: 5 to 24 days) and five subsequent deaths in hospital (range: 33 to 140 days). All participants survived long enough to undergo neurological assessment. Thirty-day mortality rates for participants in the control and CSFD groups were 2.7% (2/74) and 4.9% (4/82), respectively ($P = 0.68$). In-hospital mortality rates were also similar in both groups: 6.8% (5/74) for participants in the control group and 7.3% (6/82) for participants in the CSFD group ($P = 1.0$). In the CSFD group, 64.1 ± 42.9 ml of CSF (range: 10 to 250 ml) were drained during surgery and 260.9 ± 190.5 ml of CSF (range: 40 to 864 ml) were drained during the postoperative period. In this trial, CSF pressure in all cases was maintained at 10 mmHg or less. See additional tables for results of trials (Table 2).

The 30-day survival rate in the three studies was comparable; 96% (Crawford 1991), 96.9% (Svensson 1998), and 95.9% (Coselli 2002), respectively. Overall, meta-analysis shows an odds ratio (OR) of

0.48 (95% confidence interval (CI) 0.25 to 0.92). For CSFD trials only the OR was 0.57 (95% CI 0.28 to 1.17) and for intention to treat in CSFD-only studies, the OR remained unchanged.

DISCUSSION

Spinal cord ischaemia remains unpredictable and a major cause of morbidity after thoracoabdominal aortic aneurysm (TAAA) repair. Although the aetiology of spinal cord neurological injury is multifactorial, ischaemia remains the principal factor. No single intervention has been able to reduce this potentially disabling condition. The hypothesis of cerebrospinal fluid drainage (CSFD) in order to reduce the CSF pressure and enhance spinal cord perfusion pressure (SCPP) to prevent ischaemia, has not been adequately tested in humans. Clamping of aorta during aortic aneurysm surgery increases CSF pressure and decreases distal aortic systolic pressure. This results in decreased perfusion pressure of the spinal cord. Hence CSFD is expected to lessen the CSF pressure and improve blood flow to spinal cord. Indirect evidence from canine models has shown improved neurological outcome using CSFD in spinal cord ischaemia (Blaisdell 1962; Bower 1989; Dasmahapatra 1988; McCullough 1988).

Although there have been multiple experimental studies, only three randomised controlled trials have come to light. A number of non-randomised observational cohort studies, non-randomised studies with historical controls and case series have also been reported (Acher 1990; Acher 1994; Acher 1998; Hollier 1992; Murray 1993; Safi 1998; Svensson 1988). Due to the heterogeneity in methodological design of various studies, it was not appropriate to pool all the data for meta-analysis.

An early trial from Houston (Crawford 1991) failed to show a reduction in neurological deficit using CSFD. The hypothesis of improving spinal cord perfusion by CSFD was not adequately tested in this study. To reduce CSF pressure to less than 10 mmHg, removal of up to 500 ml of CSF may be required. The intraoperative CSFD in this trial was limited to 50 ml. CSF pressure was reduced to less than 10 mmHg in only 20 (43%) of the 46 participants. Although this study had sufficient statistical power to show a reduction in neurological injury, the assumption of a possible reduction in the event rate by 80% in the treatment group was an unlikely outcome.

The Massachusetts study (Svensson 1998) concluded that a combination of CSFD and intrathecal papaverine reduced the risk of neurological injury after high-risk thoracoabdominal surgery. This study was terminated early by the institution review board after one third ($n = 33$) of the estimated sample size ($n = 100$) was entered because of a statistically significant difference in the rate of postoperative neurological deficit ($P = 0.039$). It is difficult to rule out the possibility of type I error as a result and the conclusion may be biased. Moreover it is difficult to interpret the role of CSFD alone as the therapeutic effect against intrathecal papaverine has not been addressed. In the Massachusetts trial (Svensson 1998), the control group had more emergency and urgent cases (7/16; 44%) than did the CSFD group (4/17; 25%).

The second trial from Houston (Coselli 2002) did not limit the CSFD to volume. Instead it targeted to maintain a CSF pressure of 10 mmHg or less. This adequately tested the pressure perfusion theory. The efficacy analysis performed after applying operative exclusion criteria showed a significant evidence of neurological protection in participants receiving CSFD as

compared to controls. There were no significant differences between intraoperative variables, including clamp and ischaemic times, body temperatures, and re-attachment of intercostal arteries, and the two groups were at similar overall risk for neurological deficits. A blinded examiner to perform postoperative neurological assessment, however, would have improved the study design.

The overall mortality rates are comparable in all the three trials. Similar rates have been reported by Coselli 1997, (92.6% in-hospital), Cambria 1997, (90% 60-day survival), Safi 1994, (96% 30-day survival), and Kouchoukos 1995, (91.2% 30-day survival).

Monitoring of CSF pressure in the control groups would have provided comparative data and results that could be extrapolated more objectively. In all the studies, adjunct methods such as distal aortic perfusion, re-attachment of intercostal and lumbar arteries and active cooling to a bladder temperature of 29°C to 31°C before aortic cross-clamping by atriocaval bypass were also employed. Their role in prevention of neurological insult remains unresolved.

It is important to develop a uniform method of scoring in evaluation of postoperative neurological deficit. An independent assessor for example, a neurologist may be more appropriate to assess any neurological injury.

Spinal cord ischaemia may lead to oedema following TAAA repair leading to delayed spinal cord injury. The role of CSFD for prevention of delayed-onset neurological injury in such cases also remains undetermined. The immediate initiation of multimodality treatment makes it impossible to attribute subsequent recovery to any individual manoeuvre. The type of treatment, length of aortic clamp times and the intercostal artery ischaemia time influences the predicted risks of neurological injury. Postoperative hypotension may also increase the risk of neurological injury (Crawford 1991).

It must be mentioned that the first two trials had some drawbacks. In the first Houston trial (Crawford 1991), CSFD was limited to a maximum of 50 ml in excess of loss during insertion of catheter and withdrawal of catheter at the end of surgery. The second study (Svensson 1998) was stopped early and CSFD participants also had intrathecal insertion of papaverine. Only the second Houston study (Coselli 2002) shows CSFD benefit in TAAA surgery.

It is appropriate to mention here that many non-randomised studies have looked at the role of CSFD as an adjunct to other modalities for their benefits in spinal cord protection in high risk thoracoabdominal aneurysm surgery (Acher 1990; Acher 1994; Acher 1998; Hollier 1992; Murray 1993; Safi 1998; Svensson 1988). It is difficult to conclude the relative benefits of each of the therapies given the confounding nature of the intervention itself and the weakness inherent in the non-randomised observational design employed in these trials. Small sample size and insufficient power to detect differences in the event rate were other technical difficulties with these trials.

CSFD has been shown to benefit for Types I and II TAAAs, but the results cannot be extrapolated to the repair of less extensive aneurysms. There is currently no clinical evidence that show that CSFD prevents deficits during the repair of Type III or IV TAAAs.

Cases of spinal cord dysfunction following endovascular repair have also been reported. The role of CSFD in such a setting remains to be determined.

AUTHORS' CONCLUSIONS

Implications for practice

There is limited evidence that perioperative cerebrospinal fluid drainage (CSFD) appears to reduce the rate of paraplegia after repair of Type I and II TAAA. CSFD is recommended as a component of the multimodality approach for prevention of neurological injury. Use of CSFD alone as protection has not been established from the available evidence.

Implications for research

It is difficult to employ CSFD alone for spinal cord protection in clinical practice. Hence more experimental work is required to understand better the mechanisms of neurological injury. There is also a need to develop better techniques for measuring spinal cord perfusion directly. There is a need for more clinical trials and perhaps multicentre collaboration to establish the role of CSFD in prevention of neurological injury.

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REFERENCES

References to studies included in this review

Coselli 2002 {published data only}

Coselli JS, Lemaire SA, Koksoy C, Schmittling ZC, Curling PE. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: results of a randomized clinical trial. *Journal of Vascular Surgery* 2002;**35**(4):631-9.

Crawford 1991 {published data only}

Crawford ES, Svensson LG, Hess KR, Shenaq SS, Coselli JS, Safi HJ, et al. A prospective randomized study of cerebrospinal fluid drainage to prevent paraplegia after high-risk surgery on the thoracoabdominal aorta. *Journal of Vascular Surgery* 1991;**13**(1):36-45; discussion 45-6.

Svensson 1998 {published data only}

Svensson LG, Hess KR, D'Agostino RS, Entrup MH, Hreib K, Kimmel WA, et al. Reduction of neurologic injury after high-risk thoracoabdominal aortic operation. *Annals of Thoracic Surgery* 1998;**66**(1):132-8.

References to studies excluded from this review

Acher 1990 {published data only}

Acher CW, Wynn MM, Archibald J. Naloxone and spinal fluid drainage as adjuncts in the surgical treatment of thoracoabdominal and thoracic aneurysms. *Surgery* 1990;**108**(4):755-61.

Acher 1994 {published data only}

Acher CW, Wynn MM, Hoch JR, Popic P, Archibald J, Turnipseed WD. Combined use of cerebral and spinal fluid drainage naloxone reduces the risk of paraplegia in thoracoabdominal aneurysm repair. *Journal of Vascular Surgery* 1994;**19**(2):236-46; discussion 247-8.

Acher 1998 {published data only}

Acher CW, Wynn MM, Hoch JR, Kranner PW. Cardiac function is a risk factor for paralysis in thoracoabdominal aortic replacement. *Journal of Vascular Surgery* 1998;**27**:821-8; discussion 829-30.

Hollier 1992 {published data only}

Hollier LH, Money SR, Naslund TC, Procter CD Sr, Buhrman WC, Marino RJ, et al. Risk of spinal cord dysfunction in patients undergoing thoracoabdominal aortic replacement. *American Journal of Surgery* 1992;**164**(3):210-3; discussion 213-4.

Murray 1993 {published data only}

Murray MJ, Bower TC, Oliver WC Jr, Werner E, Gloviczki P. Effects of cerebrospinal fluid drainage in patients undergoing thoracic and thoracoabdominal aortic surgery. *Journal of Cardiothoracic and Vascular Anesthesia* 1993;**7**(3):266-72.

Safi 1998 {published data only}

Safi HJ, Winnerkvist A, Miller CC 3rd, Iliopoulos DC, Reardon MJ, Espada R, et al. Effect of extended cross-clamp time during

thoracoabdominal aortic aneurysm repair. *Annals of Thoracic Surgery* 1998;**66**(4):1204-9.

Svensson 1988 {published data only}

Svensson LG, Stewart RW, Cosgrove DM 3rd, Lytle BW, Antunes MD, Beven EG, et al. Intrathecal papaverine for the prevention of paraplegia after operation on the thoracic or thoracoabdominal aorta. *Journal of Thoracic and Cardiovascular Surgery* 1988;**96**(5):823-9.

Additional references

Agee 1991

Agee JM, Flanagan T, Blackburne LH, Kron IL, Tribble CG. Reducing postischemic paraplegia using superoxide dismutase. *Annals of Thoracic Surgery* 1991;**51**(6):911-4; discussion 914-5.

Blaisdell 1962

Blaisdell FW, Cooley DA. The mechanism of paraplegia after temporary thoracic aortic occlusion and its relationship to spinal fluid pressure. *Surgery* 1962;**51**(3):351-5.

Bolton 1939

Bolton B. Blood supply of human spinal cord. *Journal of Neurology and Psychiatry* 1939;**2**:137-148.

Bower 1989

Bower TC, Murray MJ, Gloviczki P, Yaksh TL, Hollier LH, Pairolero PC. Effects of thoracic aortic occlusion and cerebrospinal fluid drainage on regional spinal cord blood flow in dogs: correlation with neurologic outcome. *Journal of Vascular Surgery* 1989;**9**(1):135-44.

Cambria 1997

Cambria RP, Davison JK, Zanetti S, L'Italien G, Brewster DC, Gerther JP, et al. Clinical experience with epidural cooling for spinal cord protection during thoracic and thoracoabdominal aneurysm repair. *Journal of Vascular Surgery* 1977;**25**(2):234-41; discussion 241-3.

Coselli 1997

Coselli JS, LeMaire SA, de Figueiredo LP, Kirby RP. Paraplegia after thoracoabdominal aortic aneurysm repair: is dissection a risk factor?. *Annals of Thoracic Surgery* 1997;**63**(1):28-35; discussion 35-6.

Cox 1992

Cox GS, O'Hara PJ, Hertzner NR, Piedmonte MR, Krajewski LP, Beven EG. Thoracoabdominal aneurysm repair: a representative experience. *Journal of Vascular Surgery* 1992;**15**(5):780-7; discussion 787-8.

Crawford 1986

Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, et al. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients. *Journal of Vascular Surgery* 1986;**3**(3):389-404.

Dasmahapatra 1988

Dasmahapatra HK, Coles JG, Wilson GJ, Sherret H, Adler S, Williams WG, et al. Relationship between cerebrospinal fluid dynamics and reversible spinal cord ischemia during experimental thoracic aortic occlusion. *Journal of Thoracic and Cardiovascular Surgery* 1988;**95**(5):920-3.

de Mol 1990

de Mol BA, Boezeman EH, Hamerlijnck RP, de Geest R. Experimental and clinical use of somatosensory evoked potentials in surgery of aneurysms of the descending thoracic aorta. *Thoracic and Cardiovascular Surgery* 1990;**38**(3):146-50.

Gillian 1958

Gillian LA. The arterial blood supply of the human spinal cord. *Journal of Comparative Neurology* 1958;**110**(1):75-104.

Grace 1997

Grace RR, Mattox KL. Anterior spinal artery syndrome following abdominal aortic aneurysmectomy. Case report and review of the literature. *Archives of Surgery* 1997;**112**(7):813-5.

Gray 1973

Gray H. Gray's Anatomy. 5th Edition. Edinburgh: Longman, 1973.

Grubbs 1988

Grubbs PE Jr, Marini C, Toporoff B, Nathan I, Basu S, Acinapura AJ, et al. Somatosensory evoked potentials and spinal cord perfusion pressure are significant predictors of postoperative neurologic dysfunction. *Surgery* 1988;**104**(2):216-23.

Joseph 1989

Joseph MG, Langsfeld MA, Lusby RJ. Infrarenal aortic aneurysm: unusual cause of paraparesis. *Australian and New Zealand Journal of Surgery* 1989;**59**(9):743-4.

Kazama 1994

Kazama S, Masaki Y, Maruyama S, Ishihara A. Effect of altering cerebrospinal fluid pressure on spinal cord blood flow. *Annals of Thoracic Surgery* 1994;**58**(1):112-5.

Kouchoukos 1995

Kouchoukos NT, Daily BD, Rokkas CK, Murphy SF, Bauer S, Abboud N. Hypothermic bypass and circulatory arrest for operations on the descending thoracic and thoracoabdominal aorta. *Annals of Thoracic Surgery* 1995;**60**(1):67-76; discussion 76-7.

Laschinger 1984

Laschinger JC, Cunningham JN Jr, Cooper MM, Krieger K, Nathan IM, Spencer FC. Prevention of ischemic spinal cord injury following aortic cross-clamping: use of corticosteroids. *Annals of Thoracic Surgery* 1984;**38**(5):500-7.

Marsala 1993

Marsala M, Vanicky I, Galik J, Radonak J, Kundrat I, Marsala J. Panmyelic epidural cooling protects against ischemic spinal cord damage. *Journal of Surgical Research* 1993;**55**(1):21-31.

McCullough 1988

McCullough JL, Hollier LH, Nugent M. Paraplegia after thoracic aortic occlusion: influence of cerebrospinal fluid drainage. Experimental and early clinical results. *Journal of Vascular Surgery* 1988;**7**(1):153-60.

Mehrez 1962

Mehrez IO, Nabseth DC, Hogan EL, Deterling RA Jr. Paraplegia following resection of abdominal aortic aneurysm. *Annals of Surgery* 1962;**156**(6):890-8.

Miyamoto 1960

Miyamoto K, Ueno A, Wada T, Kimoto S. A new and simple method of preventing spinal cord damage following temporary occlusion of the thoracic aorta by draining the cerebrospinal fluid. *Journal of Cardiovascular Surgery* 1960;**1**:188-97.

North 1991

North RB, Drenger B, Beattie C, McPherson RW, Parker S, Reitz BA, et al. Monitoring of spinal cord stimulation evoked potentials during thoracoabdominal aneurysm surgery. *Neurosurgery* 1991;**28**(2):325-30.

Okamoto 1992

Okamoto Y, Murakami M, Nakagawa T, Murata A, Moriya H. Intraoperative spinal cord monitoring during surgery for aortic aneurysm: application of spinal cord evoked potential. *Electroencephalography and Clinical Neurophysiology* 1992;**84**(4):315-20.

Qayumi 1994

Qayumi AK, Janusz MT, Dorovoni Zis K, Lyster DM, Jamieson WR, Poostzideh A, et al. Additive effect of allopurinol and deferoxamine in the prevention of spinal cord injury caused by aortic crossclamping. *Journal of Thoracic and Cardiovascular Surgery* 1994;**107**(5):1203-9.

Rosen 1988

Rosen SA. Painless aortic dissection presenting as spinal cord ischemia. *Annals of Emergency Medicine* 1988;**17**(8):840-2.

Safi 1994

Safi HJ, Bartoli S, Hess KR, Shenaq SS, Viets JR, Butt GR, et al. Neurologic deficit in patients at high risk with thoracoabdominal aortic aneurysms: the role of cerebral spinal fluid drainage and distal aortic perfusion. *Journal of Vascular Surgery* 1994;**20**(3):434-44; discussion 442-3.

Schepens 1995

Schepens MA, Defauw JJ, Hamerlijnck RP, Vermeulen FE. Use of left heart bypass in the surgical repair of thoracoabdominal aortic aneurysms. *Annals of Vascular Surgery* 1995;**9**(4):327-38.

Shiia 1995

Shiia N, Yasuda K, Matsui Y, Sakuma M, Sasaki S. Spinal cord protection during thoracoabdominal aneurysm repair: results of selective reconstruction of the critical segmental arteries guided by evoked spinal cord potential monitoring. *Journal of Vascular Surgery* 1995;**21**(6):970-5.

Suzuki 1994

Suzuki S, Ogane K, Souma M, Ohkuma H, Iwabuchi T. Efficacy of steroid hormone in solution for intracranial irrigation during aneurysmal surgery for prevention of the vasospasm syndrome. *Acta Neurochirurgica* 1994;**131**(3-4):184-8.

Svensson 1990

Svensson LG, Grum DF, Bednarski M, Cosgrove DM 3rd, Loop FD. Appraisal of cerebrospinal fluid alterations during aortic surgery with intrathecal papaverine administration and cerebrospinal fluid drainage. *Journal of Vascular Surgery* 1990;**11**(3):423-9.

Vanicky 1993

Vanicky I, Marsala M, Galik J, Marsala J. Epidural perfusion cooling protection against protracted spinal cord ischemia in rabbits. *Journal of Neurosurgery* 1993;**79**(5):736-41.

Zull 1988

Zull DN, Cydulka R. Acute paraplegia: a presenting manifestation of aortic dissection. *American Journal of Medicine* 1988;**84**(4):765-70.

References to other published versions of this review

Khan 2002

Khan SN, Stansby GP. Cerebrospinal fluid drainage for thoracic and thoracoabdominal aortic aneurysm surgery (Protocol). *Cochrane Database of Systematic Reviews* 2002, Issue 2. [DOI: [10.1002/14651858.CD003635](https://doi.org/10.1002/14651858.CD003635)]

Khan 2003

Khan SN, Stansby GP. Cerebrospinal fluid drainage for thoracic and thoracoabdominal aortic aneurysm surgery. *Cochrane Database of Systematic Reviews* 2003, Issue 4. [DOI: [10.1002/14651858.CD003635.pub2](https://doi.org/10.1002/14651858.CD003635.pub2)]

CHARACTERISTICS OF STUDIES

Characteristics of included studies [ordered by study ID]

Coselli 2002

Methods	Randomisation: concealed randomisation using computer-generated assignment in sequentially numbered opaque envelopes. Blinding: surgical team was not blinded to group assignment. Intention: to prevent complications.
Participants	Location: Houston, Texas, United States. Total no. of participants: 156; 82 treatment; 74 control. Withdrawals: 6 treatment; 5 control. Sex: 46 males, 30 females treatment; 44 males, 25 females. Age: 65.5 ± 10.2 years treatment; 65.5 ± 10.9 years control. Inclusion criteria: Type I or II TAAA.
Interventions	Treatment: CSF drainage + moderate heparinisation, permissive mild hypothermia, left heart bypass, and re-attachment of patent critical intercostal arteries. Controls: moderate heparinisation + permissive mild hypothermia + left heart bypass + re-attachment of patent critical intercostal arteries.
Outcomes	Daily evaluation of each patient's neurologic status until discharge and classified as 'deficit' or 'no deficit'. No blinding of evaluation mentioned.
Notes	Efficacy analysis was done separately after withdrawal of 11 patients who did not meet the operative inclusion criteria.
Risk of bias	
Bias	Authors' judgement Support for judgement

Coselli 2002 (Continued)

Allocation concealment (selection bias)	Low risk	A - Adequate
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Crawford 1991

Methods	<p>Randomisation: concealed randomisation using computer-generated sequence in opaque, sealed envelopes.</p> <p>Blinding: Observers documenting outcome were blinded only in cases initially documented by the operating team to have a neurological deficit.</p> <p>Intention: to prevent complications.</p>
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Participants	<p>Location: Houston, Texas, United States.</p> <p>Total no. of participants: 100; 47 treatment; 52 control (46 treatment and 52 control evaluated).</p> <p>Withdrawals: 1 (pulmonary oedema). Death: 1.</p> <p>Sex: Males and females.</p> <p>Inclusion criteria: Type I or II TAAA.</p>
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Interventions	<p>Treatment: moderate heparinisation, permissive mild hypothermia, left heart bypass, and re-attachment of patent critical intercostal arteries. CSF drainage peroperatively only +/- atriofemoral bypass +/- re-attachment of intercostal and lumbar vessels.</p> <p>Controls: distal atriofemoral bypass +/- re-attachment of intercostal and lumbar vessels.</p>
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Outcomes	Reported neurological deficit and if present, graded by a neurologist.
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Notes	Average CSF drainage was 52.5 ml only and was regardless of CSF pressures maintained intraoperatively.
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Risk of bias

Bias	Authors' judgement	Support for judgement
Allocation concealment (selection bias)	Low risk	A - Adequate

Svensson 1998

Methods	<p>Randomisation: concealed randomisation using computer-generated assignment and the use of folded cards in serially numbered opaque envelopes.</p> <p>Blinding: observers documenting the outcome were not blinded.</p> <p>Intention: to prevent complications.</p>
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Participants	<p>Location: Burlington, Massachusetts, United States.</p> <p>Total no. of participants: 33; 17 treatment; 16 control.</p> <p>Sex: 23 males; 8 females.</p> <p>Age: Median age 66 years (range 34 to 79 years).</p>
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Svensson 1998 (Continued)

Inclusion criteria: Type I or II TAAA.

Interventions	Treatment: CSF drainage + Intrathecal papaverine +/- distal atriofemoral bypass using active cooling +/- re-attachment of intercostal and lumbar vessels. Controls: distal atriofemoral bypass using active cooling +/- re-attachment of intercostal and lumbar vessels.
Outcomes	Neurological deficit and motor scores reported by blinded neurologist.
Notes	Initial plan to recruit 100 patients had to be dropped in view of significant results. Only 33 patients re-recruited.

Risk of bias

Bias	Authors' judgement	Support for judgement
Allocation concealment (selection bias)	Low risk	A - Adequate

CSF = cerebrospinal fluid

CSFD = cerebrospinal fluid drainage

TAAA = thoracoabdominal aortic aneurysm

Characteristics of excluded studies [ordered by study ID]

Study	Reason for exclusion
Acher 1990	Non-randomised study using historical controls. Small numbers. Twelve out of 24 patients in the CSFD group also received intravenous naloxone for 48 hours postoperatively. Relative contribution of each in reducing deficit could not be determined.
Acher 1994	A retrospective observational cohort study. Both naloxone and CSFD used.
Acher 1998	A prospective observational cohort study. Used both naloxone and CSFD as interventions.
Hollier 1992	A non-randomised historical control study. CSFD up to three days postoperatively, avoiding solutions containing glucose, passive hypothermia plus a bolus of thiopental sodium before cross-clamp, use of mannitol and nimodipine, Non protocol group also had three patients with CSFD. Intraoperative details for non protocol group are not clear.
Murray 1993	A non-randomised study using historical controls. Extent of disease differed in the two groups. Variable surgical technique between the two groups, the use of mild hypothermia (passive cooling to 34°C) in the intervention group and variable amount of CSFD were the main reasons for exclusion of results.
Safi 1998	A non-randomised historical control study mainly looking at the effect of cross-clamp time greater than 30 min in all patients with TAAA or TA. A group of patients did not receive CSFD.
Svensson 1988	Non randomised study with concurrent control group.

CSFD = cerebrospinal fluid drainage

TA = thoracoabdominal

TAAA = thoracoabdominal aortic aneurysm

DATA AND ANALYSES

Comparison 1. Cerebrospinal fluid drainage vs No drainage

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Development of lower extremity neurologic deficits	3	287	Odds Ratio (M-H, Fixed, 95% CI)	0.48 [0.25, 0.92]

Analysis 1.1. Comparison 1 Cerebrospinal fluid drainage vs No drainage, Outcome 1 Development of lower extremity neurologic deficits.

Comparison 2. Cerebrospinal fluid drainage vs No drainage in CSFD only trials

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Development of lower extremity neurologic deficits	2	254	Odds Ratio (M-H, Fixed, 95% CI)	0.57 [0.28, 1.17]
1.1 Coselli and Crawford Studies	2	254	Odds Ratio (M-H, Fixed, 95% CI)	0.57 [0.28, 1.17]

Analysis 2.1. Comparison 2 Cerebrospinal fluid drainage vs No drainage in CSFD only trials, Outcome 1 Development of lower extremity neurologic deficits.

Comparison 3. Intention to treat

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Intention to treat	2	256	Odds Ratio (M-H, Fixed, 95% CI)	0.57 [0.28, 1.17]

Analysis 3.1. Comparison 3 Intention to treat, Outcome 1 Intention to treat.

ADDITIONAL TABLES

Table 1. Crawford Classification of Aortic Aneurysms

Type	Extent
I	Most or all of the descending thoracic aorta to the celiac artery
II	Most or all of the descending thoracic and abdominal aorta
III	Most or all of the distal descending thoracic and abdominal aorta
IV	Most or all of the abdominal aorta up to and including the celiac artery

Table 2. Results of trials

Study	Cases	Controls	CSFD vol / pressure	Postop drainage
Crawford	14/46 (30%)	17/52 (33%)	50 mls only	No
Svensson	2/17 (11.8%)	7/16 (43.8%)	7 - 10 cm H2O	Up to 48 hours
Coselli	2/82 (2.7%)	9/74 (12.2%)	< 10 mmHg	Up to 48 hours

APPENDICES

Appendix 1. CENTRAL search strategy

#1	MeSH descriptor Aortic Aneurysm explode all trees	687
#2	aneurysm* near4 (abdom* or thoracoabdom* or thoraco-abdom* or aort*)	993
#3	AAA* or TAAA or TEVAR	1142
#4	(aort* near3 (ballon* or dilat* or bulg* or expan*))	67
#5	(#1 OR #2 OR #3 OR #4)	1873
#6	MeSH descriptor Cerebrospinal Fluid explode all trees	128
#7	MeSH descriptor Cerebrospinal Fluid Pressure, this term only	44
#8	MeSH descriptor Spinal Cord explode all trees with qualifier: BS	12
#9	MeSH descriptor Paraplegia explode all trees with qualifiers: CO,PC	34
#10	MeSH descriptor Paraparesis explode all trees	12
#11	MeSH descriptor Neurologic Manifestations, this term only	39
#12	spinal near3 (isch* or damage* or injur*)	1425
#13	paraplegia or paraparesis	410
#14	neurologi* near3 (damag* or injur*)	316
#15	(cerebrospin* or CSF) near3 (drain* or pressure)	175
#16	intrathecal near3 pressure	14
#17	(#6 OR #7 OR #8 OR #9 OR #10 OR #11 OR #12 OR #13 OR #14 OR #15 OR #16)	2191
#18	(#5 AND #17)	22

FEEDBACK

Conclusions: Effects of CSF drainage

Summary

It is deeply confusing why this meta-analysis has completely the opposite conclusion on the effects of CSF drainage on the incidence of paraplegia for repair of thoraco-abdominal aortic surgery from that of Cina CS, Abouzahr L, Arena GO, et al. Cerebrospinal fluid drainage to prevent paraplegia during thoracic and thoracoabdominal aortic aneurysm surgery: a systematic review and meta analysis. J Vasc Surg 2004;40:36-44. This is most especially so when the core analysis is on the same three RCTs! Some rationale needs to be added to the discussion for this divergence.

Reply

The Journal of Vascular Surgery article (Cina et al) included the same three randomised controlled trials used in our systematic review as well as 11 retrospective cohort controlled studies which we did not include as these are usually prone to bias. Based on the randomised data, we have stated that there are limited data supporting the use of CSF drainage and our conclusion in "Implications for practice" was that "it is recommended as a component of the multimodality approach". This is not so different from the conclusions of Cina, et al that it should be used "as an adjunct to prevent paraplegia". We do not agree that our conclusions are "completely the opposite" of theirs.

Contributors

Feedback: Peter Alston, Anaesthetist

Reply: Gerard Stansby, Shaukat Khan

WHAT'S NEW

Date	Event	Description
19 September 2012	Review declared as stable	No new studies have been identified since the review was first published in 2004. The review has been marked as stable and will only be updated when new studies are identified.

HISTORY

Protocol first published: Issue 2, 2002

Review first published: Issue 1, 2004

Date	Event	Description
31 May 2012	New citation required but conclusions have not changed	Searches were updated. No new trials were found. Conclusions not changed.
31 May 2012	New search has been performed	Searches were updated. No new trials were found.
13 May 2008	Amended	Converted to new review format.
13 November 2007	New search has been performed	Re-ran searches; no new trials found. Conclusions remain unchanged.
14 November 2006	New search has been performed	Added plain language Summary and acknowledgements. Made minor copy edits to Characteristics of included studies table, reference list, and text. Clarified comparisons in Comparisons and data table. Updated search strategy for CENTRAL. Updated dates of changes. No new trials found in literature search; conclusions remain unchanged.
15 November 2005	Feedback has been incorporated	Comment and response to comment added.

Date	Event	Description
12 August 2004	Amended	Synopsis added to the review. No new trials found. Minor revisions made in accordance with Cochrane Style Guide

CONTRIBUTIONS OF AUTHORS

SNK identified trials, determined eligibility and quality of trials, extracted data and wrote the review.
GS identified trials, determined eligibility and quality of trials, verified the data, and contributed to the writing of the review.

DECLARATIONS OF INTEREST

None known

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Internal sources

- No sources of support supplied

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INDEX TERMS

Medical Subject Headings (MeSH)

*Cerebrospinal Fluid; Aortic Aneurysm, Abdominal [*surgery]; Aortic Aneurysm, Thoracic [*surgery]; Cerebrospinal Fluid Pressure; Drainage [*methods]; Ischemia [complications] [*prevention & control]; Papaverine [administration & dosage]; Paraparesis [etiology]; Paraplegia [etiology]; Postoperative Complications [etiology]; Spinal Cord [*blood supply]; Vasodilator Agents [administration & dosage]

MeSH check words

Humans