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LITERATURE REVIEW

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Compressive Pressure *Versus* Time in Cauda Equina Syndrome

A Systematic Review and Meta-Analysis of Experimental Studies

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Study Design. Systematic review and meta-analysis.

Objective. To examine the relationship between compressive pressure and its duration in cauda equina compression, and the effects of subsequent decompression, on neurophysiological function, and pathophysiology in animal studies. We further aim to investigate these relationships with systemic blood pressure to assess whether a vascular component in the underlying mechanism may contribute to the clinical heterogeneity of this disease.

Summary of Background Data. The complex relationship between preoperative factors and outcomes in cauda equina syndrome (CES) suggests heterogeneity within CES which may inform better understanding of pathophysiological process, their effect on neurological function, and prognosis.

Methods. Systematic review identified 17 relevant studies including 422 animals and reporting electrophysiological measures (EP), histopathology, and blood flow. Modeling using meta-regression analyzed the relationship between compressive pressure, duration of compression, and electrophysiological function in both compression and decompression studies.

Results. Modeling suggested that electrophysiological dysfunction in acute cauda equina compression has a sigmoidal response, with particularly deterioration when mean arterial blood pressure is exceeded and, additionally, sustained for approximately 1 hour. Accounting for pressure and duration may help risk-stratify patients pre-decompression. Outcomes after decompression appeared to be related more to the degree of compression, where exceeding systolic blood pressure tended to result in an irreversible lesion, rather than duration of compression. Prognosis was most strongly associated with residual pre-decompression function.

Conclusion. Compressive pressure influences effects and outcomes of cauda equina compression. We suggest the presence of two broad phenotypic groups within CES defined by the degree of ischaemia as a potential explanatory pathophysiological mechanism.

Key words: animal models, biomechanics, cauda equina syndrome, electrophysiology, lumbar disc hernia, meta-regression, neurophysiology, outcomes, pathophysiology, predictive factor, prognosis, spinal surgery.

Level of Evidence: 1

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The relationship between preoperative factors and outcomes in patients with acute cauda equina syndrome (CES) is unclear and has been identified as a research priority.[1] Meta-analyses of human studies suggested that neurological outcomes are not improved when decompression is performed within 24 to 72 hours after onset or urinary incontinence[2,3] but more recent studies have not supported this correlation.[4,5] It has been suggested that neurological deterioration, which appears to be a continuous rather than a step-wise phenomenon, may be a more important determinant of prognosis than the duration of compression.[6] Other examined predictive factors, such as rate of symptom onset[5,7–9] and size of the herniating disc[10,11] have yielded contradictory or non-significant results, respectively.

The variability in findings suggests that there is a large heterogeneity within CES and further knowledge about the pathophysiological process and its effect on neurological function and prognosis might help guide most effective

management. One potential source of heterogeneity is the compressive pressure exerted by the herniating disc on the cauda equina.

A meta-analysis of animal studies testing spinal cord decompression suggested that higher compressive pressures and longer duration are associated with smaller treatment effects.[12] A power law relationship was found when the compressive pressure was plotted against duration that resulted in paraplegia, with higher pressures resulting in paraparesis faster compared with lower pressures, possibly due to variation in the degree of secondary ischaemia. Therefore, compressive pressure may have importance for both the management and the prognosis of CES. Animal models of cauda equina compression allow for controlled onset of compression *in vivo* and study of pathophysiological progression.

Aims

We aimed to examine any relationship of both compressive pressure and duration in cauda equina compression, and subsequent decompression, with neurophysiological function and pathophysiology in animal studies using systematic review and meta-analysis. Further, we aimed to investigate any relationship with systemic blood pressure to assess whether a vascular contribution in the underlying mechanism might contribute to the clinical heterogeneity of this disease.

MATERIALS AND METHODS

Protocol

The *a priori* protocol was registered on the CAMARADES platform (http://www.dcn.ed.ac.uk/camarades).

Study Eligibility Criteria

Studies underwent two-stage screening to identify animal models that used constant, single-level, paracentral compression defined in mmHg of the cauda equina for a maximum 1 week duration with or without subsequent decompression (Supplementary Text 1, http://links.lww.com/BRS/B422).

Information Sources and Search

We searched MEDLINE, EMBASE, Web of Science and PubMed on June 24, 2017 using a broad, inclusive search strategy (Supplementary Text 2, http://links.lww.com/BRS/B422).

Data Extraction

We extracted study design and outcome measures for electrophysiology, compression-zone blood flow, and histology (Supplementary Text 3, http://links.lww.com/BRS/B422).

Risk of Bias

Risk of bias assessment in individual studies was performed using an adapted version of the 10-point CAMARADES checklist[13–15] (Supplementary Text 4, http://links.lww.com/BRS/B422).

Data Analysis

Effect Size

For compression studies, we defined effect size as the percentage loss of function after compression compared with precompression or sham operated control. For decompression studies, we calculated two measures of effect: an absolute measure, the percentage recovery with normal function set at 100% and no function at 0%; and a mean difference, the difference between pre- and post-decompression,[16] both at 90 minutes recovery.

Modeling

We fitted linear and non-linear mixed-effects models using the restricted maximum likelihood method (Supplementary Text 5, http://links.lww.com/BRS/B422). We explored the relations of pressure, duration, pressure × duration, pre-decompression function, electrophysiological measures and mean arterial/systolic blood pressure (MABP/SBP) with effects on neurophysiological function with our without decompression. Non-independence of points within a time series was accounted for by using continuous autoregression of order 1 (CAR1) structures.

Model Selection and Fit

We fitted models using the maximum likelihood approach, then used the Akaike and Bayesian Information Criteria (AIC and BIC, respectively) approaches to assess model fit during model selection. After model selection we calculated standard deviations of the population-level residuals to assess deviation from the model. I^2 and pseudo-R^2 values were also calculated (Supplementary Text 5, http://links.lww.com/BRS/B422). Analysis was conducted using the *nlme* and *metafor* packages and results presented as bubble plots *ggplot2*, *scales*, *gridExtra* packages, with the size of the points corresponding to the weight assigned to that point, in R (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Study Selection

We identified 6393 unique English-language studies; 66 used animal models of acute cauda equina compression; 17 of these satisfied the inclusion criteria for this study[17–33] (Supplementary Figure 1, http://links.lww.com/BRS/B422).

Study Characteristics

A total of 422 animals were included: nine studies used canine models (218 animals) and eight used porcine models (204 animals). Characteristics of the included studies are summarized in Table 1.

Risk of Bias

Median study quality was 3/10, interquartile range 3 to 4 (Supplementary Figure 2, http://links.lww.com/BRS/B422).

TABLE 1. Characteristics of the Included Studies

Study ID	Animal	Level	Pressure, mmHg	Duration, min	Recovery End Time, min	BP (SD; mmHg)	Histology	Electrophysiology	Blood Flow
Sekiguchi 2008[17]	Canine	L7	10	120	90	–	–	MNCV	–
Sekiguchi 2004[18]	Canine	L7	10	10080	–	SBP - 104 (16)	–	–	Yes
Takahashi 2003[19]	Canine	S1	10	1, 10080	–	SBP - 145 (25)	Morphology	SNCV, SEP (amplitude)	–
Sekiguchi 2002[20]	Canine	L7	10	10080	–	–	Morphology	–	Yes
Konno 2001[21]	Canine	L7	10	10080	–	–	–	MNCV	–
Otani 2001[22]	Canine	L7	10	10080	–	–	–	–	Yes
Kikuchi 1996[23]	Canine	L7	10, 50, 100	120, 10080	–	–	–	MNCV	–
Konno 1996[24]	Canine	L7	100	120	90	–	–	MNCV, MEP (area)	–
Sato 1995[25]	Canine	L7	50, 100, 200	120, 10080	90	–	Morphology	MNCV, MEP (area)	–
Baker 1995[26]	Porcine	Co1/2	15	24	–	–	–	–	Yes
Olmarker 1992[27]	Porcine	Co1/2	10, 50	120	90	–	–	MEP (amplitude)	–
Pedowitz 1992[28]	Porcine	Co1/2	50, 100, 200	240	90	–	–	MEP (amplitude), SEP (amplitude)	–
Rydevik 1991[29]	Porcine	Co1/2	50, 75, 100, 200	120	90	–	Morphology	MEP (amplitude), SEP (amplitude)	–
Garfin 1990[30]	Porcine	Co1/2	50, 100, 200	120	90	MABP – 92 (4), 60	Morphology	MEP (amplitude), SEP (amplitude), MNCV, SNCV	–
Olmarker 1990[31]	Porcine	Co1/2	50, 100, 200	120	90	–	–	MEP (amplitude)	–
Olmarker 1990b[32]	Porcine	Co1/2	10, 50, 200	30	–	–	Glucose transport	–	–
Olmarker 1989[33]	Porcine	Co1/2	50, 200	120	–	–	Morphology	–	–

Note: Co indicates coccygeal; Fast, 0.05–0.1 seconds; L, lumbar; MABP, mean arterial blood pressure; MEP, motor evoked potential; MNCV, motor nerve conduction velocity; S, sacral; SBP, systolic blood pressure; SD, standard deviation; SEP, sensory evoked potential; slow, 10–20 seconds; SNCV, sensory nerve conduction velocity.

Analysis

Histology

Briefly, short compression (2–120 min) at high pressure (50–200 mmHg) resulted in edema, which increased with both higher pressure and longer duration[25,29,30,33] (Supplementary Table 1, http://links.lww.com/BRS/B422).

Blood Flow

Low pressure compression (10–15 mmHg) at either 24 minutes or 7 days did not significantly reduce mean blood flow (Supplementary Table 2, http://links.lww.com/BRS/B422).

Electrophysiology (EP)

Global Effect Size

CE compression is significantly reduced EP measures and decompression with 90 minutes recovery significantly improved EP measures (Table 2). There was substantial heterogeneity across studies (Supplementary Table 3, http://links.lww.com/BRS/B422).

Modeling of Compression Studies

The maximum predicted effect was a 94.3% (95% confidence interval [CI]: 86.8%–>100.0%) decline in electrophysiological function (Table 3). For duration of compression, the model suggests near maximal effects after 90 minutes, and a linear increase in deficit between 30 and 60 minutes (Figure 1A). For pressure, the model suggested that the near-maximum effect was reached at 140 mmHg; there was little to no effect below 50 mmHg; and the effect increased near-linearly from around 80 to 115 mmHg (Figure 1B). Incorporating MABP and SBP, as largely externally imposed constants onto the data, resulted in a mostly additive transformation but showed that with MABP the mid-point was near 0 suggesting that exceeding it largely increases effect size (Supplementary Figure 3, http://links.lww.com/BRS/B422).

Both the linear and univariate models performed poorly compared with the models above ($P < 0.0001$) and had poor predictive validity (Supplementary Table 4, and Figure 4, http://links.lww.com/BRS/B422).

The Pressure × Duration model performed poorer by all measures compared with the main models ($P < 0.001$, Table 3). Incorporating MABP and SBP resulted in an

TABLE 2. Global Effect Size of Compression and Decompression Studies

	Effect Size	95% CI	*k*	*P*
Compression	34.77	20.91–48.63	28	<0.0001
Decompression—absolute measure	50.91	65.28–79.65	27	<0.0001
Decompression—mean differences	12.23	4.623–19.83	27	0.0027

Note: CI indicates confidence interval.

TABLE 3. Parameters of Main Models for Compression and Decompression Studies

	Parameter	Estimate	95% CI	*P*	σ	I^2 Param	I^2 Overall	R^2	AIC	BIC	SD Residuals
Compression	Asym:	94.3	86.8–>100	<0.001	9.91	98.3%	95.7%	70.0%	2442.0	2473.0	14.1
	Dmid:	44.9	37.5–52.3	<0.001	10.7	92.2%					
	Pmid:	96.2	89.0–103.3	<0.001	12.8	98.4%					
	Scal:	10.1	9.0–11.2	<0.001	–	18.5%					
Decompression—absolute measure	Intercept:	152.2	125.9–178.6	<0.001	15.9	99.1%	99.1%	5.83%	448.5	457.4	16.7
	D:	−0.21	−0.38–0.04	0.018	–	98.3%					
	P:	−0.53	−0.65–0.42	<0.001	–	97.2%					
Decompression—mean difference	Intercept:	−51.9	−87.6–16.3	0.006	14.8	98.3	98.3%	0%	544.4	553.3	14.8
	P:	1.27	0.60–1.93	0.001	–	98.5					
	P^2:	−0.00	−0.01–0.00	0.001	–	98.5					

Note: AIC indicates akaike information criterion; BIC, Bayesian information criterion; CI, confidence interval; D, duration; P, pressure; SD, standard deviation.

additive transformation revealing grouping of studies based on whether the aforementioned pressures were exceeded by compression (Figure 2; Supplementary Figure 5, http://links.lww.com/BRS/B422).

Modeling of Decompression Studies

The absolute measure model suggested that each minute delay to decompression reduced recovery of function by 0.21% (95% CI: 32.7–62.4, $P = 0.018$; Table 3, Supplementary Figure 6A, http://

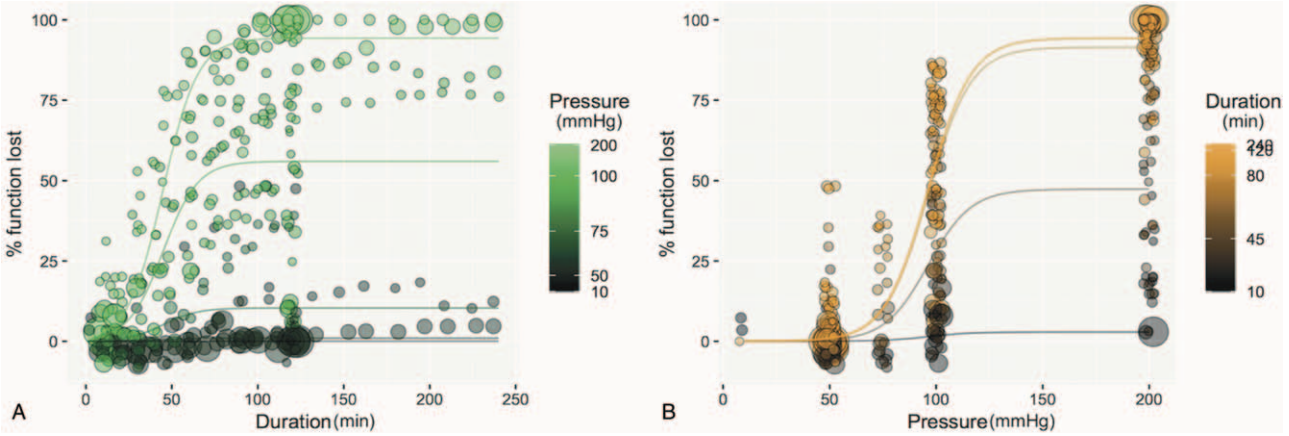

Figure 1. Models of compression studies. (**A**) By duration; (**B**) by pressure.

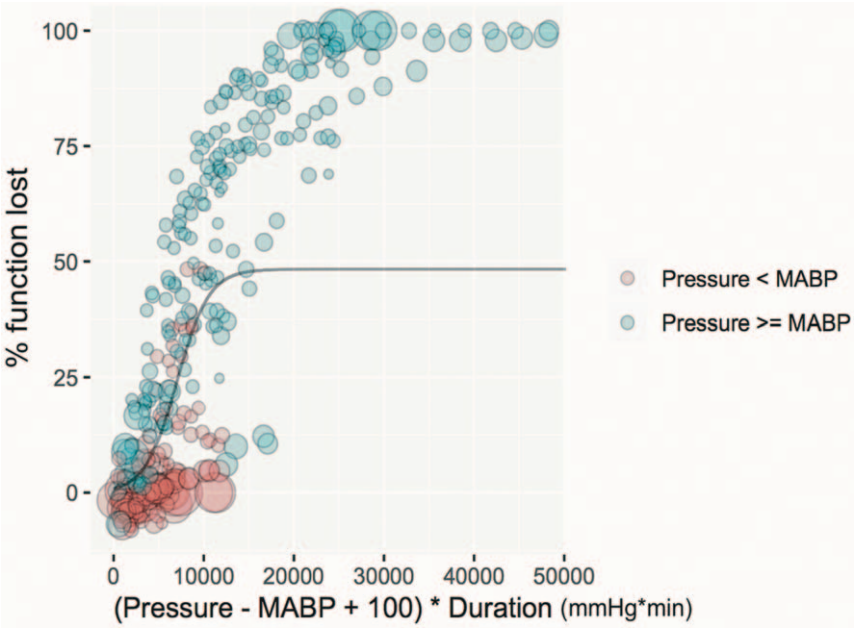

Figure 2. Pressure × duration models of compression studies accounting for MABP. MABP indicates mean arterial blood pressure.

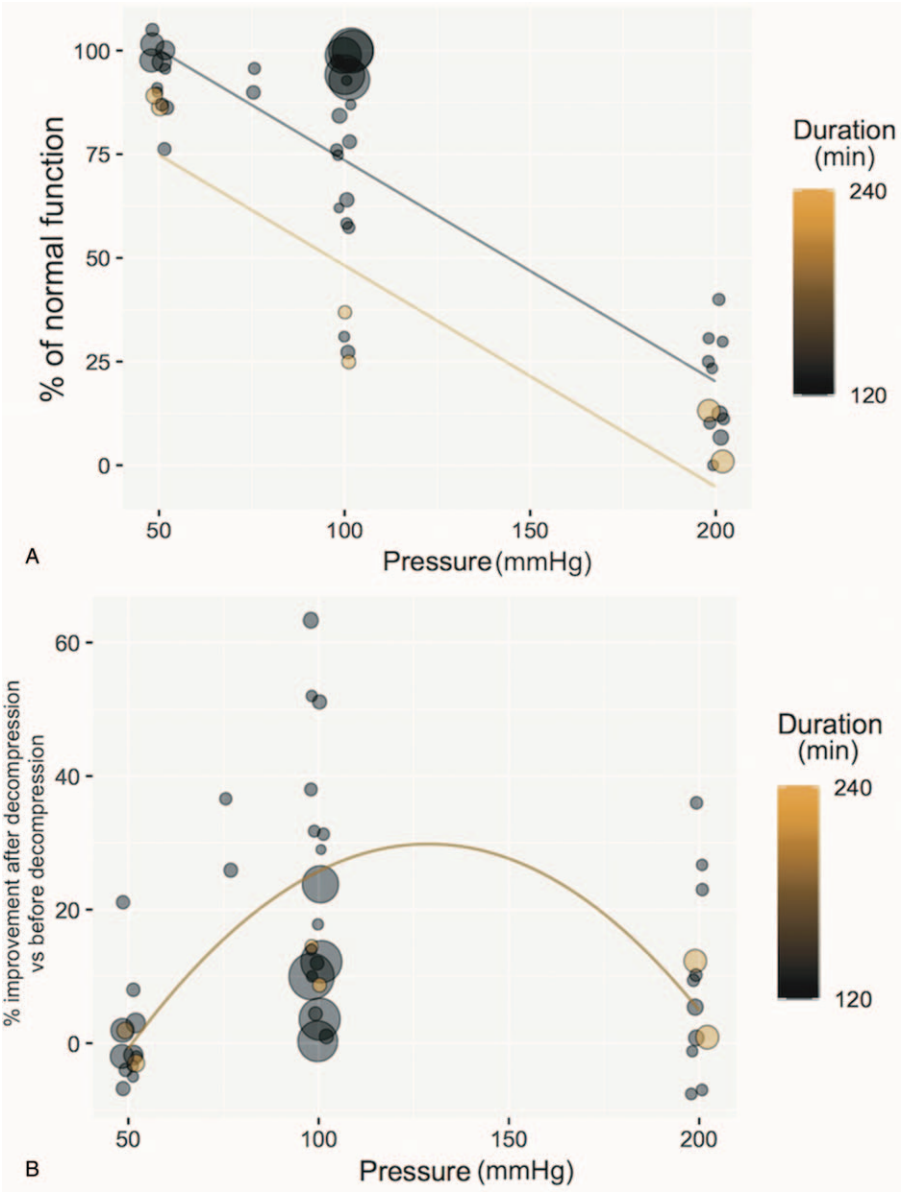

Figure 3. Models of decompression studies after 90 minutes recovery. (**A**) Using absolute measure by pressure; (**B**) using mean difference by pressure.

links.lww.com/BRS/B422). Each additional mmHg of compression was predicted to reduce function by 0.53% of normal performance (95% CI: 0.42–0.65, $P < 0.0001$, Figure 3A). For mean differences, the maximum improvement was at 128.9 mmHg, and there were no effects below 51.0 mmHg and above 206.7 mmHg (Figure 3B). Duration of compression was not a significant predictor of effect ($P = 0.44$), and including it as moderator worsened AIC/BIC (Supplementary Figure 6B, http://links.lww.com/BRS/B422). The mean differences model incorporating MABP shifted the vertex of the curve closer towards zero (Supplementary Figure 7, http://links.lww.com/BRS/B422).

The Pressure × Duration model for decompression also performed poorer than the main model ($P < 0.0001$, Table 4, Supplementary Figure 8A–B, http://links.lww.com/BRS/B422) and including MABP and SBP again resulted in a mostly additive transformation (Supplementary Figure 8C–F). The univariate models performed poorer compared with main model ($P < 0.0001$, Supplementary Table 4, http://links.lww.com/BRS/B422).

Incorporating the precise electrophysiological measure used in compression and decompression studies led to a significant improvement in model fit ($P < 0.0001$) but not in predictive utility (Supplementary Table 5, http://links.lww.com/BRS/B422).

Pre-decompression function was strongly related to recovery, more so than the pressure and duration models (Table 5, Figure 4A, B, Supplementary Figure 9, http://links.lww.com/BRS/B422).

DISCUSSION

Compressive Pressure, Duration, and Electrophysiological Function

Compression

Our findings show that low compressive pressure had little effect on EP function but that once pressure is increased, EP function deteriorates near-linearly. Furthermore, once

TABLE 4. Parameter of Pressure × Duration Models for Compression and Decompression Studies

	Parameter	Estimate	95% CI	*P*	σ	I^2 Param	I^2 Overall	R^2	AIC	BIC	SD Residuals
Compression	Asym:	47.57	32.72–62.43	<0.001	37.3	99.5%	99.5%	68.9%	2530.0	2553.2	23.6
	Mid:	6598.5	5295.8–7901.3	<0.001	1948.6	97.6%					
	Scal:	1471.3	1683.6–1896.0	<0.001	–	55.2%					
Decompression—Abs measure	Intercept:	137.9	115.9–159.9	<0.001	16.7	98.0%	98.0%	<0%	491.3	500.3	17.2
	PxD:	−0.006	−0.009–0.004	<0.001	–	97.8%					
	$(PxD)^2$:	7.0 e-8	2.2e−8–1.2e−7	0.0065	–	98.4%					
Decompression—mean diff	Intcp:	3.3	−21.4–28.0	0.79	18.9	98.4%	98.4%	<0%	587.5	596.4	16.8
	P:	0.001	−0.001–0.004	0.35	–	98.3%					
	P^2:	−3.0e−8	−8.5e−8–2.5e−8	0.27	–	98.8%					

Note: AIC indicates akaike information criterion; BIC, Bayesian information criterion; CI, confidence interval; D, duration; P, pressure; SD, standard deviation.

TABLE 5. Parameters of Pre-decompression Function Models

	Parameter	Estimate	95% CI	*P*	I^2 Param	I^2 Overall	R^2	AIC	BIC	SD Residuals
Absolute measure	Asym:	100.5	96.3–104.8	<0.001	72.0%	72.1%	13.9%	424.5	433.7	14.5
	lrc:	−3.49	−3.8–03.1	<0.001	93.9%					
Mean differences	Intercept:	4.3	−8.6–17.2	0.50	98.3%	98.3%	20.4%	433.7	442.6	14.5
	ES:	1.2	0.8–1.7	<0.001	95.0%					
	Intercept∗ (ES^2):	−0.014	−0.02–−0.01	<0.001	92.0%					

Note: AIC indicates akaike information criterion; BIC, Bayesian information criterion; CI, confidence interval; D, duration; ES, effect size/% function pre-decompression; P, pressure; SD, standard deviation.

compression exceeds MABP a large effect size is more likely, even at pressures less than SBP. Longer durations of compression also have a strong effect on deteriorating EP function and the product of duration and compressive pressure too shows a sigmoid relationship. There were still low effect sizes once MABP was exceeded but these data points had short durations of compression suggesting that duration may determine extent of the underlying pathological process that results in EP dysfunction. Our data suggest that once compression exceeds a certain limit deterioration occurs rapidly in under 1 hour. Conversely, at a low compressive pressure it appears that a lower level of dysfunction is reached that is unlikely to progress from longer duration. This is supported by the fit of the Pressure × Duration model which extrapolates the data points to achieve the asymptote around 50% and reveals an unmeasured group of low pressure/long duration not present in the included studies (Supplementary Figure 10, http://links.lww.com/BRS/B422). Accounting for pressure and duration may help risk-stratify patients for decompression: those who are unlikely to deteriorate further, those about to deteriorate rapidly and those for whom it is likely too late to recover sufficiently.

In patients undergoing discectomy for lumbar disc herniation compression pressures varied from 7 to 256 mmHg (53 mmHg mean) and it was significantly higher in those

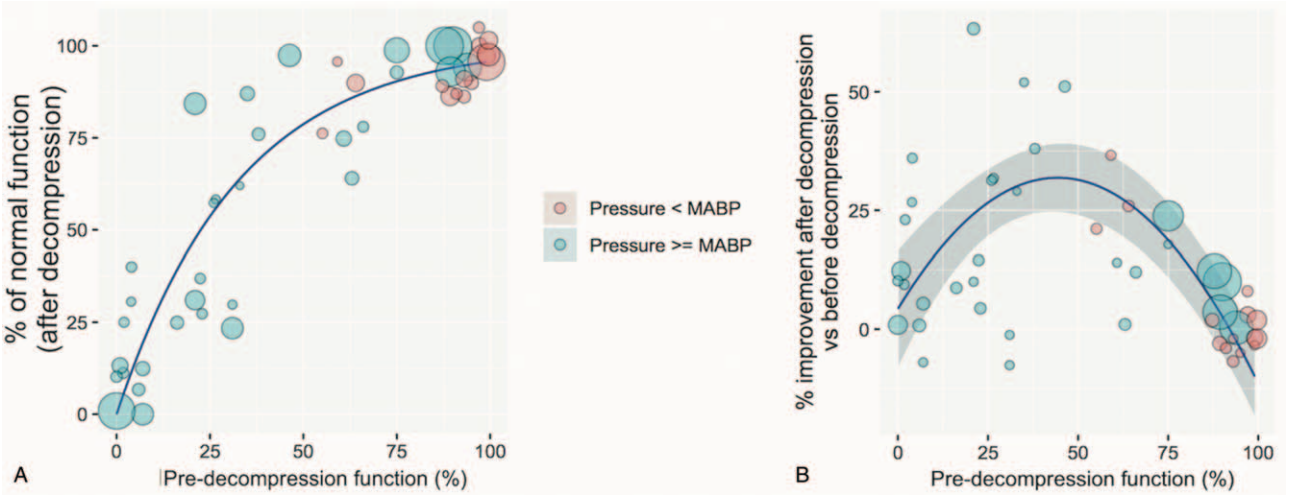

Figure 4. Models of pre-decompression function *versus* recovery, with relationship to MABP displayed. (**A**) Using absolute measure; (**B**) using mean difference, with 95% confidence intervals. MABP indicates mean arterial blood pressure.

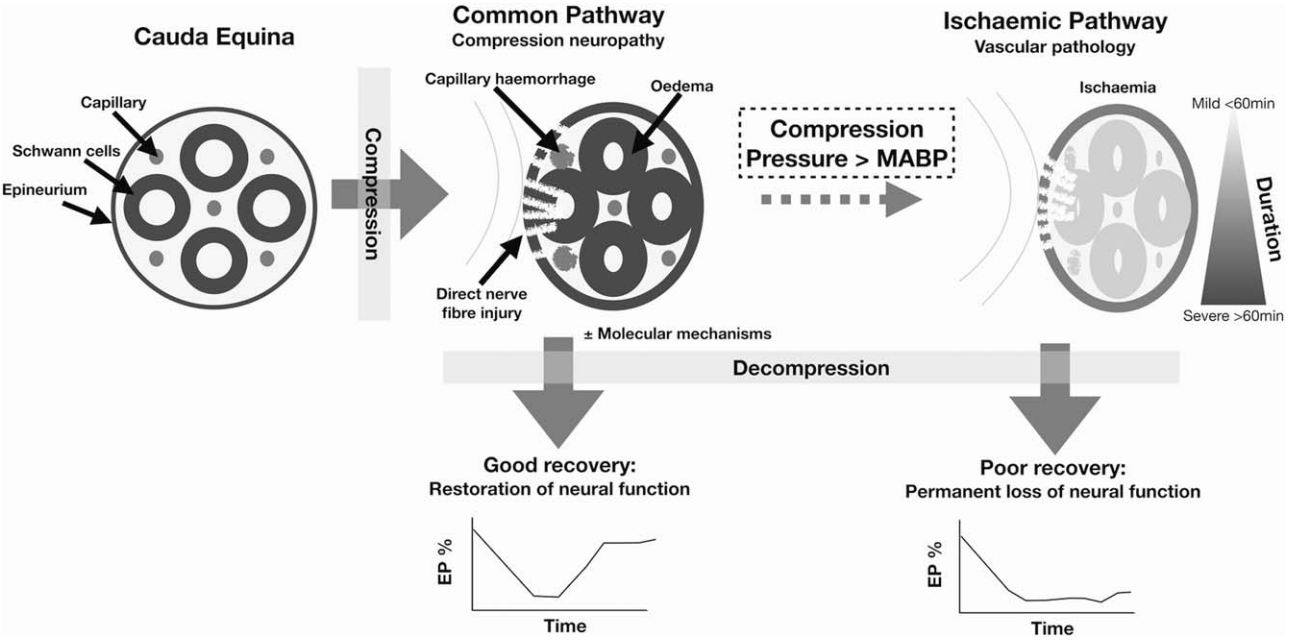

Figure 5. Schematic of proposed pathophysiology of acute cauda equina compression. EP indicates electrophysiological function; MABP, mean arterial blood pressure.

who had neurologic deficits.[34] The pressure was especially high—mean 161 mmHg, range, 104 to 256 mmHg—in patients with severe paralysis such as foot drop or bladder dysfunction. Similarly, CES symptoms occurred in patients with lumbar stenosis at epidural pressures of 116.5 ± 38.4 mmHg.[35] One study found that once the cauda equina is constricted to a certain size ($60–80\ mm^2$) then further constriction results in sharp increases of intrathecal pressure that normalize quickly until a size is reached where the pressure is sustained.[36] This potentially suggests a maximal limit of adaptation and fits with our findings above.

Decompression

Longer durations and higher pressure were both significant predictors of the degree of post-decompression EP function. The difference between pre- and post-decompression function was minimal at low (due to minor initial lesioning) and high pressures. Duration was not a significant predictor of the pre- and post-decompression difference.

Taken together, this indicates that decompression after a low pressure event has better outcomes as the decompression halts progression when little function has been lost, rather than by recovering the lost function. Decompression after a medium pressure event improves outcomes by both halting progression and also recovering the lost function. Decompression after a high pressure event has poor outcomes as much of the function has already been lost, and decompression is unable to recover the lost function. Earlier decompression improves outcomes by halting progression. Overall, it suggests that a reliably large lesion is produced above MABP, but that this can be reversible unless SBP is exceeded, which might be mostly independent of duration of compression.

This finding is similar to studies using other compression methodologies, for example, Valone *et al*[37] used forceps with 1 N or 2 N of force on a porcine lumbar root (approximately 75 and 150 mmHg assuming 1 cm^2 forceps area) and found that the higher pressure resulted in a drastically larger reduction of MEP amplitude which did not recover after 10 minutes unlike with the lower pressure.

Our model, however, did not support the idea that earlier decompression leads to greater recovery of lost function, which may be attributed to a lack of data and power at durations above 120 minutes. Pre-decompression function appeared to be a stronger predictor of prognosis after recovery than either duration or pressure repeating the finding by Chau *et al*.[6]

Relation With Neurobehavioral Function

It is difficult to correlate our models with neurobehavioral measures though they resemble those of motor function by Batchelor *et al*.[12] Studies assessing neurobehavioral outcomes in CE compression use mostly murine models and/or circumferential compression and/or long duration simulating chronic spinal stenosis, for example, Ma *et al*,[38] rather than CES where neurologic deterioration occurs rapidly.[39]

In decompression studies, two studies showed that motor function recovery after decompression occurred faster with shorter durations of CE compression,[40,41] but both used imprecise compression methods and only recorded large deficits. Recovery may also be a longer process than that measured by our study, for example in one rat study motor function normalized at 4 weeks after decompression.[42]

Pathophysiology and Proposed Integrated Model

The cauda equina's blood supply possibly results in an area of relative hypovascularity[43,44] and the microscopic

anatomy of nerve roots makes them especially sensitivity to compression.[45] The anatomy of the CE in canine[46] and porcine models[47] closely resembles a human's as does the pathology—intraradicular edema has been found in both patients and animal models with lumbar disc herniation.[48,49] Circulation disruption with consequential venous congestion has been proposed as a mechanism for neurogenic claudication in spinal stenosis[50] and in post-spinal-surgery CES in patients with pre-existing spinal stenosis.[51] Similarly, a cadaveric study of lumbar stenosis found pathological neural changes associated with venous obstruction even in the absence of direct compression.[52] Animal studies suggest that vasodilators may be neuroprotective in CE compression.[21,24]

Using graded compression, Olmarker *et al*[45] found a significant correlation between MABP and the compressive pressure required to stop flow within arterioles, but not in capillaries or venules. Balloon pressures that stopped arteriolar blood flow tended to be lower than MABP and much lower in capillaries/venules. This agrees with our results and may explain the variability between studies. Additionally, reduction in blood flow sufficient to initiate ischaemia, without cessation of flow, could result in a similar effect size at longer durations.

Decompression has been shown to completely restore circulation[33] because blood flow proximal to CE compression is not affected.[17] Our results may have underestimated the extent of recovery by measuring it at 90 minutes post-decompression and reperfusion edema may explain some variation in our models.

It may be that primary injury is caused by the disc through direct pressure, hemorrhage, and myelin sheath damage (±initiated molecular signaling pathways[23,53–56]) whereas secondary injury to the cauda equina occurs through inflammatory and edematous changes, including ischaemia if circulation is compromised. Our finding that low effect sizes still occur at high compressive pressures but low durations suggests that duration may determine the extent of ischaemia; a process similar to that in spinal cord injury.[57] Our study suggests that a greater deterioration occurs when the compression pressure disrupts vascular supply and differences in this may explain the phenotypic heterogeneity of CES. Broadly, two separate groups may result from the presence/absence of ischaemia (Figure 5).

Clinical Implications

Though measuring directly pressure is currently unfeasible in patients with CES, other techniques may be used as surrogate measures, such as diffusion tensor imaging (DTI), which in spinal stenosis and lumbar disc prolapse has identified parameters[58,59] that correlate with neurophysiological measures, functional measures, and outcomes.[60–62] To our knowledge, DTI of the CE has only been evaluated in a goat model of CE transection.[63]

Better understanding of the pathophysiology of CE compression may unveil a window period for adjuvant therapy, such as vasodilators like lipoprostaglandin E1,[64] or anti-neuroinflammatory agents like S-nitrosoglutathione and methylprednisolone.[65,66]

Limitations

The time points employed may not be applicable to human CES due to the short durations and 90 minutes recovery time but may be too early to determine maximum benefit. Furthermore, our study is not able to predict effects past 240 minutes. Though it is the first study to model the relationship with BP, few studies measured it and a constant was applied to simulate it. It also lacks neurobehavioral measurements therefore the implications for CES, which is identified through clinical features, are limited.

Conclusions

This systematic review and meta-analysis suggests that electrophysiological dysfunction in acute cauda equina compression occurs in a sigmoidal pattern with particularly deterioration when mean arterial blood pressure is exceeded and, additionally, sustained for approximately 1 hour. Accounting for pressure and duration may help risk-stratify patients prior to decompression. Outcomes after decompression appeared to be related more to the degree of compression, where exceeding systolic blood pressure tended to result in an irreversible lesion, rather than duration of compression. Prognosis was most strongly associated with residual pre-decompression function. We suggest the presence of two broad phenotypic groups within CES defined by the degree of ischaemia as a potential explanatory pathophysiological mechanism.

➢ Key Points

- ❑ Electrophysiological dysfunction in acute cauda equina compression has a sigmoidal response.
- ❑ Electrophysiological function particularly deteriorates when mean arterial blood pressure is exceeded.
- ❑ Compressive pressure has a larger effect than compression duration on electrophysiological outcomes after decompression.
- ❑ Electrophysiological outcome is most strongly associated with residual pre-decompression function.
- ❑ Neural ischaemia is suggested as an important mechanism in cauda equina syndrome pathophysiology.

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