Does screening for diabetes in at-risk patients improve long-term outcomes?

EVIDENCE-BASED ANSWER

No randomized clinical trials or prospective studies have demonstrated adequate evidence to screen individuals for diabetes mellitus. A recently published meta-analysis for the United States Preventative Services Task Force (USPSTF) stated that "until we have better evidence about its benefits, harms, and costs, the role of screening as a strategy to reduce the burden of suffering of diabetes will remain uncertain" (strength of recommendation [SOR]: **B**, based on inconclusive studies).

The group of patients most likely to benefit from diabetes screening are patients with hypertension (SOR: **B**), or those whose risk for coronary heart disease is such that a diagnosis of diabetes would mandate addition of aspirin or lipid-lowering agents (SOR: **C**).

EVIDENCE SUMMARY

It is estimated that by the year 2010 approximately 216 million individuals worldwide will be affected with diabetes; 90% of these people will have type 2.[1] In addition, it is well documented that diabetes significantly increases the risk of morbidity and mortality, especially due to retinopathy, nephropathy, neuropathy, and coronary artery disease.[2]

For screening to be effective, the disease of interest must have an easily detectable asymptomatic state, and a treatment that improves outcomes by intervening before symptoms develop. Diabetes does have an asymptomatic state, which is of uncertain duration (likely years), and is detectable with simple, inexpensive tests: specifically, either a fasting blood glucose or a 2-hour post-glucose-load blood glucose. In order to be useful, a screening program must also lead to an intervention that reduces morbidity or mortality. The data are much less clear whether any interventions during the presymptomatic period improve patient outcomes.

No randomized trials have tested whether screening provides any benefits.[3] In a thorough systematic review using USPSTF methodologies, several potential postscreening interventions were evaluated.[3] While tight glycemic control reduces progression of albuminuria and retinopathy, it is unclear how large the long-term clinical benefit would be, or at what cost. Reasonable evidence supports more aggressive control of blood pressure for patients with diabetes to reduce adverse cardiovascular outcomes. It is

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What are Clinical Inquiries?

Clinical Inquiries answer real questions that family physicians submit to the Family Practice Inquiries Network (FPIN), a national, not-for-profit consortium of family practice departments, residency programs, academic health sciences libraries, primary care practice-based research networks, and other specialists.

Questions chosen for Clinical Inquiries are those that family physicians vote as most important through a web-based voting system.

Answers are developed by a specific method:

- FPIN medical librarians conduct systematic and standardized literature searches in collaboration with an FPIN clinician or clinicians.
- FPIN clinician authors select the research articles to include, critically appraise the research evidence, review the authoritative sources, and write the answers.
- Each Clinical Inquiry is reviewed by 4 or more peers and editors before publication in *JFP*.
- FPIN medical librarians coauthor Type I Clinical Inquiries that have required a systematic search.
- Finally, a practicing family physician writes an accompanying commentary.

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Evidence supports more aggessive control of blood pressure for patients with diabetes

important to note that the data for these interventions (aggressive blood sugar and blood pressure control) were derived in studies of patients with established diabetes; no studies have tested these interventions for patients who had early diagnosis by screening.

Since undiagnosed diabetes doubles the risk of coronary artery disease, there is the potential that intervention with prophylactic aspirin and lipid-lowering agents could reduce coronary artery disease, although this has not been tested. There is no evidence that the diagnosis of diabetes per se alters individual patients' behavior in response to lifestyle counseling, particularly about smoking cessation, diet, and exercise. It is unlikely that screening for foot ulcers would provide any benefit in those with an early diagnosis of diabetes.

There is reasonable evidence that aggressive counseling and behavioral interventions can postpone the diagnosis of diabetes for patients with glucose intolerance. The studies were too small and short to detect any meaningful difference in morbidity or mortality. In addition, it is unknown if this postponing of the onset of diabetes is cost-effective.

The risks of screening include false-positive diagnosis, labeling effect, and subjecting patients to potentially harmful medications. There is little data to estimate the size of these effects.

Using a best-case scenario, the number needed to screen (NNS) is 500 to prevent cardiovascular outcomes by aggressive hypertension therapy. This assumes a baseline rate of 6% undetected diabetes, with a 5-year lead-time benefit to screening, and 50% increase in the rate of aggressive hypertension control. Assuming the baseline rate is 3% and the lead time is 2.5 years, the NNS is 3600.

The NNS for preventing monocular blindness is higher, even using best-case assumptions. The calculations for blindness rely on greater extrapolations of the data; the other potential interventions described above had inadequate data even to make such calculations.

RECOMMENDATIONS FROM OTHERS

The USPSTF concludes that the evidence is insufficient to recommend for or against routinely screening asymptomatic adults for type 2 diabetes, impaired glucose tolerance, or impaired fasting glucose. The USPSTF recommends screening for type 2 diabetes in adults with hypertension or hyperlipidemia. They report that it is likely that more aggressive treatment of hypertension, hyperlipidemia, and other cardiovascular risk factors could reduce cardiovascular morbidity and mortality.[4]

The American Diabetes Association (ADA) recommends that health care providers consider screening patients at age 45 years and continue screening in 3-year intervals. The ADA also notes that individuals who are overweight or considered to be at higher risk should be screened at a younger age and more frequently.

The ADA recommends routine screening in "high-risk" patients, defined as those with a positive family history of type 2 diabetes (in first- and second-degree relatives), or who are Native Americans, African-Americans, Hispanic Americans, or Asians/South Pacific Islanders.

The ADA also recommends screening for patients who have signs of insulin resistance or conditions associated with insulin resistance, such acanthosis nigricans, hypertension, dyslipidemia, and polycystic ovary syndrome. They note that this advice is based on expert opinion and should be carried out at the discretion of the health care provider.[5]

Danielle S. Nordone, DO, Dyanne Westerberg, DO, Diane Wolf, MLS, Department of Family Medicine, Christiana Care Health System, Wilmington, Del

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CLINICAL COMMENTARY: Evidence for universal screening is not there

Many of my patients lead unhealthy lifestyles; they become obese and often develop hypertension, diabetes, dyslipidemia, and heart disease. Further, the incidence of diabetes in the United States has grown by one third in the last decade, and the urge to screen is great. However, the evidence for a significant benefit from screening for diabetes is not there. In fact, the meta-analysis by Harris et al suggests that the number needed to screen in the most favorable group, hypertensives, would still be 900 to prevent 1 cardiovascular event. Furthermore, that estimate results from extrapolation and conjecture; no randomized controlled trial of screening for diabetes has been done. Accordingly, the recommendations by the ADA and USPSTF to screen high-risk patients are likely as aggressive as can be supported at this time—regardless of the drive to do something.

Jim Holt, MD, East Tennessee State University, Johnson City

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APPLIED EVIDENCE
"Strategies to reduce complications of type 2 diabetes," *page 366*

What is the best treatment for diabetic neuropathy?

EVIDENCE-BASED ANSWER

Tricyclic antidepressants, anticonvulsants, and capsaicin reduce the pain of diabetic neuropathy; limited data suggests that lidocaine patches may also be efficacious. Both tricyclic antidepressants and anticonvulsants are superior to placebo in relieving painful diabetic neuropathy. Compared with placebo, patients taking tricyclic antidepressants report reduced pain (number needed to treat [NNT] for at least 50% reduction= 3.5) (strength of recommendation [SOR]: **A**). Similarly, patients taking anticonvulsants report reduced pain (NNT for at least 50% reduction in pain=2.7) (SOR: **A**).

Limited evidence suggests that selective serotonin reuptake inhibitors (SSRIs) are no more efficacious than placebo (SOR: **C**). Both antidepressants and anticonvulsants have a high rate of minor adverse effects (number needed to harm [NNH]=2.7 for both). Tricyclic antidepressants have an NNH of 17 for side effects severe enough that patients withdrew from the study.

Compared with placebo, topical capsaicin also reduces pain (NNT=4) (SOR: **A**); however, there are no systematically collected data on side effects for capsaicin. A single case series demonstrates that lidocaine patches are efficacious for neuropathic pain, though expensive (SOR: **B**). Almost no trials comparing different classes of treatments have been performed.

EVIDENCE SUMMARY

A recent well-done meta-analysis[1] summarized available randomized placebo-controlled trials of antidepressants (including tricyclics and SSRIs) and anticonvulsants (including phenytoin, carbamazepine, and gabapentin). Almost all trials compare individual agents against placebo, and there have been no head-to-head trials that address functional outcomes, quality of life, patient

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TABLE

Efficacy of drug treatments for diabetic neuropathy

Drug	Number of controlled trials	NNT (95% CI) for 50% pain reduction	NNH (95% CI)	Efficacious dose	Typical cost
Antidepressants	**16**	**3.4 (2.6–4.7)**	**2.7 (2.1–3.9)**		
Tricyclics	8	3.5 (2.5–5.6)	3.2 (2.3–5.2)	Amitryptiline 50–100 mg/d; Nortryptiline 50–75 mg/d	$12
SSRIs	3	Not efficacious		N/A	N/A
Anticonvulsants*	**3**	**2.7 (2.2–3.8)**	**2.7 (2.2–3.4)**		
Phenytoin	1	Not available	3.2 (2.1–6.3)	300 mg/d	$18
Carbamezapine	1	Not available	Not available	400 mg 2x daily	$28
Gabapentin	2	Not available	2.6 (2.1–3.3)	600–900 mg 3x daily	$333
Valproate†	1	Not available	Rare	400 mg 3x daily	$36
Topical capsaicin	**4**	**4 (2.9–6.7)**	Not available	0.075% 4x daily	$39
Lidocaine patch	0	Not available	Not available	1 patch each foot, daily	$272

Costs based on 30 days of typical efficacious dose. Retail prices from www.drugstore.com, December 2003, except for capsaicin, which was obtained from Walmart.
*This summary does not include results from Kochar et al.
†Data from this trial cannot be summarized within this framework; however, results were statistically significant and similar in magnitude to other trials.
NNT, number needed to treat; NNH, number needed to harm; CI, confidence interval

satisfaction, or cost. Most trials do not describe diagnostic criteria, consider causes of pain other than diabetes or address diabetic control, which is known to predict frequency of neuropathy. Finally, very few trials include typical primary care patients in a primary care setting or control for important confounding variables such as over-the-counter medications or comorbid illnesses.

Within the constraints of this literature, placebos have a substantial impact, with an aggregate 32% of patients receiving placebo reporting at least 50% reduction in pain. A total of 16 trials have addressed the efficacy of antidepressants for diabetic neuropathy. Compared with placebo, tricyclic antidepressants have an aggregate NNT of 3.5 (95% confidence interval [CI], 2.6–4.7) for patients reporting at least 50% reduction of pain, along with an NNH of 2.7 (95% CI, 2.1–3.9) for minor adverse effects (typically the muscarinic effects of dry mouth, constipation, and blurred vision) and 17 (95% CI, 10–43) for side effects severe enough to cause withdrawal from a trial. Dosages were in the low to middle range of those used to treat depression; there was no significant difference in efficacy between trials less than 3 weeks and those greater than 3 weeks. No evidence supports differences among different tricyclic agents, and limited evidence suggests that SSRIs are no more efficacious than placebo.

A total of 4 randomized placebo-controlled

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Both tricyclics and anticonvulsants are superior to placebo for relieving painful diabetic neuropathy

trials (1 each for phenytoin [Dilantin], carbamazepine [Tegretol], gabapentin [Neurontin], and valproate [Depakote]) have extractable data about the efficacy of anticonvulsants for the pain of diabetic neuropathy. As a class, the NNT for patients reporting at least a 50% reduction in pain was 2.7 (95% CI, 2.2–3.8); the NNH for minor adverse effects (typically transient central nervous system effect such as dizziness, somnolence, or disturbance in gait) was 2.7 (95% CI, 2.2–3.4).

These summary estimates do not include the valproate trial,[2] which was reported after the meta-analysis was completed; the report did not allow calculation of NNT, but the findings were consistent with these results. Phenytoin dosage was 300 mg/d; carbamazepine dosage was titrated to 200–600 mg/d, gabapentin from 300–3600 mg/d, and valproate 1200 mg/d. Patients taking anticonvulsants did not have a higher rate of withdrawal compared to those taking placebo. Limited evidence suggests no significant differences among anticonvulsants; there is insufficient evidence to determine optimal dosage of any of these agents.

Studies involving topical agents are also limited. According to an information summary,[3] a total of 4 trials have addressed the efficacy of topical capsaicin for neuropathic pain. Compared with placebo, capsaicin reduces pain (NNT=4; 95% CI, 2.9–6.7), but no pooled information is available on side effects or rate of study withdrawal. Finally, 1 case series has suggested that lidocaine patches are efficacious for diabetic neuropathy.[4]

A variety of other interventions have been reported for diabetic neuropathy, including nonsteroidal anti-inflammatory drugs, transcutaneous electrical nerve stimulation (TENS), angiotensin-converting enzyme inhibitors, and Tramadol, but there have been no published systematic evaluations of them.

The **Table** characterizes the agents, the number of trials that address each, the NNT, NNH, typical effective dose, and approximate retail cost per month with the average effective dose.

RECOMMENDATIONS FROM OTHERS

American Diabetes Association practice guidelines do not address neuropathy; UptoDate emphasizes prevention through glycemic control, with initial treatment using amitriptyline or nortriptyline, followed by capsaicin and anticonvulsants.[5]

Warren P. Newton, MD, MPH, *Department of Family Medicine, University of North Carolina at Chapel Hill;*
Linda Collins, MSLS, *Health Sciences Library, University of North Carolina at Chapel Hill*

CLINICAL COMMENTARY: Anticonvulsants and antidepressants effective at reducing perception of pain

The management of patients with chronic pain requires a combination of artistry and skill. As each individual's perceptions, expectations and response to therapy differ, dynamic treatment approaches are required. The relative dearth of evidence supporting effective treatments for chronic pain compounds the problem. This evidence review helps to lessen some of the guesswork for patients with diabetic neuropathy. Anticonvulsants and antidepressants are impressively effective at reducing patients' perceptions of pain at a favorable benefit to significant harm ratio, NNT of 2–4 vs. NNH of 18. Several things however, aren't clear from the literature: as these were all placebo comparisons, which drug is more effective? As well, were reductions in functional limitation and disability measures or improvements in quality of life scores demonstrated? Will other newer agents prove to be superior? Despite these unanswered questions, for patients with diabetic neuropathy good evidence now supports what has likely been many clinicians' preference for the treatment of most chronic pain conditions; any alternative to narcotics.

Charissa Fotinos, MD, *Seattle–King County Public Health, Seattle, Wash*

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What is the best way to treat patients with white-coat hypertension?

EVIDENCE-BASED ANSWER

Evidence is conflicting regarding the risk of cardiovascular complications from white-coat hypertension. Some but not all studies show lower cardiovascular event rates for patients with white-coat hypertension compared with those with sustained hypertension (strength of recommendation [SOR]: **B**, cohort studies with conflicting results and methodological problems).

Little information is available about the use of antihypertensive medication for white-coat hypertension. In 1 small randomized trial, the difference in stroke incidence and cardiovascular complications between active treatment and placebo did not reach statistical significance (SOR: **B**, based on an underpowered randomized controlled trial). Some experts recommend that patients with white-coat hypertension should be evaluated for evidence of target organ injury and monitored for the development of sustained hypertension (SOR: **C**, expert opinion).

Little evidence is available about use of antihypertensive medication for white-coat hypertension

EVIDENCE SUMMARY

A prospective cohort study compared cardiovascular events among patients with white-coat hypertension vs those with sustained hypertension. The study evaluated 479 patients with persistently elevated clinic systolic blood pressures of 140 to 180 mm Hg. Using 24-hour intra-arterial ambulatory blood pressure monitoring (ABPM), they found that 126 patients had ambulatory blood pressures below 140/90 mm Hg (white-coat hypertension) while 353 patients maintained pressures above 140/90 mm Hg (sustained hypertension). On average, white-coat hypertension patients were younger than sustained hypertension patients (44 vs 52 years) but were otherwise similar. Over the next 9 years, patients with white-coat hypertension had significantly fewer cardiovascular events than patients with sustained hypertension (**Table**).[1]

Another prospective cohort study compared fatal and nonfatal cardiovascular event rates among patients who had white-coat hypertension, sustained hypertension, or were normotensive. Investigators performed 24-hour ABPM on 1187 patients who had clinic blood pressures over 140/90 on three visits. They found that 228 patients had white-coat hypertension, defined as mean ambulatory blood pressures below the 90th percentile of a normotensive population, and 959 patients had sustained hypertension. They followed these patients, along with 205 normotensive controls, for a mean of 3.2 years. Cardiovascular event rates did not differ significantly between normotensive and white-coat hypertension patients (P=.83; see **Table**), but the difference in event-free survival between the sustained hypertension group and both the white-coat hypertension and normotensive groups was highly significant (P=.002).[2]

TABLE

Cohort studies of patients with white-coat hypertension

Patients	Outcome	Total number of events			*P* value
		NT	WCH	SH	
479 patients, mean age of 64[1]	Cardiovascular events	N/A	15 (11.9%)	83 (23.5%)	*P*<.001
1392 patients, mean age of 51[2]	Cardiovascular events	4 (1.9%)	3 (1.3%)	37 (5.3%)	NT vs WCH: *P*=.83
					WCH vs SH: *P*<.0001
566 patients, mean age of 48[3]	Cardiovascular events	10 (6.8%)	14 (18.4%)	56 (16.3%)	Overall *P*=.03
					NT vs WCH: *P*=.03
					NT vs SH: *P*=.01

NT, normotensive; WCH, white-coat hypertension; SH, sustained hypertension

In contrast, a recent 10-year longitudinal study of 146 normotensive people, 76 people with white-coat hypertension, and 344 with sustained hypertension showed that cardiovascular event rates were similar for patients with white-coat and sustained hypertension, and were significantly higher than in the normotensive group (*P*=.03 overall, *P*=.03 between white-coat hypertension and normotension and *P*=.01 between sustained hypertension and normotension).[3]

One randomized trial evaluated outcomes of antihypertensive therapy for white-coat hypertension for patients aged >60 years. Ninety-nine patients with white-coat hypertension were identified on the basis of systolic blood pressure greater than 160 mm Hg in clinic and normal 24-hour ABPM and were randomized to either placebo or drug therapy. Active treatment did not significantly lower ambulatory blood pressure in white-coat hypertension, but it did reduce blood pressure measured in clinic. After a year, medication produced an absolute reduction in cardiovascular events of 8.6%, and in stroke of 4.2%. Neither result was statistically significant due to the small sample size.[4]

RECOMMENDATIONS FROM OTHERS

The American College of Cardiology and American Academy of Family Physicians have made no specific recommendations about white-coat hypertension. The Blood Pressure Monitoring Task Force V concluded that a significant number of white-coat hypertension patients become truly hypertensive over years of follow-up.[5]

Experts agree that patients with white-coat hypertension should be indefinitely monitored for the development of sustained hypertension.[6] Treatment is not needed unless the patient has sustained hypertension, evidence of cardiovascular disease, or signs of target organ injury.[7,8] Typically, expert opinion recommends confirming the diagnosis of white-coat hypertension with home blood pressure records or ambulatory blood pressure monitoring.

Shobha Rao, MD, Chun-Tsai Liu, MD, Laura Wilder, MLIS, *University of Texas Southwestern, Dallas*

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CLINICAL COMMENTARY:

White-coat hypertension represents one point along the continuum of hypertension

Unfortunately, the best available clinical evidence provides an unfulfilling answer to the question posed by this Clinical Inquiry. It requires inductive reasoning and logic to derive a treatment plan from the evidence presented. Perhaps it is because the diagnosis of white-coat hypertension remains poorly defined and clinically elusive.

Nevertheless, application of the simple principle of "where there's smoke, there's fire" fits best here. Clinicians should be aware that white-coat hypertension represents one point along the continuum of hypertensive disease. When diagnosed, patients with white-coat hypertension should at a minimum be followed for associated morbidities and treated when systemic hypertension is identified.

Mark B. Stephens, MD, MS, *Uniformed Services University, Bethesda, Md*

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Does a short symptom checklist accurately diagnose ADHD?

EVIDENCE-BASED ANSWER

Several abbreviated checklists perform well in distinguishing children with attention deficit/hyperactivity disorder (ADHD) from those without ADHD under ideal conditions and in research settings. While many guidelines and experts recommend using these checklists as an efficient method to collect data from multiple sources (strength of recommendation: **B**, based on extrapolation from cohort studies to define test characteristics and consensus opinion), experts point out the subjective nature of responses on behavior rating scales, and the limitations in using checklists as the sole source of information.

The Swanson, Nolan, and Pelham (SNAP) checklist from the *Diagnostic and Statistical Manual of Mental Disorders*, revised 3rd edition (*DSM-III-R*) has been shown to have a sensitivity and specificity in excess of 94% to distinguish hyperactive, inattentive, and impulsive children with ADHD from those without ADHD. This was based on criteria in the *DSM-III-R*. The *DSM-IV* SNAP checklist (available at www.adhd.net/snap-iv-form.pdf; scoring at www.adhd.net/snap-iv-instructions.pdf), based on the newer diagnostic criteria, has not been adequately evaluated. The ADHD Rating Scale-IV (in DuPaul et al, *ADHD Rating Scale IV—Checklists, Norms, and Clinical Interpretations,* available from Guilford Press) and the ADD-H Comprehensive Teacher/Parent Rating Scale (ACTeRS; available from MetriTech, Inc at www.metritech.com) are useful for their brevity, but they do not perform as well in differentiating children with ADHD from those without ADHD.

EVIDENCE SUMMARY

A variety of brief ADHD-specific rating scales are used for both parent and teacher assessment of child behavior. Rating scales are generally

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TABLE

Descriptive characteristics of abbreviated symptom checklists for ADHD

Scale	Minutes	# Items	Age	Effect size		
				Hyperactivity	Inattention	Impulsivity
ACTeRS Parent Version	5–10	25	5–12	1.5	2.0	NA
ACTeRS Teacher Version	5–10	24	5–12	NA	NA	NA
***DSM-IV* SNAP**	5–10	40	6–12	NA	NA	NA
***DSM-III–R* SNAP**	5–10	38	6–12	3.1–5.1	3.5–4.2	4.0–5.5
ADHD Rating Scale-IV	5	18	5–18	1.1	1.2	1.1
Conners Rating Scale,Revised (1997, Short Version)[11,12,13]	5–10	27	3–17	NA	NA	NA

Numbers reported in ranges indicate multiple studies.
ACTeRS, ADD-H Comprehensive Teacher Rating Scales; *DSM, Diagnostic and Statistical Manual of Mental Disorders*; SNAP, Swanson, Nolan, and Pelham; ADHD, attention deficit/hyperactivity disorder; NA, not available.

evaluated to establish mean scores for affected and unaffected children. Many scales publish such normative data in commercially available manuals. Some scales have been evaluated by 1 or more independent studies to compare children with and without ADHD. Rating scales have not been evaluated as a sole tool for the diagnosis of ADHD.

The test characteristics of a particular scale depend on the cut points for a positive or negative test. The usefulness of psychological tests in discriminating normal from abnormal behavior is often reported as "effect size." The effect size is the difference in mean scores between 2 populations divided by an estimate of the individual standard deviation.[1] An effect size of 4.0 means that abnormal subjects and normal controls are separated 4 standard deviations and thus almost completely separated. An effect size of 1.0 shows significant overlap between the 2 populations. An effect size of 4.0 is roughly equivalent to a sensitivity and specificity of 97%. An effect size of 1.0 is roughly equal to a sensitivity and specificity of 71%.

Table 1 outlines the characteristics and effect size of several available brief ADHD-specific checklists.[2–4,6,11–13] Typically, the gold standard was a clinical diagnostic interview, usually conducted by a clinical psychologist, as well as supporting data from schools and parents.

RECOMMENDATIONS FROM OTHERS

The American Academy of Pediatrics states that the use of ADHD-specific checklists is a clinical option when evaluating children for ADHD. They caution that the ADHD scales may function less well in clinicians' offices than suggested by reported effect size and, in addition, rating scales are subject to bias and may convey a false sense of validity. They also state that it is not known if these scales provide additional information beyond a careful clinical assessment.[7]

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Often scales miss more passive, less disruptive ADHD children and overdiagnose high-energy children

The Institute for Clinical Systems Improvement recommends use of at least 1 ADHD-specific rating scale to be administered to parents and teachers. This information should be used as part of the overall historical database for the child and should not be used as the sole criteria for diagnosis of ADHD.[8]

Many sources agree that ADHD-specific rating scales allow a rapid and consistent collection of information from multiple sources. However, the information they provide is necessary, but not sufficient, to make a definitive diagnosis of ADHD. In addition to assisting in diagnosis, checklists can be helpful in monitoring treatment changes once a diagnosis has been established.

Adam J. Zolotor, MD, MPH, *Department of Family Medicine, University of North Carolina, Chapel Hill;*
Jill Mayer, MLS, *Health Sciences Library, University of North Carolina, Chapel Hill*

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CLINICAL COMMENTARY:

Gather data from multiple sources

Sorting out children with ADHD, bipolar disorder, or learning disabilities from lively or distractible children is not a simple matter. Often the objective rating scales miss the more passive, less disruptive, inattentive ADHD children while overdiagnosing high-energy children as having ADHD. Perhaps the new *DSM-IV* SNAP will provide the objective sensitivity and specificity we desire as clinicians. However, this checklist requires further evaluation.

Information from ACTeRS scales has helped me treat these children, but I prefer to have both parents, if possible, independently complete the form. Obtaining scales from a Special Education teacher or psychologist, when available, in addition to the primary classroom teacher, is invaluable. Still, it often comes down to how a child responds to medication. Proceed with caution if there is a family history of bipolar disorder, as these children often do worse on stimulants and are better treated by our colleagues in child psychiatry.

John Hill, MD, *Rose Family Medicine Residency/ University of Colorado Health Sciences Center, Denver*

CONTINUED

Should home apnea monitoring be recommended to prevent SIDS?

EVIDENCE-BASED ANSWER

While home apnea monitoring may find an increased incidence of apnea and bradycardia in preterm infants compared with term infants, no association links these events with sudden infant death syndrome (SIDS). Apnea of prematurity is not a proven risk factor for SIDS. Since apnea of prematurity has not been shown to be a precursor to SIDS, home apnea monitoring for the purpose of preventing SIDS cannot be recommended (strength of recommendation [SOR]: **B**, based on a single prospective cohort study and multiple case-control studies). Neonates with significant neurologic or pulmonary disease may benefit from apnea monitoring (SOR: **C**, expert opinion).

EVIDENCE SUMMARY

Multiple case-control studies have identified risk factors for SIDS, which are presented along (with odds ratios) in **Table 1**.[1–6] None of these case-control studies found apnea of prematurity to be a risk factor for SIDS.

A prospective cohort study of 1079 infants monitored for cardiorespiratory events, the Collaborative Home Infant Monitor Evaluation (CHIME) study, demonstrated that prior to 43 weeks postconceptional age, preterm infants had a statistically significant greater risk of extreme events (apnea or bradycardia longer than 30 seconds) compared with healthy term infants (**Table 2**). After 43 weeks postconceptional age, there were no differences in incidence of apnea or bradycardia, comparing preterm and term infants. Neither preterm infants nor infants with apnea, bradycardia, or apparent life-threatening events had increased incidences of SIDS.[7]

Significant financial costs are associated with home monitoring. The average monthly cost is $300 to $400, not including physician fees. This would lead to an estimated annual cost of $24 million dollars if every infant <1500 grams in the United States were monitored.[8]

The psychological costs of home apnea monitoring have also been studied. One hundred and four parents of monitored and unmonitored infants were enrolled in a questionnaire study to determine emotional distress and family functioning. As is common among families in the postpartum period, all experienced increased stress. But parents of monitored infants, compared with parents of unmonitored infants, had an increased incidence of subjective depression (number needed to harm [NNH]=7) and hostility (NNH=12) at 2 weeks postpartum. Interestingly, at 1-year follow-up interviews, 83% of parents who had consistently used the monitor reported feeling more secure for having used it and 69% believed that monitor use had been helpful.[9]

RECOMMENDATIONS FROM OTHERS

The American Academy of Pediatrics (AAP) acknowledges that no established predictive or precursor relationship exists between prolonged apnea and SIDS, stating that the "prevention of SIDS is not an acceptable indication for home cardiorespiratory monitoring." They issue a weak recommendation that home cardiorespiratory monitoring may be necessary for recurrent apnea, recurrent bradycardia, hypoxemia, chronic lung disease, and technology-dependent infants. Finally, they state that monitoring should be discontinued at 43 weeks postconceptional age or after cessation of extreme cardiorespiratory events, whichever occurs last. The AAP recommends proven practices such as supine sleeping position, a safe sleeping environment, and elimination of prenatal and postnatal exposure to tobacco smoke to decrease the risk of SIDS.[8]

Michael Shoemaker, MD, Mark Ellis, MD, MSPH,
Cox Family Practice Residency, Springfield, MO;
Susan Meadows, MLS, *Department of Family and Community Medicine, University of Missouri-Columbia*

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TABLE 1

Risk factors for SIDS

Risk factor	Odds ratio (95% CI)
Maternal factors	
Transport problems for prenatal care[1]	11.8 (2.7–52.7)
Education ≤12 years[1]	4.2 (1.1–15.5)
Prenatal smoke exposure[3]	3.7 (2.9–4.6)
<7 prenatal visits[1]	3.3 (1.1–9.8)
Unmarried[3]	2.0 (1.6–2.5)
Paternal factors	
Education ≤12 years[1]	8.8 (1.1–70.8)
Parental factors	
Parental smoking[4]	5.19 (2.26–11.91)
Passive smoke exposure—all sources[5]	3.50 (1.81–6.75)
Maternal consumption of alcohol	
First trimester[1]	6.7 (2.2–20.1)
Any trimester[1]	3.4 (1.4–10.9)
Binge drinking—first trimester[1]	6.3 (1.8–22.8)
Binge drinking—any trimester[1]	3.9 (1.4–10.9)
Infant care	
<3 well-child visits[1]	13.8 (1.7–109.9)
Sleeping prone[4]	6.96 (1.51–31.97)
≥2 layers of clothing[1]	3.9 (1.4–10.9)
Routine use of reused mattress[2]	3.1 (1.5–6.2)
Drug treatment in previous week[4]	2.33 (1.10–4.54)
Infant demographics	
Low birth weight (≤2500 g)[3]	3.6 (2.4–5.2)
Black[3]	2.5 (1.6–3.9)
Male gender[6]	1.47 (1.26–1.70)

Table adapted from multiple case-control studies.

CLINICAL COMMENTARY:

Apnea monitors are not the answer

An episode of SIDS is devastating to parents and leaves physicians questioning what more could have been done to prevent the tragedy. Apnea monitors, however, are not the answer. There are clearly downsides to apnea monitors and the added stress they place on parents. I do not think anyone would argue this would be a small price to pay if they helped to prevent SIDS; unfortunately, this is not the case.

I find it interesting that although apnea monitors add stress to parents, most would use them again and many felt they were helpful. This highlights the importance of education and clear communication with parents about SIDS and its prevention. Anecdotally, I have yet to have parents who did not stop using apnea monitor early because of the constant false alarms.

Matthew Gannons, MD, *Departments of Family and Community Medicine and Orthopaedic Surgery, Medical College of Wisconsin*

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