both unnecessary warfarin dose adjustments and vitamin K administration remain guiding principles of anticoagulation management. Patients should be encouraged to monitor their INR at a consistent cave site, thereby avoiding unstable INRs introduced when different locations use different monitoring devices or reagents.

High-quality anticoagulation management services typically know these risks and have standard operating procedures to confirm elevated POC INR results and monitor agreement between CL INR and POC INR. For example, at our institution we confirm with CL INR all POC INR results >5.0, and base all warfarin adjustments on the CL INR value. In addition, we perform annual CL INR to POC INR correlation studies, as well as competency testing for healthcare providers performing POC INR testing. These quality assurance measures heightened our awareness of INR discordance within our institution and led us to develop a POC INR correction factor.

**POC INR Correction Factors**

In spite of efforts to harmonize PT assays with the advent of the INR, inter-laboratory variability likely will persist until the medical community reaches consensus on a standardized thromboplastin preparation. Although most institutions consider the CL INR as the “gold standard” or off reference standard, the absence of large clinical trials demonstrating superiority of one INR method over the other adds to the controversy. Until further standards or clinical trials become available, CL INR and POC INR discrepancies will remain, leaving healthcare providers wondering which to believe.

In an attempt to remove some of this confusion and harmonize INR results, some anticoagulation management services have derived correction factors that can be applied to POC INR values to predict the CL INR value (9, 10). These correction factors seek to minimize the difference between POC INR and CL INR results and clarify subsequent clinical decisions. For example, within our institution applying a correction factor to POC INRs >3.0 improved agreement to the CL INR (within ±15%) in more than 70% of tests (9). A Bland-Altman plot comparing uncorrected (Figure 1) and corrected POC INRs highlights the effectiveness of this approach (Figure 2). Applying this correlation factor in our institution reduced differences in clinical decision making by 43%.

These findings have altered our clinical approach to POC INRs, and for values between 3.0-5.0, we routinely apply a correction factor to estimate the CL INR. Similarly, Richter et al. demonstrated the feasibility of applying a POC INR correction factor to guide warfarin dosing among patients with supratherapeutic POC INRs (>4.0), compared to confirmatory venipuncture samples. The differences in warfarin dosing decisions were negligible and the corrected POC INR approach was more efficient (10).

The methodology used to derive a correction factor is simple (linear regression) and has been published elsewhere (9, 10). POC INR devices may overestimate or underestimate the CL INR. This means that correction factors are device- and institution-specific and cannot be directly applied across institutions with different POC devices and lab analyzers. In addition, changes in reagent lots, lab analyzers, POC devices, or test strip lots may influence the correction factor equation. Laboratories should recalculate their correlation factor every 6-12 months and following any significant changes in lab equipment.

**Clinical Case Continued**

The patient described at the beginning of this review has multiple stroke risk factors in the setting of atrial fibrillation. Based on the stroke risk prediction scoring system, called CHA2DS2-VaSc, she has 6 points, placing her in the moderate-high risk group with an approximate 9.7% annual risk of stroke. The pharmacist is appropriately concerned about the patient’s elevated POC INR of 4.0, and must balance the small increased risk of bleeding with the patient’s underlying stroke risk. An inappropriate warfarin dose reduction could result in a subtherapeutic INR and inadequate protection from a stroke or systemic embolism. The additional history obtained from the patient did not uncover any new medications that would interfere with the POC INR device (e.g., low molecular-weight heparin). The patient’s hematocrit was normal at