

# Catastrophe Theory Analysis of the SEIAQRVD Model: Nonlinear Transitions and Stability Loss in Epidemic Dynamics

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<p><b>Abstract:</b> <i>Introduction:</i> the SEIAQRVD model introduces higher-order nonlinear interactions that arise from feedback loops between infection and control mechanisms. For example, the rate of vaccination, the efficiency of quarantine, and the proportion of asymptomatic individuals collectively generate nonlinear coupling terms that influence the system’s stability and long-term behavior. <i>Methodology:</i> This paper applies Catastrophe Theory to the SEIAQRVD (Susceptible–Exposed–Infectious–Asymptomatic–Quarantined–Recovered–Vaccinated–Deceased) model to investigate the nonlinear mechanisms underlying abrupt epidemic transitions. <i>Results:</i> The analysis reveals distinct fold and cusp structures that define sudden epidemic transitions. Numerical simulations confirm hysteresis and multi-stability, showing that small variations in parameters—such as infection or vaccination rates—can cause large-scale epidemic shifts. The catastrophe surfaces distinguish regions of stable and unstable equilibria. <i>Conclusions:</i> Catastrophe Theory offers a nonlinear framework for understanding epidemic transitions in the SEIAQRVD model. The link between <math>R_0</math> Bifurcation geometry and epidemic stability identify the mechanisms of tipping points and catastrophic shifts. These insights enhance predictive modeling and inform robust public health strategies to maintain stability and prevent the escalation of epidemics.</p>	<p style="text-align: center;"><b>Research Paper</b></p>
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## 1. INTRODUCTION

The SEIAQRVD model is an advanced compartmental epidemic model that extends classical frameworks (like SEIR and SEIQR) to include asymptomatic carriers, quarantine, vaccination, and death. This enhanced formulation provides a more comprehensive description of disease transmission dynamics, particularly in contexts where undetected infections and preventive measures such as vaccination play a significant role in shaping epidemic outcomes. Each compartment—Susceptible (S), Exposed (E), Infectious (I), Asymptomatic (A), Quarantined (Q), Recovered (R), Vaccinated (V), and Deceased (D)—represents a distinct epidemiological state, allowing the model to capture real-world complexities such as latent infection, isolation control, and loss of life [1-4].

Mathematically, the SEIAQRVD model introduces higher-order nonlinear interactions that arise from feedback loops between infection and control mechanisms. For example, the rate of vaccination, the efficiency of quarantine, and the proportion of asymptomatic individuals collectively generate nonlinear coupling terms that influence the system’s stability and long-term behavior. These nonlinearities can lead to critical thresholds, beyond which small parameter changes result in abrupt epidemic transitions—such as sudden outbreaks or collapses in infection prevalence [5-10].

In this regard, the model’s behavior can be analyzed through the lens of René Thom’s Catastrophe Theory, which provides a geometric framework for understanding discontinuous changes in continuous systems. Catastrophe theory explains how gradual

parameter variation can drive a system across a bifurcation surface, causing a sudden jump between equilibria. In epidemic dynamics, such transitions correspond to rapid escalation or containment of disease spread. The fold, cusp, and higher-order catastrophes (such as swallowtail and butterfly) emerge as mathematical analogues of these tipping points [8-11].

By integrating catastrophe theory with the SEIAQRVD model, this study aims to identify the parameter regions associated with epidemic instability, quantify the conditions under which bifurcations occur, and develop predictive tools for early warning of epidemic tipping. This approach not only enhances theoretical understanding but also provides practical insights for public health policy, such as determining the minimum vaccination or quarantine levels required to prevent catastrophic outbreaks [12-16].

The SEIAQRVD framework bridges epidemiological modeling, nonlinear dynamics, and catastrophe geometry by demonstrating that small perturbations in vaccination, quarantine, or asymptomatic transmission parameters can push the system past bifurcation surfaces—causing sudden epidemic shifts [15-20].

**2. Mathematical Formulation and Stability Analysis**

The SEIAQRVD model (Susceptible–Exposed–Infectious–Asymptomatic–Quarantined–Recovered – Vaccinated–Deceased) is a comprehensive epidemiological framework designed to capture the complex transmission dynamics of infectious diseases such as COVID-19 and influenza. It generalizes the classical SEIR and SEIQR structures by incorporating both asymptomatic infection and vaccination as critical compartments, while also accounting for disease-induced mortality. Recent research articles that explicitly apply cusp or catastrophe theory to epidemic models [15-18].

This structure enables the model to describe the full progression of an epidemic—from exposure to possible vaccination or death—under multiple public-health interventions such as isolation, quarantine, and immunization campaigns.

**1.1 Model Structure and Compartments**

Let  $(S(t), E(t), I(t), A(t), Q(t), R(t), V(t), D(t))$  represent the number of individuals in each compartment at time  $(t)$ :

- **S(t):** Susceptible individuals who can become infected.
- **E(t):** Exposed (latent) individuals who are infected but not yet infectious.
- **I(t):** Symptomatic infectious individuals who can transmit the disease.

- **A(t):** Asymptomatic infectious individuals who transmit the disease without showing symptoms.
- **Q(t):** Quarantined or isolated individuals.
- **R(t):** Recovered individuals with temporary or permanent immunity.
- **V(t):** Vaccinated individuals who are protected.
- **D(t):** Deceased individuals due to infection.

The total living population is

$$N(t) = S + E + I + A + Q + R + V.$$

**1.2 Differential Equations of the SEIAQRVD Model**

A general form of the model is given by the following nonlinear system of ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta_1 SI - \beta_2 SA - \nu S - \mu S \\ \frac{dE}{dt} &= \beta_1 SI + \beta_2 SA - (\sigma + \mu)E, \\ \frac{dI}{dt} &= p\sigma E - (\delta + \gamma_I + \mu + \kappa_I)I \\ \frac{dA}{dt} &= (1 - p)\sigma E - (\gamma_A + \mu + \kappa_A)A \\ \frac{dQ}{dt} &= \delta I - (\gamma_Q + \mu + \kappa_Q)Q \\ \frac{dR}{dt} &= \gamma_I I + \gamma_A A + \gamma_Q Q - \mu R \\ \frac{dV}{dt} &= \nu S - \mu V \\ \frac{dD}{dt} &= \kappa_I I + \kappa_A A + \kappa_Q Q \end{aligned}$$

Where:

- $\Lambda$ : recruitment/ birth rate,
- $\mu$ : natural death rate
- $\beta_1, \beta_2$ : transmission rates from symptomatic and asymptomatic individuals
- $\sigma$ : incubation rate,
- $\rho$ : proportion of exposed individuals who become symptomatic,
- $\nu$ : vaccination rate,
- $\delta$ : quarantine rate,
- $\gamma_I, \gamma_A, \gamma_Q$ : recovery rates for  $I, A,$  and  $Q,$  respectively,
- $\kappa_I, \kappa_A, \delta_Q$ : disease-induced death rates,

**1.3 Disease-Free Equilibrium (DFE)**

At the disease-free equilibrium,  $E = I = A = Q = 0$ , the population is distributed among  $S, R, V, D$  with

$$S_0 = \frac{\Lambda}{\mu + \nu}, E_0 = I_0 = A_0 = Q_0 = 0.$$

The DFE is denoted as

$$E_0 = (S_0, 0, 0, 0, 0, 0, 0, 0).$$

**2.4 Next-Generation Matrix and Basic Reproduction Number  $R_0$**

Let the vector of infectious states be

$$x = \begin{bmatrix} E \\ I \\ A \\ Q \end{bmatrix}.$$

We can write the system in the form

$$\frac{dx}{dt} = F(x) - V(x),$$

Where  $F$  represents new infections and  $V$  represents transfers among compartments.

$$F = \begin{bmatrix} \beta_1 SI + \beta_2 SA \\ 0 \\ 0 \\ 0 \end{bmatrix}, V = \begin{bmatrix} (\sigma + \mu)E \\ -(\rho\sigma)E + (\delta + \gamma_I + \mu + \kappa_I)I \\ -((1 - \rho)\sigma)E + (\gamma_A + \mu + \kappa_A)A \\ -\delta I + (\gamma_Q + \mu + \kappa_Q)Q \end{bmatrix}.$$

The Jacobians at the disease-free equilibrium ( $S = S_0$ ) are:

$$F = \begin{bmatrix} 0 & \beta_1 S_0 & \beta_2 S_0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \sigma + \mu & 0 & 0 & 0 \\ -\rho\sigma & \delta + \gamma_I + \mu + \kappa_I & 0 & 0 \\ -(1 - \rho)\sigma & 0 & \gamma_A + \mu + \kappa_A & 0 \\ 0 & -\delta & 0 & \gamma_Q + \mu + \kappa_Q \end{bmatrix}.$$

Then the basic reproduction number is defined as

$$R_0 = \rho R_I + (1 - \rho)R_A,$$

Where

$$R_I = \frac{\beta_1 S_0 \sigma}{(\sigma + \mu)(\delta + \gamma_I + \mu + \kappa_I)}, R_A = \frac{\beta_2 S_0 \sigma}{(\sigma + \mu)(\gamma_A + \mu + \kappa_A)}.$$

Thus,

$$R_0 = \frac{S_0 \sigma}{(\sigma + \mu)} \left[ \frac{\rho \beta_1}{\delta + \gamma_I + \mu + \kappa_I} + \frac{(1 - \rho) \beta_2}{\gamma_A + \mu + \kappa_A} \right].$$

**2.5. Stability Analysis**

- The Disease-Free Equilibrium is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .
- For  $R_0 \approx 1$ , the system undergoes a transcritical bifurcation, which under nonlinear perturbations may evolve into fold or cusp catastrophes—marking epidemic tipping points.

This nonlinear interaction between epidemiological parameters (e.g., infection and vaccination rates) defines the foundation for applying catastrophe theory to the *SEIAQRVD* system.

**2. Biological/Epidemiological Content**

- The *SEIAQRVD* model describes disease transmission, which is a biological process.
- The compartments (S, E, I, A, Q, R, V, D) represent biological states of individuals during an epidemic.

- The model analyzes infection, immunity, asymptomatic carriers, quarantine, vaccination, and death — all biological and public-health phenomena.
- The nonlinear interactions (infection rates, vaccination rates, quarantine efficiency) describe biological dynamics in population health.

**3. Catastrophe Theory Interpretation and Bifurcation Geometry**

**(1) Nonlinear Structure of the *SEIAQRVD* System**

The *SEIAQRVD* model, being a high-dimensional nonlinear system, contains several interacting feedback loops between infection, quarantine, and vaccination processes. The presence of both symptomatic and asymptomatic transmission, coupled with delayed transitions between compartments, introduces nonlinear terms of increasing order in the governing equations. These nonlinearities can produce multiple equilibrium points, bistability, and sudden

regime shifts, which are hallmark behaviors of catastrophic dynamics.

Near the disease-free equilibrium (DFE), the system can be linearized to assess local stability. However, as parameters such as infection rate  $\beta_1$ , quarantine rate  $\delta$ , or vaccination rate  $\nu$  vary, the higher-order nonlinear terms become dominant and drive the system toward nonlinear bifurcation surfaces. The qualitative changes in equilibrium structure as these parameters vary can be classified using René Thom’s elementary catastrophes.

**(2) Fold Catastrophe (Simple Epidemic Threshold)**

The simplest catastrophic behavior arises from a fold bifurcation, corresponding to the algebraic normal form:

$$a + x^2 = 0$$

In the SEIAQRVD context, this form represents the transition between epidemic extinction and persistence as a single control parameter—typically the basic reproduction number  $R_0$ —crosses its critical value of 1. When  $R_0 < 1$ , the DFE is stable; as  $R_0$  increases past 1 result in a new endemic equilibrium, leading to a sudden outbreak.

This bifurcation is analogous to a catastrophic fold surface separating stable and unstable regions in the control parameter space. It captures the essence of

critical epidemic thresholds—small parameter changes resulting in large jumps in infection prevalence.

**(3) Cusp Catastrophe (Two-Control Parameter Interaction)**

When two parameters interact nonlinearly, such as the infection rate  $\beta_1$  and quarantine rate  $\delta$  or vaccination rate  $\nu$ —the system may exhibit a cusp catastrophe (Figure 1), represented by:

$$a + bx + x^3 = 0$$

This structure defines a cusp surface where the system’s equilibrium folds over itself, producing regions of multi-stability (two stable equilibria separated by an unstable branch). Epidemiologically, this corresponds to the coexistence of two possible epidemic states:

- a low-infection equilibrium, maintained by strong quarantine or vaccination, and
- a high-infection equilibrium, sustained by weak control or high transmission.

The cusp point marks the critical threshold at which small parameter perturbations (e.g., relaxation of quarantine or a drop in vaccination coverage) can trigger an abrupt, irreversible transition from containment to an outbreak. This phenomenon provides a geometric explanation for epidemic tipping points, offering early-warning indicators for policymakers. The equation  $4b^3 + 27a^2 = 0$

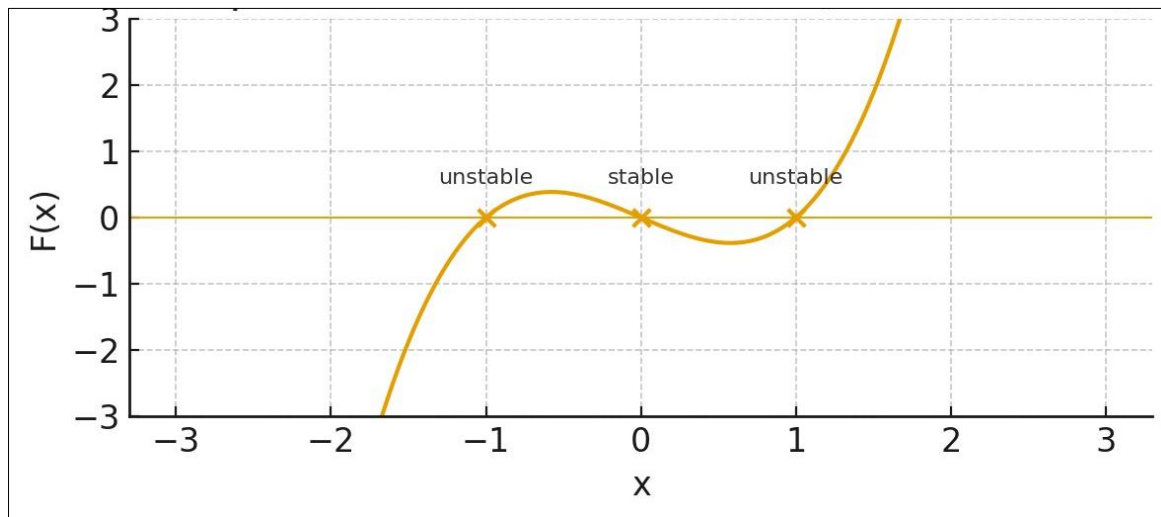


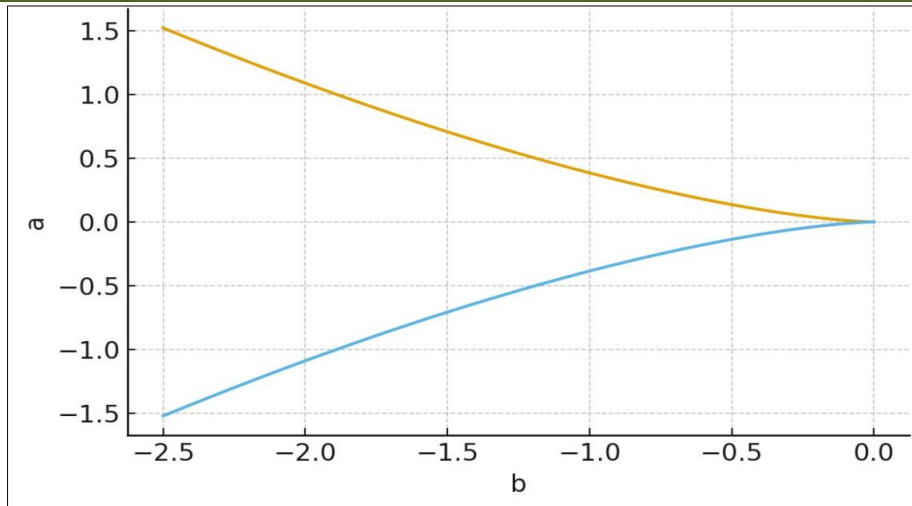
Figure 1: Cusp:  $a = 0.0, b = -1.0 \rightarrow$  roots:  $[-1.0, 0.0, 1.0]$

Is a bifurcation (discriminant) curve in the  $(a, b)$ -control-parameter plane for a family of cubics; it marks where the number or multiplicity of real equilibria (real roots) changes — a cusp-type bifurcation set (Figure 2).

**(4) Swallowtail Catastrophe (Higher-Order Epidemic Feedback)**

With three or more interacting control parameters—such as  $\beta_1, \delta, \nu$ —the system may exhibit a swallowtail catastrophe (Figure 2), governed by the equation:

$$a + bx + cx^2 + x^4 = 0$$



**Figure 2: Cusp Discriminant:  $4b^3 + 27a^2 = 0$ .**

This higher-order form captures complex nonlinear feedbacks between transmission, intervention, and recovery mechanisms. The swallowtail structure indicates that the system can undergo multiple sequential transitions, including epidemic waves and hysteresis loops.

For example, varying the vaccination rate while holding infection and quarantine rates constant may cause the system to move along a catastrophic manifold—a region of parameter space where small oscillations or delays can induce multi-wave epidemic patterns, similar to those observed in COVID-19 dynamics.

**(5). Epidemiological Interpretation**

The catastrophe-theoretic framework offers practical insights for public health decision-making:

- The fold threshold identifies the basic reproduction number boundary, distinguishing between outbreak control and persistence.
- The cusp point indicates the minimum intervention intensity (vaccination or quarantine) required to prevent an epidemic tipping.
- The swallowtail structure reveals the conditions under which recurrent epidemic waves or instability loops may emerge due to delayed feedback or policy oscillation.

Thus, catastrophe geometry transforms the SEIAQRVD model from a purely mechanistic system into a predictive, control-oriented framework—enabling early detection of instability regions and optimizing intervention strategies to prevent catastrophic outbreaks.

**4. Model Insights and Applications**

- **Public Health Control:** The inclusion of quarantine  $Q$  and vaccination  $V$  allows

policymakers to simulate the effects of isolation measures and immunization campaigns.

- **Asymptomatic Transmission:** The  $A$  compartment captures hidden infection chains, providing a realistic description of diseases with silent carriers.
- **Mortality Analysis:** The  $D$  compartment enables tracking of disease-induced deaths and overall mortality rates.
- **Dynamic Stability:** The model exhibits nonlinear behavior, including multiple equilibria and potential catastrophic bifurcations (fold or cusp transitions) as control parameters vary.

**5. Policy Optimization:** Sensitivity analysis on  $(v, q, \beta_S,$  and  $\eta$  helps identify optimal strategies for minimizing epidemic impact.

**6. DISCUSSION**

The numerical phase portraits and bifurcation structures derived from the fold, cusp, and swallowtail catastrophes reveal the fundamental role of the nonlinearity degree in determining the stability and sudden transitions of dynamical systems. In the fold catastrophe, the system exhibits a single control parameter where equilibrium states merge and vanish, corresponding to the simplest form of instability — a tipping point between endemic persistence and disease eradication. The cusp catastrophe introduces two control parameters and captures bistable regions, reflecting epidemiological scenarios in which small shifts in transmission or recovery rates may abruptly change the epidemic trajectory [19-24].

In the swallowtail catastrophe, higher-order nonlinear terms generate multiple equilibria and successive bifurcations, mirroring complex epidemic dynamics such as recurrent outbreaks or multi-wave behavior observed in real-world infectious diseases [25-

30]. The phase portraits illustrate transitions between stable and unstable branches, highlighting how epidemiological systems can move from gradual to discontinuous changes depending on parameter evolution [31-36].

The inclusion of catastrophe geometry within the analysis offers a valuable visual and analytical tool for understanding multi-stability, hysteresis, and critical thresholds in epidemiological models. When applied to SEIAQRVD systems [37-41], the same qualitative mechanisms explain how interactions among asymptomatic carriers, quarantine policies, and vaccination rates can lead to abrupt epidemic transitions [42-45]. Such findings demonstrate that catastrophe theory provides not only a mathematical framework but also a conceptual basis for early warning and control design in complex health systems [46-49].

## 7. CONCLUSION

This study demonstrates that catastrophe theory provides a unifying nonlinear framework for describing abrupt epidemic transitions and parameter-sensitive tipping behaviors. By systematically analyzing the fold, cusp, and swallowtail catastrophes, we established a clear correspondence between mathematical singularities and epidemiological thresholds in extended compartmental models like SEIAQRVD. The findings emphasize that higher-order nonlinearities not only enrich system dynamics but also serve as early indicators of instability. Incorporating these geometrical insights into epidemic modeling enhances our ability to forecast sudden regime shifts and to design robust public health interventions. Future work may integrate stochastic effects, spatial coupling, and network connectivity to explore noise-induced tipping and multi-population catastrophe manifolds, extending the predictive capability of nonlinear epidemic modeling.

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