The impulsive vaccination strategies of the epidemic SIR models with nonlinear incidence rates $\beta I^p S^q$ are considered in this paper. Using the discrete dynamical system determined by the stroboscopic map, we obtain the exact periodic infection-free solution of the impulsive epidemic system and prove that the periodic infection-free solution is globally asymptotically stable. In order to apply vaccination pulses frequently enough so as to eradicate the disease, the threshold for the period of pulsing, i.e. $\tau_{\text{max}}$, is shown. Furthermore, by bifurcation theory, we obtain a supercritical bifurcation at this threshold, i.e. when $\tau > \tau_{\text{max}}$ and is closing to $\tau_{\text{max}}$, there is a stable positive periodic solution. Throughout the paper, we find impulsive epidemiological models with nonlinear incidence rates $\beta I^p S^q$ show a much wider range of dynamical behaviors than do those with bilinear incidence rate $\beta SI$ and our paper extends the previous results, at the same time, theoretical results show that pulse vaccination strategy is distinguished from the conventional strategies in leading to disease eradication at relatively low values of vaccination, therefore impulsive vaccination strategy provides a more natural, more effective vaccination strategy.

1. Introduction. Although vaccines against measles have been routinely applied over a quarter of a century, measles is still persistent in a number of developed countries at present, with major epidemics roughly every 5 years. Currently, the guidelines for measles immunization in many areas of the west word are based on the conventional concept of time-constant immunization strategies, for example, the first vaccination dose at 15 months of age and second dose at around 6 years. The conventional vaccination strategies lead to epidemic eradication if the proportion of the successfully vaccinated individuals is higher than a certain critical value, which is approximately equal to 95% for measles (see Anderson & May [2]), while in practice, it is both difficult and expensive to implement vaccination for such a large population coverage. We are therefore led to examine the potential of other strategies that epidemics can be more efficiently controlled, such as pulse vaccination, i.e. by a vaccination effort that varies significantly and abruptly in time. Theoretical results show that the pulse vaccination strategy can distinguished from the conventional strategies in leading to disease eradication at relatively low values of vaccination (see Agur etc. [1]).

Pulse vaccination has gained in prominence as a result of its highly successfully application in the control of poliomyelitis and measles throughout Central and South
America (see Dequadros, Andrus & Olive [5], Sabin [13]). Another example for the application of this strategy is the United Kingdom, where, during November 1994, children aged 5 to 16 years were offered a single vaccination pulse, in the form of a combined measles and rubella (MR) vaccine. Coverage of 90% or more was achieved in 133 of 172 district health authorities (77%), and the mean coverage in England and Wales was 92%. As a result of this policy, the number of cases of measles notified to the Office of Population Censuses and Surveys has fallen significantly. As a consequence, it was concluded that pulse vaccination of all children of school age is likely to have a dramatic effect on the transmission of measles for several years and prevent a substantial toll of morbidity and mortality.

The epidemic models have been studied by many authors (see Bailey [3], Brauer [4], Derrick & van den Driessche [6] and Jung, Lenhart & Feng [9]), recently, the impulsive vaccination of epidemic models has also been discussed by some authors (see Shulgin, Stone & Agur [14]). Most of the papers consider the situation that the incidence rate (the rate of new infections) is bilinear in the infective fraction $I$ and the susceptible fraction $S$, i.e. $\beta IS$. But the standard bilinear form may require modification, the reason is that the assumption of homogeneous mixing may be invalid; in this case, it is best to introduce the necessary population structure and represent heterogeneous mixing directly. However, incidence rates that increase more gradually than linear in $I$ and $S$ can also arise from saturation effects; for example, if the number of infections is very high, so that exposure to the disease agent is virtually certain, the incidence rate will respond more slowly than linearly to increases in $I$. In contrast, a rate of increase faster than linear would be observed under various conditions, for example, if multiple exposures to the disease vector were necessary before infection occurred. So the epidemiological models with nonlinear incidence rates show a much wider range of dynamical behaviors than do those with bilinear incidence rate $\beta IS$. Locally, such nonlinearities can be approximated by a variety of forms, such as $\beta IS^p$, $\beta IS^p/(1 + Vp^{-1})$ and $\beta H(I, S)I$, which were discussed by Liu, Levin & Iwasa [12] and Liu, Hethcote & Levin [11].

In this paper, we mainly consider the pulse vaccination strategy in the SIR epidemic model with nonlinear incidence rate $\beta IS^2$, the following mathematical model is established:

\[
\begin{aligned}
\dot{S} &= -\beta IS^2 + \mu - \mu S, \\
\dot{I} &= \beta IS^2 - (\gamma + \mu)I, \\
\dot{R} &= \gamma I - \mu R, \\
S(n\tau^+) &= (1-p)S(n\tau^-), \\
I(n\tau^+) &= I(n\tau^-), \\
R(n\tau^+) &= R(n\tau^-) + pS(n\tau^-),
\end{aligned}
\]

where, the fractions of the population that are susceptible, infectious, and recovered with immunity, are denoted by $S$, $I$ and $R$ respectively. We assume that the population is in equilibrium, which is normalized to unity, i.e. $S + I + R = 1$, thus, the birth rate is equal to the death rate, and is denoted by $\mu$. Newborn individuals are susceptible to the infection. The parameter $\beta$ is the contact rate, $\gamma$ is the rate that the infective population becomes recovered and $p$ is the proportion of those vaccinated successfully, the impulsive vaccination is applied every $\tau$ years (with $0 < p < 1, \mu, \beta, \gamma > 0, n = 0, 1, 2, \ldots$). We are concerned about the meaningful domain: $\{(S, I, R)|S \geq 0, I \geq 0, R \geq 0, S + I + R = 1\}$.

The paper is arranged like this: In section two, the simple SIR model with nonlinear incidence rate $\beta IS^2$ and constant vaccination strategy are considered re-
respectively, we establish the conditions under which the corresponding equilibria are
globally asymptotically stable; using representative parameters of measles dynamics,
we can estimate the critical vaccination proportion of the successfully vaccinated
individuals is approximately equal to 78% which is much less than 95% (see Shulgin,
Stone & Agur [14]). In section three, using the discrete dynamical system deter-
determined by the stroboscopic map, we obtain the exact periodic infection-free solution
of the impulsive vaccination system and the threshold for the period of pulsing, i.e.
\( \tau_{\text{max}} \). Further, we prove the infection-free solution is globally asymptotically stable
if \( \tau < \tau_{\text{max}} \). In section four, by bifurcation theory, we prove that there is a stable
positive periodic solution if \( \tau > \tau_{\text{max}} \) and is closing to \( \tau_{\text{max}} \). However, Shulgin,
Stone & Agur [14] only provided numerical simulations to obtain the global asymp-
totical stability and the existence of positive periodic solution. In section five, we
extend the incidence rate \( \beta IS^2 \) to \( \beta IS^q \) \((p > 1, q > 0)\); by comparing the constant
vaccination and impulsive vaccination, we know the impulsive vaccination strategy
is more natural, more effective.

2. The Standard SIR Model and Constant Vaccination. The standard
SIR model with nonlinear incidence rate \( \beta IS^2 \) is the following:

\[
\begin{align*}
\dot{S} &= -\beta IS^2 + \mu - \mu S, \\
\dot{I} &= \beta IS^2 - (\gamma + \mu)I, \\
\dot{R} &= \gamma I - \mu R.
\end{align*}
\]

(2.1)

For the system (2.1), we denote \( \sigma \equiv \frac{\beta}{\mu + \mu} \), which is the basic reproductive rate of the
epidemic. The system (2.1) always has an infection-free equilibrium: \((S_0, I_0, R_0) = (1, 0, 0)\). If \( \sigma < 1 \), \((S_0, I_0, R_0)\) is globally asymptotically stable (see Liu, Hethcote & Levin [11]); if \( \sigma > 1 \), there is the unique epidemic equilibrium: \((S_1, I_1, R_1) = \left(\sqrt{\frac{2}{\sigma}}, H(1 - \sqrt{\frac{2}{\sigma}}), (1 - H)(1 - \sqrt{\frac{2}{\sigma}})\right)\), where \( H \equiv \frac{\sigma}{\mu + \mu} \); in this case, \((S_1, I_1, R_1)\)
is globally asymptotically stable (see Hethcote [8]), while \((S_0, I_0, R_0)\) is unstable
saddle (see Liu, Hethcote & Levin [11]).

We note for future reference that in this article we use typical parameters that
are representative of measles dynamics, as follows (see Engbert & Drepper [7]):
\( \mu = 0.02 \), \( \beta = 1800 \), \( \gamma = 100 \), then, \( \sigma > 1 \), from the analysis above, we know
measles can not be eradicated, thus we have to consider the vaccination strategy.

According to the conventional constant vaccination strategy, all newborn infants
should be vaccinated, and \( p \) is the proportion of those vaccinated successfully (with
0 < \( p < 1 \). The model is the following:

\[
\begin{align*}
\dot{S} &= -\beta IS^2 + \mu(1 - p) - \mu S, \\
\dot{I} &= \beta IS^2 - (\gamma + \mu)I, \\
\dot{R} &= \gamma I - \mu R + p\mu.
\end{align*}
\]

(2.2)

The system (2.2) always has an infection-free equilibrium: \((S_0', I_0', R_0') = (1-p, 0, p)\).
If \( 0 < \sigma \leq \frac{1}{(1-p)} \), i.e. \( p \geq 1 - \sqrt{\frac{2}{\sigma}} \), there is a nontrivial equilibrium, whereas
if \( \sigma > \frac{1}{(1-p)} \), i.e. \( p < 1 - \sqrt{\frac{2}{\sigma}} \), there is the unique nontrivial positive epidemic
equilibrium: \((S_1', I_1', R_1') = \left(\sqrt{\frac{2}{\sigma}}, H(1 - p - \sqrt{\frac{2}{\sigma}}), (1 - H)(1 - \sqrt{\frac{2}{\sigma}}) + Hp\right)\), where \( H \),
\( \sigma \) are defined as above. Increasing the vaccination proportion \( p \), linearly reduces the
equilibrium number of infectious individuals and linearly increases the equilibrium number of immune individuals, but the number of susceptible individuals remains unaffected.

For the stabilities of the infection-free equilibrium and epidemic equilibrium, we have the following theorems:

**Theorem 2.1.** If \( p > 1 - \sqrt{\frac{1}{\sigma}} \), the infection-free equilibrium \((S'_0, I'_0, R'_0)\) is globally asymptotically stable; inversely, if \( p < 1 - \sqrt{\frac{1}{\sigma}} \), \((S'_0, I'_0, R'_0)\) is unstable.

**Theorem 2.2.** If \( p < 1 - \sqrt{\frac{1}{\sigma}} \), the epidemic equilibrium \((S'_1, I'_1, R'_1)\) is globally asymptotically stable.

Using Lyapunov-Lasalle theorem and Poincaré-Bendixson theory respectively, we can easily prove Theorem 2.1 and Theorem 2.2, here we omit the proof. From Theorem 2.1 and Theorem 2.2, we know \( p_c = 1 - \sqrt{\frac{1}{\sigma}} \) is the critical vaccination proportion; using typical parameters as above, we can estimate the critical vaccination proportion as below: \( p_c \approx 0.78 \). This means that, for the SIR model with nonlinear incidence rate \( \beta IS^2 \), constant vaccination scheme is successful, i.e. the infection free equilibrium is stable, it is necessary to immunize at least 78% of all children soon after birth. This data is much less than the result in [14], so the constant vaccination of SIR model with nonlinear incidence rate \( \beta IS^2 \) is more effective than that of SIR model with bilinear incidence rate \( \beta IS \).

In the system (2.2), we assume the vaccination is continuous, but in practice, it is often the case that the vaccination is in regular pulses, the continuous vaccination is then removed from the system (2.2) and replaced with the impulsive vaccination, which provides a more natural description, so we consider below the pulse vaccination.

3. SIR Model with Pulse Vaccination. The pulse vaccination proposes to vaccinate a fraction \( p \) of the entire susceptible population in a single pulse, applied every \( \tau \) years, the system (2.1) evolves from its initial state without being further affected by the vaccination schemes until the next pulse is applied, when the pulse vaccination is incorporated into the system (2.1), the system may be rewritten as (1.1). In practice, the equation for \( R \) is redundant because \( R \) can be obtained from the relation \( S(t) + I(t) + R(t) = 1 \), so we only consider the following system:

\[
\begin{align*}
\dot{S} &= -\beta IS^2 + \mu - \mu S, \\
\dot{I} &= \beta IS^2 - (\gamma + \mu)I, \\
S(n\tau^+) &= (1 - p)S(n\tau^-), \\
I(n\tau^+) &= I(n\tau^-),
\end{align*}
\]

As paper [1] has shown, we may maintain \( \dot{I} < 0 \) for all time, i.e. \( S(t) < \sqrt{\frac{1}{\tau}} \geq S_c \) (epidemic threshold), so as to prevent the infectious population from ever growing. For this threshold method, periodic pulsing can maintain \( S(t) \) below \( S_c \) as long as the period of pulsing \( \tau \) is kept below a fixed critical value \( \tau_{max} \). One of the main goals of this paper is to find a suitable technique for estimating this parameter, it is essential for applying vaccination strategy efficiently.

We first demonstrate the existence of an infection-free solution, in which infectious individuals are entirely absent from the population permanently, i.e. \( I(t) = 0 \)
0, \ t \geq 0. Under this condition, the growth of susceptible individuals must satisfy:

\[
\begin{cases}
\dot{S} = \mu - \mu S, & t \neq n\tau, \\
S(n\tau^+) = (1 - p)S(n\tau^-), & t = n\tau.
\end{cases}
\] (3.2)

We show below that the susceptible population S oscillates with period \(\tau\), in synchronization with the periodic pulse vaccination. It is easy to obtain the solution of the first equation in (3.2): \(\tilde{S}(t) = 1 + (S_{n\tau} - 1)e^{-\mu(t-n\tau)}\), \(n\tau < t \leq (n+1)\tau\), here, \(S_{n\tau}\) is the number of susceptible population immediately after the \(n\)-th vaccination pulse at time \(t = n\tau\). Using the second equation of the system (3.2), we deduce the stroboscopic map such that: \(S_{(n+1)\tau} = (1 - p)[1 + (S_{n\tau} - 1)e^{-\mu\tau}]\). The map has the unique positive fixed point: \(S^* = \frac{(1-p)(e^{\mu\tau}-1)}{e^{\mu\tau} + p - 1}\). The fixed point \(S^*\) of the stroboscopic map implies there is a corresponding cycle of period \(\tau\) in the susceptible population, i.e.

\[
\tilde{S}(t) = 1 - \frac{pe^{\mu\tau}}{e^{\mu\tau} + p - 1}e^{-\mu(t-n\tau)}, \quad n\tau < t \leq (n+1)\tau.
\] (3.3)

In the section that follows we determine the stability conditions of this periodic infection-free solution \((\tilde{S}(t), 0)\) of the system (3.1).

3.1. Local Stability of the Periodic Infection-Free Solution. Let \(S(t) = \tilde{S}(t) + s(t)\), \(I(t) = i(t)\), the linearized equations corresponding to the system (3.1) read:

\[
\begin{cases}
\dot{s} = -\mu s - \beta S^2 i, \\
\dot{i} = (\beta S^2 - \gamma - \mu)i, \\
s(n\tau^+) = (1 - p)s(n\tau^-), \\
i(n\tau^+) = i(n\tau^-),
\end{cases}
\] (3.4)

Denote matrices \(A(t)\) and \(B\) respectively as following:

\[
A(t) = \begin{bmatrix}
-\mu & -\beta S^2 \\
0 & \beta S^2 - \gamma - \mu
\end{bmatrix}, \quad B = \begin{bmatrix}
1 - p & 0 \\
0 & 1
\end{bmatrix}.
\]

The monodromy matrix \(M(\tau)\) of the system (3.4) is

\[
M(\tau) = Be^{\int_0^\tau A(t)dt} = \begin{bmatrix}
(1 - p)e^{-\mu\tau} & 0 \\
0 & \exp\left\{\int_0^\tau (\beta S^2 - \gamma - \mu)dt\right\}
\end{bmatrix},
\]

there is no need to calculate the exact form of \(\exp\) as it is not required in the analysis that follows. The Floquet multipliers are defined as the eigenvalues of the monodromy matrix \(M(\tau)\), i.e. \(\lambda_1 = (1 - p)e^{-\mu\tau}, \lambda_2 = e^{\int_0^\tau (\beta S^2 - \gamma - \mu)dt}\). It is obvious that \(\lambda_1 < 1\), so the stability of \((\tilde{S}(t), 0)\) is decided by whether \(\lambda_2 < 1\). The infection-free solution \((\tilde{S}(t), 0)\) is locally stable if \(\int_0^\tau (\beta S^2 - \gamma - \mu)dt < 0\), i.e.

\[
\frac{1}{\tau} \int_0^\tau \tilde{S}^2 dt < \frac{\gamma + \mu}{\beta} = \frac{1}{\sigma},
\] (3.5)

here

\[
\int_0^\tau \tilde{S}^2 dt = \tau + \frac{p^2(e^{2\mu\tau} - 1)}{2\mu(e^{\mu\tau} - 1 + p)^2} - \frac{2p(e^{\mu\tau} - 1)}{\mu(e^{\mu\tau} - 1 + p)}.
\]
substituting it into (3.5), we have:

\[ \sigma [1 + \frac{p^2 (e^{2\mu \tau} - 1)}{2\mu \tau (e^{\mu \tau} - 1 + p)^2} - \frac{2p (e^{\mu \tau} - 1)}{\mu \tau (e^{\mu \tau} - 1 + p)}] < 1. \] (3.6)

Denote \[ R_0 = \sigma [1 + \frac{p^2 (e^{2\mu \tau} - 1)}{2\mu \tau (e^{\mu \tau} - 1 + p)^2} - \frac{2p (e^{\mu \tau} - 1)}{\mu \tau (e^{\mu \tau} - 1 + p)}], \] (3.7)
and we have the following theorem:

**Theorem 3.1.** If \( R_0 < 1 \), then the periodic infection-free solution of the system (3.1), i.e. \( \tilde{S}(t), 0 \) is locally stable; if \( R_0 > 1 \), \( \tilde{S}(t), 0 \) is unstable; \( R_0 = 1 \) is the critical value.

In order to simplify (3.7), we can use Taylor expansions by reasonably assuming the period of pulses is much shorter than half of the mean life-time, i.e. \( \tau << \frac{1}{2\mu} \), neglecting higher order terms, we know \( R_0 \) is an increasing function of \( \tau \), so the maximum allowable period of the pulse, \( \tau_{max} \) occurs when \( R_0 = 1 \), thus we obtain the estimation of \( \tau_{max} \):

\[ \tau_{max} \approx \frac{p}{\mu(\sqrt{\sigma} - 1)}, \quad (\sigma > 1). \]

**Remark 3.1.** In section two, when \( \sigma > 1 \), the infection-free equilibrium point \( (S_0, I_0, R_0) \) is unstable, whereas by using pulse vaccination strategy, as long as \( R_0 < 1 \), the infection-free solution \( \tilde{S}(t), 0 \) is stable although \( \sigma > 1 \). The constant vaccination is ineffective if the successfully vaccinated proportion \( p \) is less than \( p_c \approx 78\% \), noteworthy is the fact that the pulse vaccination leads to an infection-free population even at relatively small values of \( p \) if the period of pulse satisfies: \( \tau < \tau_{max} \).

**3.2. Global Stability of the Periodic Infection-Free Solution.** In section 3.1, we have shown that, if \( R_0 < 1 \), then the periodic infection-free \( \tilde{S}(t), 0 \) is locally asymptotically stable, we will prove below the solution is globally asymptotically stable under the condition \( R_0 < 1 \). We have the following theorem:

**Theorem 3.2.** If \( R_0 < 1 \), the periodic infection-free solution \( \tilde{S}(t), 0 \) is globally asymptotically stable.

**Proof.** Since \( R_0 < 1 \), we can choose \( \epsilon_1 > 0 \) sufficiently small such that

\[ \frac{1}{\tau} \int_0^\tau (\tilde{S} + \epsilon_1)^2 dt < \frac{\gamma + \mu}{\beta}. \] (3.8)

From the system (3.1), we have \( \tilde{S} \leq \mu - \mu S \). Then we consider the comparison system with pulse

\[
\begin{cases}
\dot{x} = \mu - \mu x, & t \neq n\tau, \\
x(n\tau^+) = (1 - p)x(n\tau^-), & t = n\tau.
\end{cases}
\] (3.9)

Integrating and solving the first equation of the system (3.9) between pulses, we have

\[ x(t) = 1 + (x_n - 1)e^{-\mu(t - n\tau)}, \quad n\tau < t \leq (n + 1)\tau, \] (3.10)
using the second equation of the system (3.9), we obtain the following difference equation:

\[ x_{(n+1)\tau} = (1 - p)[1 + (x_{n\tau} - 1)e^{-\mu\tau}]. \]  

(3.11)

The difference equation (3.11) has the unique positive equilibrium:

\[ \pi = \frac{(1 - p)(e^{\mu\tau} - 1)}{e^{\mu\tau} + p - 1} > 0. \]  

(3.12)

The difference equation (3.11) can be rewritten as

\[ x_{(n+1)\tau} = d + \overline{d}x_{n\tau}, \]

where

\[ d = \frac{1 - p}{1 - e^{-\mu\tau}}, \quad \overline{d} = \frac{1 - p}{1 - e^{-\mu\tau}}, \]

using iterative step by step, we have:

\[ x_{(n+1)\tau} = d^n + d^{n+1} + \ldots + d^{n+1}x_0, \]

where \( x_0 = x(0^+) > 0 \), since \( 0 < \overline{d} < 1 \), therefore \( \lim_{n \to \infty} x_{(n+1)\tau} = \frac{d}{1 - \overline{d}} = \frac{(1 - p)(e^{\mu\tau} - 1)}{e^{\mu\tau} + p - 1} = \pi \), i.e. \( \pi \) is globally asymptotically stable, thus, the periodic solution of the system (3.9) i.e.

\[ \tilde{x}(t) = 1 - \frac{pe^{\mu\tau}}{e^{\mu\tau} - 1 + p}e^{-\mu(t-n\tau)}, \quad n\tau < t \leq (n+1)\tau, \]

is globally asymptotically stable.

Let \( x(t) \) be any solution of the system (3.9) with initial value \( x_0 = x(0^+) > 0 \), by comparison theorem in impulsive differential equation, we know that for any solution \((S(t), I(t))\) of the system (3.1) with initial values \( S_0 = S(0^+) = x_0, I_0 = I(0^+) > 0 \), there exists a nonnegative integer \( m_1 \) such that the following inequality holds:

\[ S(t) < \tilde{x}(t) + \epsilon_1, \quad n\tau < t \leq (n+1)\tau, \quad n\tau > m_1\tau. \]

i.e.

\[ S(t) < \tilde{S}(t) + \epsilon_1, \quad n\tau < t \leq (n+1)\tau, \quad n\tau > m_1\tau. \]  

(3.13)

where \( \tilde{S}(t) \) is denoted in (3.3). Further, from the second equation of the system (3.1), we know (3.13) implies:

\[ \dot{I}(t) \leq \beta I(\tilde{S} + \epsilon_1)^2 - (\gamma + \mu)I, \quad t > n\tau, \quad n\tau > m_1\tau. \]

then we consider the following comparison system with pulse:

\[ \begin{cases} 
\dot{y}(t) = \beta y(\tilde{S} + \epsilon_1)^2 - (\gamma + \mu)y, & t \neq n\tau, \\
\dot{y}(n\tau^+) = y(n\tau^-), & t = n\tau.
\end{cases} \]  

(3.14)

Integrating the system (3.14) between pulses \((n\tau, (n+1)\tau]\), we have

\[ y_{(n+1)\tau} = y_{n\tau} \int_{n\tau}^{(n+1)\tau} [\beta(\tilde{S} + \epsilon_1)^2 - \gamma - \mu] dt = y_{n\tau} \int_{n\tau}^{(n+1)\tau} [\beta(\tilde{S} + \epsilon_1)^2 - \gamma - \mu] dt, \]
then using iterative step by step,
\[ y_{n\tau} = y_{(n-1)\tau} e^{\int_{n\tau}^{(n-1)\tau} [\beta(S+\epsilon_1)^2 - \gamma - \mu] dt} \]
\[ = y_{(n-2)\tau} e^{2\int_{n\tau}^{(n-2)\tau} [\beta(S+\epsilon_1)^2 - \gamma - \mu] dt} \]
\[ \vdots \]
\[ = y_0 e^{\int_0^{n\tau} [\beta(S+\epsilon_1)^2 - \gamma - \mu] dt}, \tag{3.15} \]
where \( y_0 = y(0^+) > 0 \), from (3.15), using the condition (3.8), we obtain \( \lim_{n \to \infty} y(n\tau) = 0 \).

On the other hand, integrating and solving the first equation of the system (3.14) between pulses, it is easy to know
\[ y(t) = y_{n\tau} e^{\int_{n\tau}^{t} [\beta(S+\epsilon_1)^2 - \gamma - \mu] dv}, \quad n\tau < t \leq (n+1)\tau, \]
in incorporing into the boundedness of \( e^{\int_0^t [\beta(S+\epsilon_1)^2 - \gamma - \mu] dv} \), we have \( \lim_{t \to \infty} y(t) = 0 \).

Let \((S(t), I(t))\) be any solution of the system (3.1) with initial value \( S_0 = S(0^+) > 0, I_0 = I(0^+) = y_0 > 0 \), according to the comparison theorem in impulsive differential equation, then we have
\[ \lim_{t \to \infty} \sup I(t) \leq \lim_{t \to \infty} \sup y(t) = 0. \]
Incorporating into the positivity of \( I(t) \), we know \( \lim_{t \to \infty} I(t) = 0 \). Then, for any \( \epsilon_2 > 0 \) (sufficiently small), there exists a nonnegative integer \( m_1 > m_1 \), such that \( I(t) < \epsilon_2 \) \((t > m_2 \tau > m_1 \tau)\).

For the first equation of the system (3.1), we have
\[ \dot{S}(t) \geq -\beta \epsilon_2 S^2 + \mu - \mu S, \]
in incorporing into the condition \( 0 < S(t) \leq 1 \), so we obtain
\[ \dot{S}(t) \geq -\beta \epsilon_2 + \mu - \mu S. \]

Consider comparison impulsive differential equation for \( t > m_2 \tau \),
\[ \begin{cases} \dot{z} = -\beta \epsilon_2 + \mu - \mu z, & t \neq n\tau, \\ z(n\tau^+) = (1-p)z(n\tau^-), & t = n\tau. \tag{3.16} \end{cases} \]
It is analogous to the system (3.9), we have the following difference equation:
\[ z_{(n+1)\tau} = (1-p)[(1 - \frac{\beta}{\mu} \epsilon_2) + (z_{n\tau} - 1 + \frac{\beta}{\mu} \epsilon_2) e^{-\mu \tau}], \tag{3.17} \]
Further,
\[ z = \frac{(1-p)(e^{\mu \tau} - 1)(1 - \frac{\beta}{\mu} \epsilon_2)}{e^{\mu \tau} - 1 + p}, \]
is the unique positive equilibrium of (3.17) which is globally asymptotically stable.

Therefore the corresponding periodic solution of the system (3.16), i.e.
\[ \tilde{z}(t) = (1 - \frac{\beta}{\mu} \epsilon_2)[1 - \frac{p e^{\mu \tau}}{e^{\mu \tau} + p - 1} e^{-\mu (t-n\tau)}], \quad n\tau < t \leq (n+1)\tau, \]
is globally asymptotically stable.
Let $z(t)$ be any solution of the system (3.16) with initial values $z_0 = z(0^+) > 0$, by comparison theorem of impulsive differential equation, we know that for any solution $(S(t), I(t))$ of the system (3.1) with initial values $S_0 = S(0^+) = z_0 > 0$, $I_0 = I(0^+) > 0$, there exists a nonnegative integer $m_1 > m_2 > m_3 > m_1$, such that

$$S(t) > \tilde{z}(t) - \epsilon_2, \quad n\tau < t \leq (n + 1)\tau, \quad n\tau > m_3\tau,$$

(3.18)

Because $\epsilon_1$ and $\epsilon_2$ are sufficiently small, it follows from (3.13) and (3.18) that

$$\tilde{S}(t) = 1 - \frac{pe^{\mu t}}{e^{\mu t} + p - 1} e^{\mu(t-n\tau)}, \quad n\tau < t \leq (n + 1)\tau,$$

is globally attractive. Thus, infection-free solution $(\tilde{S}(t), 0)$ is globally asymptotically stable. \(\square\)

### 4. Existence of Positive Periodic Solution.

In this section, we use the bifurcation theory in [8] to study the existence of positive periodic solution. Using the same notations as [8], we change $S(t), I(t)$ into $x_1(t), x_2(t)$ respectively, thus

$$F_1(x_1, x_2) = -\beta x_1^2 x_2 + \mu - \mu x_1, F_2(x_1, x_2) = \beta x_1^2 x_2 - (\gamma + \mu) x_2, \Theta_1(x_1, x_2) = (1 - p) x_1, \Theta_2(x_1, x_2) = x_2, \zeta(t) = (x_s(t), 0)^T = (\tilde{S}(t), 0)^T.$$  

From $\epsilon_0 = 0$, it is easy to obtain

$$\frac{1}{\tau} \int_0^{\tau} \tilde{S}^2 dr = \frac{2B}{K} = \frac{1}{2},$$

i.e., $R_0 = \sigma [1 + \frac{\omega^2 (e^{\mu\tau} - 1)}{\mu \mu (e^{\mu\tau} - 1 + \omega^2)}] = 1$, so $\tau_0$ is $\tau_{\text{max}}$ in section 3.1 making $R_0 = 1$.

We can calculate that

$$a'_0 = 1 - (1 - p) e^{-\mu \tau_0} > 0,$$

$$b'_0 = -(1 - p) \int_0^{\tau_0} \exp[-\mu(\tau_0 - u)] [-\beta \tilde{S}^2(u)] \exp(\int_0^u (\beta \tilde{S}^2(r) - \gamma - \mu dr) du > 0,$$

$$\frac{\partial^2 \Phi_2(\tau_0, X_0)}{\partial \tau \partial x_2} = [\beta \tilde{S}^2(\tau_0) - \gamma] \exp(\int_0^{\tau_0} (\beta \tilde{S}^2(\tau) - \gamma - \mu) dr) > 0,$$

$$\frac{\partial^2 \Phi_2(\tau_0, X_0)}{\partial x_1 \partial x_2} = \int_0^{\tau_0} \exp(\int_0^{\tau_0} (\beta \tilde{S}^2(\tau) - \gamma - \mu dr) du \beta \tilde{S}^2(r) - \gamma - \mu dr du > 0,$$

$$\frac{\partial \Phi_1(\tau_0, X_0)}{\partial \tau} = \tilde{S}(\tau_0) = \frac{\mu p}{e^\mu - 1 + p} > 0,$$

$$\frac{\partial^2 \Phi_2(\tau_0, X_0)}{\partial x_1^2} = \int_0^{\tau_0} \exp(\int_0^{\tau_0} (\beta \tilde{S}^2(\tau) - \gamma - \mu dr) du \beta \tilde{S}^2(r) - \gamma - \mu dr) du \{ \int_0^u \exp(\int_p^{u-p} (-\beta \tilde{S}^2(p)) \exp(\int_0^P (\beta \tilde{S}^2(r) - \gamma - \mu dr) dr = 0,$$

since

$$\frac{\partial \Phi_2}{\partial x_1} = 1 - p, \frac{\partial \Phi_2}{\partial x_2} = 1, \frac{\partial \Phi_2}{\partial x_1 \partial x_2} = 0, \frac{\partial \Phi_2}{\partial \tau} = 0, \text{so}$$

$$B = -\frac{\partial \Theta_2}{\partial x_2} (\frac{\partial^2 \Phi_2(\tau_0, X_0)}{\partial \tau \partial x_2} + \frac{\partial \Phi_2(\tau_0, X_0)}{\partial x_1 \partial x_2} \frac{1}{a_0} \frac{\partial \Phi_1(\tau_0, X_0)}{\partial \tau}) < 0,$$

$$C = 2 \frac{b'_0}{b_0} \frac{\partial^2 \Phi_2(\tau_0, X_0)}{\partial x_1 \partial x_2} - \frac{\partial \Phi_2(\tau_0, X_0)}{\partial x_2^2} > 0,$$
then $BC < 0$, so we have the following theorem:

**Theorem 4.1.** The system (3.1) has a stable positive periodic solution which bifurcates from $R_0 = 1$. i.e. when $\tau > \tau_{\text{max}}$ and is closing to $\tau_{\text{max}}$, there is a stable positive periodic solution of the system (3.1).

5. Discussion. We have analyzed the SIR model with nonlinear incidence rate $\beta IS^2$, let’s recall what we have done. Firstly, we consider the simple SIR model and show that the epidemic will die out if the basic reproductive rate of the epidemic is less than one, i.e. $\sigma < 1$, that is, each infected individual on average, infects less than one member of the population; whereas if each infected individual infects more than one other member of the population, i.e. $\sigma > 1$, the epidemic can not be eradicated. According to the typical parameters that are representative of measles dynamics, we know measles can not be eradicated as $\sigma > 1$, thus, we have to consider the vaccination strategies. There are two cases for the vaccination strategies. One case is the constant vaccination, i.e. all newborn infants should be vaccinated and vaccination is continuous, by Lyapunov-Lasalle theorem and Poincaré-Bendixson theory respectively, we show the infection-free equilibrium is globally asymptotically stable if the proportion $p$ of those vaccinated successfully is greater than $1 - \sqrt{1/\sigma}$, i.e. $p > 1 - \sqrt{1/\sigma}$, whereas the epidemic equilibrium is globally asymptotically stable if $p < 1 - \sqrt{1/\sigma}$, thus, $p_c = 1 - \sqrt{1/\sigma}$ is the critical vaccination proportion, using the typical parameters of measles dynamics, we estimate $p_c \approx 78\%$, which is much less than the corresponding result of the SIR model with bilinear incidence rate $\beta IS$ (see Shulgin, Stone & Agur [14]), that is to say, the constant vaccination of the SIR model with nonlinear incidence rate $\beta IS^2$ is easier to be carried out than that with bilinear incidence rate $\beta IS$. Considering the vaccination is in regular pulses in practice but not continuous, so we study the other case of the vaccination in section three, that is the impulsive vaccination which provides a more natural description of the vaccination strategies. The impulsive vaccination strategy of the SIR model with nonlinear incidence rate $\beta IS^2$ is the main aim of our paper.

Using the stroboscopic map we obtain the exact periodic infection-free solution $(\tilde{S}(t), 0)$ which is globally asymptotically stable if $\int_0^{\tau} \tilde{S}^2 dt < \frac{1}{\sigma}$ i.e. $R_0 < 1$ (where $R_0$ is denoted in section 3.1; from the critical value $R_0 = 1$ we obtain the estimation of the maximum allowable period of the pulses, i.e. $\tau_{\text{max}} \approx \frac{\mu}{\mu(\sqrt{\sigma} - 1)}$, from the expression, we know, as long as $R_0 < 1$ (or $\tau < \tau_{\text{max}}$) the infection-free solution $(\tilde{S}(t), 0)$ is globally asymptotically stable although $\sigma > 1$, whereas for the standard SIR model, the infection-free equilibrium is unstable if $\sigma > 1$; on the other hand, the constant vaccination is ineffective if $p < p_c \approx 78\%$, noteworthy is the fact that the pulse vaccination leads to an infection-free population even at relatively small values of $p$ if $\tau < \tau_{\text{max}}$. Therefore, the impulsive vaccination strategy is more natural, more effective. We also show there is a stable nontrivial periodic solution which bifurcates from $R_0 = 1$ i.e. $\tau = \tau_{\text{max}}$. For the global stability of the infection-free solution and the existence of the stable nontrivial periodic solution, paper [11] only provided numerical simulations, while the theoretical proofs are shown in our paper.

Finally, the nonlinear incidence rate $\beta IS^2$ can be extended to $\beta IS^pS^q (p > 1, q > 0)$, the corresponding impulsive system has the same periodic infection-free solution as in section 3, which is always locally asymptotically stable. There is still a
tremendous amount of work to do in this area, for example, it would be interesting to study whether the infection-free solution is globally asymptotically stable and whether there is a nontrivial periodic solution bifurcating from the threshold $\tau_{\text{max}}$, and so on, we leave these for future consideration.

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