



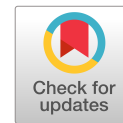
## **Distribution-Theoretic Basis for Hidden Deltas in Frequency-Domain Structural Modeling**

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# Distribution-Theoretic Basis for Hidden Deltas in Frequency-Domain Structural Modeling

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**Abstract:** Frequency-domain modeling is a core tool for the analysis of linear time-invariant structures. In a process that has been unclear, additional Dirac delta distributions can arise in the frequency-domain transfer functions of certain structures, beyond those seemingly given by the structural model—e.g., in the mechanical impedance of a linear spring. Previous analyses have manually appended these “hidden deltas” to the relevant transfer functions in to ensure that they remain causal, but questions remain as to their exact origin and behavior in noncausal models. Here, we demonstrate that these hidden deltas arise from the theory of distributions and the solution of the distributional division equation. We demonstrate a rigorous and reliable method for deriving these hidden deltas in which the role of causality constraints are made clear. Furthermore, we demonstrate that the appropriate frequency-domain conditions for causality in such systems are generalized—not classical—Hilbert transform relations, and that the process of appending delta distributions is related to the analysis of causality via these generalized relations. DOI: [10.1061/JENMDT.EMENG-7898](https://doi.org/10.1061/JENMDT.EMENG-7898). This work is made available under the terms of the Creative Commons Attribution 4.0 International license, <https://creativecommons.org/licenses/by/4.0/>.

## Introduction

Several decades of research (Titchmarsh 1948; Makris and Efthymiou 2020) have shed significant light on the relationship between frequency-domain models of structural phenomena, and the causality of these phenomena: their relationship to the directional nature of time, and whether they respect it. The constraints of causality provide insight into the behavior of viscoelastic constitutive models—including in exact or approximate rate-independent damping models (Keivan et al. 2017; Makris 1997a; Pons 2023), fractional-order models (Enelund and Olsson 1999; Makris and Efthymiou 2020), and power-law media (Gulgowski and Stefański 2021; Kelly and McGough 2009). They also allow the identification of viscoelastic loss moduli based only on storage moduli behavior (Madsen et al. 2008). Analyses of structural model causality are relevant to seismology (Meza-Fajardo and Lai 2007; Deng and Morozov 2018), seismic analysis (Keivan et al. 2017), rheology (Shanbhag and Joshi 2022; Makris and Efthymiou 2020), biomechanics (Kelly and McGough 2009; Madsen et al. 2008; Pons 2023), hydrodynamic wave-energy conversion (Faedo et al. 2017), aeroelasticity (Park et al. 2014), and the study of metamaterials (Srivastava 2021).

It has also been known for several decades (Crandall 1991; Makris 1997b) that under certain conditions, an unusual phenomenon can arise within these lines of analysis. In certain simple, causal, models, the well-established derivation of frequency-domain transfer functions leads to model formulations that are noncausal—a contradiction with the known behavior of the model, and an apparent error in established derivations. The widespread conventional approach (Crandall 1991; Falnes 1995; Makris 1997b, 2017, 2018;

Faedo et al. 2017) is to manually append Dirac delta distributions to these transfer functions so as to ensure causality—the “hidden deltas” (Makris 1997b). This process resolves the causality violation, but it raises several questions. Why is manual correction required? What is missing in the analysis such that these deltas do not arise naturally? Will similar hidden deltas arise in more complex transfer functions?

Here, we use the theory of distributions (Schwartz 1957) to resolve these questions. We demonstrate that the hidden deltas arise from the solution of the distributional division equation: a rigorous basis for these terms that predicts their presence in general transfer functions. Distribution division connects these hidden deltas in causal structural models with the nonunique deltas that are observed in noncausal models (Makris 1997b); both arise from the nonuniqueness of distributional division, with causality a constraint forcing uniqueness. In addition, we show how distributional division is closely connected to frequency-domain causality analysis. Distributional analogues of Titchmarsh’s theorem and the Kramers–Kronig relations allow causality analysis in the frequency domain, for a restricted space of distributional transfer functions, but we identify that the generalization of this theorem due to Beltrami and Wohlers (1966) significantly extends the space—including, to the case of constant or improper transfer functions that typically present challenges for frequency-domain causality analysis (Carcione et al. 2019; Makris 2018; Waters et al. 2000). In this way, distribution-theoretic principles not only elucidate aspects of frequency-domain structural analysis that have previously been opaque, but also provide new analysis routes for the study of causality in frequency-domain structural systems.

## Transfer Functions and Hidden Deltas

Following Makris (1997b, 2017), consider one of the simplest conceivable structures—a linear spring, in the time ( $t$ ) domain

$$F(t) = kx(t) \quad (1)$$

with force output  $F$  proportional to displacement input  $x$  via stiffness  $k$ . Note that if we redefined the input variable  $x$  to be velocity

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or acceleration, we would have a linear damper or inerter, respectively (Makris 2017); these structures can all be analyzed along the same lines. Taking the Fourier transform  $\mathcal{F}\{\cdot\}$ —that is

$$\begin{aligned}\mathcal{F}\{f(t)\} &= \hat{f}(\omega) = \int_{-\infty}^{\infty} f(t)e^{-i\omega t} dt, \\ \mathcal{F}^{-1}\{\hat{f}(\omega)\} &= \frac{1}{2\pi} \int_{-\infty}^{\infty} \hat{f}(\omega)e^{i\omega t} d\omega\end{aligned}\quad (2)$$

—of Eq. (1), we obtain the transfer function (TF) between force and displacement:

$$\hat{F}(\omega) = k\hat{x}(\omega) \quad (3)$$

Eq. (3) defines the dynamic stiffness of the spring as  $\hat{Q}_0(\omega) = \hat{F}/\hat{x} = k$ . Note that certain works, notably Nussenzveig (1972), reverse the sign of  $\omega$  in the Fourier transform, and so are sign-flipped with respect to this analysis. Based on Eqs. (1)–(3), we pose a pair of apparently simple questions. What is the mechanical impedance of the spring—the TF between force and velocity? (Findeisen 2000). And, the TF between force and acceleration? To define these TFs, we have the following well-established Fourier transform of a derivative:

$$\mathcal{F}\{\dot{x}(t)\} = i\omega\hat{x}(\omega) \quad (4)$$

Applying this relation to Eq. (3) in a normal manner leads to the TFs in:

$$\begin{aligned}\text{force/velocity: } \hat{Q}_1(\omega) &= \frac{\hat{F}}{i\omega\hat{x}} = -\frac{ik}{\omega}, \\ \text{force/acceleration: } \hat{Q}_2(\omega) &= \frac{\hat{F}}{-\omega^2\hat{x}} = -\frac{k}{\omega^2}\end{aligned}\quad (5)$$

in which we observe a problem, in that  $\hat{Q}_1(\omega)$  and  $\hat{Q}_2(\omega)$  are apparently *noncausal*—they do not respect the directionality of time, and the principle that effect should follow cause.

This noncausality can be observed directly in their inverse Fourier transforms, which represent the structure’s time-domain response to an impulse in the associated variable. Representing an impulse input with a Dirac delta distribution at  $t = 0$ ,  $\delta(t)$  (which we use without, as of yet, considering any deeper properties of distributions), then  $\mathcal{F}\{\delta(t)\} = 1$ , and via the inverse Fourier transform, we compute the time-domain responses,  $Q(t)$ , to the following:

$$\begin{aligned}\text{a velocity impulse: } Q_1(t) &= \frac{1}{2}k\text{sgn}(t), \\ \text{an acceleration impulse: } Q_2(t) &= \frac{1}{2}k\text{tsgn}(t)\end{aligned}\quad (6)$$

where  $\text{sgn}(t)$  is the signum function. Per Makris (1997b, 2017, 2018), these responses are noncausal: the impulse occurs at  $t = 0$ ; whereas nonzero response occurs back to  $t \rightarrow -\infty$ .

Where did the well-established analysis of Eqs. (1)–(5) go wrong? Previous studies have not addressed this question directly, but instead have manually modified the TFs of Eq. (1) to maintain causality (Crandall 1991; Faldes 1995; Makris 1997b, 2017, 2018; Faedo et al. 2017). With the arguments that one can add an impulse,  $\delta(t)$ , into the singularity of the TF without “an observer noticing” (Crandall 1991), and a motivation based on the derivative of the logarithm (Makris 1997b), these studies append additional distributional terms:

$$\begin{aligned}\hat{Q}_{1,\text{mod}}(\omega) &= -\frac{ik}{\omega} + \pi k\delta(\omega), \\ \hat{Q}_{2,\text{mod}}(\omega) &= -\frac{k}{\omega^2} + i\pi k\delta^{(1)}(\omega)\end{aligned}\quad (7)$$

where  $\delta^{(1)}(\omega)$  is the distributional first derivative of the Dirac delta. These appended terms are the “hidden deltas” (Makris 1997b), specifically formulated to solve causality violation:

$$\begin{aligned}Q_{1,\text{mod}}(t) &= kH(t), \\ Q_{2,\text{mod}}(t) &= ktH(t)\end{aligned}\quad (8)$$

for Heaviside step function  $H(t)$ . In practical terms, this modification restores causality—although it does not elucidate the error over Eqs. (1)–(5), nor does it indicate whether these hidden deltas might appear in other transfer functions. Interestingly, in the case of Eq. (7), these deltas may also be derived from a loose application of Titchmarsh’s theorem (the Kramers–Kronig relations) (Makris 1997b; Nussenzveig 1972), which expresses conditions for causality in a square-integrable TF in terms of the Hilbert transform. However, as Beltrami and Wohlers (1966) allude to, TFs such as Eq. (7) are neither square integrable ( $1/\omega$ ) nor ordinary functions ( $\delta$ ,  $\delta^{(1)}$ ), and thus are not admissible to a classical analysis, despite its correct results. The prevalence of distributions ( $\delta$ ,  $H$ ,  $\text{sgn}$ ) throughout this process suggests that distribution-theoretic principles are at work—to these we now turn.

## Hidden Deltas and the Distributional Division Equation

### Properties and Spaces of Distributions

In Eqs. (6) and (7), when we introduced the delta distribution,  $\delta(t)$ , we did so blithely. Distributions, in the sense of Schwartz (1957), do not map values in the sense of an ordinary function (e.g.,  $\mathbb{R} \rightarrow \mathbb{R}$ ). Instead, they approximate this mapping via an integral on a space of test functions—in the manner of a weak formulation. For details, see Pandey (2011) and Friedlander and Joshi (1998). By convention, we write distributions as functions, e.g.,  $\delta(x)$ , but they do not inherit all properties of ordinary functions—notably, in distributional differentiation ( $D^n$ ), which can be applied to singular functions; and in multiplication and division, which are not always defined, and may produce nonunique results. Various well-behaved functions, such as  $x$ ,  $x^2$ , etc., themselves define equivalent distributions, but the space of distributions also involves objects that do not correspond to any function—notably,  $\delta(x)$  and  $D^n\delta(x) = \delta^{(n)}(x)$ . Various singular or discontinuous functions can be given greater utility via distributional formulation:  $H(x)$ ,  $\text{sgn}(x)$ , and  $1/x$ ; the latter, with integration defined via Cauchy principal value, defines the distribution denoted p.v.( $1/x$ ). To analyze the causality of distributional TFs, we must define several spaces of distributions.

- The space of all distributions— $\mathcal{D}'$  (Nussenzveig 1972) or  $D'$  (Beltrami and Wohlers 1966).
- The space of *tempered distributions*— $\mathcal{L}' \subset \mathcal{D}'$  (Nussenzveig 1972), or  $S'$  (Beltrami and Wohlers 1966; Pfaffelhuber 1971). Tempered distributions are continuous in a distributional sense, which permits certain singularities; grow no faster than polynomial as  $x \rightarrow \infty$ ; and are the natural domain of the Fourier transform:  $\mathcal{F}$  maps a tempered distribution to another tempered distribution. Within  $\mathcal{L}'$  are:  $\delta(x)$ ,  $H(x)$ , p.v.( $1/x^n$ ) for all  $n$ , all polynomials, and all  $L^p$ -integrable functions with  $p \geq 1$  (King 2009).

- The space of *summable distributions*— $\mathcal{D}'_{L^1} \subset \mathcal{L}'$  (Beltrami and Wohlers 1966; Pandey 2011), or  $\mathcal{D}'_L$  (Nussenzweig 1972),  $\mathcal{D}'_{L^0}$  (Ishikawa 1987). Summable distributions can be expressed as a finite sum of the distributional derivatives of ordinary integrable ( $L^1$ ) functions—in analogy with the Sobolev space  $W^{n,1}$  for some  $n$ . They can also be defined in other  $L^p$  norms: We follow Nussenzweig's (1972) treatment of  $L^1$ ; but Beltrami and Wohlers (1966) and Ishikawa (1987) provide generalizations.  $\mathcal{D}'_{L^1}$  contains  $\delta(x)$  and any continuous function that decays at least as fast as  $\mathcal{O}(x^{-2})$ . It does not contain p.v.( $1/x$ ),  $H(x)$ , or a constant ( $c$ ).
- A set of spaces— $\mathcal{D}'_{L^1(n+1)} \subset \mathcal{L}'$  for integer  $n \geq 0$  (Nussenzweig 1972), containing any distribution  $g(x)$  that satisfies

$$g(x)(1+x^2)^{-\frac{n+1}{2}} \in \mathcal{D}'_{L^1} \quad (9)$$

It follows that  $g(x)$  is now allowed to show growth of  $\mathcal{O}(x^\alpha)$ ,  $\alpha < n$ . Distributions within  $\mathcal{D}'_{L^1(1)}$  (i.e.,  $n = 0$ ) can always be convolved with p.v.( $1/x$ ), and so always have a well-defined Hilbert transform (Nussenzweig 1972), although generalizations outside this space are possible (Pandey 2011). Practically,  $n$  can be assessed for a given  $g(x)$  by incrementally testing whether  $g(x)(1+x^2)^{-\frac{n+1}{2}}$  is integrable. As structurally-relevant examples, cf. Faedo et al. (2017) and Keivan et al. (2017): at minimum  $n$ , p.v.( $1/x$ ) is in  $\mathcal{D}'_{L^1(1)}$ ,  $H(x)$ ;  $\text{sgn}(x)$  and a constant ( $c$ ) are in  $\mathcal{D}'_{L^1(2)}$ ; and a polynomial of order  $m$  is in  $\mathcal{D}'_{L^1(m+2)}$ .

### Distributional Transfer Functions

Consider then a distributional representation of Eqs. (1)–(5), within which we may identify the role of delta distributions. If  $x(t) \in \mathcal{L}'$ , with the only practical restriction being polynomial growth as  $t \rightarrow \infty$ , then  $F(t) \in \mathcal{L}'$ . Under the Fourier transform,  $\hat{x}(\omega)$ ,  $\hat{F}(\omega)$ , and the TF  $\hat{Q}_0 = \hat{F}/\hat{x} = k$  are all in  $\mathcal{L}'$ . Eq. (4), the Fourier transform of a derivative, is identical—but the final operation, the division by  $i\omega$ , is not. Division can only be defined for distributions in restricted cases, and may lead to nonunique solutions (Friedlander and Joshi 1998). In the case of division of  $k$  by  $(i\omega)^N$ , to determine the TF with respect to the  $N$ th derivative of  $x$ , we can guarantee that the quotient  $\hat{Q}_N(\omega) \triangleq k/(i\omega)^N$  exists, and we can compute it by solving the following distributional division equation (Beltrami and Wohlers 1966, 1967; Nussenzweig 1972):

$$(i\omega)^N \hat{Q}_N(\omega) = k \quad (10)$$

That is, to define division, we seek distributions that recover  $k$  under multiplication. Eq. (10) has a well-established nonunique solution (Beltrami and Wohlers 1966, 1967; Nussenzweig 1972), as follows:

$$\begin{aligned} \hat{Q}_N(\omega) &= \left[ \frac{k}{(i\omega)^N} \right] + \sum_{m=1}^{N-1} b_m \delta^{(m)}(\omega) \\ &= \frac{k}{i^N} \text{p.v.} \left( \frac{1}{\omega^N} \right) + \sum_{m=1}^{N-1} c_m \delta^{(m)}(\omega) \end{aligned} \quad (11)$$

where  $b_m$  and  $c_m$  are arbitrary complex-valued constants, representing the fact that  $\omega^m \delta^{(m-1)}(\omega) = 0$ ; and thus, adding any delta derivative up to  $\delta^{N-1}(\omega)$  to  $\hat{Q}_N(\omega)$  will still lead to  $k$  being recovered under multiplication [Eq. (10)]. The term  $[k/(i\omega)^N]$  denotes the *particular* solution to the division equation, which we are here free to express as a factor of p.v.( $1/\omega^N$ ).

The  $c_m \delta^{(m)}(\omega)$  of Eq. (11) are the hidden deltas of Makris (1997b), and the distributional division equation is the mechanism by which they arise. Distributional division formalizes the intuition of Crandall (1991), that the  $\delta(\omega)$  is not “noticed” in p.v.( $1/\omega$ ), although it also qualifies this intuition. These deltas are not specifically connected to the presence of a singularity in the quotient (they arise in any distributional division by  $\omega^N$ ), but rather by the fact that this division is uniquely determined only up to  $\delta^{(N-1)}(\omega)$ . Makris (1997b) made a distinction between the causally motivated hidden deltas and the presence of a nonunique delta term in a noncausal rate-independent damping model, but both arise from the same source: distributional division. However, there is an additional connection between these delta terms and causality.

### Causality in Distributions

#### Causality Constraints on the Division Equation

Per ordinary TFs, the response of a distributional TF to an impulse  $D^N x(t) = \delta(t)$  is  $Q_N(t) = \mathcal{F}^{-1}\{\hat{Q}_N(\omega)\}$ , because  $\mathcal{F}\{\delta(t)\} = 1$ . For causality to be respected,  $Q_N(t)$  cannot represent a response prior to the impulse at  $t = 0$ . Because a distribution acts on test functions rather than values, we require that its support ( $\text{supp}\{\cdot\}$ )—the set of points around which the distribution maps any test function to a nonzero value (Nussenzweig 1972)—be located in  $[0, \infty)$ , as in

$$\text{supp}\{Q_N(t)\} \subseteq [0, \infty), \text{ for causality} \quad (12)$$

In a certain limited sense, the coefficients,  $c_m$ , of the hidden deltas in Eq. (11) determine whether  $Q_N(t)$  is causal: the terms  $\delta^{(m)}(\omega)$  transform to factors of  $t^m$  in the time domain. However, by the uniqueness results of Beltrami and Wohlers (1966) (Theorem 1.37), we know that if the original TF  $\hat{Q}_0(\omega)$  is causal, then, for any  $\hat{Q}_N(\omega)$ , the set  $\{c_m\}$  ensuring causality necessarily exists; whereas if  $\hat{Q}_0(\omega)$  is not causal, then no such set exists.

Assessing causality can proceed in one of two ways. In cases such as the linear spring, we can use time-domain analysis directly. The inverse Fourier transform of Eq. (11) (Kammler 2008) is

$$Q_N(t) = \frac{k}{2(N-1)!} \text{sgn}(t) + \frac{1}{2\pi} \sum_{m=1}^{N-1} c_m \frac{t^m}{i^m} \quad (13)$$

From Eq. (13), we make the following determinations. (1) To ensure  $Q_N(t)$  is real-valued, then if  $m$  is even,  $c_m$  must be purely real, and if  $m$  is odd,  $c_m$  must be purely imaginary. We may conveniently define real-valued coefficients  $d_m$  as  $i^m d_m = c_m$  to satisfy this condition. (2) To satisfy causality, only the highest-order term  $t^{N-1}$  can be nonzero. Setting all coefficients to zero other than  $c_{N-1}$ , we can compute this remaining coefficient as  $c_{N-1} = i^{N-1} \pi k / (N-1)!$ , and confirm that

$$\begin{aligned} \hat{Q}_N(\omega) &= \frac{k}{i^N} \text{p.v.} \left( \frac{1}{\omega^N} \right) + \frac{i^{N-1} \pi k}{(N-1)!} \delta^{(N-1)}(\omega), \\ Q_N(t) &= k \frac{t^{N-1}}{(N-1)!} H(t) \end{aligned} \quad (14)$$

which is causal. This is the solution for the hidden delta in any TF of the linear spring. For  $N = 1$  we recover  $\pi k \delta(\omega)$ , and for  $N = 2$ ,  $i \pi k \delta^{(1)}(\omega)$ , per Eq. (7). Time-domain causality analysis of this form is useful for assessing the causality of TFs defined a priori. However, in cases where we wish to identify TF properties or

parameters that proceed from causality—e.g., loss moduli from storage moduli (Madsen et al. 2008), or casual approximations of hysteretic damping (Makris 1997a)—then causality analysis directly in the frequency domain can be preferable.

### Causality Analysis by Distributional Hilbert Transform

Frequency-domain causality analysis for distributional TFs rests on the finer spaces of distribution we have outlined previously. Initially, let us assume that  $\hat{Q}_N(\omega) \in \mathcal{D}'_{L^1}$ , which is true for  $N \geq 1$  in the linear spring, but *not* for the original  $\hat{Q}_0(\omega) = k$ . Distributions in  $\mathcal{D}'_{L^1}$  can always be convolved (\*) with p.v.(1/x), and following Theorem 1.8.5 of Nussenzveig (1972), we can construct the following relation:

$$\mathfrak{F}^{-1} \left\{ \hat{Q}_N(\omega) * \left( \text{p.v.} \left( \frac{1}{\omega} \right) + i\pi\delta(\omega) \right) \right\} = 2\pi i H(t) Q_N(t) \quad (15)$$

Per Eq. (12),  $H(t)Q_N(t)$  is causal, and when  $Q_N(t)$  itself is causal,  $H(t)Q_N(t) = Q_N(t)$ . Using this in Eq. (15) yields the fundamental theorem for causality in distributions [Beltrami and Wohlers (1965), Theorem 2; (Nussenzveig 1972), Theorem 1.8.6]:

$$\hat{Q}_N(\omega) = \frac{1}{i\pi} \hat{Q}_N(\omega) * \text{p.v.} \left( \frac{1}{\omega} \right) = \frac{1}{i} \mathcal{H}\{\hat{Q}_N(\omega)\}, \text{ for causality} \quad (16)$$

where  $\mathcal{H}\{\cdot\}$  is the distributional Hilbert transform, defined via the convolution in Eq. (16). Hilbert transforms can be evaluated via tabulated results (King 2009) or by the Fourier transform of a convolution (Pandey 2011). Splitting  $\hat{Q}_N(\omega)$  into real and imaginary parts reveals that these parts must be Hilbert transforms pairs, but we will operate directly on  $\hat{Q}_N(\omega)$ . Applying Eq. (16) to Eq. (14), we confirm causality of the hidden delta solution. Given King (2009), we have

$$\begin{aligned} \mathcal{H}\left\{ \text{p.v.} \left( \frac{1}{\omega^N} \right) \right\} &= \frac{(-1)^N \pi}{(N-1)!} \delta^{(N-1)}(\omega), \\ \mathcal{H}\{\delta^{(N-1)}(\omega)\} &= \frac{(-1)^{N-1} (N-1)!}{\pi} \text{p.v.} \left( \frac{1}{\omega^N} \right) \end{aligned} \quad (17)$$

Then

$$\begin{aligned} \mathcal{H}\{\hat{Q}_N(\omega)\} &= \frac{k}{i^N} \mathcal{H}\left\{ \text{p.v.} \left( \frac{1}{\omega^N} \right) \right\} + \frac{i^{N-1} \pi k}{(N-1)!} \mathcal{H}\{\delta^{(N-1)}(\omega)\} \\ &= \frac{i^N \pi k}{(N-1)!} \delta^{(N-1)}(\omega) + \frac{k}{i^{N-1}} \text{p.v.} \left( \frac{1}{\omega^N} \right) \\ &= i \hat{Q}_N(\omega) \therefore \text{causal} \end{aligned} \quad (18)$$

As noted by Beltrami and Wohlers (1966), Eq. (16), which is valid for distributions in  $\mathcal{D}'_{L^1}$  and thus ordinary functions in  $L^1$ , is equivalent to Titchmarsh's theorem for ordinary functions in  $L^2$ . The distributional formulation thus extends the validity of an ordinary-function analysis to  $L^1$ , provided that certain statements are interpreted in a distributional sense.

### Causality Analysis by Generalized Hilbert Transform

Nevertheless, the restriction to  $\mathcal{D}'_{L^1}$ , including  $L^1$  and  $L^2$ , excludes a range of relevant structural models. Constant transfer functions, such as the dynamic stiffness of the spring,  $\hat{Q}_0(\omega) = k$ , are one immediate case (Carcione et al. 2019). As an ordinary function,

$k \notin L^2$ ; as a distribution,  $k \notin \mathcal{D}'_{L^1}$ ; and we may confirm violation of Eq. (16):  $\mathcal{H}\{k\}/i = 0 \neq k$ . Other more complex inadmissible transfer functions can be found in viscoelastic power-law media (Szabo 1994; Gulgowski and Stefański 2021; Waters et al. 2000). There is, however, an extension of the causality condition of Eq. (16) to a wider space of distributions, as derived by Beltrami and Wohlers (1966). For any distribution  $\hat{Q}_N(\omega) \in \mathcal{D}'_{L^1}$ , we may define a generalized Hilbert transform as follows:

$$\mathcal{H}_{(n)}\{\hat{Q}_N(\omega)\} = \omega^n \mathcal{H}\left\{ \left[ \frac{\hat{Q}_N(\omega)}{\omega^n} \right] \right\} = \frac{\omega^n}{\pi} \left( \left[ \frac{\hat{Q}_N(\omega)}{\omega^n} \right] * \text{p.v.} \left( \frac{1}{\omega} \right) \right) \quad (19)$$

Then, per Beltrami and Wohlers (1966), Theorem 3.13, and Nussenzveig (1972), Eq. 1.8.40, we have

$$\hat{Q}_N(\omega) = \frac{1}{i} \mathcal{H}_{(n)}\{\hat{Q}_N(\omega)\} + \mathcal{P}_{n-1}(\omega), \text{ for causality} \quad (20)$$

$[\hat{Q}_N(\omega)/\omega^n]$  again represents the particular solution of this distributional quotient, and  $\mathcal{P}_{n-1}(\omega)$  represents an arbitrary polynomial of order  $n-1$  in  $\omega$ , accounting for delta distributions introduced by division [Eq. (11)]: these deltas become polynomial under convolution and multiplication by  $\omega^n$ . If Eq. (20) is satisfied for some  $\mathcal{P}_{n-1}(\omega)$ , then  $\hat{Q}_N(\omega)$  is causal.

Using Eq. (20), if we know that a distributional TF is in some  $\mathcal{D}'_{L^1}$ , then we may rapidly assess its causality by computing  $\mathcal{H}_{(n)}\{\cdot\}/i$  and observing whether this differs from the original TF by more than  $\mathcal{P}_{n-1}(\omega)$ . This allows a direct causality analysis of any improper or not strictly proper TF, with a numerator of order greater than or equal to that of the denominator. For instance, for the dynamic stiffness of a spring,  $\hat{Q}_0(\omega) = k \in \mathcal{D}'_{L^1}$  (i.e.,  $n=1$ ), we have

$$\begin{aligned} \frac{1}{i} \mathcal{H}_1\{k\} &= \frac{\omega}{i\pi} \left( \left[ \frac{k}{\omega} \right] * \text{p.v.} \left( \frac{1}{\omega} \right) \right) = \frac{k\omega}{i\pi} \text{p.v.} \left( \frac{1}{\omega} \right) * \text{p.v.} \left( \frac{1}{\omega} \right) \\ &= ki\omega\delta(\omega) = 0 \\ &= k + \mathcal{P}_0 \therefore \text{causal} \end{aligned} \quad (21)$$

The same approach is applicable to the dynamic stiffnesses of linear dampers [Makris (1997b) and inerters  $\hat{Q}_0(\omega) = -m\omega^2$ , Makris (2018)], which are improper TFs. Indeed, several representation theorems—including Theorem 2 of Ishikawa (1987), Theorem 1.28 of Beltrami and Wohlers (1966), and Theorem 2 of Pfaffelhuber (1971)—indicate that *any* TF in  $\mathcal{L}'$  can be analyzed via this method. For instance, we can directly confirm the non-causality of the classical rate-independent damper, with dynamic stiffness  $\hat{Q}_0(\omega) = i\text{sgn}(\omega) \in \mathcal{D}'_{L^1}$ . Computing the convolution via Fourier transform ( $\omega$  to  $\Omega$ ), and denoting the Euler–Mascheroni constant by  $\gamma$ , gives

$$\begin{aligned} \frac{1}{i} \mathcal{H}_{(1)}\{i\text{sgn}(\omega)\} &= \frac{\omega}{\pi i} \text{p.v.} \left( \frac{i}{|\omega|} \right) * \text{p.v.} \left( \frac{1}{\omega} \right) \\ &= \frac{\omega}{\pi} \mathcal{F}^{-1} \left\{ \mathcal{F} \left\{ \text{p.v.} \left( \frac{1}{|\omega|} \right) \right\} \mathcal{F} \left\{ \text{p.v.} \left( \frac{1}{\omega} \right) \right\} \right\} \\ &= 2i\omega \mathcal{F}^{-1} \{ \ln |\Omega| \text{sgn}(\Omega) + \gamma \text{sgn}(\Omega) \} \\ &= \frac{2}{\pi} \ln |\omega| \neq i\text{sgn}(\omega) + \mathcal{P}_0(\omega) \therefore \text{non-causal} \end{aligned} \quad (22)$$

We may verify with a few further steps that the addition of this residual term ( $2/\pi \ln |\omega|$ ) to the rate-independent damping model

causes it to become causal, per Makris (1997a). Indeed, Eq. (22) elucidates one final paradoxical causality result in the literature. Makris (1997a) derived the following causal rate-independent damping model with dynamic stiffness:

$$\hat{Q}_0(\omega) = i \operatorname{sgn}\left(\frac{\omega}{\varepsilon}\right) + \frac{2}{\pi} \ln \left| \frac{\omega}{\varepsilon} \right| \quad (23)$$

where  $\varepsilon$  is an arbitrary positive constant. The real and imaginary parts of Eq. (23) are an exact generalized Hilbert transform pair [Eq. (22)], and thus the model is causal for all  $\varepsilon$ . However, while  $\operatorname{sgn}(\omega/\varepsilon) = \operatorname{sgn}(\omega)$  always,  $\operatorname{sgn}(\omega)$  and  $2/\pi \ln |\omega/\varepsilon|$  are not exact generalized Hilbert transform pairs. The difference is, indeed, a polynomial  $\mathcal{P}_0$ , as  $\ln |\omega/\varepsilon| = \ln |\omega| - \ln \varepsilon$ , and thus for any  $\varepsilon$ , a polynomial residual  $\mathcal{P}_0$  will exist in Eq. (22), satisfying causality. In this way, the hidden deltas allow us to derive the following equivalent simplified model:

$$\hat{Q}_0(\omega) = i \operatorname{sgn}(\omega) + \frac{2}{\pi} \ln \left| \frac{\omega}{\varepsilon} \right| = i \operatorname{sgn}(\omega) + \frac{2}{\pi} \ln |\omega| + c \quad (24)$$

and confirm that it is causal for all  $\varepsilon$  and all  $c$ .

Eq. (20) is a powerful condition to assess causality in linear systems, but it also has a key physical connection. The generalized Hilbert transform involves dividing a transfer function  $\hat{Q}(\omega)$  by  $\omega^n$ , convolving it, and then multiplying again by  $\omega^n$ . This is equivalent to integrating the impulse response  $Q(t)$   $n$  times, multiplying by a step function to force causality, and then differentiating  $n$  times back again. This causality assessment works because, if a system is causal with respect to any kinematic variable, then it is causal with respect to any derivative or integral of this variable. It is not possible via differentiation or integration to propagate causal signals to before  $t = 0$ , and thus we are free to choose the differential/integral order ( $n$ ) at which to perform the causality analysis—we have only to choose  $n$  to reach  $\mathcal{D}_{L^1}^{(1)}$ . This process underpins the implicit choice of Makris (1997b, 2017) to analyze the spring's causality in mechanical impedance ( $n = 1$ ) rather than dynamic stiffness ( $n = 0$ ); it is here that we reach  $\mathcal{D}_{L^1}^{(1)}$ , and the fundamental theorem [Eq. (16)] is applicable. The representation theorems of Ishikawa (1987) and others further support this choice by indicating that a suitable  $n$  exists for any distribution in  $\mathcal{L}'$ , including any slowly growing function.

## Concluding Remarks

Distribution-theoretic principles not only provide the basis for the presence of the hidden deltas, but also justification for choices made by current studies to analyze causality in specific higher-derivative transfer functions, such as mechanical impedance. Distributional analysis predicts exactly which higher derivative is required for conventional causality analysis to be valid, providing a direct method for assessing causality at any initial derivative order that does not require computation of hidden deltas.

## Data Availability Statement

No data, models, or code were generated or used during the study.

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