Stabilization of Homogeneously Precessing Domains by Large Magnetic Fields in Superfluid ^{3}He-B

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We have studied the catastrophic relaxation in superfluid ^{3}He-B as a function of magnetic field for a sample pressure of 31 bars. "Catastrophic relaxation" refers to a novel magnetic relaxation process which rapidly disrupts the homogeneous precession of nuclear spins in NMR experiments on the B phase. The catastrophe was observed through its effect on the evolution of a long-lived coherent dynamic state, the homogeneously precessing domain. Our measurements reveal that the onset of catastrophic relaxation is suppressed to lower temperatures by a strong magnetic field.

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When a closed volume of superfluid ^{3}He-B in a magnetic field is subjected to a nuclear magnetic resonance (NMR) pulse of large tipping angle ($\theta_{\rm rf} \sim 104°$), a free induction decay signal is observed with a duration much greater than that expected from the dephasing by the inhomogeneity of the applied magnetic field alone. Under typical experimental conditions, the time constant of the decay in the normal Fermi liquid is limited by field inhomogeneities to $T_2^* = 1/(\gamma\Delta H) \approx 6-10$ ms, but in the B phase signals lasting hundreds of milliseconds were observed long ago in large tipping angle NMR experiments [1,2]. Application of an additional linear gradient of several G/cm reduces the T_2^* of the normal liquid in proportion to $1/\Delta H$ across the sample but reduces the duration of the B phase signal rather weakly. This phenomenon results from the existence of spin supercurrents, as described by Fomin [3] and demonstrated in an important series of experiments by Borovik-Romanov *et al.* [4]. After the rf tipping pulse, the spins begin to dephase due to the field gradient. However, gradients in the phase of the order parameter drive spin supercurrents which carry longitudinal and transverse components of the magnetization toward opposite ends of the cell, respectively, redistributing magnetization until a stable two-domain structure is formed. On the high field side of the cell, the static domain (SD) forms with the net magnetization pointing along the field, producing no induction signal. On the low field side, the magnetization precesses coherently at a deflection angle $\theta \sim 104°$ in spite of the field gradient; this is the homogeneously precessing domain (HPD). This dynamic precessing state is stable because the B phase frequency shift for deflection angles greater than 104° compensates for the applied gradient such that the precession frequency is equal to the Larmor frequency at the boundary between the SD and HPD. Unless continuous rf energy is supplied, the domain will subsequently lose energy through various dissipation processes and will shrink, with the domain wall moving towards the low field end of the sample space.

It has been shown in previous work that the homogeneously precessing domain is a useful tool for the study of spin dynamics in the isotropic superfluid. For example, two independently driven HPDs have been used to demonstrate macroscopic quantum coherence and phase slips through a weak link [5]. HPD spectroscopy has also been used to measure the spin diffusion coefficient $D_\perp$ as a function of temperature and pressure, which is not possible with simple spin echo techniques [6].

The study of HPDs appears limited, however, at lower temperatures where a sudden, vast increase in the magnetic relaxation rate arises. Other experiments at a single value of magnetic field have shown that the catastrophic relaxation anomaly occurs at a temperature $T_{\rm cat}$ between about $0.3T_{\rm c}$ and $0.5T_{\rm c}$, depending on the pressure [7]. In spite of its sharp onset, catastrophic relaxation is not fully characterized with respect to experimentally accessible parameters, in the sense that there is not precise agreement on the exact value of $T_{\rm cat}/T_{\rm c}$ even at a single pressure [7–9].

In this work we report the first evidence that the homogeneously precessing domain can be stabilized to lower temperatures by a strong magnetic field. Previous experiments over a narrow range of low fields observed no field dependence to the onset of catastrophic relaxation [8]. In extending the range of fields studied, however, we find a monotonic dependence on external field which is not expected from current ideas regarding the catastrophe. This may provide useful information in developing a theory for catastrophic relaxation. Also, the suppression of catastrophic relaxation should allow lower temperature measurements of the spin diffusion constants and of the effective Leggett-Takagi relaxation time in the future [6].

Our experimental sample cell, shown in Fig. 1, was constructed from two nested coaxial tubes of epoxy. This allowed us to maintain as large a filling fraction as possible ($\eta = 0.14$) within the NMR coils while also having a vibrating wire thermometer outside of the rf region. The outer tube was cast onto a coin silver flange which bolted onto an array of annealed silver rods attached to our Cu nuclear demagnetization stage. At the end opposite

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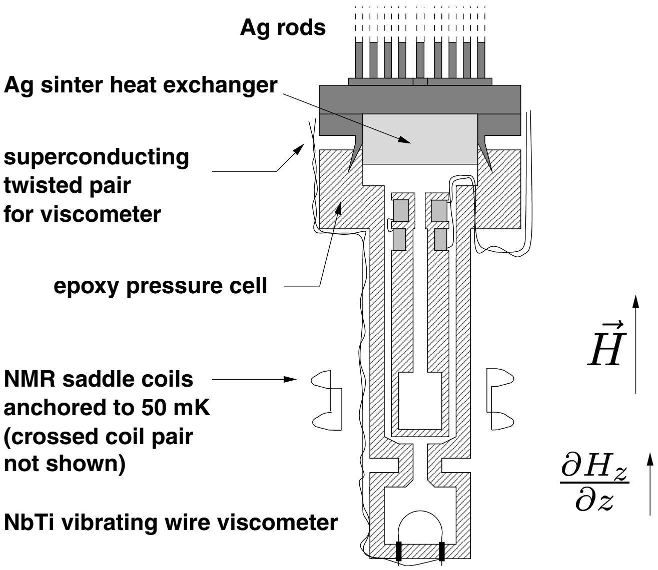

FIG. 1. The epoxy cell with NMR chamber and viscometer.

this flange was mounted a NbTi vibrating wire thermometer. The vibrating wire ($d = 0.127$ mm) was chosen as the main thermometer in these experiments for several reasons, the most important of which is that its resonance width is rather independent of magnetic field for the range of fields studied [10]. The viscometer also measures the temperature of the liquid directly, without the added time lag from a thermal impedance between the liquid and a sintered metal. The annular volume of helium between the tubes gave the viscometer a thermal time constant of approximately 3 min at 1 mK.

The inner tube of epoxy forming the HPD region had a diameter of 0.95 cm and a length of 1.01 cm, and was closed on the end nearest the vibrating wire. The tube necked down to a diameter of 3.17 mm at the other end of the NMR space. This column of liquid helium provided substantial thermal contact to the Ag sinter heat exchanger above it. The inner cell was manufactured by casting the epoxy around a mold of polished aluminum, in order to minimize the surface roughness. The Al mold was subsequently removed by etching in a concentrated solution of NaOH. The resulting finish was smooth to about 1 μm.

The NMR apparatus consisted of a pair of crossed saddle coils mechanically and thermally mounted on the 50 mK shield of the dilution refrigerator. For fields of 519 G and below, the coils were tuned with capacitors at the head of the cryostat. Above 519 G, the coils were untuned, but the large sample and higher frequency compensated somewhat for the loss in sensitivity. A home-built pulsed NMR homodyne spectrometer was used which provided in-phase and quadrature outputs.

At high temperatures in the B phase, the NMR pulse length was adjusted to generate the longest duration HPD. The optimal pulses were of approximately 120°, which corresponded to 30 μs pulses when the coils were tuned at 344 G and to as much as 680 μs when the coils were left untuned at 951 G. The copper refrigerant was then demagnetized down to the lowest temperature while we recorded the HPD signal. This allowed us to obtain an initial rough value of the temperature at which catastrophic relaxation occurs as the sample cooled. Because of imperfect compensation of the demagnetization field in the sample region, it was necessary to vary the NMR frequency by a few hundred Hz in order to obtain the optimal HPD signal, given the narrow spectral width of our rf pulses. Because of the $1/T^2$ dependence of the specific heat of the copper refrigerant, it was more convenient to sweep the temperature back up through a series of short, slow remagnetizations rather than to heat the sample directly. In this way the region of catastrophic relaxation was carefully studied for each value of the magnetic field as the sample was warmed.

Our original intent was to investigate the properties of HPDs at substantially higher fields than had been previously studied, where the order parameter becomes anisotropic. The first run was attempted in a static field of 1.6 kG with a linear gradient of 0.2 G/cm parallel to the field. As we have recently reported [11], the decay signals steadily increased in lifetime with decreasing temperature up to 6 s at our lowest temperature (Fig. 2). This is about an order of magnitude longer than typical decays at similar pressures in previous work [4]. Because the decays are nonexponential, corresponding to shrinkage of the domain along the direction of the field, the lifetime was measured from the beginning of the signal to the point where it vanished into the noise [12]. Down to $0.25T_c$, the lowest temperature we achieved, there was no clear evidence of catastrophic relaxation; the signal length seemed to reach a plateau at 6 s, but this may have been associated with our inability to cool the ^{3}He further. Modifying our spectrometer to work as a heterodyne detector, we found

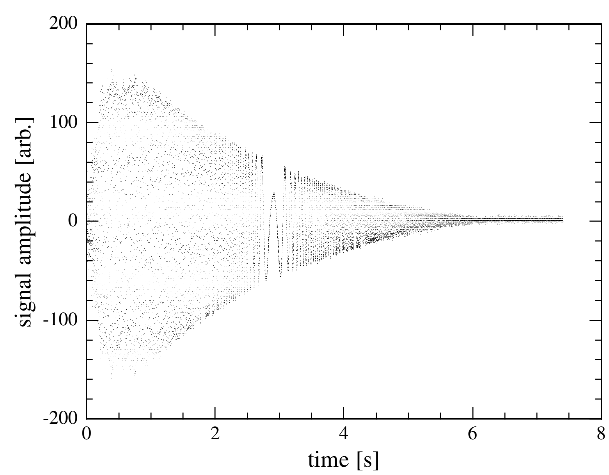

FIG. 2. A long decay signal at $0.25T_c$, 1.6 kG. The Larmor frequency decreases during the decay, and a zero beat is seen at $\sim$3 s where the signal frequency crosses the detector frequency of the spectrometer.

that the frequency of the signal decreased during the decay, ensuring that we were observing the HPD and not the persistent induction signal which has been discovered at lower temperatures [12].

Because the absence of catastrophic relaxation is unexpected given previous work placing its onset between $0.3T_c$ and $0.5T_c$, we decided to return to lower fields in order to restore the feature. We measured the HPD lifetime versus temperature for seven lower fields from 207 to 951 G, all of which exhibited catastrophic relaxation within our restricted temperature range. The results for all fields are shown together in Fig. 3. In each case, the onset of the catastrophe is abrupt, and the HPD does not recover in lifetime at all below the catastrophe temperature, which is in agreement with the high pressure data of other groups [7]. Above the catastrophe temperature, the curves of HPD lifetime follow the profile calculated assuming that the main mode of dissipation is spin diffusion across the SD-HPD boundary. In this case the lifetime should be proportional to $(1 - T/T_c)^{1/3}/D_{zz}^{\perp}(T)$ when the static field, the field gradient, and the size of the HPD are constant throughout the temperature sweep [6,13]. The new feature, though, is that the temperature for catastrophic relaxation moves to lower temperatures as the static field is increased. If one takes as the catastrophe temperature the point on each curve at which the lifetime has decreased by $1/2$ from its maximum value (which is relatively insensitive in temperature to the exact value of this maximum, because the onset is so sharp), one obtains the curve in Fig. 4. To verify that this falloff of the HPD lifetime is due to catastrophic relaxation, we also studied free induction decay signals for lower tipping angles ($\theta = 30°$) at several of the fields. The onset of more rapid relaxation approximately coincided with the destruction of the HPD, as was observed before by the Moscow group [8].

One interesting model for catastrophic relaxation involved a cross relaxation of precessing modes in the superfluid [7,14]. It was noted in previous experiments that the onset of catastrophic relaxation occurred near the temperature at which the magnitude of the molecular field equals that of the applied field. In the collisionless regime the magnetizations of the condensate and of the quasiparticles may decouple and undergo a mutual precession about the molecular field $\mathbf{H}_L = -F_0^a \mathbf{M}/\chi_{n0}$, where $\mathbf{M}$ is the net magnetization and χ_{n0} is the bare susceptibility without Fermi liquid corrections. This internal precession could absorb energy from the precession of the total magnetization if the frequencies of these motions were equal, but the

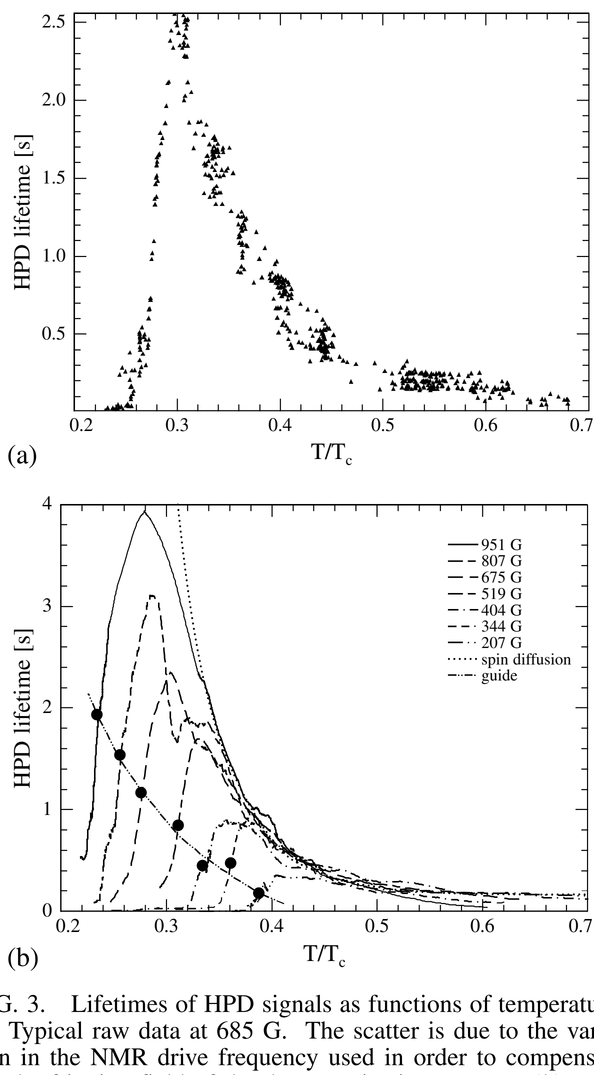

FIG. 3. Lifetimes of HPD signals as functions of temperature. (a) Typical raw data at 685 G. The scatter is due to the variation in the NMR drive frequency used in order to compensate for the fringing field of the demagnetization magnet. (b) Temperature sweeps for all fields. The curves nearly overlap for temperatures above their respective points of catastrophic relaxation. This common envelope agrees with the signal lifetimes calculated assuming that spin diffusion across the domain wall is the dominant cause of relaxation of the HPD [6,13]. The half-height points of the catastrophe are shown along with a curve to guide the eye.

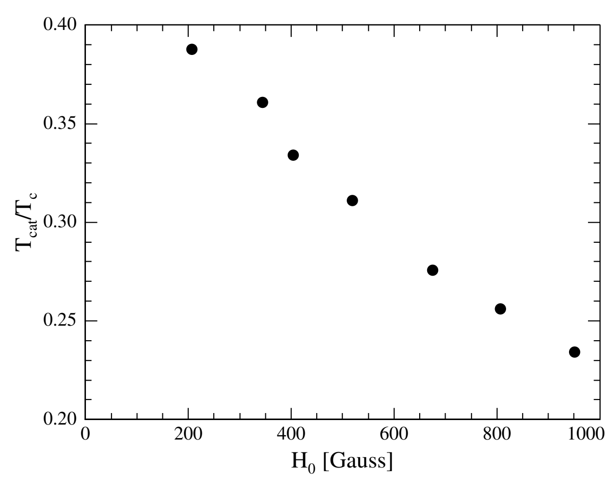

FIG. 4. Plot of the relative temperature of catastrophic relaxation as a function of static field. T_{cat} is taken as the point at which the lifetime has fallen by half from its maximum.

internal precession would be highly damped by Leggett-Takagi relaxation. The expression for the ratio of fields or frequencies in terms of the Fermi liquid parameters is

$$\frac{H_L}{H} = -\frac{F_0^a(2 + Y(T))}{3 + F_0^a(2 + Y(T))}. \quad (1)$$

For the accepted values of F_0^a at pressures above about 15 bars, H_L/H approaches but never crosses unity as $T/T_c \rightarrow 0$, and for the 31 bars used in our experiments $H_L/H \geq 1.01$ for all temperatures. In that case, the theory can account only for our scans if there is some field dependent threshold value of H_L/H greater than unity at which the rapid relaxation sets in. However, if F_0^a is at least 1% smaller than the accepted value for 31 bars so that a crossing can occur, then our experiment does not rule out the Landau field model; H_L/H would increase with field above the value given by Eq. (1) because of the enhancement of susceptibility through distortion of the *B* phase energy gap [15].

Another possibility is that catastrophic relaxation occurs due to the development of instabilities [16] which nucleate at the cell walls by the mechanism originally proposed by Ohmi *et al.* [17]. At equilibrium in a magnetic field, we expect our sample to be in the well-known flare-out texture [18]. In the center of the cell, $\hat{\mathbf{n}}$ will line up parallel to the magnetic field. Within a magnetic healing length l_H of the wall, $\hat{\mathbf{n}}$ will bend such that at the wall it forms an angle of 63.5° with respect to the radial direction and 60° from the static field. Inside the HPD, though, $\hat{\mathbf{n}}$ is precessing in the plane perpendicular to the applied field. The order parameter in the HPD may dephase at the walls where $\hat{\mathbf{n}}$ relaxes back to the original boundary conditions, and the rate of this dephasing or relaxation may therefore be sensitive to the value of the bending length, which varies with the field as $1/H$.

While the lifetime of the HPD is not a fundamental property and depends on the conditions of formation, these data do demonstrate that catastrophic relaxation dramatically alters the decay rates of the signals and that the temperature at which it occurs is strongly dependent on magnetic fields. As we were concerned that we might be observing a change dependent on the conditions under which we were attempting to form HPDs, we varied the rf pulse length and amplitude. No combination of these parameters was able to restore the long decay times below the point of catastrophe, demonstrating that there is a true onset of rapid relaxation in the ^{3}He itself. The truncated decay signals observed by pulsed NMR of small tipping angles also reinforces this result, ruling out a systematic error or measurement artifact.

In conclusion, we have demonstrated that HPDs can be observed at higher fields than those at which they have previously been systematically studied. In addition we have observed that a strong applied field suppresses the catastrophic relaxation. This opens up phase space for the study of spin supercurrents through HPDs to lower temperatures and to a region in which the order parameter becomes anisotropic as a result of the gap distortion induced by high magnetic fields. It is hoped that further experimental and theoretical study will reveal the correct explanation for catastrophic relaxation and its suppression by **H** observed in this work.

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