Quantitative Sensory Testing (QST)

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PancreasFest

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Disclosures

- No conflicts of interest to declare

Pain in Chronic Pancreatitis (CP)

- Most disabling symptom
- Affects up to 90% of patients
- Stigma: alcohol, tobacco, opioids
- No curative treatment for CP as there is for other painful abdominal conditions (i.e. peptic ulcer disease, inflammatory bowel disease)
- Invasive therapy is offered to patients whose pain is thought to be due to obstructive disease (pancreatic duct stones, strictures)
- Pain response to invasive treatment is unpredictable

Pain Response Rates to Treatment

Table 1 Highlights of studies on pain treatment in chronic pancreatitis

Study	Design	N	Interventions	Follow-up	Pain relief (%)*
Endoscopy					
Rosch *et al*[12]	Cohort	1018	Endoscopy (including ESWL)	4.9 years†	65
Tadenuma *et al*[71]	Cohort	57	Endoscopy (including ESWL)	1 year	63
Dumonceau *et al*[72]	RCT	55	ESWL with/without subsequent endoscopy	2 years	55 vs 62 (p=0.651)
Conventional surgery					
Dite *et al*[61]	RCT	72	Endoscopy (without ESWL) versus surgery	5 years	61 vs 86 (p=0.002)
Cahen *et al*[13]	RCT	39	Endoscopy (including ESWL) versus surgery	2 years	32 vs 75 (p=0.007)
Cahen et al[26]	Long-term results RCT	31	Endoscopy (including ESWL) versus surgery	>6 years	38 vs 80 (p=0.042)
TPIAT					
Bellin *et al*[82]	Cohort	215	TPIAT	10 years	82
Neuropathic pain medication					
Olesen et al[67]	RCT	64	Pregabalin versus placebo	3 weeks	

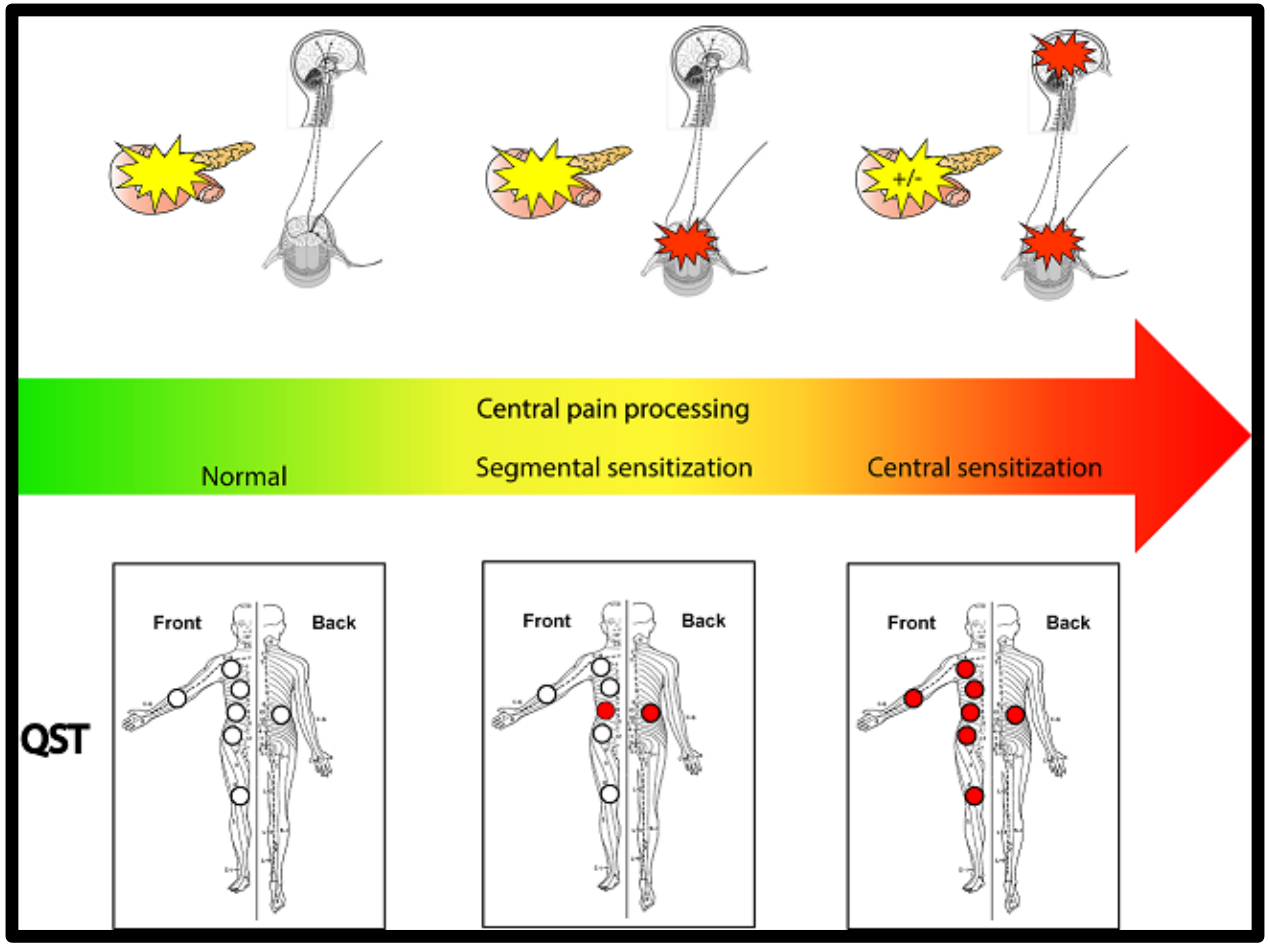
*Complete and partial pain relief combined.
†Mean.
CP, chronic pancreatitis; ESWL, extracorporeal shockwave lithotripsy; RCT, randomised controlled trial; TPIAT, total pancreatectomy with islet autotransplantation.

Drewes AM et al, Gut, 2019

Suboptimal pain response to existing therapies

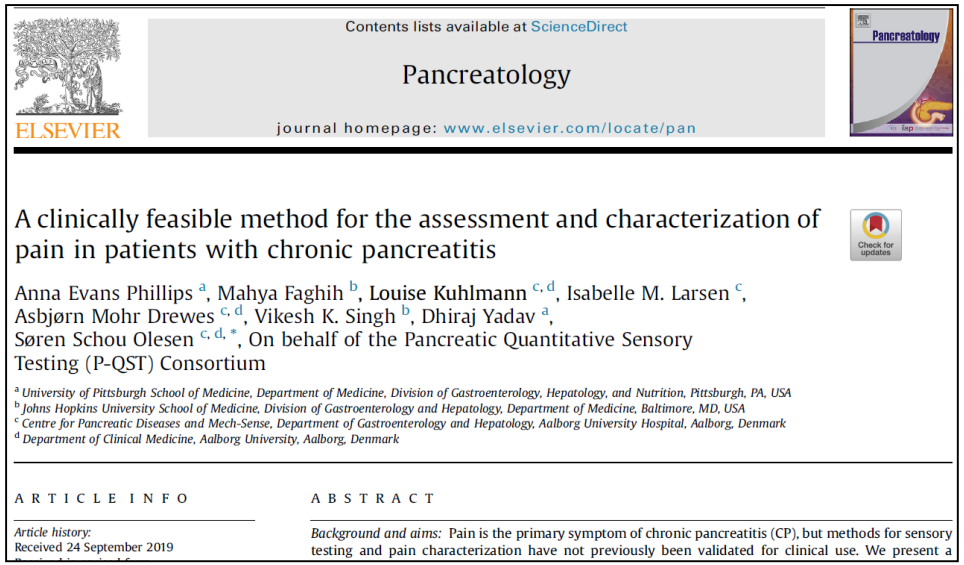
- CP pain results from multiple mechanisms
- Central sensitization: hyper-excitability in the central nervous system
  - functional reorganization of cerebral cortex
  - Neuroplastic and neuropathic changes occur in response to persistent visceral and somatic pain stimuli
  - Incomplete pain response to local CP therapies is thought to be at least partially due to central sensitization

Nociceptive Continuum in Painful CP



Phillips et al, Pancreatology, 2020

Pancreatic QST (P-QST)



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A clinically feasible method for the assessment and characterization of pain in patients with chronic pancreatitis

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ABSTRACT

Background and aims: Pain is the primary symptom of chronic pancreatitis (CP), but methods for sensory testing and pain characterization have not previously been validated for clinical use. We present a

- New and simplified testing protocol
- Tailored specifically for use in patients with CP
- Utilizes convergence of visceral nerves and somatic nerves in the spinal cord root
- Facilitates surface testing of the pancreatic viscerotome (T10)

P-QST Technique

Temporal Summation

Pin-Prick Stimulator

Pressure Pain Detection

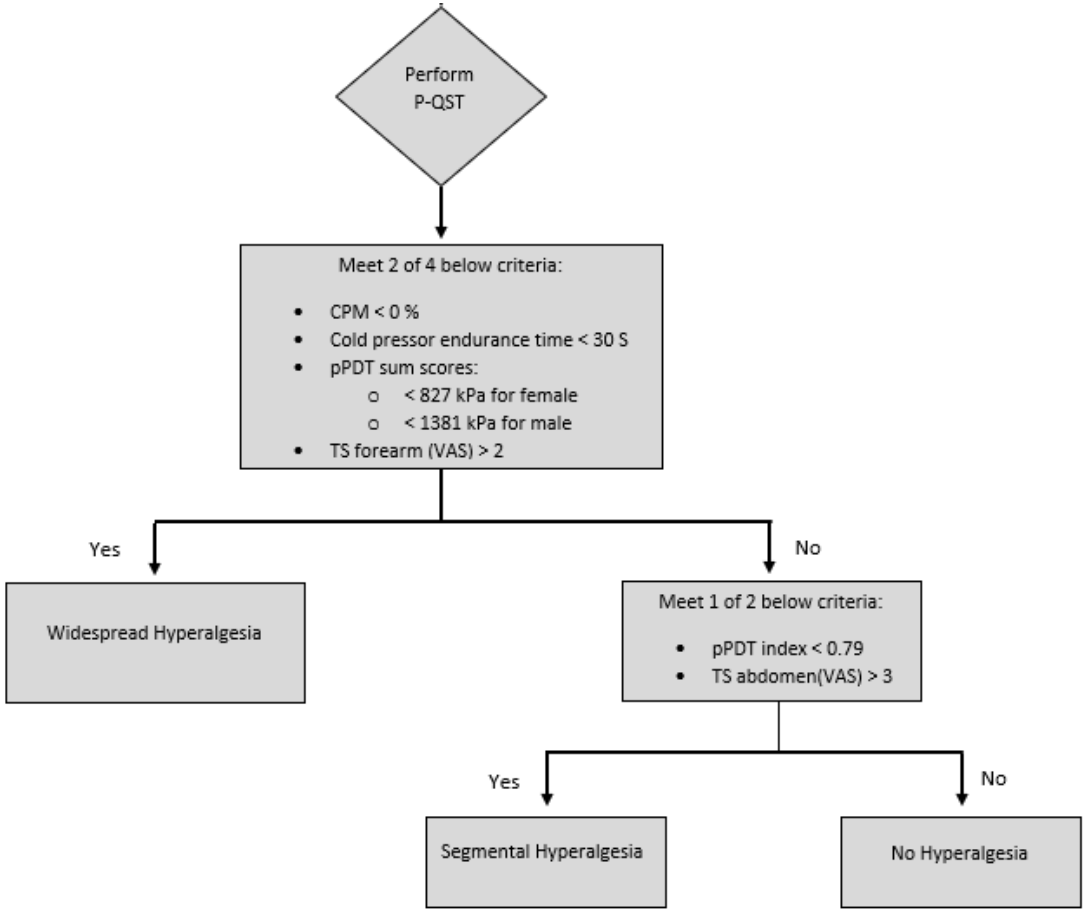
Pressure Algometer

Conditioned Pain Modulation

Ice Water Bath

https://youtu.be/seO3QSrRiuo

P-QST Testing Algorithm



Faghih et al, Clin Gastro Hep, 2021

P-QST Phenotype Distributions

- Higher percentage of patients with widespread hyperalgesia in patients with current pain
- Constant pain is more prevalent in patients with segmental or widespread hyperalgesia

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PANCREAS, BILIARY TRACT, AND LIVER

Pancreatic QST Differentiates Chronic Pancreatitis Patients into Distinct Pain Phenotypes Independent of Psychiatric Comorbidities

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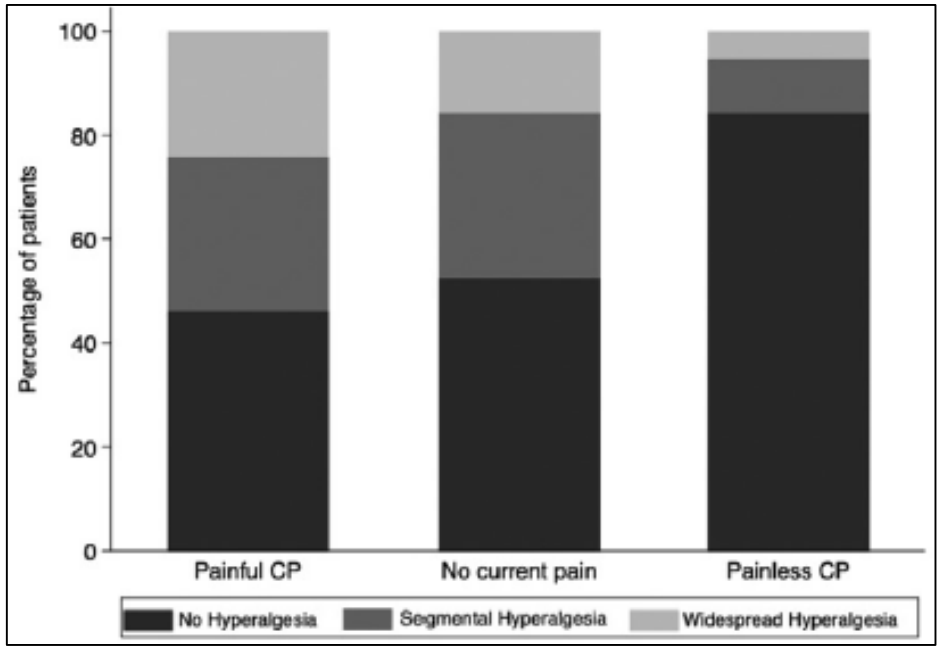
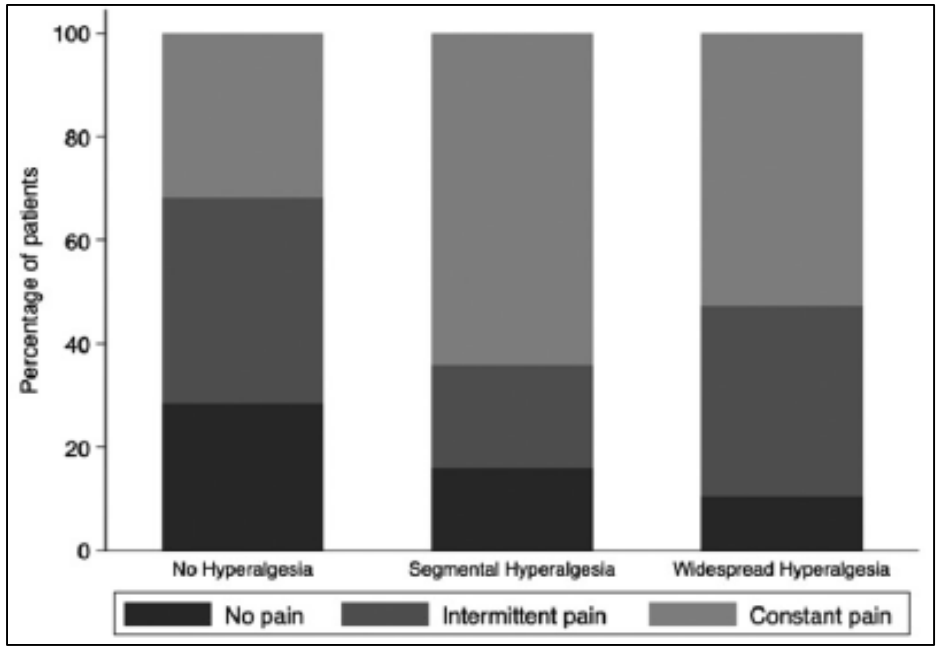




Faghih et al, Clin Gastro Hep, 2021

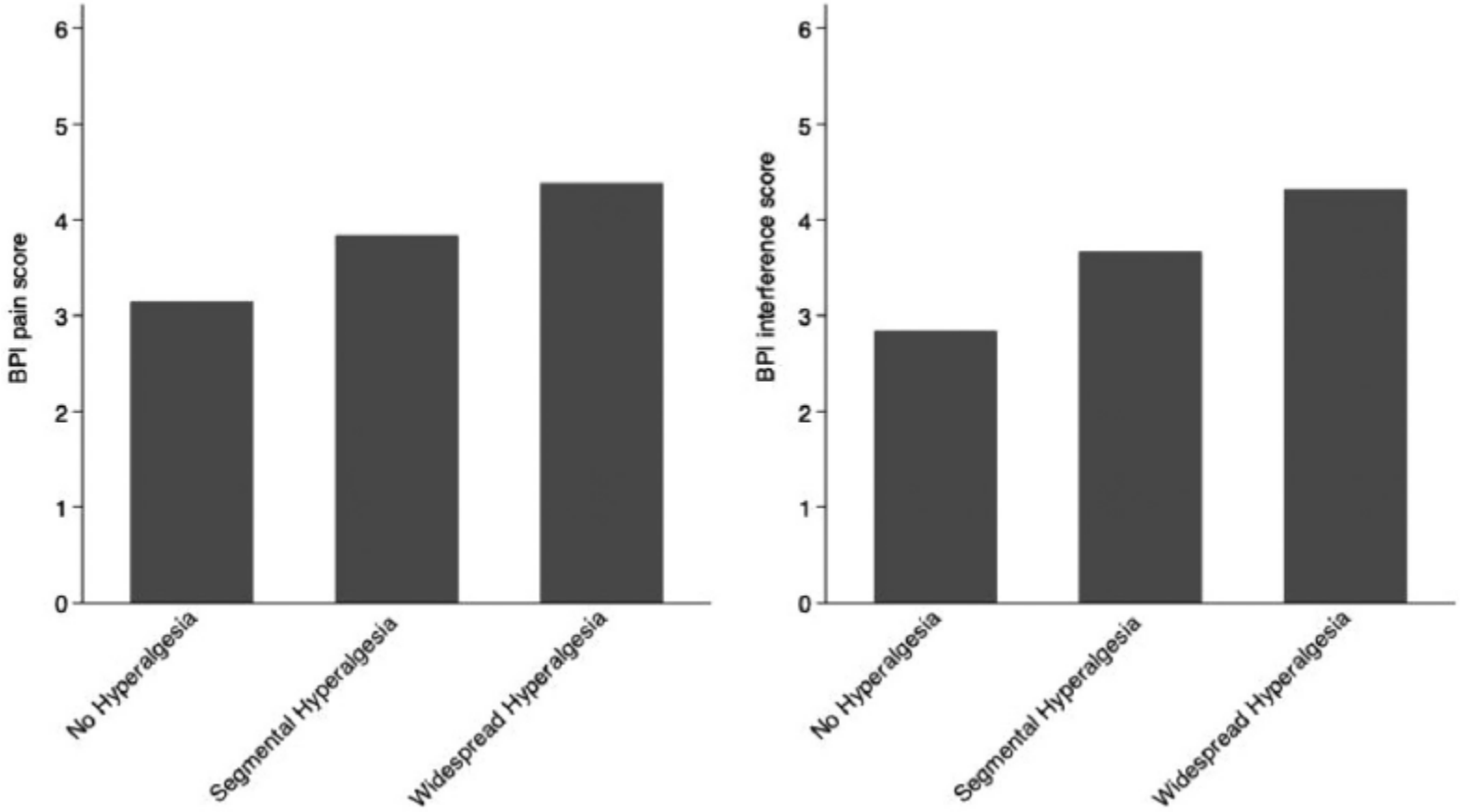
Independent of Psychiatric Comorbidity

Table 2. Comparisons Between QST Phenotypes, Clinical Pain Characteristics, and Psychiatric Assessment Parameters

	All patients (n = 179)	No hyperalgesia (n = 91)	Segmental hyperalgesia (n = 50)	Widespread hyperalgesia (n = 38)	*P* value
Demographic and clinical characteristics					
Gender, n (%)					.88
Women	73 (41)	36 (40)	22 (44)	15 (40)	
Men	106 (59)	55 (60)	28 (56)	23 (61)	
Mean age, *y*	54.1 ± 13.6	53.9 ± 14.7	53.0 ± 13.3	55.9 ± 12.0	.60
Diabetes, n (%)	67 (39)	32 (36)	22 (45)	13 (36)	.59
Alcohol etiology, n (%)	75 (42)	31 (34)	26 (52)	18 (47)	.09
Clinical pain characteristics					
Pain pattern, n (%)					.002
No current pain	38 (21)	26 (29)	8 (16)	4 (11)	
Intermittent pain	60 (34)	36 (40)	10 (20)	14 (37)	
Constant pain	81 (45)	29 (32)	32 (64)	20 (53)	
Pain severity					
BPI pain score	3.6 ± 2.5	3.1 ± 2.6	3.8 ± 2.7	4.4 ± 1.9	.03
BPI interference score	3.4 ± 2.9	2.8 ± 2.8	3.7 ± 3.0	4.3 ± 2.6	.02
Analgesic use, n (%)					
Opioids	87 (49)	35 (38)	31 (62)	21 (55)	.02
Adjuvant analgesics	77 (43)	37 (41)	25 (50)	15 (39)	.53
Psychiatric variables					
Pain catastrophizing score					
Conditional	19.5 ± 13.6	18.0 ± 13.8	20.2 ± 13.8	22.2 ± 12.5	.31
Situational	22.5 ± 13.4	21.2 ± 13.2	23.1 ± 13.9	25.0 ± 13.1	.34
HADS, n (%)[a]					
Depression	67 (38)	27 (31)	21 (42)	19 (50)	.11
Anxiety	80 (46)	36 (41)	23 (46)	21 (55)	.36
HADS, n (%)[b]					
Depression	28 (16)	11 (13)	9 (18)	8 (21)	.43
Anxiety	47 (27)	20 (23)	13 (26)	14 (37)	.27

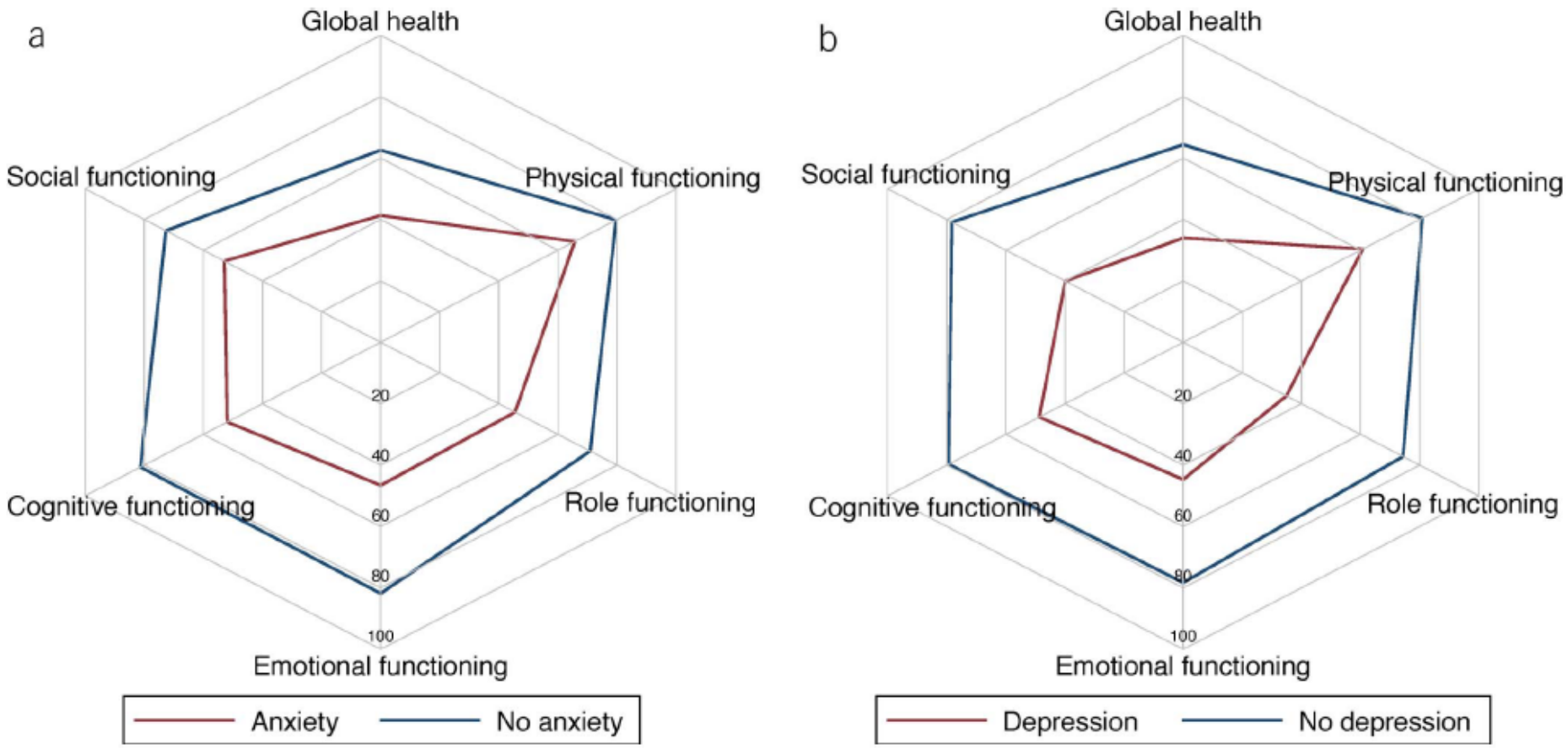
Faghih et al, Clin Gastro Hep, 2021

Higher Pain Intensity and Interference with Hyperalgesia



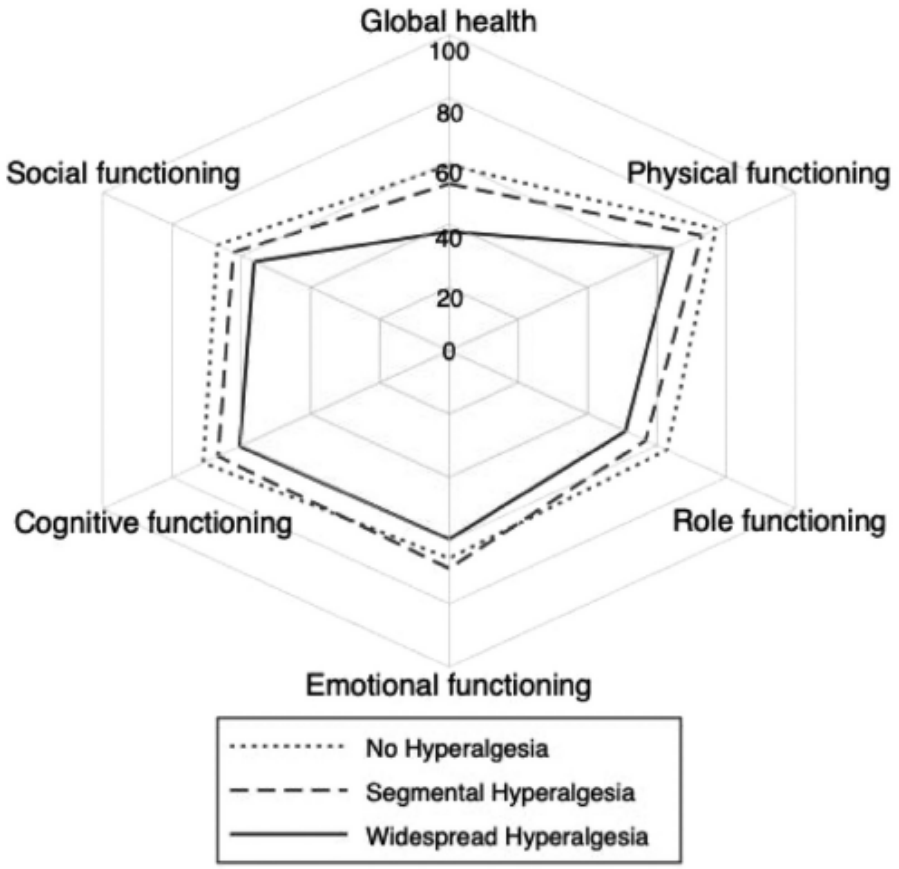
Faghih et al, Clin Gastro Hep, 2021

Lower QOL in patients with CP and Psychiatric Comorbidity

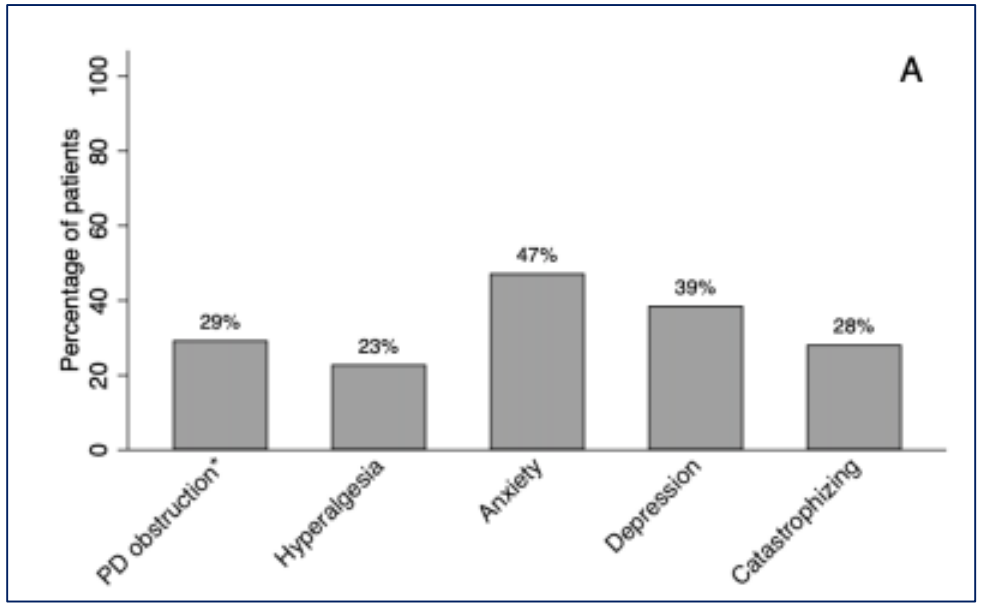


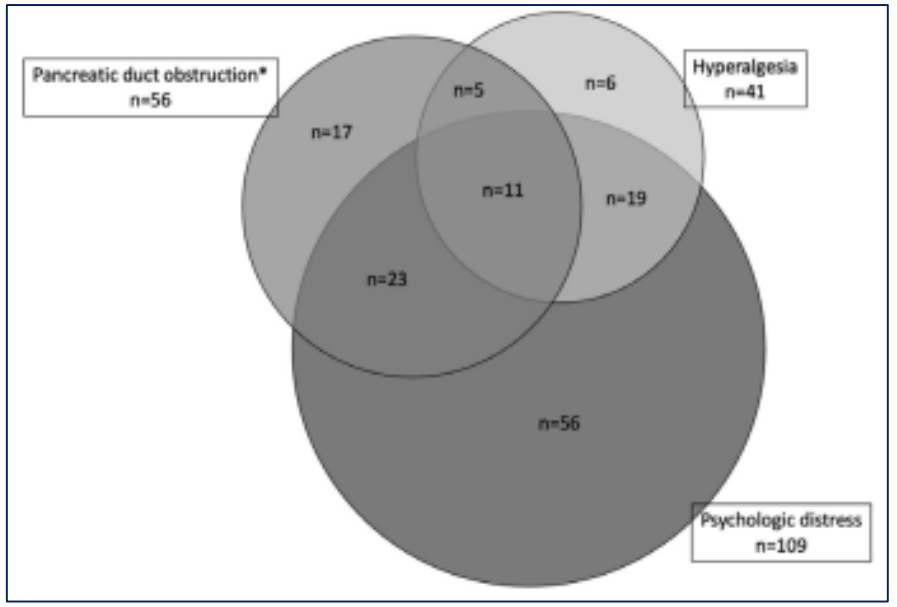
Phillips et al, Am J Gastro, 2020

Also lower QOL in patients with widespread hyperalgesia



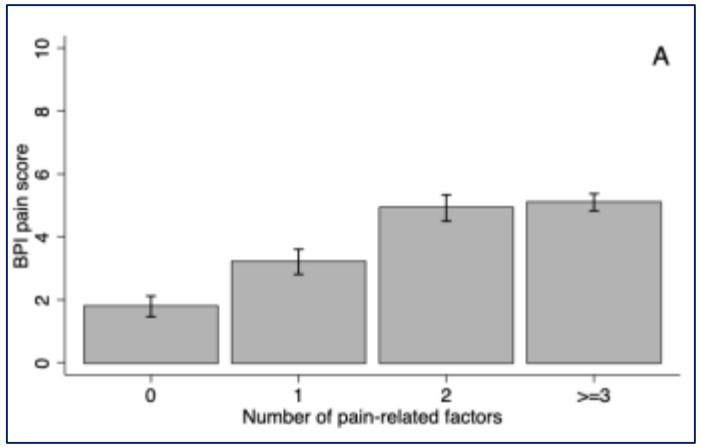
Overlapping Factors in CP

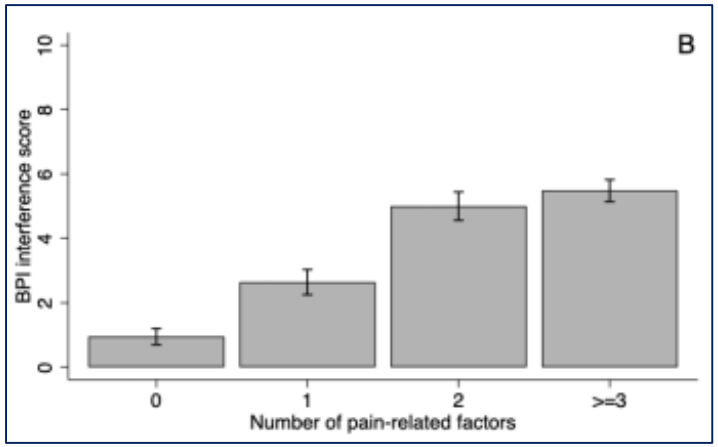


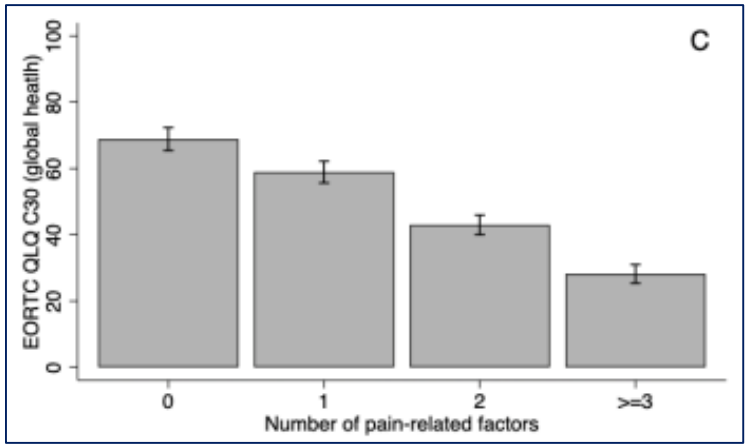


Olesen et al, Gut, 2021

Cumulative Effect of Multiple Overlapping Factors







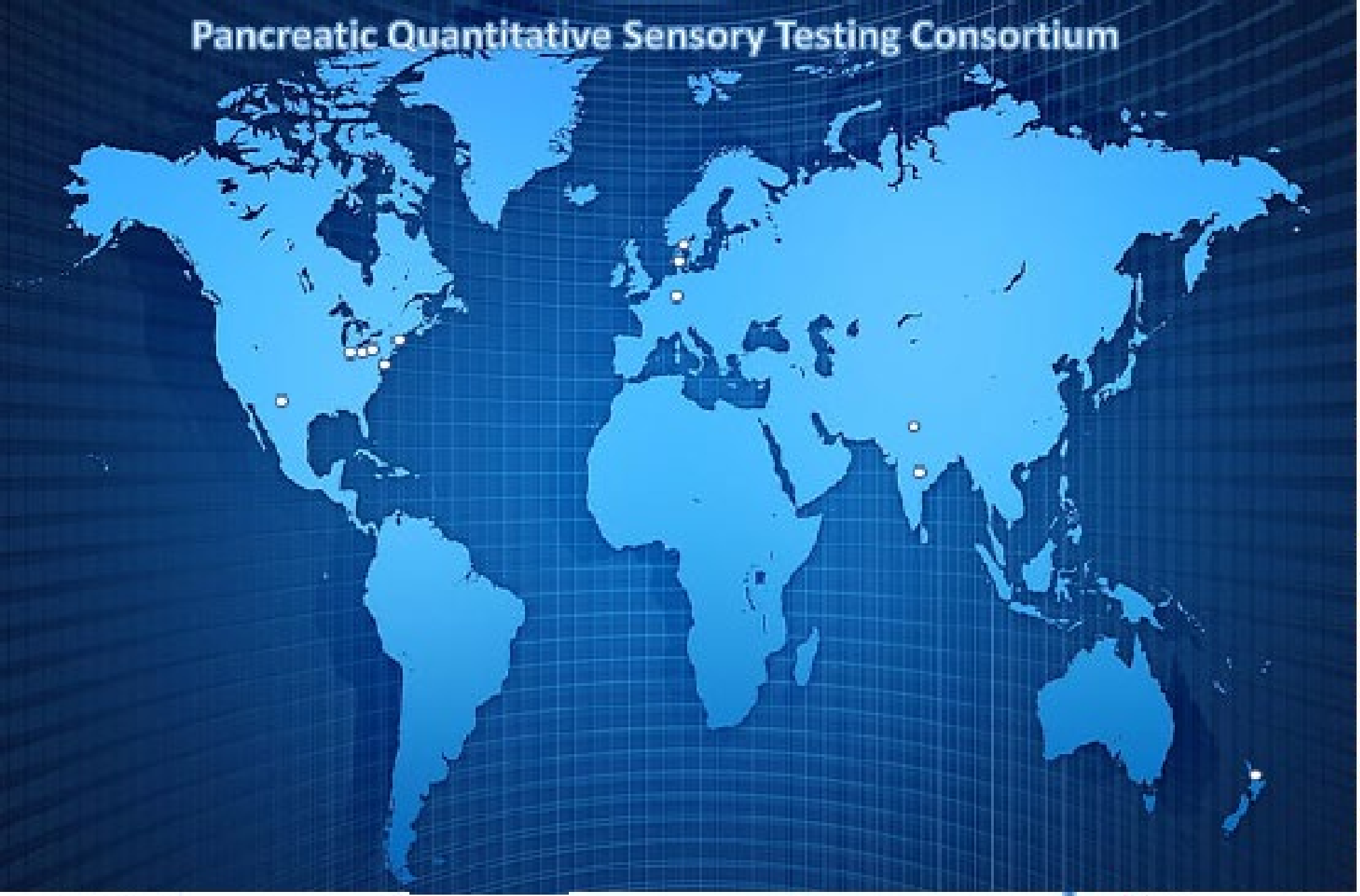
- Linear increase in pain severity and pain interference, and linear decrease in QOL with accumulation of pain-related factors

Olesen et al, Gut, 2021

Conclusions

- P-QST characterizes the sensory profiles independently of psychological status (anxiety, depression, catastrophizing)
- Proxy of pain processing or nociception
- P-QST as phenotyping tool for presence of hyperalgesia has the ability to contribute another dimension to pain assessments in CP
- Potential as predictor of outcome to therapy

Pancreatic Quantitative Sensory Testing Consortium



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