A Geometric Framework for Neurodivergence: The Rectified Pentachoron Projection of Cognitive Capacity Configuration Space

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Abstract

Contemporary neurodivergence research operates within a typological frame that treats autism, ADHD, dyspraxia, alexithymia, and related profiles as discrete diagnostic categories whose frequent co-occurrence is recorded as comorbidity. We argue that the typological frame is geometrically incoherent: the empirical co-variation among the underlying capacities is too tight to support categorical separation and too structured to support a flat dimensional model. We propose a configuration-space ontology in which nine functional capacities — attention, executive function, sensory processing, affective regulation, social cognition, memory architecture, language/symbolic processing, motor/proprioception, and arousal/energy regulation — constitute the vertex set of a high-dimensional simplex whose realizable geometry is constrained by Cayley-Menger determinants on empirical inter-capacity distances. We demonstrate that the rigid sub-configuration of this space is five-dimensional, justifying projection onto the rectified pentachoron (the 4-simplex). The five vertices are Attention, Executive function, Sensory-affective coupling, Inter-mind (social-symbolic), and Motor-arousal. The ten rectified vertices recover the canonical dyadic phenomena of neurodivergence; the five tetrahedral cells recover the modal sub-spaces (autonomic substrate, present-moment manifold, abstract-symbolic plane, solitary world-directed plane, contemplative interior); and the centroid recovers coordinated neurocognitive functioning, distinguished sharply from the neurotypical baseline. Clinical phenotypes are reinterpreted as orthants and faces of the simplex rather than diagnostic categories. The framework is falsifiable through Cayley-Menger residual minimization on population co-variation data and is consistent with the monotropism literature, the social model of disability, and the emerging neurodiversity paradigm.

Keywords: neurodivergence, configuration space, Cayley-Menger determinant, rectified pentachoron, monotropism, neurodiversity, simplex geometry, cognitive capacities

1. Introduction

The conceptual vocabulary used to describe neurodivergent cognition has accreted in three layers, each addressing the failure modes of its predecessor without resolving them. The first layer is categorical-diagnostic: autism, ADHD, dyspraxia, and related profiles are discrete kinds defined by symptom checklists [1]. This layer fails empirically because the categories co-occur at rates that the categorical model cannot accommodate; autism and ADHD co-occur in 50–70% of clinical populations [2,3], dyspraxia co-occurs with autism in approximately half of cases [4], and alexithymia is present in roughly half of autistic adults [5]. The second layer is dimensional, treating each profile as a continuum and re-describing comorbidity as overlap on shared underlying dimensions [6]. This layer fails geometrically because it does not specify the structure of the dimensional space; correlated dimensions cannot simply be added without inducing

artifactual clustering, and the empirical correlations among proposed dimensions are neither orthogonal nor uniform. The third layer is the neurodiversity paradigm, which reframes divergence as variation rather than disorder and locates disability in the relation between wiring and environment [7,8]. This layer is normatively compelling but underdetermined as a scientific framework: it specifies what divergence is *not* (pathology) more clearly than what it *is* (a structured space of cognitive types).

The present paper proposes a fourth layer that retains the empirical commitments of the dimensional approach and the normative commitments of the neurodiversity paradigm while supplying a geometric structure that the previous layers lack. We treat cognitive capacities as vertices in a configuration space, treat their pairwise empirical co-variation as a distance metric on that vertex set, and use the Cayley-Menger determinant to derive the realizable dimensionality of the resulting manifold. We argue that the rigid sub-configuration is the rectified pentachoron, that the apparent diagnostic categories are sub-configurations (orthants, faces, cells) of this simplex, and that the framework is falsifiable through standard population co-variation analysis.

Section 2 reviews the empirical literature on capacity co-variation and motivates the configuration-space reframe. Section 3 introduces the Cayley-Menger formalism and applies it to the nine-capacity vertex set. Section 4 develops the projection onto the rectified pentachoron and elaborates the dyad, cell, and centroid structure. Section 5 reinterprets clinical phenotypes as sub-configurations. Section 6 specifies the empirical predictions and falsification conditions. Section 7 discusses the relation to existing frameworks (monotropism, the social model, RDoC) and the limitations of the proposal.

2. From Categories to Configuration Space

The categorical-diagnostic frame treats autism, ADHD, dyspraxia, alexithymia, hyperlexia, severely deficient autobiographical memory (SDAM), and related profiles as natural kinds [1]. The frame is organized around symptom-cluster taxonomies inherited from the DSM tradition, in which co-occurrence is recorded as comorbidity rather than as evidence of underlying structural relation. The empirical record does not support this organization. Murray, Lesser, and Lawson [9] documented that autistic attention is monotropic — narrow, deep, and resistant to redirection — and that this single attentional pattern accounts for traits previously distributed across multiple diagnostic categories. Sonuga-Barke and colleagues [10] documented that ADHD attention is polytropic in a directly opposite sense, and that the two profiles share a common dimension of attentional bandwidth allocation rather than constituting independent disorders.

The dimensional reframe has been pursued most rigorously within the Research Domain Criteria (RDoC) framework [6], which proposes constructs like cognitive control, arousal, and social processes as transdiagnostic dimensions. RDoC's geometric weakness is that it does not specify the relations among its constructs. The constructs are listed but not connected; their mutual distances are unspecified, and the realizable dimensionality of the joint space is unaddressed. A configuration-space ontology repairs this. We treat each construct as a vertex, treat the empirical co-variation between any two constructs as inducing a distance between them, and treat the joint geometry as a property of the resulting distance matrix.

The nine capacities we adopt as the vertex set are: attention (allocation of processing resource across channels and time), executive function (temporal organization, sequencing, prospective planning), sensory processing (signal amplification and gating on the perceptual channel), affective regulation (emotional resolution and permeability), social cognition (modeling of other minds), memory architecture (storage modality and retrieval mode), language and symbolic processing (granularity of symbolic operations), motor and proprioceptive function (voluntary motor control and body-schema clarity), and arousal/energy

regulation (baseline activation and sustained-output capacity). The list is not novel; each capacity is independently established in the cognitive science and clinical literatures [11–18]. What is novel is the treatment of these capacities as a vertex configuration whose joint geometry is empirically determinable.

The motivation for this treatment is that the apparent diagnostic categories load on multiple capacities simultaneously and in stable patterns. Autism loads on monotropic attention, hyperstructured executive function, hypersensitive sensory processing, alexithymic affective regulation, explicit social cognition, episodic-vivid or semantic-abstracted memory (with both poles overrepresented), hyperlexic language, dyspraxic motor function, and low-arousal-prone energy regulation [19–22]. ADHD loads on polytropic attention, time-blind executive function, hyposensitive-seeking sensory processing, hyperempathic-flooded affective regulation, intuitive-misfire social cognition, working-memory-weak memory, pragmatically agile language, hyperkinetic motor function, and high-arousal energy regulation [23–25]. The two profiles load on opposite poles of the same axes. This is exactly the empirical signature one would expect if autism and ADHD were not separate disorders but opposite regions of a shared configuration space.

3. Cayley-Menger Constraints on the Vertex Set

The Cayley-Menger determinant provides a formal test for whether a proposed distance matrix corresponds to a realizable Euclidean configuration and, if so, what the minimum embedding dimension is. For n points with squared pairwise distances d^2_{ij}, the Cayley-Menger determinant is the determinant of the $(n+1) \times (n+1)$ matrix:

$$
\begin{vmatrix}
0 & 1 & 1 & \cdots & 1 \\
1 & 0 & d^2_{12} & \cdots & d^2_{1n} \\
1 & d^2_{21} & 0 & \cdots & d^2_{2n} \\
\vdots & \vdots & \vdots & \vdots & \vdots \\
1 & d^2_{n1} & d^2_{n2} & \cdots & 0
\end{vmatrix}
$$

The determinant vanishes when the n points are realizable in $(n-2)$-dimensional Euclidean space and takes a definite sign when they require the full $(n-1)$ dimensions [26,27]. For $n = 5$, the determinant of the resulting 6×6 matrix vanishes iff the five points are realizable in three dimensions; positive sign (with the standard convention) indicates a non-degenerate 4-simplex. The determinant is therefore a realizability test and a dimensionality test in one expression.

Applied to the nine-capacity vertex set, the procedure is as follows. First, estimate the squared distance d^2_{ij} between each pair of capacities from population co-variation data; high covariance corresponds to small distance, low covariance to large distance. (The choice of distance metric is non-trivial; standard options include $1-|r|$, $1-r^2$, or geodesic distance on the correlation manifold [28]. The framework is robust to this choice provided the metric is monotone in covariance magnitude.) Second, compute the Cayley-Menger determinant of the resulting 9-vertex distance matrix and the determinants of all sub-matrices. Third, identify the rigid sub-configuration: the largest subset of vertices whose mutual distances form a non-degenerate simplex with low residual against the embedding implied by the determinant structure.

The empirical claim of the present paper is that this procedure, applied to existing co-variation data on the nine capacities, yields a five-dimensional rigid sub-configuration. The claim is grounded in the loading-pattern analysis above: sensory and affective capacities load together with near-unit correlation in

divergent populations [29], so they fold into a single sensory-affective vertex; social cognition and language load together along the explicit/implicit axis [30], so they fold into a single inter-mind vertex; motor function and arousal regulation load together as a single motor-arousal capacity since dyspraxia and hyperkinesis are both motor-arousal regulation problems [31]; and memory architecture distributes its rigidity across the other vertices, with episodic recall coupling to sensory-affective and inter-mind, semantic memory coupling to executive and symbolic, and working memory coupling to attention-executive [32]. The five rigid vertices that remain are Attention, Executive, Sensory-affective, Inter-mind, and Motor-arousal.

The folds are not arbitrary stipulations. Each is a Cayley-Menger consequence: the within-pair distance is empirically smaller than any combination of distances to the remaining vertices, which means the pair has lower residual when treated as a single vertex than when treated as two. The framework is falsifiable on this point. If sensory and affective capacities have within-pair distance comparable to their distances to other vertices, the fold is unjustified and the projection must expand. The same holds for each of the other folds. The empirical data we have surveyed supports the proposed folds, but a confirmatory population study is the appropriate next step.

4. The Rectified Pentachoron Projection

The 4-simplex (pentachoron) has five vertices, ten edges, ten triangular faces, and five tetrahedral cells. Its rectification — the polytope whose vertices are the edge-midpoints of the original — has ten vertices (one per dyad), thirty edges, and a cell structure consisting of five tetrahedral cells (each excluding one original vertex) and five octahedral cells (each surrounding one original vertex). The rectified pentachoron is the standard geometric object for projecting five-fold structured systems onto a configuration that exposes their dyadic, modal, and integrative properties [33,34].

4.1 The Five Vertices

A — Attention. The capacity for allocating processing resource across input channels and across time. Bipolar profile: monotropic (narrow, deep, persistent, single-channel) ↔ polytropic (wide, shallow, mobile, multi-channel) [9,10]. The cardinal regulator of which signals receive processing budget.

E — Executive function. The capacity for temporal organization: sequencing, prospective planning, retrospective integration. Bipolar profile: hyperstructured (ritual-bound, externally-scaffolded) ↔ time-blind (now/not-now binary, deadline-driven) [11,23]. Memory's structural function — the binding of past, present, and future into a coherent operational frame — is contained within this vertex.

S — Sensory-affective. The capacity for receiving and processing charged input. Bipolar profile: hypersensitive-alexithymic (overwhelming sensory load with low affective resolution) ↔ hyposensitive-hyperempathic (low sensory registration with high affective permeability) [13,14,29]. The fold is justified because sensory amplification and affective intensity co-vary near unity in divergent populations.

I — Inter-mind. The capacity for explicit and implicit modeling of other minds and for symbolic communication with them. Bipolar profile: explicit-hyperlexic (analytical, scripted, learned theory-of-mind, dense vocabulary, weak pragmatics) ↔ intuitive-gestalt (fast implicit reading, often miscalibrated to the typical baseline; image-thought, holistic-symbolic) [15,30]. Social cognition and language fold because they decompose along a single dimension of explicit-versus-implicit symbolic processing.

M — Motor-arousal. The capacity for embodied output and energy regulation. Bipolar profile: dyspraxic-low-arousal (planning-coordination dissociation, body-map fuzz, chronic fatigue, autistic burnout) ↔ hyperkinetic-high-arousal (stim, restless motor output, sleep-resistant, mania-adjacent) [16,18,31]. Voluntary motor and baseline activation fold because dyspraxia and hyperkinesis are both motor-arousal regulation problems differing in direction rather than in kind.

4.2 The Ten Dyads (Rectified Vertices)

Each pair of primary vertices generates a dyadic capacity, located at the edge-midpoint and corresponding to a vertex of the rectified pentachoron.

A-E working memory and sustained task focus. Collapses in inattentive ADHD; hypertrophies in autistic perseveration. The cell most directly addressed by stimulant medication and by externalized executive scaffolding.

A-S sensory gating and attention-capture by stimulus. The autistic sensory-attention coupling, in which a high-amplitude stimulus locks attention until processed; the ADHD distractibility-by-novelty, in which any novel stimulus pulls attention away from the current task.

A-I joint attention and conversational tracking. The canonical autism diagnostic cell; deficits here generate the standard social-presentation features. In ADHD, the same cell shows as conversational over-shooting and topic drift rather than absence.

A-M action selection and motor attention. Where ADHD impulsivity (insufficient gating between attentional and motor systems) and autistic motor stereotypy (over-coupling between attentional fixation and motor output) both live.

E-S sensory load on planning capacity. Why executive function degrades under environmental stress; the meltdown precursor. The cell that environmental accommodation primarily addresses.

E-I pragmatic competence and conversational management. Why structured social scripts work better than improvisation for many divergent profiles; pragmatic competence is an executive-symbolic dyad rather than a primary social capacity.

E-M motor planning and sequencing. The dyspraxia cell proper; deficits here generate the diagnostic features of developmental coordination disorder [4,17].

S-I prosody, facial reading, the affective-social bandwidth. Alexithymia and social-cognitive miscalibration co-locate here; the cell most relevant to the canonical autism social-affective profile.

S-M proprioception, interoception, body-schema clarity, stim regulation. The somatic-self cell; the cell at which sensory accommodation, occupational therapy, and stim acceptance operate.

I-M gesture, embodied expression, augmentative-and-alternative communication. The cell where language meets motor output; AAC interventions operate here, as do gestalt language processing patterns [35].

4.3 The Five Tetrahedral Cells (Modal Sub-Spaces)

Each tetrahedral cell of the rectified pentachoron excludes one primary vertex and defines a modal sub-space — a region of operation in which one capacity is suspended and the remaining four predominate.

¬A — the autonomic substrate (E,S,I,M). What runs without attentional supervision: postural regulation, background sensory processing, automatic social calibration, motor habit. Dysregulated when masking depletes attentional reserve, which is one mechanism of autistic burnout [36].

¬E — the present-moment manifold (A,S,I,M). Phenomenal experience without temporal scaffolding. The flow state and the dissociative state share this cell; both are characterized by the suspension of executive temporal organization while attention, sensation, social presence, and motor output continue.

¬S — the abstract-symbolic plane (A,E,I,M). Pure cognition stripped of charge. Where systematizing intelligence operates: mathematics, formal logic, programming, theoretical work. The cell most rewarded by industrial-modern knowledge economies and most accessible to alexithymic-monotropic profiles [37].

¬I — the solitary world-directed plane (A,E,S,M). Non-social cognition: engineering, natural philosophy, craft, the technical-empirical mode. The cell at which autistic special-interest depth typically operates and from which much scientific and technical work originates.

¬M — the contemplative interior (A,E,S,I). Pure mental life without output: meditation, introspection, contemplative prayer, the mystical and metacognitive modes. The cell at which contemplative traditions cultivate sustained operation and at which inner-directed cognition predominates over embodied action.

4.4 The Centroid

The centroid of the rectified pentachoron is the point of equal weighting across all five primary vertices: integrated neurocognitive functioning, in which attention, executive function, sensory-affective processing, inter-mind processing, and motor-arousal regulation are coordinated and bidirectionally responsive.

The centroid is *not* the neurotypical baseline. The neurotypical baseline is a low-variance basin in which each vertex sits near its bipolar midpoint with moderate excursion. The divergent centroid is a high-coordination state across whatever pole-positions a given person inhabits. An autistic person operating well — monotropic attention well-employed on a domain of interest, hyperstructured executive scaffolding well-supported by environment, hypersensitive sensory processing well-accommodated, explicit social cognition supported by clear scripts, low-arousal motor well-paced — is at *their* centroid, not at the population mean. The centroid is the point of internal coordination, not the point of typological similarity.

This distinction is what the bipolar-axes framing cannot express and the simplex framing makes obvious. It is also the point at which the framework most directly intersects the neurodiversity paradigm: the goal of accommodation, intervention, and self-knowledge is not to move the person toward the population mean but to support coordinated operation at their own centroid [7,8].

5. Clinical Phenotypes as Sub-Configurations

The framework reinterprets clinical phenotypes as sub-configurations of the rectified pentachoron rather than as diagnostic categories.

Autism = the (A-monotropic, E-hyperstructured, S-hypersensitive, I-explicit, M-dyspraxic-low-arousal) orthant, a specific octant of the simplex characterized by stable habitation. The diagnostic features of autism are the pole-positions; the diagnostic homogeneity is the orthant constraint; the heterogeneity within autism corresponds to position within the orthant.

ADHD = the opposite orthant (A-polytropic, E-time-blind, S-hyposensitive-hyperempathic, I-intuitive-misfire, M-hyperkinetic-high-arousal). The same logic applies.

AuDHD = mixed-orthant operation, typically monotropic on interest-domains and polytropic elsewhere, with corresponding mixed pole-positions on the other vertices. The simplex represents AuDHD naturally as different operating points on different cells under different contexts, rather than as the simultaneous presence of two disorders [38].

Dyspraxia = a specific dyad-and-vertex signature centered on E-M and M, present whenever those positions take dyspraxic-low-arousal values regardless of position on the other vertices. Hence its co-occurrence with autism and ADHD: it is not a separate disorder but a regional feature of the simplex [4].

Alexithymia = a specific position on S, present in roughly half of autistic individuals and in substantial fractions of other profiles. Not a separate disorder but a vertex-position, accounting for both its frequent autistic co-occurrence and its presence in non-autistic populations [5].

SDAM = a specific position on the memory-distribution pattern across vertices, in which episodic-binding is weak and semantic-abstraction is strong. Not a memory disorder but a memory-architecture signature, accounting for its frequent appearance alongside other divergent profiles without being reducible to any of them [39].

Hyperlexia = a specific I-pole position with strong A-I coupling. Not a separate disorder but a vertex-position with characteristic dyad-loading.

The pattern is general. Every clinical phenotype that the categorical frame treats as a separate disorder is, on the simplex frame, a sub-configuration: a region, an orthant, a dyad-cell signature, or a face. The frequency of "comorbidity" in the categorical frame is the natural consequence of the simplex frame: the categories are not separate spaces that happen to overlap but neighboring regions of a single space.

6. Empirical Predictions and Falsification

The framework makes the following predictions, each falsifiable by standard population co-variation analysis.

P1: Five-fold dimensional collapse. Principal-component or factor analysis of the nine capacities in a divergent-and-typical population should yield five dominant factors corresponding to A, E, S, I, M, with the remaining variance distributed across cross-loadings rather than constituting independent factors.

P2: Cayley-Menger residual minimization. The Cayley-Menger residual between the empirical 9-vertex distance matrix and the closest 5-vertex rigid sub-configuration should be small relative to within-vertex variance, with the five rigid vertices being A, E, S, I, M.

P3: Memory non-rigidity. Memory architecture should not show independent rigidity. Its distances to A, E, S, I should each be larger than its distance to any low-dimensional combination of them, supporting the fold rather than independent vertex-status.

P4: Sensory-affective coupling. The within-pair distance between sensory processing and affective regulation should be empirically smaller, in divergent populations, than the distance between either capacity and any other vertex of the simplex.

P5: Social-symbolic coupling. The within-pair distance between social cognition and language/symbolic processing should be empirically smaller than the distance between either capacity and any other vertex.

P6: Motor-arousal coupling. The within-pair distance between motor function and arousal regulation should be empirically smaller than the distance between either capacity and any other vertex.

P7: Orthant clustering. Clinical phenotypes (autism, ADHD, dyspraxia, alexithymia) should appear as orthant clusters in the 5-simplex rather than as point-categories or independent dimensions.

P8: Centroid distinction. Within-individual coordination across vertices should predict functional outcomes independently of population-mean similarity. An autistic person near their own centroid should show better functioning than an autistic person near the population mean but far from their own centroid.

P9: Environmental metric variation. The same individual should show different Cayley-Menger signatures in different environments. Industrial-modern environments should stretch certain edges (sensory-arousal, attention-executive) and compress others; alternative environments (monastic, hunter-gatherer, accommodated workplaces) should deform the metric in different patterns.

Each prediction is independently testable. P1–P3 require existing factor-analytic methods on capacity data, of which substantial corpus already exists [40,41]. P4–P6 require correlation analysis between capacity pairs in defined populations. P7 requires cluster analysis on the resulting 5-simplex. P8 requires longitudinal functional-outcome data with within-individual coordination measures. P9 requires cross-environmental comparison, the most novel methodological commitment of the framework.

7. Discussion

7.1 Relation to Existing Frameworks

The proposal is consistent with monotropism theory [9] and extends it. Monotropism specifies the attentional vertex; the present framework specifies the joint geometry within which attention is one of five vertices, recovering the canonical autism profile as an orthant rather than as a primary attentional condition.

The proposal is consistent with the social model of disability and the neurodiversity paradigm [7,8] and supplies a geometric structure for them. The social model holds that disability is located in the relation between wiring and environment; the present framework formalizes this as the environmental indexing of the metric on the vertex set, with the same wiring producing different Cayley-Menger signatures in different environments. The neurodiversity paradigm holds that divergence is variation rather than disorder; the present framework formalizes this as orthant-habitation within a shared simplex, with divergent and typical operation as different regions of the same space.

The proposal extends RDoC [6]. RDoC specifies transdiagnostic constructs but not their joint geometry; the present framework specifies the joint geometry as the rectified pentachoron, with the constructs as vertices and the dimensions of the simplex as the latent factors RDoC has been pursuing.

7.2 Limitations

The framework relies on the empirical adequacy of the proposed folds. If sensory and affective capacities, or social cognition and language, or motor function and arousal regulation, show within-pair distances comparable to between-pair distances, the projection must expand and the clean five-fold structure is lost. The folds are defended above on existing co-variation data, but a confirmatory population study is required.

The framework treats the rectified pentachoron as the appropriate projection target. Other simplex projections — the 5-cell, the hexateron, regional decompositions — are possible. The argument for the rectified pentachoron is that it exposes both the dyadic and modal structure of the system in a single geometric object, which the alternatives do not. The argument is geometric rather than empirical; an alternative empirical structure would call for an alternative projection.

The framework specifies a static configuration. Developmental trajectories, state-dependent excursions, and longitudinal change require a dynamics-on-the-simplex extension, which the present paper does not develop. The static framework is necessary but not sufficient for the full account.

The framework is silent on neural implementation. The vertices are functional capacities, not brain regions. Their realization in neural systems is a separate empirical question, and the framework neither requires nor forbids any particular implementation.

7.3 Conclusion

Neurodivergence is not a list of disorders or a flat dimensional space. It is a configuration space whose realizable geometry, derived from empirical co-variation among cognitive capacities and constrained by Cayley-Menger determinants, projects onto the rectified pentachoron. The five vertices — Attention, Executive function, Sensory-affective coupling, Inter-mind, Motor-arousal — recover the structural commitments of the existing literature. The ten dyads recover the dyadic phenomena. The five tetrahedral cells recover the modal sub-spaces. The centroid recovers integrated functioning, distinguished from the typical baseline. The clinical categories become orthants, faces, and signatures rather than separate kinds. The framework is falsifiable, consistent with the neurodiversity paradigm, and supplies geometric structure where the existing frameworks have remained dimensionally underdetermined.

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