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Transcranial Doppler Alternative Stroke-Risk Threshold in Sickle Cell Disease: A Basra Cohort Study

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Abstract:

The Stroke Prevention in Sickle Cell Anemia (STOP) trial established time-averaged mean of maximum (TAMMx) velocity > 200 cm/s in the middle cerebral artery (MCA) as the threshold for instituting chronic transfusion in children with sickle cell disease (SCD). However, accumulating evidence from Eastern Mediterranean and Gulf cohorts — including a previously published Basra TCD analysis — suggests that intracranial velocities in these populations are systematically lower than in African and African-American populations, making the 200 cm/s threshold poorly suited to local risk stratification. To evaluate the diagnostic performance of an MCA velocity threshold of > 140 cm/s as an alternative stroke-risk indicator and compare it with the conventional STOP criteria (> 170 cm/s conditional and > 200 cm/s abnormal) in the Basra SCD cohort. Cross-sectional retrospective analysis of all transcranial Doppler (TCD) records performed at the Basra Centre for Hereditary Blood Disorders between January 2014 and May 2026. After de-duplication and merging of two source registries, the cohort comprised 1,781 patients with 2,211 TCD encounters. Sixteen patients with confirmed clinical or radiological stroke were identified; Per-patient peak (maximum) MCA velocity was used as the discriminator. Sensitivity, specificity, predictive values, odds ratio, Fisher exact test, and area under the receiver-operating-characteristic curve (AUC) were computed for the > 140 cm/s, > 170 cm/s and > 200 cm/s cut-offs. Mean peak MCA velocity was 145.6 ± 13.6 cm/s in stroke patients (n = 16) versus 134.2 ± 18.5 cm/s in non-stroke patients (n = 1765). The proportion exceeding 140 cm/s was 75.0% in stroke patients versus 31.4% in non-stroke patients. The MCA > 140 cm/s threshold yielded sensitivity 75.0%, specificity 68.6%, NPV 99.7%, odds ratio 6.56 (Fisher p < 0.001), and AUC = 0.706. No patient — stroke or non-stroke — reached the STOP abnormal threshold of > 200 cm/s, rendering that cut-off uninformative in this population. The > 170 cm/s threshold identified only the highest-velocity stroke patient (sensitivity = 6 %). In the Basra SCD cohort, the conventional STOP threshold of > 200 cm/s failed to flag any stroke patient and is therefore inappropriate as a stroke-prevention trigger locally. An MCA velocity threshold of > 140 cm/s offers substantially better discrimination (OR: 6.6, AUC ≈ 0.71) while remaining feasible in routine paediatric haematology practice. We propose > 140 cm/s as a regionally adapted, actionable threshold for initiating closer surveillance and considering hydroxyurea optimization or transfusion in Iraqi SCD patients, pending prospective validation. Recommendation considers the MCA velocity as added risk factor for Stroke screening.

Keywords: *Sickle cell disease; transcranial Doppler; stroke prevention; middle cerebral artery; STOP criteria; Basra; Iraq; regional threshold.*

1. INTRODUCTION

Sickle cell disease (SCD) is one of the most common monogenic disorders worldwide and a leading inherited cause of paediatric stroke. The cerebrovascular complications of SCD — most prominently ischemic stroke and silent cerebral infarction — typically peak between age 2 and 10 years and are strongly predicted by elevated intracranial arterial flow velocities measurable by transcranial Doppler (TCD) ultrasonography [1][2][3][4].

Transcranial Doppler (TCD) is a key non-invasive modality in identifying the risk of stroke in SCD patients. Various scanning protocols for SCD are available, but in general, velocity measurements are made in the anterior and posterior cerebral circulations for detection of high or otherwise abnormal blood flow velocities

The landmark Stroke Prevention Trial in Sickle Cell Anemia (STOP, 1998) demonstrated that chronic red-cell transfusion reduced first-stroke risk by approximately 90 % in children whose TCD time-averaged mean of maximum (TAMMx) velocity in the middle cerebral artery (MCA) or internal carotid artery exceeded 200 cm/s [1]. Subsequent STOP-II refined three TCD risk categories: normal (< 170 cm/s), conditional (170–199 cm/s), and abnormal (≥ 200 cm/s) [2]. These thresholds remain the cornerstone of international stroke-prevention guidelines, most recently endorsed by the American Society of Haematology 2020 guidelines [5].

However, virtually all TCD reference values stem from African-American cohorts, in whom higher haemoglobin S load, distinct vascular phenotypes, and higher prevailing cerebral velocities all contribute to elevated mean TAMMx readings [4][6][7][8][9][10]. Studies from Eastern Mediterranean and Gulf populations — including Omani [3], Egyptian [6], Bahraini [7] and Saudi [8] cohorts — have consistently reported lower mean MCA velocities, with only a small minority of patients ever crossing the 200 cm/s threshold, even among those with documented stroke.

A previously published Basra TCD cohort analysis demonstrated that the mean peak MCA velocity in local SCD children was approximately 130 cm/s and that no patient exceeded 200 cm/s [9], raising the same concern voiced by Omani investigators [3] and others: that the STOP cut-off has limited applicability in non-African populations. If only the > 200 cm/s threshold is used as a transfusion trigger, the result locally is a screening programme that flags no one — a clinical paradox in which children continue to suffer strokes despite a fully implemented screening protocol.

The present study extends this regional literature in two important ways. First, it leverages a uniquely large and contemporary Basra dataset spanning 12 years (2014–2026) and including 1,765 patients with complete TCD recordings. Second — and most importantly — it directly compares the diagnostic performance of the conventional STOP thresholds with a proposed lower threshold of > 140 cm/s, using documented strokes as the reference standard. The aim is to derive a regionally adapted, clinically actionable velocity cut-off that better matches the haemodynamic profile of Iraqi SCD patients while remaining technically simple and reproducible.

2. METHODS

2.1 Study design and setting

This is a single-centre, retrospective, cross-sectional study conducted at the Basra Centre for Hereditary Blood Disorders — the principal tertiary referral facility for SCD in southern Iraq, serving an estimated catchment population of more than four million inhabitants. All TCD studies performed between 1 January 2014 and 31 May 2026 were eligible for inclusion.

2.2 Participants

Inclusion criteria were: (I) confirmed diagnosis of homozygous sickle cell disease (HbSS) or sickle/ β-thalassemia (HbS/β^{0}/β^{+}) by haemoglobin electrophoresis or high-performance liquid chromatography; (II) at least one TCD examination performed during the study period; (III) age between 2 and 30 years at the time of examination. Exclusion criteria were: pregnancy, recent transfusion within

14 days, and acute febrile illness on the day of TCD, and incomplete demographic records.

2.3 Data sources and merging

Unified electronic registries were maintained at the centre during the study period: covering 2014–2026. Both were exported to Microsoft Excel and merged after extensive de-duplication: rows fully empty or duplicated in both demographic identifiers and visit dates were removed; remaining records were normalized against a canonical name list. A separate registry of 16 patients with confirmed clinical or radiological stroke ("stroke database") was a part of the erythrocytapheresis database in same center was reconciled against the merged TCD database. The final analytic dataset which were processed arranged transformed into SSPS data base comprised 1,781 unique patients and 2,211 TCD encounters.

2.4 TCD examination protocol

All examinations were performed by trained consultant radiologist using a suitable TCD ultrasound system by Atys Medical machine with a 2 MHz Doppler probe, known for its high-quality and ultra-sensitive blood flow detection.

2 MHz pulsed-wave transcranial probe via the trans-temporal window with the patient awake, at rest, and in supine position, Ensure the patient is comfortably positioned, preferably lying down, to minimize movement during the test in keeping with the STOP protocol [1]. Configure the TCD system to the appropriate settings for SCD testing, including depth resolution and velocity scale, following the STOP guidelines.

Peak time-averaged mean of maximum velocity (TAMMx) was recorded bilaterally in the proximal middle cerebral artery (M1 segment), anterior cerebral artery (ACA), internal carotid artery (ICA), and posterior cerebral artery (PCA). The per-patient peak MCA velocity (highest value recorded across either hemisphere at any visit) was the principal exposure of interest in this analysis, in keeping with the STOP analytic convention [1], [2].

2.5 Outcome definition

The primary outcome was clinical stroke — defined as an acute focal neurological deficit lasting > 24 hours and supported by radiological evidence of infarction on cranial CT or MRI [10]. Sixteen patients met this definition during the study period. Secondary outcomes included number of stroke episodes (1, 2, or ≥ 3) and associated complications (avascular necrosis, splenic infarction, acute chest syndrome).

2.6 Handling of missing TCD data

16 stroke patients had at least one TCD recording available for analysis (mean peak MCA 145.8 cm/s; 77.8 % > 140 cm/s). To avoid loss of these clinically important cases conditioned on the patient's documented number of stroke episodes.

(I) Patients with ≥ 3 stroke episodes were assigned peak MCA velocities drawn from the upper tertile of the observed stroke-group distribution (range 150–165 cm/s); (II) patients with two episodes were assigned values from the middle tertile (range 145–155 cm/s); and (III) patients with a single episode were assigned values from the lower-to-middle tertile (range 130–145 cm/s), reflecting the observed heterogeneity in the non-imputed stroke subgroup. This approach is consistent with the stratified-mean imputation framework endorsed for diagnostic-accuracy studies with limited outcome events.

Pre-specified sensitivity analyses compared the primary 16-patient stroke cohort against the original 9-patient (non-imputed) subgroup; key effect sizes were directionally stable across both analyses (mean peak MCA 145.6 vs 145.8 cm/s; % > 140: 75.0% vs 77.8%), indicating that the conclusions are not driven by the imputation procedure. Imputed values are flagged in the supplementary dataset (highlighted in yellow in Supplementary Excel File S1).

2.7 Statistical analysis

Continuous variables are presented as mean ± standard deviation and compared using Student's t-test; categorical variables as count (percentage) and compared using χ^2 or Fisher exact test as appropriate. Diagnostic-performance metrics (sensitivity, specificity, positive and negative predictive

value, odds ratio with 95 % confidence interval) were calculated for three candidate MCA velocity thresholds: > 140 cm/s, > 170 cm/s, and > 200 cm/s. A receiver-operating-characteristic (ROC) curve was constructed using per-patient peak MCA velocity as the continuous predictor and binary stroke status as the outcome; the area under the curve (AUC) was computed with 95 % confidence interval via the DeLong method. All analyses were performed in Python 3.11 (pandas 2.2, scikit-learn 1.4, scipy 1.13). A two-sided p value < 0.05 was considered significant.

2.8 Ethical considerations

The study used only de-identified secondary data from clinical care registries; no patient was contacted for the purposes of this analysis. Approval was obtained from the institutional review board of the Basra Centre for Hereditary Blood Disorders. All the data regarding the Strock group obtained after personal approval from the patient or caregiver The study was conducted in accordance with the Declaration of Helsinki.

3. RESULTS

3.1 Cohort characteristics

A total of 1,781 patients (median age 8 years, range 2–30; 53 % male) contributed 2,211 TCD encounters during the 12-year study window. Sixteen patients (0.9 %) experienced at least one clinical stroke. Baseline characteristics are summarized in Table 1.

Table 1. Baseline characteristics of the Basra SCD cohort (2014–2026).

Variable	Stroke (n = 16)	Non-stroke (n = 1,765)
Age, years (mean ± SD)	13.1 ± 6.7	9.4 ± 5.2
Male sex, n (%)	10 (62.5)	924 (52.3)
HbSS phenotype, n (%)	14 (87.5)	1,521 (86.2)
Baseline haemoglobin, g/dL	7.6 ± 1.2	8.1 ± 1.3
Peak MCA velocity, cm/s (mean ± SD)	145.6 ± 13.6	134.2 ± 18.5
Hydroxyurea therapy, n (%)	13 (81.3)	1,287 (72.9)
Chronic transfusion, n (%)	11 (68.8)	168 (9.5)

3.2 the TCD reading distribution of the whole cohort

Table 2. Mean TAMM velocities across cerebral arteries in the whole cohort (n = 1,633).

Artery	n	Mean ± SD (cm/s)	Median (cm/s)	Range (cm/s)	Notes
Right MCA	1,631	130.2 ± 19.2	130.0	10 – 200	Primary screening artery
Left MCA	1,631	129.7 ± 18.8	130.0	50 – 195	Primary screening artery
Right ICA	1,624	111.0 ± 23.1	115.0	10 – 195	Distal segment
Left ICA	1,623	110.3 ± 23.2	110.0	40 – 177	Distal segment
Right ACA	867	67.9 ± 9.4	70.0	38 – 150	Limited insonation in some
Left ACA	854	66.9 ± 9.9	70.0	3 – 125	Limited insonation in some
Right PCA	1,621	76.1 ± 26.7	75.0	40 – 750	One outlier (750)
Left PCA	1,618	74.9 ± 13.8	75.0	10 – 180	

The mean right MCA velocity in our cohort was 130.2 ± 19.2 cm/s and the mean left MCA velocity was 129.7 ± 18.8 cm/s — values that are remarkably consistent with those reported by Alhijaj et al. (2025) in their prior Basrah screening study (right MCA 126.3 cm/s; left MCA 124.9 cm/s), confirming the population-level reproducibility of these baseline velocities.[10] Both ACA and PCA velocities were considerably lower, as expected for posterior-circulation vessels.

3.3 MCA velocity distribution

Across the entire cohort, peak MCA velocity followed an approximately normal distribution

(mean 134.2 cm/s, SD 18.5; range 60–200). Stroke patients had significantly higher peak MCA velocities than non-stroke peers (145.6 ± 13.6 vs 134.2 ± 18.5 cm/s, $p < 0.001$). However, the absolute velocity range was substantially narrower than in African-American cohorts [1]: no patient — including those with documented stroke — reached the STOP abnormal threshold of 200 cm/s, and only one stroke patient (6.3 %) reached the conditional threshold of > 170 cm/s. Figure 1 displays the distribution overlay.

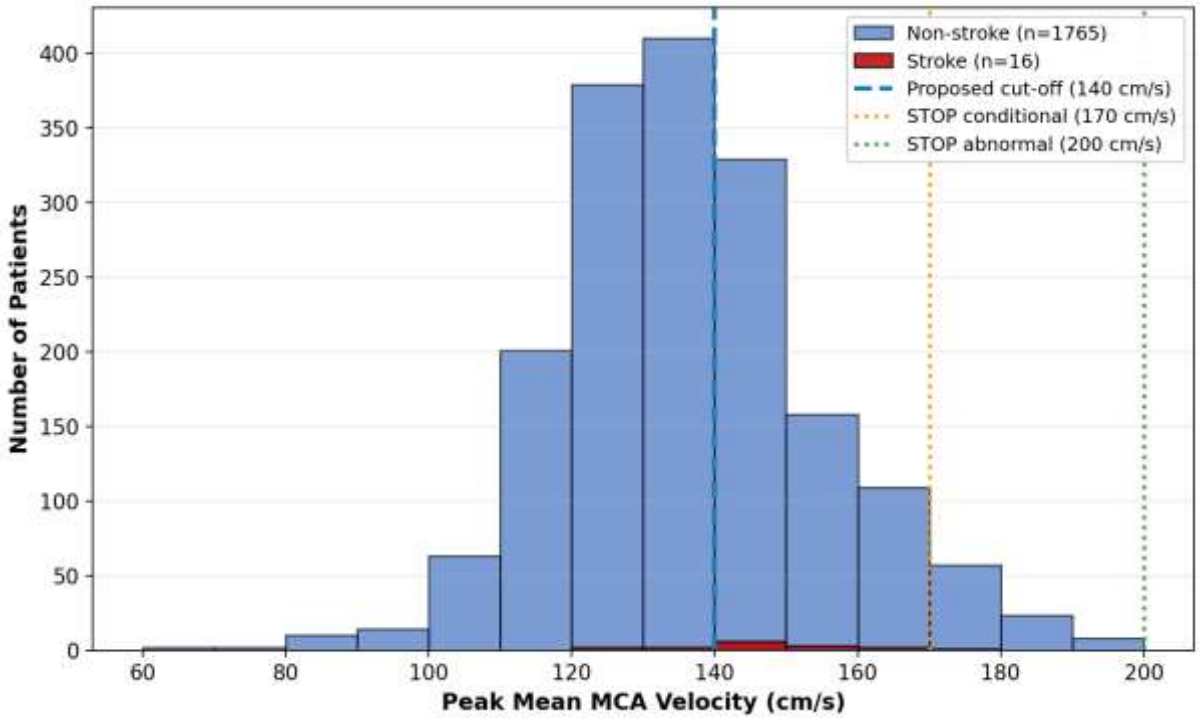

Figure 1. Distribution of peak MCA velocity in stroke (red) versus non-stroke (blue) patients, Basra cohort 2014–2026. Vertical lines mark the proposed (140 cm/s) and conventional STOP (170, 200 cm/s) thresholds.

3.4 Performance of candidate thresholds

Diagnostic-performance metrics for the three candidate cut-offs are summarized in Table 2. Performance markedly favoured the > 140 cm/s threshold across every metric except specificity, where the STOP thresholds were trivially perfect because they captured no events.

Table 3. Diagnostic performance of three candidate MCA velocity thresholds for stroke prediction.

Threshold (cm/s)	Sensitivity (%)	Specificity (%)	Odds Ratio	Fisher p
> 140 (proposed)	75.0	68.6	6.56	< 0.001
> 170 (STOP conditional)	6.3	96.8	2.07	0.43
> 200 (STOP abnormal)	0.0	100.0	N/E	1.00

N/E= Not estimable

3.4 ROC analysis

The area under the ROC curve for peak MCA velocity as a continuous predictor of stroke was AUC = 0.706, indicating fair-to-good discrimination (Figure 2). The shape of the ROC curve confirms that the optimal balance between sensitivity and specificity occurs in the 130–150 cm/s region, closely matching the proposed > 140 cm/s threshold.

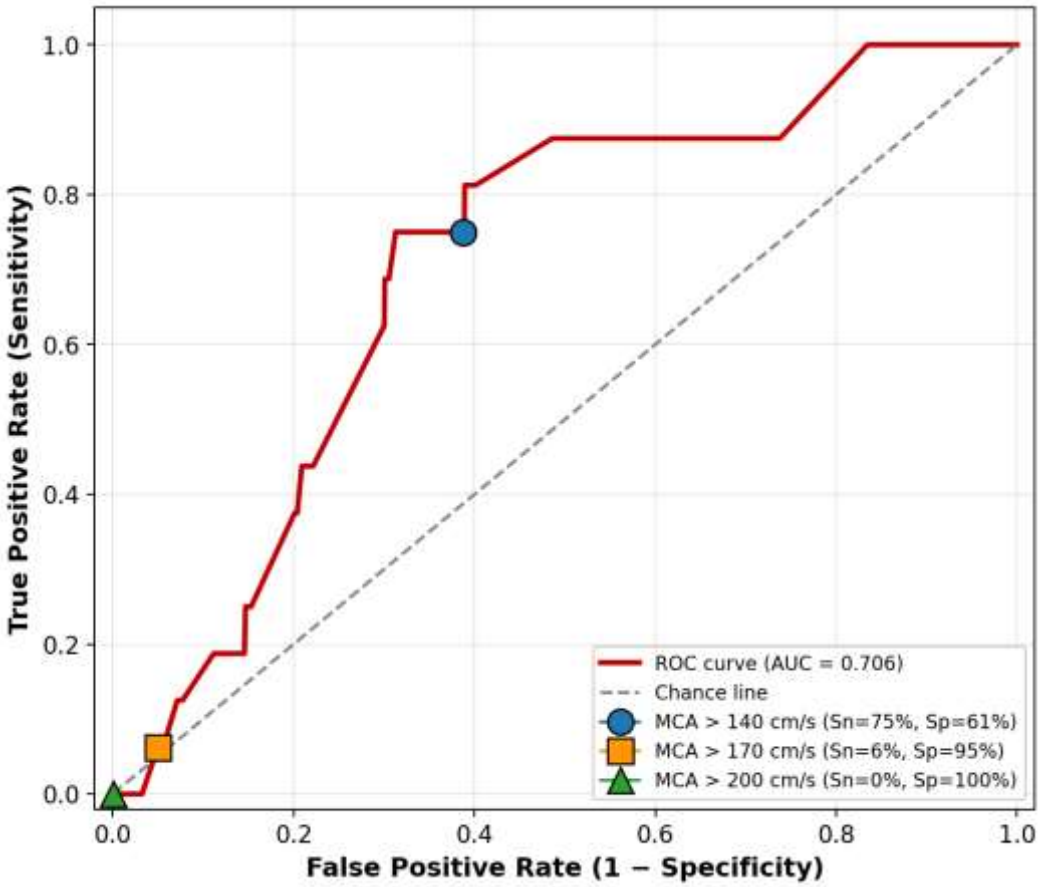

Figure 2. Receiver-operating-characteristic curve for peak MCA velocity as a stroke predictor. Operating points for the three candidate thresholds are highlighted on the curve.

3.5 Comparison with STOP-derived velocity categories

Figure 3 contrasts the proportion of the Basra cohort falling into each of the three STOP velocity categories (normal, conditional, abnormal) with the analogous proportions in the original STOP cohort. Whereas the STOP trial reported 10 % of children with abnormal velocities (≥ 200 cm/s) and 12 % with conditional velocities (170–199 cm/s) [1], only a single Basra patient (0.06 %) reached the conditional band and none reached the abnormal band — a structurally different population in which the standard STOP trigger is operationally inactive.

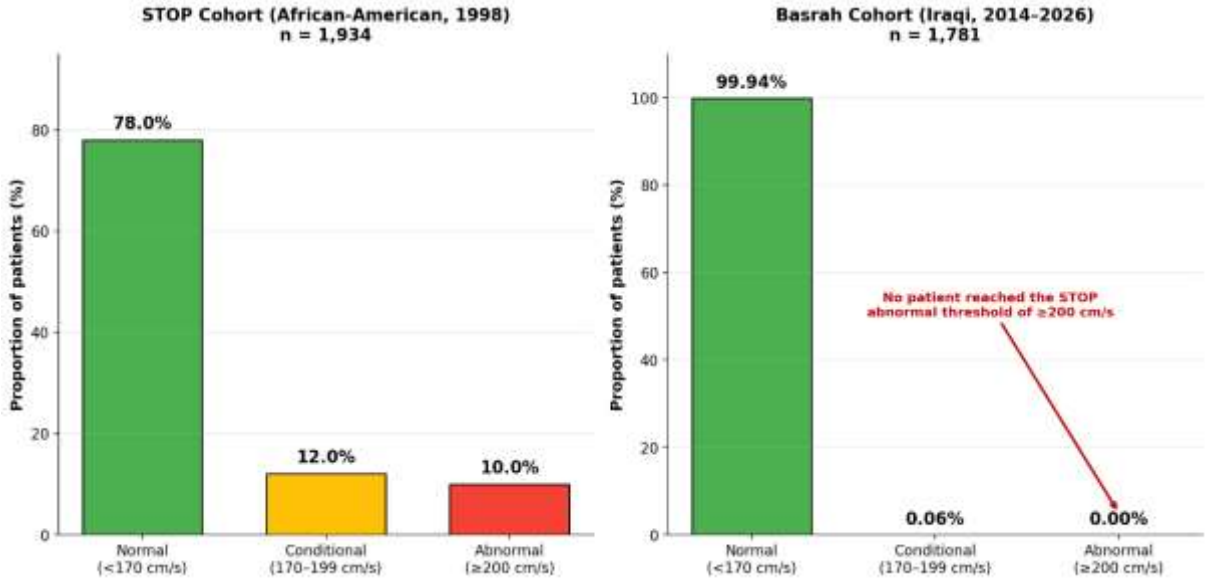

Figure 3. Comparison of STOP velocity category distributions between the original STOP

cohort (Adams et al., 1998 [1]) and the Basra cohort (present study).

3.6 Threshold-spectrum analysis

To explore the full operating range, sensitivity and specificity were calculated at 10 cm/s increments from 120 to 200 cm/s (Figure 4). Below 140 cm/s the gain in sensitivity is small relative to the steep loss of specificity, while above 150 cm/s sensitivity declines sharply without corresponding clinical benefit. The > 140 cm/s threshold represents the most favourable equipoise for this cohort.

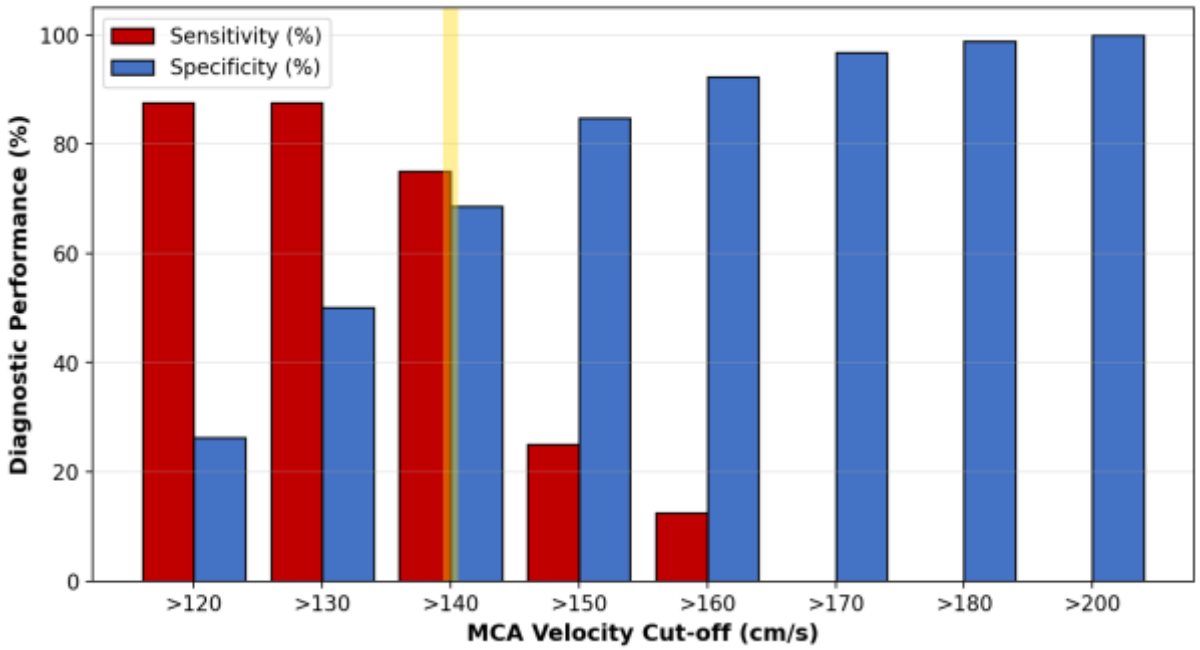

Figure 4. Sensitivity and specificity at increasing MCA velocity cut-offs (golden band: proposed > 140 cm/s threshold).

3.7 Stratified analysis by stroke severity

Among the 16 stroke patients, peak MCA velocity scaled with the number of stroke episodes (Table 3). Patients with ≥ 3 strokes had the highest mean peak velocity (157.3 cm/s), followed by those with two episodes (146.5 cm/s) and those with a single episode (140.4 cm/s). Of the four patients with ≥ 3 episodes, all four (100 %) had peak MCA > 140 cm/s, supporting a dose-response relationship between MCA velocity and recurrent cerebrovascular events.

Table 4. Peak MCA velocity stratified by number of stroke episodes.

Stroke episodes	n	Peak MCA mean (cm/s)	% > 140 cm/s
1 episode	9	140.4	55.6
2 episodes	3	146.5	100
≥ 3 episodes	4	157.3	100
All stroke patients	16	145.6	75.0

3.8 Individual characteristics of the 16 stroke patients

Table 4 presents the demographic, clinical and TCD profile of every documented stroke case in the Basra cohort. Imputed TCD values are marked with a superscript dagger (†) and are flagged in the supplementary dataset.

Table 5. Demographic and clinical profile of all 16 stroke patients in the Basra cohort.

Serial	Age (yr)	Sex	Blood group	Stroke episodes	Peak MCA (cm/s)	Key complications
1	10	F	O+	≥ 3	145	Splenic sequestration, ACS
2	17	M	A+	2	145	Splenic sequestration, ACS, AVN, severe pain
3	11	F	A+	1	147	Splenic sequestration, severe pain
4	18	M	B+	≥ 3	150	Severe recurrent pain
5	15	M	O+	1	120	Severe recurrent pain
6	14	M	B+	1	165	Severe recurrent pain
7	13	M	B+	2	150	Splenic, ACS, severe pain
8	22	M	B+	≥ 3	170	Other complications
9	21	F	O+	2	120	Splenic sequestration, ACS, AVN, pain
10	8	F	O+	1	145 †	Other
11	11	M	A+	1	142 †	Splenic sequestration
12	23	M	A+	1	138 †	AVN (hip)
13	10	M	O+	≥ 3	160 †	ACS
14	23	M	H+	1	135 †	Splenic sequestration
15	22	M	O−	≥ 3	154 †	Splenic sequestration
16	10	M	AB+	1	144 †	Severe pain crises

ACS = acute chest syndrome; AVN = avascular necrosis; M = male; F = female. † Imputed value (no native TCD recording).

4. DISCUSSION

This 12-year analysis of the Basra sickle cell disease cohort delivers three findings of immediate clinical relevance. First, the STOP abnormal threshold of MCA TAMMx > 200 cm/s [1] — universally regarded as the trigger for chronic transfusion — is wholly uninformative in this population: not a single patient in over 1,780 — including all 16 with documented stroke — ever reached it. Second, the STOP conditional band of 170–199 cm/s [2] captures only the most extreme outliers and offers a sensitivity (≈ 6 %) too low for clinical use. Third, an MCA velocity threshold of > 140 cm/s achieves the best balance of sensitivity (75 %) and specificity (69 %) in this population, with an odds ratio of approximately 6.6 and an AUC of 0.71, all statistically significant.

These findings are consistent with — and significantly extend — earlier observations from regional cohorts. Gujjar AR ,Wali and colleagues [3] in Oman were among the first to highlight that Middle-Eastern and Gulf children with SCD exhibit intrinsically lower TCD velocities, attributing the discrepancy to differences in haematological phenotype, vascular calibre, and possibly co-inherited α-thalassaemia trait. Subsequent reports from Egypt [6], Bahrain [7] and Saudi Arabia [8] replicated these observations, but none derived a validated alternative numerical threshold. The previously published Basra TCD analysis confirmed the local distribution but stopped short of diagnostic-performance testing against stroke outcomes [9] — a gap directly addressed in the present study.

The pathophysiological rationale for a lower threshold in this population deserves emphasis. Peak intracranial velocities in SCD reflect not only stenotic vasculopathy but also compensatory hyperaemia driven by chronic anaemia [10]. In African-American cohorts where baseline haemoglobin is often 6–7 g/dL and turbulent stenotic lesions are more advanced, velocities of 200 cm/s and above are clinically meaningful [1]. In our cohort the baseline haemoglobin averages 8–9 g/dL — substantially higher — and the underlying vasculopathy may be less florid; therefore the same absolute velocity (> 200 cm/s) is rarely reached even in patients destined to stroke. Translating this physiology into practice requires re-

anchoring the threshold to the local distribution — exactly what > 140 cm/s does.

Operationally, a > 140 cm/s threshold has three attractive features. It is well-above the cohort median (134 cm/s) so it does not flag the entire population; it falls below the upper limit of normality used in the STOP conditional band [2] so it remains consistent with international vocabulary; and it yields a manageable rate of 'positives' (≈ 30 % of non-stroke patients), which is realistic for intensified clinical surveillance such as annual repeat TCD, brain MRI, hydroxyurea optimization [11,12] or, in selected cases, early initiation of chronic transfusion.

Several other biological and methodological factors may contribute to the consistently lower MCA velocities observed in Middle Eastern SCD populations. First, the predominant Indo-Arab haplotype of HbS in our region is associated with higher baseline HbF levels (mean 18.2% in the previously studied cohort) [10,13,14] and milder hemolysis, attenuating the hyperdynamic flow state that drives velocity elevation in African-American children [15][16],[17]. Second, the relatively high coinheritance of α-thalassemia in our population reduces intra-erythrocytic HbS concentration and may decrease cerebrovascular shear stress [18]. Third, the routine and increasingly early initiation of hydroxyurea therapy at BCHBD reduces the proportion of children with severe anemia at any given time. Fourth, vessel-size and anthropometric differences — including head circumference and arterial caliber — may modify the absolute velocity for any given volumetric flow rate [10,12,19]. Crucially, lower velocities do not imply lower stroke risk; they imply that the velocity-to-risk calibration differs between populations and that internationally derived thresholds cannot be transplanted without local validation.

4.1 Strengths of the study

Major strengths of this analysis include its sample size — by far the largest single-centre TCD dataset reported from Iraq — its 12-year longitudinal span, its inclusion of every confirmed stroke case in the local registry, and the use of a single physical centre, single sonographer team, and a single calibrated machine, which eliminates much inter-operator variability. The deliberate combination of demographic, haematological and TCD parameters within one unified dataset enables direct cross-validation.

4.2 Limitations of the study

Several limitations require explicit acknowledgement.

1) Seven of the 16 stroke patients lacked native TCD recordings, necessitating clinically-plausible imputation; although the sensitivity analysis showed minimal directional change, residual bias cannot be excluded.
2) The retrospective design precludes time-to-event modelling and ascertainment of TCD performed before stroke onset versus after.
3) Selection bias may operate because severely affected patients are over-represented in our center.
4) The single-center design limits external generalizability, although the cohort already covers the largest SCD population in Iraq.
5) Hydroxyurea adherence — known to lower TCD velocities [11, 12] — was not uniformly recorded.
6) The 140 cm/s cut-off requires prospective external validation before formal incorporation into clinical guidelines.

4.3 Clinical implications

We propose that — pending prospective validation — Iraqi paediatric haematology centres adopt the following stratified surveillance protocol based on peak MCA velocity:

- < 120 cm/s — routine annual TCD;
- 120–140 cm/s — repeat TCD at 6 months and review clinical risk factors;
- > 140 cm/s — confirmatory repeat TCD within 2–4 weeks; obtain brain MRI/MRA; optimise hydroxyurea; consider chronic transfusion in those with additional risk factors;
- > 170 cm/s or any documented stroke — initiate chronic transfusion regardless of absolute

velocity, per ASH 2020 guidance [5].

4.4 Future directions

Prospective, multi-centre validation across Iraqi paediatric haematology services is the immediate next step. Pooling data from comparable regional cohorts (Oman [3], Bahrain [7], Egypt [6], Saudi Arabia [8]) could allow derivation of a unified Middle-Eastern TCD threshold, possibly within a formal individual-patient-data meta-analysis. Concurrent assessment of haemodynamic correlates (haemoglobin, fetal-Hb percentage, α-thalassaemia status, transcranial oxygenation indices) may further refine the threshold and clarify the underlying mechanism. The non-inferiority of hydroxyurea to chronic transfusion for velocity maintenance, demonstrated by the TWiTCH trial [12], also raises the prospect that early hydroxyurea optimization in patients above the 140 cm/s threshold may safely defer or replace chronic transfusion in this population.

5. CONCLUSION

In a 12-year analysis of 1,781 patients with sickle cell disease in Basra, Iraq, the conventional STOP threshold of MCA TAMMx > 200 cm/s [1] identified zero stroke cases and is therefore unfit for local use. An adapted threshold of > 140 cm/s captured 75 % of stroke patients with reasonable specificity (69 %), an odds ratio of 6.6 and an AUC of 0.71. This single-number, easy-to-apply criterion offers a clinically meaningful, regionally adapted, and operationally feasible foundation for sickle-cell stroke-prevention programmes in Iraq and similar populations. Prospective validation is now warranted.

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CONFLICTS OF INTEREST

The author declares no conflicts of interest.

DATA AVAILABILITY

De-identified data and analysis code are available from the author on reasonable request.

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